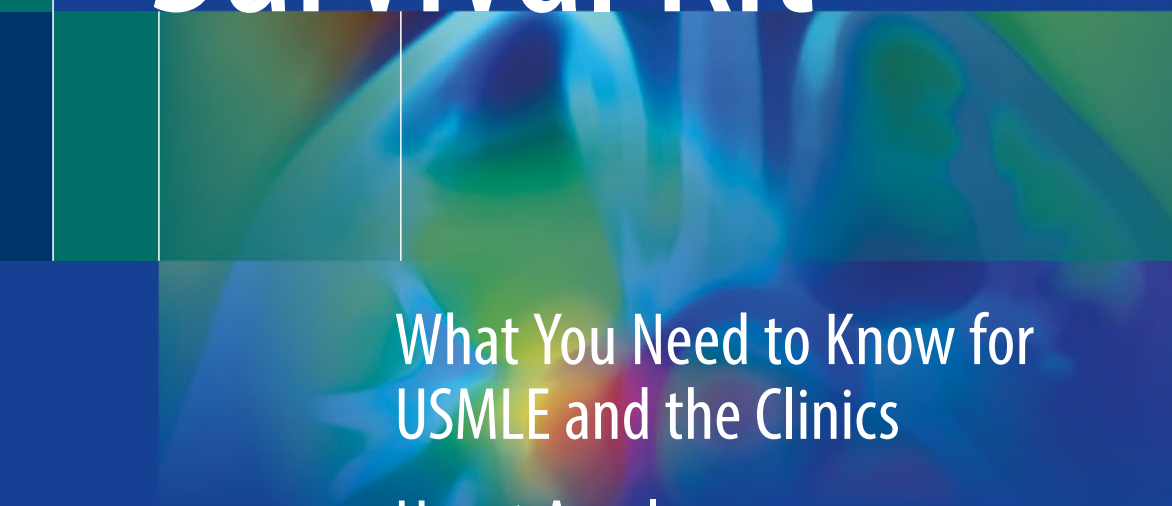
The Radiology Survival Kit



What You Need to Know for USMLE and the Clinics

Hayet Amalou
Robert D. Suh
Bradford J. Wood
Editors

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To precious Alice Coco:
"Dream with passion,
listen to your inner fire,
laugh hard like a child,
be a hero for those you love."

Disclaimer

Dr. Wood contributed to this book in his personal capacity. The views expressed are his own and do not necessarily represent the views of the National Institutes of Health or the United States Government.

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Part I

General

The Radiology and Images Survival Kit: What You Need to Know for Boards and Clinics

Hayet Amalou, Robert D. Suh, Bradford J. Wood, Gianpaolo Carrafiello, and Chiara Floridi

Contents

No concise image-based cookbook introductory review exists that summarizes foundational basic images from *radiology, interventional radiology, dermatology, and ophthalmology*, all together in one source. To create this resource, we gathered up friends and cooked up a bare-bones minimum of the basics for each chapter that reflects typical material that commonly shows up on Medical Student and Intern USMLE Exams Steps 1–3. Each chapter presents a perspective of what you never ever want to miss on boards or on call. This is what the boards question writers really want to know anyway: that you are safe and won't miss major dangerous stuff. This story is told with images and covers just the basics, super simple with no

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extra fluff. If it is in this book, it is worth committing to memory. We do throw in some rare and weird facts to which the examiners love to circle back, for some reason. We include helpful memory aids and mnemonics for differential diagnoses that are passed down from class to class.

Chapters are devoted to the "Philosophy of Board Prep," including "Silly Things They Love to Ask You," hot new topics of "Women's Imaging," "Interventional Radiology and Interventional Oncology," and minimally invasive image-guided therapies. The chapter on sample questions has typical USMLE-type clinical case scenarios, with accompanying images, question bank multiple choice, and paired matchings, K-type questions, and typical sample questions that include images (similar to USMLE step 2 and step 3).

The "Google Image Self-Trainer" chapter lists "Google Image" search terms. These Google Image search terms yield rapid-fire image familiarity training using "Google Image." This is the little link just below the main Google search engine where you can limit your search to images only. This takes advantage of the continuously updated Google Image search engine to show you the latest web-based typical images for each search term. What better way to recognize all types of emergencies than to look at a few hundred super quickly? Use these search terms to pull up images of common boards scenarios, and become familiar with usual imaging appearances of common boards diagnoses. But don't stare too long at any single image. They are meant to be absorbed fast, so take a mental picture and move on.

1 Summary: Aims and Scope

Radiology exams are the modern stethoscope. Imaging is often one of the first diagnostic tests during initial patient encounters. For USMLE, let the key features of the clinical story guide you. Images cannot be interpreted in a void. In fact radiology reports guide you, but the physician who knows the patient's story will be the one to make the diagnosis and correlate the clinical significance of the image. Imaging cannot replace the physical exam, and an image may have a different meaning with a high white count, pleuritic chest pain, or hypotension. Luckily, anatomy does not change, and diseases are slow to change. However, radiology is the fastest changing medical discipline, reliant upon rapidly evolving and often confusing and complex technologies. As radiology becomes broader in scope and impact, it becomes more difficult to boil down to the bare basics of just what images you need to know for the USMLE steps 1–3, licensure, and certification, and no more.

Approach board questions as if you were working with a real patient. The most common mistake is to get into an unrealistic surreal question mode and try to outsmart the question writer. Use common sense and narrow down the answers if needed, but imagine it as a real person in an actual clinic. Same for Step 2 CS and Step 3 CCS: treat the patients as real.

The intern, medical student, or subintern may just want to survive a night on call, looking at PACS alone, with the attending and the patient's family waiting for your

interpretation. All this school, time and training! All the classes, tests, and books! Now, you are on the spot. The good news is that it is better to miss your first case here, where boards or real patients will not pay the price. So join us in missing cases, hurting a few virtual patients here, where it doesn't matter. You will recall better if you treat each case as a real patient. If you intentionally miss free air here in these pages, it will stick with you, no one gets hurt, and no one but you knows it. Sharpen your skills for boards and wards. Imaging has become an essential component of modern medicine, and basic radiology is essential to physicians and students in training and will be used throughout most physicians' careers, no matter what type of medicine or specialty.

As radiology, dermatology, and ophthalmology technologies evolve, textbooks get bigger and more detailed. We hope to shine light on the critical foundations of imaging and images, without getting lost in the complexities of the details. Information is power, but too much information fills the brain with needless debris, so we will ignore the details and focus only on what you need to know to score well on USMLE steps 1, 2, and 3, as well as to be better able to order, understand, and sometimes interpret imaging. Typical and relevant cases are reviewed that are commonly seen in clinic and on tests. Certain questions and variations can be expected and predicted with high likelihood.

This brief book is neither comprehensive nor detailed but includes essential definitions and critical images for students and residents in any specialty. Too many medical books assume students already know something about the subject. No background knowledge of radiology is expected here, just anatomy and basic medicine. Written largely by radiology, dermatology, and ophthalmology residents and recent residents who understand the basic elements you need to survive USMLE and internship and who want to share that information in a practical and concise format. Key references are kept to a minimum or not referenced at all, with details left to look up or learn later, or never, rather than clutter your brain. This basic introduction to radiology and imaging lets you start by bypassing large textbooks, by boiling it down to the bare minimum of what you need to know from a practical clinical perspective, and no more. Organization and chapters cover the usual basics of chest, musculoskeletal, abdominopelvic, and interventional radiology, with emphasis on signs, symptoms, history, disease, image findings, clinical service/specialty, and finally typical USMLE questions and images. Two chapters cover imaging topics often overlooked while preparing for USMLE: imaging in ophthalmology and imaging in dermatology. These images always pop up on USMLE and are challenging to find in a single book. We pick a core set of images and explanations that will start to make you a safe physician. There are no shortcuts, but it is easier to add new learning upon this basic foundation of images.

If rare or uncommon diseases are mentioned, it is only because they show up on USMLE or board exams. Do not waste time on them, but do remember the one thing about the disease or imaging finding that is classic or has a link to a picture or image. If you have a visual memory, you will do well with imaging and radiology. Flipping through this book very quickly will commit a few classic images and associations to memory forever.

Chapters begin by addressing early challenges faced by students, interns, and junior residents trying to absorb the bare essentials of imaging rapidly, prior to beginning a rotation or internship, and without reading a thick, long textbook. Such very brief and digestable resources are limited and scarce, with most introductory radiology textbooks either too cluttered with irrelevant details or not inclusive of other images for boards. The goal here is for USMLE step 1, 2, and 3 preparation focused just on the images in radiology, interventional radiology, ophthalmology, and dermatology. This is also a quick read before a rotation, elective, or clerkship.

This book will not make you a radiologist, dermatologist, nor ophthalmologist but will make you a better student, resident, doctor, ICU nurse, technologist, or healthcare worker. Learning normal radiology anatomy will help you to see the abnormal, like the brain-teaser puzzles.

For example, finding a hidden soccer ball in a "*Where's Waldo*"-type image of a busy street can be difficult. However, if I tell you that two twins in green shirts and blue shorts are kicking the ball in the crosswalk, then it becomes a bit easier to find. This is called structured pattern recognition. You see it once and make it stick by pretending it is a real scenario with a real patient. Feature recognition, pattern recognition, and multiple layers of such decision trees are also how deep learning and artificial intelligence operate, which will revolutionize images in medicine, assisting the physicians to practice better.

Find something that looks abnormal or just doesn't belong. Then always think of what anatomy lives nearby and what sort of common problems might go wrong with that anatomy. If it is the lung, it might be pneumonia or CHF, but don't forget the things nearby: pneumothorax, free air, and rib fracture. If it is headache with head CT, think SAH, CVA, tumor, sinusitis, or trauma with subdural or epidural hematoma plus nearby fracture. Epigastric pain? Look nearby at what lives there: pancreas, duodenum, stomach, or free air from an ulcer perforation?

Finding the abnormality requires rapid assimilation of imaging findings and clinical data. Every chest X-ray is a puzzle. Every test question is a puzzle. Attack it. Better understanding of what radiology and imaging can do for your patient builds bridges between image and disease and between anatomy, diagnosis, and treatment. Imaging can shed light by linking the history, physical, and labs. Imaging helps you to put together what you may have learned in biology, physiology, chemistry, anatomy, and clinical medicine.

Welcome to the cool world of radiology, where Superman's X-ray vision provides a direct window inside your patient! Learn the language of radiology and turn on the lights to see beyond the shadows and into the window of medicine using the stethoscope of the twenty-first century!

2 Chapter Organizational Themes

Chapters focus on the basics of what not to miss, what to order and when to order it. Chapters are generally organized by anatomy (chest, abdomen, bone/musculoskeletal MSK, IR/vascular). This general introductory chapter covers the basics of

imaging interpretation, common terminology, and things to know on day 1 of a radiology rotation. Each chapter will have more detailed tips for ordering imaging tests. After completion, you will have seen:

1. Ordering and indications for imaging tests (with images and very brief diagnosis/explanation).
2. Very brief overview of key anatomy that is commonly imaged (with images and very brief diagnosis/explanation).
3. Most common on-call scenarios and emergencies, organized by very brief history or presentation (with images and very brief diagnosis, chief complaint, or a brief one-sentence explanation).
4. Common exam-type cases/questions, with integrated USMLE case scenarios with imaging components. Images and questions are followed by explanatory connections between diseases and images, in the setting of common on-call scenarios.

The structure, flavor, and chillaxed philosophy make this book unique.
Authors were instructed to:

- Stay simple: audience is medical students, nurses, and interns. Included are only the basics of what you absolutely need to know about each modality and clinical scenario, without any fluff or extra information or detail.
- Images are organized into several categories: (1) key anatomy, (2) on-call brief cases, and (3) exam cases and questions.
- Most images will have a chief complaint or diagnosis and a sentence or two explanation of key imaging features.
- Create typical boards themes and questions with specific typical USMLE clinical images: This includes typical step 2 + step 3 clinical case scenarios that have accompanying imaging or other USMLE written questions (step 2 or step 3) with accompanying images in radiology, interventional radiology, dermatology, or ophthalmology.
- Abnormal lab values are added in when relevant (elevated WBC, fever, smoking history, acute abdomen, flank pain, hematuria, melena, hemiparesis, epigastric pain, SOB, AFIB, Chest pain, etc.).
- Modality-specific background principles and terminology (on X-ray, CT, MRI, nuclear medicine/PET, IR) are very superficially covered in the introductory chapters.
- Anatomy-specific chapters (chest, abdomen, bone/musculoskeletal MSK, vascular) may include normal images outlining normal anatomy, as well as images depicting common pathology (typically seen on boards).
- Practical tips for ordering imaging tests are included: what to order and when to order it. Also when not to order things (e.g., lumbosacral X-rays are high radiation dose and rarely help for acute assessment of typical disc disease symptoms. Knee X-rays may not be helpful if there is typical knee twist history and exam suggesting ACL tear).

- Figures or mnemonics are scattered as memory aids, demonstrate commonly tested facts, and allow one to think on one's feet in a structured fashion, even post-call.
- Example memory aids/mnemonics and short lists for memorization of common differential diagnoses for common imaging findings (i.e. "blood, pus, water, or cells" is the differential diagnosis for airspace disease in the lungs but also the same list can be used for bowel wall thickening).
- Authors present what they wish they had known before starting internship year.
- Resemblance of sample questions to actual boards questions is unintentional. No direct duplication of any USMLE questions is intended, but the content and knowledge base may be the same.

Supplementary sections like the "Google Image Self-Trainer" are also provided to assist with online learning. This new model takes advantage of "Google Image" with specific search terms for each area of imaging, as a method for augmenting conventional book training with a web-based multimedia resource that evolves automatically over time. Becoming familiar with the typical images associated with each search term is a great way to learn radiology (as long as you learn the basic significance of the search terms first).

Radiology, dermatology, and ophthalmology rely upon imaging. This can only be learned by seeing a high number of images, even briefly, and with repetition and visualizing the variations. Basic radiology and USMLE review can be taught with a multitude of images via "Google Image." In Google, the option to search just images is below the search engine. As a novel learning paradigm, we provide suggested "Google Image" search terms. In fact, this rapid image review method provides a better learning method than possible in any book, and it evolves and auto-updates. Google image search terms are provided in an independent "Google Image Self-Trainer" chapter, divided by sections. Smartphone image search sessions can start you off in seconds.

Introduction and Basic Principles of Radiology

Hayet Amalou and Bradford J. Wood

Contents

To fully appreciate the relevance of any one image, do not jump to immediate conclusions. Take your time. Slow the analysis down, and approach any image methodically. You usually will be presented with additional clinical information which will often be clues as to the finding on the image. The first three chapters show how to look at an image critically and sound like you know what you are talking about. We provide you the basic tools with which to look at the puzzle of a medical image

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critically, like a scientist. This includes a section on philosophy for board preparation and some basic and sometimes silly facts which are commonly tested. Chaps. 4, 5, 6, 7, and 8 provide an anatomy-based core radiology, including common emergencies with paired images. The "Other" Chap. 9, 10, 11, and 12 then focus on hot newer topics. For those who like video games, we present the newest residency of interventional radiology (IR) and minimally invasive image-guided therapies like interventional oncology. Women's imaging combines a unique set of disciplines and is mature enough to be fair game for question-writers, who like hot and recent topics. The eye and skin chapters present a different side of medical imagery, also fair game for boards, and hard to find in one location. The book's closing three chapters disclose Google image search terms, practical clinical rotation tips, and a pool of USMLE-like questions and answers that were generated by our team of authors and "book faculty," to whom we are indebted. The team of authors and friends created what we hope is a uniquely combined resource to address an unmet study need for the rapid and simple review of images of all kinds. The human body is amazing – when it goes off track, medicine investigates with pictures and imaging tools...the stethoscopes of the twenty-first century.

Welcome the adventure of radiology, where medicine meets video games with X-ray vision. Interventional radiology is an active video-game adventure. To learn to be detectives with X-ray vision, we will first embark upon basic principles, rules of interpretation, terminology, techniques, and risks. Images of the body are made with X-rays (CT), magnetics (MRI), sound waves (ultrasound), or nuclear physics (positrons and gamma waves, PET, and nuclear medicine), optics (retina), or smartphone cameras (skin). These images generally follow some basic rules of photography with light, which allows the image to show clues and shadows of disease or pathology, which help guide diagnosis or treatment, once identifiable patterns are recognized. Like light, sharp edges of shadows have significance, such as on ultrasound, calcium shadows sharply at the margin compared to air, which has a more blurry indistinct shadow edge or border on ultrasound. Artificial intelligence, machine learning, and deep learning may help detect specific patterns, but the human doctor and brain will remain the director of this detective story of medical images. Cool stuff.

It is extremely important to always ask yourself how an ordered test might change the patient's management or inform decisions. *Every test comes with a cost* of money, time, and resources, and some have risk or radiation dose as well. Curiosity is a natural and compelling reason to want to order a test, but not an indication. Vigilance against this natural curiosity is required before clicking or calling to order a study. Incidental findings that are meaningless are scattered across radiology reports, at a tremendous cost to the healthcare system due to imaging follow-up, self-referred additional testing, and needless patient worry. Decisions driven by a fear of malpractice litigation or patient insecurity may not be the best decisions for the patient, nor for the healthcare system as a whole. "Treat the patient, not the image."

The electronic record means patients have instant access to reports, which can also create confusion and worry. In general, major diagnoses and imaging findings

should be shared at the same time as a plan. This means reading reports soon after a study is completed. Radiology reports with terms like "suggest clinical correlation" or "recommend follow-up" are ways to communicate unknown or raise concerns or questions. Pay close attention to the exact report wording: "consistent with" is fairly definitive, but "differential diagnosis includes…" implies the radiologist does not know the significance of the imaging finding. These differential diagnosis findings make the best teaching cases.

"If this patient had…": An intelligent tactic for discussing images on rounds involves the creation of clinical scenarios, such as: "*If this patient had* a fever and an elevated WBC, then infection would be high on the differential." Or "*if this patient had* a history of smoking and weight loss, then lung cancer would need to be excluded by further workup, which might include comparison to prior imaging, imaging follow-up, percutaneous biopsy, bronchoscopy, search for paraneoplastic syndromes, or attention to the adrenal glands on the CT, to looks for metastases. Talking also buys you time while your eyes can simultaneously look for secondary findings.

1 Radiation Awareness

X-rays, fluoroscopy, C-arms, angiography, cone beam CT, nuclear medicine, and CT use X-rays and are associated with ionizing radiation. Ultrasound and MRI do not use ionizing radiation and are often preferred for this reason (even more so in pediatric and reproductive-age females). However, realize there has been a lot of nonscientific media hype about the risks of CT radiation. These risks are real, but very small, however, and also hard to specifically define. It is your job to weigh the risks and benefits using science and facts. Yes, CT is the major contributor to the overall population's radiation dose, but this comes with the benefit of knowing, for example, when to take a patient to the OR for appendicitis, when to immobilize the C-spine for instability, or when to avoid thrombolytics in acute hemorrhagic stroke. Every test has a cost and/or a risk, so it is wise to always ask yourself before ordering any test (not only imaging): "how will this change my management of this problem?" How will this help the patient?

Curiosity is not an indication. Consider urine or blood pregnancy screen for any female (~10–55 years) prior to CT scans, pelvic or lumbar spine X-rays, or nuclear medicine tests. Weeks 2–15 (8–12) of pregnancy (organogenesis) are the most sensitive to the teratogenic effects of ionizing radiation. However, teratogenesis (birth defects) is not a major concern after diagnostic CT studies of the pregnant pelvis, because the radiation dose is below the threshold ("non-stochastic" effect = has a threshold). However, the risk of inducing cancer has no threshold ("stochastic"); therefore any radiation might cause a very low but measurable increase in risk, even at low levels of exposure. The benefits of finding a diagnosis or answering a clinical question often outweigh the very small radiation risk, or not, for example, a lumbar spine series is rarely indicated and uses a fair amount of radiation to nearby radiation-sensitive pelvic structures and is rarely helpful, whereas MRI provides

definitive answers about trauma, nerves, discs, and bones, without the radiation. Not everyone with back pain needs an MRI. Mammography can introduce very low elements of risk from radiation, but the benefits far outweigh the risks, when used according to guidelines.

The clinical judgment of when to order what test comes with experience and is as challenging to learn as it is useful. Clinical acumen sometimes requires following your gut feeling and ignoring the "book answer" which may be a wrong answer. For starters, concentrate on building some basic framework and recognizing the common things you never, ever want to miss.

2 IV Contrast

Consider blood creatinine prior to any administration of IV contrast (iodine for CT or gadolinium for MRI). Iodine carries the risk of acute tubular necrosis (ATN) and allergy, whereas gadolinium carries the risk of NSF (skin fibrosis) in the presence of renal insufficiency. In the presence of preexisting renal insufficiency (Cr > 1.5 or CR CL < 60), ATN, or renal failure, risk for NSF is decreased by aggressive hydration 75–100 ml/h, 12 h before, and 12 h after contrast (25–50 ml/h if CHF) (+/− acetylcysteine). If a real iodine allergy (anaphylaxis or bronchospasm or laryngospasm) is suspected, premedication with steroids and antihistamines in the 12 or 13 h before contrast can be life-saving. Similar to any scan or procedure requiring sedation, an accompanying driver is required, as even antihistamines can impair alertness. Failure to premedicate for contrast allergy will be repeated at rounds and cause delays, like forgetting to place an NPO order prior to sedation or anesthesia. No one likes to be noticed for the wrong reasons, but missed orders will get remembered just as well as the great pneumothorax or free air you saw on your own in the middle of the night.

Iodine contrast allergy premedication regimen: steroids + antihistamines: prednisone 50 mg PO 13, 7, and 1 h before iodine (OR methylprednisolone 32 mg PO 12 and 2 h before iodine), + diphenhydramine 50 mg PO, IM, or IV 1 h before iodine (+/− cimetidine 1 h before).

2.1 Renal Insufficiency and Gadolinium

Certain linear gadolinium contrast agents may in rare situations cause nephrogenic systemic fibrosis (NSF) from free gadolinium becoming dislodged, where it may cause problems. The gadolinium agents that were mostly responsible for NSF have been removed from the market. For patients with renal insufficiency, special gadolinium may be used (such as ProHance, MultiHance, or Gadavist). Gadolinium should only be given to a renal failure patient on dialysis (GFR < 30) *if* the contrast is critical to imaging/clinical management *and* after informed consent. Lowering the dose of gadolinium (for GFR < 45–60) may also decrease risk. Each hospital has specific protocols and policies on this which usually follow national guidelines (i.e., ACR).

3 Terminology

Speaking directly to the radiologist will often yield much more information than just reading a report. This is true for deciding how confident is the diagnosis, what other differential diagnoses should be considered, and if there is disagreement among radiologists. After all, they are human, so the report language may not reflect the real interpretation. Many radiologists will dictate one way but provide super useful information in person that may not be seen in the report itself. Radiologists' reports may not reflect clinical data (WBC, fever, blood loss, other organ disease, exposures, travel, or family history). Visiting radiology in person or by Zoom is less common with the PACS (picture archiving and communication system) and the webPACS, especially with telemedicine in the post-COVID paradigm, but it remains good medicine and the best way to communicate and share information about a sick patient, which requires a back-and-forth two-way street for discussion. Many multidisciplinary tumor boards, panels, and rounds occur in radiology or on Zoom with PACS screen sharing for just this reason. Protect private health information which is on every image. Consent, research ethics, regulatory, privacy, or information security questions may show up on boards.

Don't get lost with the language of radiology! It can be simplified. For example, bright and dark should have been used for every modality, but radiologists use different terms for each and each has a different mechanism.

4 Bright Versus Dark (Mechanism) (Main Example)

CT bright = high attenuation/dark = low attenuation (stopping X-rays is bright) (metal > bone > blood > water > air > fat) (if IV contrast enhanced, then bright = vascular, or if in wrong place, bright = active bleeding)

MRI bright = high signal/dark = low signal (sequence dependent: T2 = H2O bright, T1 = contrast bright)

US bright = echogenic or hyper-echoic/dark = hypoechoic (sound travels better in water, so echoes do not come back to the transducer camera, and thus cystic or fluid appears dark) (US is best modality for solid versus cystic). Kidney stones and gallstones also have bright echoes and "shadowing" behind the stones. Shadowing is the dark lines behind the stones – or below on the picture. Shadowing has sharp edges behind calcium or stones and fuzzy indistinct edges behind gas – such as in bowel or emphysematous cholecystitis (*E. coli* in a diabetic).

PET/nuclear medicine bright = high activity or SUV (standardized uptake values)

Bone bright = sclerosis/osteoblastic. Dark = osteolytic

Chest X-ray *terminology* is another great example of how words can be confusing. Even radiologists use different terms to mean different things, so don't stress! "Airspace disease" is the same as "alveolar disease" and may overlap with "infiltrate" or the more dense alveolar infiltrate called "consolidation." All these terms could mean pneumonia (or not!). Learn the radiologist culture of terms for infiltrates. The differential for airspace disease is blood, pus, water, cells, or atelectasis

(see Table 2). Infiltrates can be interstitial (stringy lines) or alveolar (fluffy cloud-like). If pneumonia is present, viral pneumonias are often interstitial, whereas bacterial pneumonias are often alveolar and fungal pneumonias are often nodular (think fungal in immunocompromised).

Pulmonary edema is often the same as CHF (same as LV failure) or pulmonary venous congestion or pulmonary venous hypertension. In pulmonary edema, such water grows in the lungs (often from leaky vessels, or increased pulmonary capillary pressures, or the pump not working well and backing up into the plumbing pipes). This pulmonary edema has various stages depending upon how severe it is and how fast it begins. First there is "cephalization" the same as "redistribution," where the upper lung vessels grow in size to be the same size as the usually larger lower lung vessels (pulmonary capillary wedge pressures PCWP = 12–18). Next severe comes interstitial edema (PCWP in 20s), where the vessels become indistinct, with fuzzy borders, so that you can't draw the exact edge with a pencil. More interstitial markings are seen, and then engorged horizontal lymphatics show up as "Kerley *B*" horizontal lines at the lung bases laterally (Kerley *A* lines up and down at apex), and fluid can be seen in the fissures or as pleural effusions. Next is alveolar edema (PCWP upper 20s–30s), with its fluffy, cloud-like, cotton ball densities, or completely white, when the lungs fill with fluid. To review, worsening CHF leads to bigger upper lobes vessels, and then fluid swells the interstitium and edges of the vessels, and when that cup overflows, the fluid leaks into the alveolar airspace until eventually causing white out and pleural spaces. The large heart (cardiomegaly) and prominent aortic knob and tortuous curved descending aorta (from hypertension) are often-associated bonus findings with cardiogenic pulmonary edema from LV failure.

4.1 Technique: Looking Good on Rounds

If you are quickly able to criticize a CXR technique, it will give you time to look further for abnormalities and also make you appear as if you know what you are talking about, even if you are clueless! A few simple comments like "not the best image, slightly under- or overpenetrated and rotated, but adequate" or "rotation or portable AP films make heart size unreliable" will do the trick, and it is always good to start with image technique before jumping to diagnoses.

4.2 Technique: Cardiomegaly or Not?

Remember that a big heart can only be diagnosed on a PA CXR and not an AP or portable or poorly inflated CXR (from poor inspiratory effort or in expiration). This is because of the way that shadows are made. Shine a light (X-ray) at your pencil (heart) with paper right behind the pencil, and you will see it casts an accurate estimate of the pencil's size. If you move the paper (X-ray film or detector) farther away from the pencil, you will no longer see an accurate size, and the

pencil will appear larger. On a usual PA CXR, the detector (paper) is placed closer to the heart; thus the X-ray can give an accurate size, and a heart greater than one-half of the lung cavity diameter is too big. "PA" means the X-ray travels "P to A," or posterior to anterior, with the heart residing in the anterior chest, so you get an accurate estimate of the heart size. In a portable or AP CXR (X-ray travels A to P), the detector is placed behind a patient in the ICU or ER while they are sitting up; thus the paper is far away from the pencil, so the shadow always appears bigger than usual.

4.3 Underinflation or Expiratory CXR

Underinflated lungs (poor effort or expiratory) can also cause a heart to falsely look big or cause lung vessels to crowd together, appearing like atelectasis or even infiltrates. Asking for a repeat inspiratory CXR is the only way to clarify this situation, so pay attention at first to roughly how many ribs are seen through the lung fields. Ten *posterior* ribs is great, eight may be underinflated, and six is expiratory/underinflated. Think of COPD if you see more than ten. Soon you will recognize underinflation without counting ribs.

CXR rotation is also easy to figure out by comparing spine to clavicles. Identify the clavicles (front of chest), identify the spinous processes (back of chest), and then compare these two to each other to see if the patient is twisted and, if so, turned to which direction. Rotated CXR can give the false appearance of mediastinal widening. Tracheal pseudo-deviation is most commonly just due to rotation.

Exposure: is the whole CXR too dark or too light? Look for faint spine shadow behind heart and retro-cardiac lung markings. The edges can tell the truth, and a light CXR can simulate infiltrates. Can it be adjusted on the PACS monitor, or are there just too few or too many X-rays to be able to fix it by adjusting the levels and width of the windows? While you are adjusting windows, remember to check for tubes and lines or foreign objects, since they can be easier seen dialing the windows darker (especially for ICU or ER).

4.4 Systematic Approach

When you first start looking at a chest or abdomen X-ray or CT, or head CT, use a systematic approach, so you don't miss findings. This makes sure you are using all the information on the image and that you do not miss the second finding or third finding, because you were stuck on the first finding. This is like saving your dessert for last. If there is an obvious mass or pneumonia, save it for later, and don't jump right to it, because you may get distracted or may not see the other findings on the image unless you approach it systematically, making sure to look for each category of findings, one at a time. This way you won't miss the tumor causing the post-obstructive pneumonia, or the axillary surgical clips that tell you the nursing home patient had breast cancer in the past, or the rib destruction that is a clue to the

microbiology. Start with technique (rotation, exposure, tubes and lines, verify name), and end with commonly missed areas like retrocardiac (airspace), the apices (pneumothorax), and the CPAs (small effusions). Put everything else in between in any order you like, as long as it is the same order each time you look at a chest X-ray (Tables 1, 2, and 3).

Table 1 Systematic approach to **CXR: ABCDEFGHI**

*A*ir: free air, pneumothorax
*A*irway: tracheal deviation to one side? Endotracheal tube too low?
*A*ssess quality: exposure, rotation, tubes and lines, name?
*B*ones and soft tissue: metastases, fractures, osteopenia
*C*ardiac: heart size < 50% if PA (not AP)
*D*iaphragm: free air, hyperinflation in COPD
*E*ffusions: blunting CPA
*F*ields: infiltrates (alveolar or interstitial), masses
*G*reat vessels: Aortic knob, pulmonary vessels
*H*ilum and mediastinum: adenopathy, ca++, masses paratracheal thickening
*I*mpression: What matters?

Table 2 A Few helpful memory aids

DDX: What could cause this finding?: think "*CINTV*" = congenital, clot, infectious, inflammatory, iatrogenic, neoplastic, trauma, vascular
*DDX alveolar airspace disease in lung (fluffy): (**blood, pus, water, cells**):*
Blood – hematoma, PE, Goodpasture's, Wegener's, coagulopathic
Pus – pneumonia (viral (interstitial [stringy], bacterial [consolidation], fungal [balls], atypical [mycoplasma, legionella])
Water – edema (redistribution - > interstitial - > alveolar - > ARDS)
Cells- cancer (mets or primary)
*DDX thickened bowel wall or folds: (**blood, pus, water, cells**):*
Blood – ischemia
Pus – location (CMV, campylobacter, C diff, salmonella, shigella)
Water - hypoalbumin
Cells – lymphoma, adenocarcinoma

Table 3 Cases you *never*, ever want to miss, ever, especially on-call or on boards!

Pneumothorax (CXR) +/− tension
Aortic dissection/mediastinal widening (CXR)
Free air (Abd/X-ray or CXR)
Hemorrhagic stroke (CT)
Midline shift/mass effect (CT)
Fracture (X-ray, CT)
C-spine instability (X-ray, CT)
Abscess (CT)
Tracheitis (X-ray)
PE (CT PA gram)
Appendicitis (CT/US in pediatrics)
Cholecystitis (US)
Ectopic pregnancy (US)
Pneumatosis (CT or Abdominal X-ray)

5 A Word on Research Versus Clinical Questions and Compliance

Curiosity is not an indication for ordering tests. Images may identify patients even without a name on the image. If you are asking a research question or intend to publish a research question using patients, you likely need IRB or OHSRP (Human Subjects Research Protection) approval or a waiver of consent (that meets strict criteria) or an IRB-approved protocol with prospective consent of patients. The IRB/OHSRP waiver for some case-based retrospective reports can be very easy and fast but should be addressed. Consult local offices and IRBs for processes and policies for clinical research. Training in research is often required (training in GCP, "good clinical practice"), as well as privacy and protected information training (PII, PHI, HIPAA). Accessing the picture archiving and communication system (PACS) may also require credentials and training or certification.

US FDA, EU EMA, or country-specific regulatory review may also be required, or an IDE (investigational device exemption), or an IND (investigational new drug) application. "Off-label use" is when a drug, device, or medical thing is used in a way that is very different from which it is FDA cleared, or on its IFU (instructions for use), which lists the cleared indications. Drugs are FDA-approved for a specific indication, whereas devices are often FDA-cleared for a certain use. Investigational devices that are being assessed by a company for marketing may have a premarket approval (PMA) or "510(K) clearance." PMA vs 510(K) pathways may depend upon the risk of the device (Class I, II, or III). 510(K) is a premarket submission to FDA that shows the device is as safe and effective (substantially equivalent) to another already cleared and marketed device (predicate device). Clinical trial drug studies have Phase 1 (first in human or dose determination and safety), Phase II (biological activity and efficacy), and Phase 3 (comparison to or improvement upon standard therapy). COVID-19 has made US FDA Emergency Use Authorization (EUA) a fair category for board questions. It may be wise to be familiar with the latest public health issues in the highest impact medical journals. You may see a chest CT with typical features of COVID-19 like ground glass opacities (GGO) or "crazy paving" of thickened inter- and intralobular septae. Knowing the different appearances of COVID-19 vs classic community acquired pneumonia may be one part of a question related to testing, isolation, transmission, source control, or treatment. Lymph nodes post-ipsilateral vaccination may show up on mammography. Patients may also show chronic sequelae from SARS-CoV-2 that have image correlates. Add COVID-19 to your Google image search. Flip through this book the week before boards, just to look at the pictures.

5.1 Closing Thoughts

Don't freak out about boards. It's just a random test, written by random people who were once sitting in your shoes. If you pretend the cases are real people, you will retain more when studying and test better. Remember why you went into medicine.

Lastly, don't compare yourself to others. Please Google and read "Desiderata"…and call Mom a lot 💻.

6 Silly Facts About Which They Love to Ask and Tips and Strategies for Evaluating Images

When you see an image (skin, eye, or radiology), the answer is also in the words. In other words, the case story guides you to the image finding. The image may not even need to be used for the answer or will be an obvious corroboration of the words in the question/text.

Histology slides are not covered here, but the same Google image term search will yield radiology images next to histology or pathology images. Review them in pairs at the same time. USMLE loves "Rad/Path" correlation questions.

Do not fear images; they can be the easiest questions if you are methodical. When you see an image, break the question down into digestible steps:

What modality is it? What body part? Why did they order the study? CT is just a way to show you anatomy. Find familiar or easy to recognize anatomy first, and then see if something else is there that shouldn't be, or if the normal anatomy looks weird. If the kidney looks weird, and you have an older male with hematuria, think RCC. If it's a head, CT leads you more likely to stroke/search for blood, whereas MRI more likely leads you to a tumor. If it is an MRI or CT, figure out how high or low the images are in the body, so you get oriented. The weird tumor or abnormality is usually easy to find and narrows down to several possibilities by just the image alone. Then go back to the words, and see where it points you. They may be asking about something totally different and have given you a normal image. Go with your first gut feeling.

If you imagine something subtle, it is probably not real! USMLE images will be obvious and will slap you in the face! Don't get stuck on just the image without integrating the words from the question as well. Read the vignette for clues, focusing on the question mark, often in the last sentence. Combine the image finding, with the case story, abnormal lab, or a weird history. Put it all together. It won't be subtle. There are super easy and obvious questions scattered throughout. Don't look too long. Take the free time back. The tricky or complex questions will be obviously complex. It may help to blow off stress by telling yourself sometimes: I can't believe somebody actually wrote this question! No way are they really asking this question! Don't get too far into the question writer's head though. They are usually straightforward in what information or connection they seek. Stay in the bell curve with responses – common sense may get one wrong, but common sense and educated guesses or narrowing down answers to two choices will get most right. Go with your first answer – the second one is less likely correct, unless it is just obvious you erred on the first.

Images are usually just to show anatomy or anatomy gone bad. When presented with a seemingly confusing image, there is likely one key finding alone on the test image and is likely corroborated by the clinical data and labs presented in the

question. It is unlikely that new information would be presented in a test image that is not reinforced in the text of the question. Therefore, avoid the temptation to look at an image too long. This time can be spent on other questions. So look quickly and search for the single and obvious finding that might be suggested by the history or physical findings from the words in the question.

Beware also of the general image that is not pathognomonic for one specific diagnosis or even intentionally negative. For example, an abdominal image and hematuria with no kidney stones, which may require a CT in an older male to look for kidney tumors, but maybe a urine culture with outpatient treatment for cystitis in a young female. Use the age and real situation to make real decisions. These are real patients – remember that and repeat that to yourself – so call them all by your friends and family names to wake yourself up into acting realistically.

Focus on familiarity with basic anatomy and common conditions that might result in major or poor outcomes if missed. This should be the goal of any question, including the question-writer who decides to add an accompanying image. In a patient with SOB and tachypnea, for instance, try not only to recognize common patterns but use a discriminating eye, such as trying to distinguish between the peribronchial cuffing seen in general fluid overload secondary to cardiogenic or renal failure, which is associated with confluent perihilar haze or airspace opacities, from that of a viral pneumonia, which is accompanied by patchy perihilar infiltrates, or the focal wedge consolidation of a "Hampton's hump" related to pulmonary infarction from a pneumonia caused by Streptococcus pneumonia or Klebsiella.

Initially, it may be helpful to super quickly look at the image to identify the body part and if anything is very obvious in 5 s. Avoid looking at the image for more than 5 s until you read the question. After fully reading the question, taking into consideration relevant history and lab abnormalities, create a short differential diagnosis in your mind of what you will look for or expect to see in the image. Learn to anticipate, since many cases are a constellation of findings that go together. This will become easier with time and practice questions. For example, when presented a chest X-ray with a history of trauma, your focus should be to review for pneumothorax, wide mediastinum related to traumatic aortic transection, and focal or diffuse intra- or extrathoracic fluid collections representing bleeding, fractures, and free intraperitoneal air. If the WBC is elevated with left shift, your focus when reviewing the chest X-ray should shift to community-acquired lobar pneumonia with airspace consolidation, a pulmonary abscess, or even septic emboli with the correct clinical history. If aspiration-prone, alcoholic, or post-anesthesia, the chest X-ray may show aspiration pneumonia, commonly within the right lower lobe, and the next step would be to cover for anaerobic bacteria. With abdominal pain and lower lobe and/or perihilar infiltrates on chest X-ray, consider atypical pneumonias, such as mycoplasma or legionella. With a right shift (lymphocytosis) in WBC count and peribronchial infiltrates on chest X-ray, consider viral pneumonia. If nodular consolidations are seen on the corresponding chest X-ray in the setting of transplantation or immunocompromise, consider fungal pneumonias, particularly *Aspergillus*. In HIV, the chest X-ray may show different pathogens corresponding with the CD4 count that is given in the body of the question. In a patient with lung cancer or other

endobronchial tumors, learn to anticipate and evaluate the chest radiograph for rapid collapse of lobe or post-obstructive pneumonia, which may require coverage with broad-spectrum antibiotics, to include anaerobic organism.

7 Silly Facts on Which They Love to Test

Question-writers love to ask relevant "questions of the times." For the next several years, this will be SARS-CoV-2 (the virus) and COVID-19 pneumonia, for example. Make certain to read up on the latest. In this case, become familiar with common appearances and terms, such ground glass opacities (GGO) favoring the lower and peripheral lung, crazy paving (intra- and interlobular septal lines within areas of ground glass), reverse halo sign, enlarged vessels within GGO, and absence of pleural effusions. Note that patients can be asymptomatic or mildly symptomatic (still hypoxic) in COVID-19/SARS-CoV-2 with an average incubation period (or serial interval) of ~5 days. Read up on the latest isolation, testing, vaccines, local variants, and chronic effects.

Read online abstracts or flip through the pictures from the last year of major medical journals, such as *NEJM*, *JAMA*, *Lancet*, *Nature Medicine*, *Postgraduate Medicine*, and *American Family Physician*.

The caudate and left lobe of the liver hypertrophy in cirrhosis, which is at risk for hepatoma, also known as hepatocellular carcinoma.

LIRADS, BIRADS, and PIRADS 1–5 are standardized risk criteria for liver, breast, and prostate cancer, respectively.

Stomach is a common site for both extranodal Hodgkin's lymphoma as well as the number one site in the GI tract for non-Hodgkin's lymphoma.

In some instances, accepted conventions are not always 100% correct but are still used in general terms. For instance, lymph nodes greater than 1 cm are generally considered abnormal on CT imaging studies, when in reality, this may not be totally true. A bunch of smaller mesenteric nodes may represent lymphoma and all can be under 1 cm each. Small internal iliac nodes can be pathologic in a patient with pelvic cancer.

The pathogenesis of diseases, such as cancer, is firmly rooted in pathophysiology. A deep understanding of pathophysiology will certainly help to appreciate how diseases present on imaging. Although cancer respects patterns, on occasion it may appear that it is not following convention. Melanoma and breast cancer metastases can pop up just about anywhere, much like tuberculosis, but after realizing that these diseases spread lymphohematogeously, it is easier to grasp their propensity for seemingly random presentations.

Lymphoma is "the great mimic" because it can be nodular or infiltrative and present in any organ or structure. In general, lymphoma is soft and grows around normal anatomy instead of invading or "taking over" as can many tumors. Hodgkin's lymphoma usually presents above the diaphragm in the thoracic cavity and spreads contiguously along lymph node stations and lymphatics, whereas non-Hodgkin's lymphoma often presents below the diaphragm with noncontiguous spread and

frequently with remote sites of involvement. The "sandwich sign" is a hamburger, in which the two buns represent lymphoma nodal masses surrounding mesentery or bowel. "Endo-exo-enteric lymphoma" ulcerates to communicate with bowel lumen.

Colorectal adenocarcinoma spreads to regional lymph nodes, liver, and lung occasionally with very large round lung metastases, known as "cannonball metastases."

Certain cancers exhibit a propensity for vascular invasion; hepatomas frequently grow into the portal vein and kidney cancer similarly into the renal vein and IVC.

Ovarian carcinoma and GI cancers can metastasize to the peritoneum causing "omental cake." Primary mesothelioma (like ovarian cancer) commonly spreads along the pleural or peritoneal lining or in dependent cavity surfaces, called ""drop metastases."

Pseudomyxoma peritonei = "jelly belly" is a belly full of loculated mucinous adenocarcinoma, often from the appendix.

Stomach cancer can metastasize to bilateral adnexa or ovaries. The most common breast cancer is invasive ductal carcinoma or infiltrating adenocarcinoma. Commonly used tumor markers are CA-125, CEA, HCG, AFP, CA19–9, and PSA. Premalignant conditions with images: Barrett's esophagus, IBD (ulcerative colitis > Crohn's), familial polyposis, undescended testicle, hemochromatosis, MEN, achalasia, hepatic cirrhosis, and asbestosis.

Adenomatous colonic polyps undergo slow malignant transformation from adenoma to adenocarcinoma and can be surveyed every 5 years with colonoscopy. On the other hand, it has recently become apparent that *sessile*, or flat, serrated colon polyps do not necessarily undergo this slow adenomatous transformation from benign to premalignant to malignant (unlike conventional adenomatous polyps) and therefore might require more frequent colonoscopy. Virtual colonoscopy and stool testing for blood are other less common methods of screening for colorectal cancer.

Most common intracranial tumors are metastases. Common brain metastases include melanoma, lung, breast, GI, kidney, and choriocarcinoma.

Epidural hematoma (arterial, lens shaped, biconvex, underlying fracture) versus subdural hematoma (venous, concave with further extension, classic elderly post fall).

Hydrocephalus = big ventricles and wet wobbly and weird (incontinence, wide gait ataxia, dementia).

Grave's disease case with thyroid + CT showing extraocular muscle enlargement at the orbital apex.

Age and bone tumors: newborn, neuroblastoma; 1–15 years old, Ewing's sarcoma; 10–30 years, osteosarcoma, 30–40 years, lymphoma/fibrosarcoma; > 50 years, metastases, multiple myeloma; > 70 years, prostate cancer. Most common bone tumor in adults is multiple myeloma. Most common bone tumor in children is osteosarcoma. Classic USMLE case is radiograph of skull with lytic destruction or holes = multiple myeloma.

A bone scan screens for bone metastases. Osteoblastic metastases appear black on bone scan and white on CT or X-ray ("BLT with a kosher pickle" = breast/

bladder, lung, thyroid, kidney, prostate). Osteolytic or osteoclastic metastases appear white on bone scan and dark on CT or X-ray.

Sclerotic white dense bone metastases: "5 Bees Lick Pollen": breast*, bowel, bronchus (carcinoid), bladder*, brain (medulloblastoma), lymphoma, prostate (most common sclerotic). (*Breast and bladder can be blastic (bone forming) or lytic (bone destroying)).

A lumbar spine series is rarely indicated and delivers a reasonably high dose of radiation to nearby radiation-sensitive organs and structures. In most cases, MRI provides more definitive answers regarding trauma, nerves, discs, and bones without radiation.

AVN = osteonecrosis: "ASEPTIC" – *a*lcohol, *s*ickle cell, *s*teroids, *e*mbolism, *p*ancreatitis, *t*rauma, *i*diopathic, collagen vascular disease (lupus).

When considering imaging, be knowledgeable about indications, kidney toxicities, and allergies from iodinated contrast. It is common for IV and oral contrast to be given for CT scans of the chest, abdomen, and pelvis. Head CT to evaluate for blood in a patient with a suspected stroke should be performed without contrast, at least initially, to be most sensitive for hemorrhagic stroke (avoid TPA / ASA).

Worst headache of life = SAH, MRA, CTA, or angiography and clipping of MCA aneurysm. Blood is bright on unenhanced CT. To help identify SAH, look for bright densities where water density CSF would be expected at the extreme edges of the brain on unenhanced CT. The very last tangential axial CT slice is often the best place to look for blood in the sulci between gyri.

DDX: Ring-enhancing brain lesion on enhanced CT or MRI = "MAGIC DR" – *m*etastasis, *a*neurysm, *a*bscess, *g*lioblastoma, *i*nfarct, *i*nfection (toxoplasmosis), *i*nflammatory, *c*ontusion (resolving), *d*emyelinating (MS), *r*adiation

Head CT before LP to exclude causes for high ICP to avoid herniation

MRI = best study for back pain

Colles-type fracture = distal radius joint surface fracture with dorsal tilt – results from fall on an outstretched hand

Anatomic snuffbox tenderness = scaphoid fracture (prone to osteonecrosis, as is Talus in ankle and femoral head in hip)

Down syndrome associated with atlanto-axial instability, duodenal atresia, and atrioventricular defects

On knee MRI torn triad = anterior cruciate ligament + medial meniscus +/− medial collateral ligament

Pediatric fractures: SALTER-Harris types = "SALTR" – *s*lipped, *a*bove growth plate, *l*ow (below growth plate), *t*hrough growth plate, *r*ammed growth plate (pay attention to the first bold letters that spell SALTR!).

Lobar pneumonia = community-acquired *Streptococcus pneumoniae*

Post-obstructive pneumonia (endobronchial cancer with distal pneumonia beyond bronchial block) = anaerobic pneumonia. Requires broad-spectrum coverage.

One round ball on chest X-ray, think primary lung cancer.

Many round balls on chest X-ray, then think metastases from any cancer (colorectal carcinoma, testicular cancer, breast cancer, melanoma).

Right lower lobe pneumonia in alcoholic patient who aspirated (right main stem bronchus is a straighter path down into the right lower lung with gravity) = *Klebsiella*

Perihilar subtle interstitial lines + patchy infiltrates = viral pneumonia

Lower lobe infiltrates +/− perihilar infiltrates and abdomen pain = atypical pneumonia/walking pneumonia = mycoplasma or legionella

Pneumonia: viral versus bacterial versus atypical pneumonia versus fungal

Causes for immunocompromise: HIV, PCP, chemotherapy, steroids, drugs, organ, or bone marrow transplantation, iatrogenic from medications for a variety of diseases (Crohn's, psoriasis, autoimmune, etc.)

IR: GI bleed, nephrostomy, biliary drainage, chest tube, paracentesis, thoracentesis, angiography, angioplasty, carotid stenting, AAA stent grafts, neuro IR, thrombolysis, tumor ablation, abscess drain, chemoembolization, cholecystostomy drain for acalculous cholecystitis, fibroid embolization, varicose vein ablation, TIPS, IVC filter, anticoagulation

Abdominal calcifications and AAA, abnormal air collections in the abdomen, colon cancer apple-core lesion and histology slide signet ring, bowel obstruction, inflammatory bowel disease, RBC GI bleed nuclear medicine scan with peristalsis of blood to localize bleeding spot, cervical spine fracture, trauma, stroke, headache

DDX: thick colon wall haustral folds = "thumb-printing." Think blood, pus (infection/C diff), water (edema), cells, ischemia obstruction

DDX: alveolar infiltrate = blood (trauma or Goodpasture's), pus (pneumonia), water (CHF), cells (cancer)

Pathology or histology slides + radiology image(s) questions: look for cancer cells – big dark nuclei. Look for fungi or obvious Gram-negative rods that direct you toward specific antimicrobial choices.

Family history and past medical history and travel should be factored in gently; in other words, do not always place significance/importance on this history, as they can sometimes serve as irrelevant distractions. This is one of the most difficult skills to master when reviewing questions – when to filter things out and when to highlight. This improves with practice: speed reading and scanning each question for relevant nuggets of information.

Know well the questions you know will be on the boards for sure:

- Acid-base questions: respiratory versus metabolic, acidosis versus alkalosis. One is the primary problem/disturbance and then the secondary/reaction as the body compensates, and you look for the clues in the labs and ABG. This one can be asked in about ten different ways, but one will definitely show up, so why not just learn them all, inside and out?
- CHF, diabetes, coronary artery disease, stroke, myocardial infarction, pneumonia, cancer, hyperlipidemia, viral-induced malignancy, vaccinations, and medical emergencies. Common things are common. Fall back on the common things again and again. Unless of course, it is the weird and rare paired findings that sometimes appear in questions that will never be seen again in real practice. Don't get distracted; it is usually one or the other.

- Pulmonary edema from CHF, fluid overload, or ARDS will be in many questions. Pulmonary edema is divided into hydrostatic and capillary leak causes. Hydrostatic pulmonary edema results from increased intravascular pressure and capillary leak edema from increased capillary permeability. Hydrostatic pulmonary edema can be caused by cardiogenic (CHF), renal, hepatic failure, and fluid overload. Capillary leak pulmonary edema can be associated with a number of causes, most commonly ARDS. Early mild CHF may just show "redistribution" or cephalization of the pulmonary vasculature, where the upper lung vessels get bigger than normal and larger relative to lower lung vessels. Remember that in normal situations, upper lung vessels are usually smaller than the lower lung vessels on upright chest X-ray. Moderate pulmonary edema then reflects interstitial edema, where the vessels appear thicker, bigger, and ill-defined at edges due to fluid leaking into the perivascular space, so much so that a line cannot be easily drawn around the vessels. Kerley B lines, or engorged lymphatics, are seen at the lateral lower edges of the lung. Peribronchial cuffing may also be seen, where bronchioles seen on end have thickened walls that look like thick circles (thick edematous walls). In advanced pulmonary edema, fluffy and cloud or cotton ball-like opacities, representing alveolar filling, appear within the perihilar lung. Fluid that spills over into the pleural space is a pleural effusion that blunts the usually sharp costophrenic angles and sulci. ARDS is the most severe form of permeability pulmonary edema, which eventually leads to total lung white out on chest radiographs from basement membrane damage and extensive leak. Whether hydrostatic or capillary leak pulmonary edema, coexistent infection may be difficult to ascertain on chest radiographs.
- Epidemiology and biostatistics: in these questions, a table is embedded within the question or needs to be physically drawn by you. These are free points on all steps 1, 2, or 3 and appear multiple times. Commit two tables to memory: one for disease versus exposure (Table 4) and one for specificity and sensitivity versus predictive values (Table 5). These tables are essentially the same table with "Test" replacing "Exposure" and vice versa. During practice and on the exam, writing the table down will avoid mistakes. By writing the table down, it becomes very easy to "fill in the blanks" appropriately and derive any missing information from the numbers that are provided in the question. Pay particular attention to exactly what is being asked, and *not* what you think is being asked. Questions requiring these tables will certainly appear, sometimes several times and on all steps. So remember these two tables! Learn them well and write them down. Do not do it in your head, or you will skip a step.

Table 4 Odds ratio table for disease vs exposure

	Disease	No disease	
Exposed	A	B	A + B
Not exposed	C	D	C + D
	A + C	B + D	

Odds ratio = odds of getting disease with or without exposure = [A/C] / [B/D] = *AD/BC*
Relative risk = [A/A + B] / [C/C + D]

Table 5 Table for disease vs test *(or truth vs test):* (just replace "Exposure" with "Test" in table above)

	Disease YES +	Disease NO −	Predictive values
Test positive +	**A** True pos	B False pos	PPV = A/A + B
Test negative -	C False neg	**D** True neg	NPV = D/C + D
	Sensitivity = A/A + C	Specificity = D/B + D	

Remember that A and D are the key boxes, because they are TRUE (TP and TN)! Because they are important, see how *A and D go on top* and become the numerators of all definitions of sensitivity, specificity, true positive, and true negative. Reproducing this table with a quick four squares/three lines box is faster than trying to do it all in your head. Just trust us on this one!

Philosophy and Approach to the Boards

Hayet Amalou and Bradford J. Wood

- *The philosophy and approach to boards is an intensely personal experience, and we are each as diverse as the day is long. There will be some who are stressed and only prepare and perform well when stressed. Others procrastinate but have always done it that way and have made it this far in school procrastinating, so why change now? These life lessons that follow may work for some, but not for others, so do it your own way. Keep it all in perspective. In 1, 10, 20, or 100 years, nobody will remember or care about this, so don't sweat too much. Don't forget who you are, what you care about, and what got you here. You are smart and can do this.*
- Plan but don't perseverate. Put in a good day's work, and it will all be fine. Make a formal study schedule by topic, but don't stick to it too closely.
- Eat, exercise, laugh, call Mom (or call anyone), study in groups sometimes if you like that, study alone if you like that, be you. Know when you are sharpest and work hard then. Focus for 2 h in a row, maximum, and then take a break. Some do better with breaks every 15 min. Know yourself and make it work.
- The biggest mistake is to read without learning, just to convince yourself that you have read or accomplished something. Fifteen minutes of super focused concentration is better than many hours of superficial study.
- Avoid blogs, avoid loud and aggressive personalities, avoid drama, avoid competitive peers, and do not compare yourself to others. Use practice tests to practice and identify areas on which to spend more time, but don't stress. Nobody

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else cares as much as you, and even you won't give a damn sometime after boards. Call Mom or anyone, again.

- It is important to decide what material and the extent of the material you plan on covering during your preparation time. Make a reasonable schedule for yourself. If you have 3 months to prepare, for example, and have a review book and two question banks, come up with a weekly and a daily schedule of what and how much you wish to cover during each period. Each daily goal may break down to 2 chapters and 50 questions. Don't be afraid to mix it up with reading, which is more passive, and questions, which are more interactive. Set realistic expectations for yourself! Don't shoot for the moon – if you pile on the expectations of covering tons of study material even though well intentioned, it will only cause more stress, disappointment, and even panic when you can't get through the material. By setting up a daily schedule, you can keep yourself true to the goal. If you get distracted and maybe get through one less day, like when your uncle comes into town for a surprise visit, you know how much to kick it up a notch the next day. We all get distracted and have distractions, but knowing your daily goals, you can easily get back on track. Just enough stress to motivate you, but no more! Know how you best operate.
- It is important to reevaluate your plan during your set preparation time, perhaps midway through and at a point 2–4 weeks before the exam. These periodic resets are important. You may find that you have less time remaining for whatever reason and, accordingly, adjust your studying to target areas on which you need more focus, instead of reviewing material you already know really well, or to increase your daily goals to cover the remainder of your anticipated study material. Perhaps, a study partner finds a new bank of review questions that you feel important to include. Swap, adjust, and recalibrate with realistic goals. Never get overwhelmed. Instead, just put in a good day's work, and it will work out.
- Be realistic! Build in planned social events to your schedule, such as your cousin's wedding or your best friend from high school being in town, and enough rest, relaxation and party, friend, or family time to make studying tolerable. You know yourself best; your tendency may be to hit the books hard 16 h each day for 4 days a week and have 3 days to chill and recharge. Others may opt to study all 7 days but maybe less hours per day. Exercise regularly, even if for short periods. Exercise and sleep allow for processing of your studies. Grandma was right!
- Life is full. Everyone has family, personal life, jobs, and responsibilities while studying. Don't treat anyone like "distractions," even if you may feel that way sometimes. They are bookmarks to strengthen your study time. They make you stronger. Factor them in realistically, but set priorities and goals for ~2 days at a time.
- Read up on the abstracts only from general topics over the last 12–36 months of *American Family Physician*, *JAMA*, *NEJM*, *Postgraduate Medicine*, and *Lancet*. Just read the abstracts; rarely waste time on the actual papers (this might change after boards).

- Switch up techniques to trick your brain into staying awake and focused: don't stay passive on learning: flashcards, Q banks, NBME, flipping through books like this fast may be better than months of videos, especially closer to test date.
- It's just a bunch of questions, and the answer is there somewhere – you know most of the answers. You are a good guesser for the rest. Get used to the discomfort of guessing and not knowing. Narrow it down when you can; who cares when you can't? Everyone is in the same boat.
- Collect a "basket of findings" from each question: There will be a lot of distracting junk information to filter through to find the relevant abnormalities on history, physical, or labs. Make a mental note of the major abnormalities, and try to cluster these abnormalities into unified groups that lead to a diagnosis or an action. Imaging will only be one thing in your basket, most often with corroborating evidence for a specific diagnosis.
- While studying question banks, get in the mind of the question writers just a bit, without overthinking it nor making it too complicated. It is often a simple tidbit of knowledge for which they are looking.
- Beware prejudging. Keep an open mind as you highlight or collect your basket of abnormalities. Look for associated findings, but do not jump without corroboration. Each story must be backed by facts and data in front of you.
- *Read the question!* Read all of the question. The answer is in there somewhere! Ignore the vast majority of data presented and search for the golden nugget. Make a mental note of abnormal values or clues in the history, exam, labs or presentation. Deciding where to focus, and where to speed read then gloss over and dismiss, is the key skillset for boards. Some longer questions may need a targeted speed-read, searching for key points.
- Know when to leave a question and move to the next. *Pace yourself* as you go. Get in the habit of a mental clock ticking as you read, so you know when to just guess and move to the next question. You are better guessing on a few questions than losing precious time reading through the longest questions that are in your weak topics or blind spots. Do not stare at a CT, MRI, US, or X-ray for more than a few seconds. Take a quick glance at the picture, go read the question, then come back to the picture, again only for 10 s. If still confused, go back to the written question for details. Do this once and then guess and move on. One question is just one question.
- When your mental timer goes off, make a best guess and cruise onwards with confidence! *Everyone guesses* a lot on these tests – who cares! Move on without baggage. The people who guess with confidence and approach the next question with the same uninjured blind confidence will do better.
- Your *first instinct is often your best answer*. Many people will answer correctly and then go back and perseverate and ruminate on the question. Avoid this; it wastes time, and the first answer was probably correct. The balance is to be thorough and careful but not obsessive with details.
- *Do not try to guess what the question writer is thinking*. If you catch yourself thinking "I wonder what they are trying to get me to say," you may get the questions wrong. If you just *deal with the facts* as presented, you will get it right. This

is a key point and the reason very smart and prepared people might trip up on some questions.

- Decide how you are reading today. Do Q-banks like U-World, reviews like Kaplan, or summaries like First-Aid, but scatter them throughout your test preparation schedule. You can approach them in at least three different ways. Do the questions as test setting prep: fast paced and timed. Then study what you missed, but don't spend too much time on one question/answer unless a weak spot or a recurrent theme. Later on, you may read through the question and answer very quickly to soak in the main teaching points. Finally, circle back again, a third time, for just questions or themes you wanted to review again and commit to memory another time.
- Each time you read a question, collect facts, and then always "circle back around" to the original facts (lab abnormalities, history clues, money, weapons, and golden nuggets). Do not go down a blind alley or out on a small branch. *Circle back around* to the main points and facts of the tree trunk!
- *Avoid "*Q-bank bias*":* Try to assign a question to a category that you have seen before, but do not be biased by similar questions you have reviewed from a prior Q-bank or review book. Often one little item is different that alters the answer. Be precise. Every single person will miss some questions by reading increased instead of decreased or making simple misreading errors. Read closely.
- *Study topics for short-term memory closer to the test date.* Any tables or numbers or review of paired links may be done closer to the test date. Images, questions, and general themes stay in memory longer, so this is fair game anytime, well before the test.
- Study by *building connections* between subjects and prior classes, between systems, or between diseases. Think in terms of the overall patient and not just one topic or class.
- An apparent zebra is more likely a striped horse: *Common things are common!* CHF, pneumonia, diabetes, heart attack, stroke. The problem is that they want to test you on the zebras too. Consider both. Sometimes comorbidities have secondary effects or lead to other diagnoses. But keep it from being far-fetched. If it looks like community-acquired pneumonia, don't get distracted by the travel or family history. Early in the question, cast a wide net to build a broad differential of what in the world this could be, and then later in the 30 s of the question, narrow things down to what information matters. You will build "stories" of what this could be early in a question but then rapidly and freely reject most of those "stories" to narrow on a data-supported answer. It is okay not to know, but don't try to make up far-fetched links when you don't know the answer. In other words, if you are confused, don't start grabbing at small irrelevant facts… if the hemoglobin is low, and the patient is a smoker, maybe this is silent hypoxia from COVID-19 with thrombotic problems, and DVT led to PE caused alterations in ACE and the patient on an ACE inhibitor that might lead to problems with renal artery stenosis because of dilation of the vein, because he has high cholesterol and is a smoker. Just *stop yourself when you get out on a limb that will break!* Just think, but don't go nuts!

- *Every case is real!* Convince yourself that each question is a real person! If you really and truly pretend that the question refers to a real alive and sick patient (picture a familiar face each question), then you will focus more and stay consistently attentive throughout a long test day. This is maybe the best advice anyone ever gave for boards. If you have a visual memory, *imagine case presentations as patients with familiar faces (of people you know)* or picture patient scenes in a familiar clinic. Occasionally smart top-notch students and residents don't test as well on boards by being too "unrealistic" or doing things they would never ever do in a real clinical setting. This is a clinic. This is an ER. This is your office or school. Fool yourself into being real. Give familiar names to each patient to trick your brain into focusing.
- There are not a lot of images on boards, but when there is an image, it may be a quick way to make up time. Be sure to read the end, the actual question, to see what is desired for an answer. Be fast, smart, accurate, decisive, and robotic. Then erase your brain, recalibrate, and reset. Next question!
- Decide which question strategy mode you are in: If test time is running down and you have 20 questions left, you may need to kick into higher gear and adopt a new faster strategy. Your *speed scale is adjustable*, counterbalanced by your attention to detail and ability to read every word closely. Get used to dismissing normal lab values and irrelevant history or physical findings and wasting little time reading and thinking about each one individually.
- One strategy which may be useful toward the end of sessions as time runs out is to *read the last sentence* of the body of the question, then visually scan and cancel wrong answers to narrow it down, and finally make an educated guess. If you have time, you can go back and speed read the question, scanning for abnormalities to place in your "mental basket." It is helpful to *practice this* "speed mode" a lot while practicing on Q-banks, so it becomes a familiar mode with which you feel comfortable. This way you can try many different speeds so you "test drive" your brain a lot. These modes are like having an end of game, fourth quarter, no-huddle offense drill in American football or pulling the goalie to play offense at the end of a losing hockey game. Get comfortable with these modes so panic is not on the radar. Stay calm, cool, and collected as you surgically slice each question. There is never a reason to panic, and the more you simulate those situations that might otherwise lead to panic, the less likely it is that you will panic. This is scientific. *Practice the panic*. Pilots simulate crash situations before flying, so you should too.
- Take a 2 s mental moment to recharge after long questions with a lot of reading. This is "mentally erasing the leftover residual effects and aftertaste of the last question. This is the ginger in between the sushi. Clean and refresh the palate. Spend two mental seconds on this as you click, and it will waste zero seconds. Practice this as you do Q-banks. Simulation of quick and automated mental erasure makes it a comfortable habit.
- USMLE scores are proven to not correlate with other measures of clinical performance or success as a physician. They are meant for licensure thresholds only.

No one but you will recall your score, and even you will forget soon. Read to learn, not to score higher.

- Focus on the next 24 h, maybe the next week, and keep only a general plan for the next months. Write down a balanced plan on a calendar and schedule a section focus for each day, so you cover all sections, and adjust this schedule slightly to spend more time on your weak spots. Days can be split into core sections + Q-banks, so variety can keep you awake.
- Testing boards and licensing authorities may know about common established Q-banks and which of their questions essentially duplicate the board question. With deep learning and artificial intelligence search engines, the board authorities will become even more adept at this over time, while looking at the standard Q-banks that everyone uses, and may keep you in the bell curve and "in the pack." Some of the more fresh and more recently released Q-banks may not have been thoroughly searched for duplicate themes and may be fertile study material. Be aware though; there are some less-informed resources out there, which may also be new. This is probably the only helpful piece of information you can get from blogs and online chats on the topic of boards. So, other than this information, stay away from blogs and chats!
- Blogs and chat rooms on boards are more trouble than they are worth. It is not worth the stress, and you are more likely to get disinformation than helpful information. Sometimes you might find the correct answer to a mainstream question. Other times, you might get the stressed ramblings of the misinformed, which will confuse you.
- Do not worry about how you are faring on percent correct practice tests! Use practice tests only to gauge your relative strengths and weaknesses or to find specific topics or sections on which you may need to spend more time. To quote Desiderata: "If you compare yourself with others, you may become vain or bitter, for always there will be greater and lesser persons than yourself."
- Chillax! Never panic; it uses time and brain power. But if you need motivation, allow just a tad of panic, just enough to get motivated, but no more. Take a deep breath and eat chocolate. Stress is normal, and this too shall pass. Always take a pause to exercise or hang out with someone who makes you laugh. Schedule downtime to chill. Everyone is in the same boat, and if you stay focused, and put in a good day's work, you will do fine.
- Don't freak out about boards. It's just a random test, written by random humans who were once sitting in your shoes. If you pretend the cases are real people, you will retain more when studying and test better. Remember why you went into medicine. Whatever got you where you are will get you through this too.

Part II

Core Radiology

Chest X-Ray and Chest CT

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Contents

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1 Systematic Approach to the Chest X-Ray

The alphabet mnemonic "ABCDEFGHI" can organize your approach to the chest X-ray (Fig. 1). There are many other approaches to viewing each part of the image, and it is important to stick with the same one that works best for you. Using the same systematic approach every time will ensure that you do not miss one thing. The trainee that does not have a systematic approach to each imaging modality will inevitably miss important pathology, which can have grave consequences for the patient.

"A" stands for air. Assess for two important air-related diagnoses that can kill the patient: pneumoperitoneum and pneumothorax. It also stands for assessment of image quality. How is the exposure (is it too dark or pale)? Is the patient rotated? Are there lines and tubes and, if so, are they in the correct position?

"B" stands for bones and soft tissues: assess for bone density (is there osteoporosis?), fractures, metastases, partially viewed shoulder abnormalities? Is there air or edema in the subcutaneous soft tissues? Is there focal soft tissue swelling?

"C" stands for cardiac: assess heart size and heart borders. Coronary calcifications can be well seen on non-contrast CT.

"D" stands for diaphragm: is the diaphragm flattened from increased lung volume (i.e., COPD)? Are they symmetric? Is there free air under the diaphragm?

"E" stands for effusions: are the costophrenic angles sharp or blunted by collection of pleural fluid between visceral and parietal layers of pleura?

"F" stands for (lung) fields: assess all five lobes of the lungs. Don't forget the tricky areas, including the lung apices, lower lobes behind the diaphragm, and left lower lobe behind the heart! Are there densities, and if so, are they hazy (alveolar), reticular, or reticulonodular (interstitial)? Are there any lucencies (emphysema, cavitation, cystic lung disease)? Is there consolidation? Pulmonary nodules, cysts, or masses?

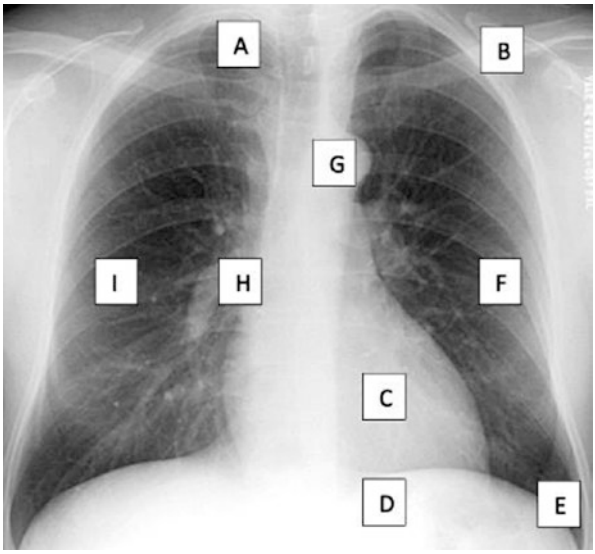

Fig. 1 A consistent pattern to evaluating the chest X-ray ensures that you review all of the anatomy so you don't miss any pathology. The alphabet mnemonic "ABCDEFGHI" is one way to review each part of the image, shown visually above. In short, the mnemonic is: "A" stands for air, "B" stands for bones (and soft tissues), "C" stands for cardiac, "D" stands for diaphragm, "E" stands for effusions, "F" stands for (lung) fields, "G" stands for great vessels, "H" stands for hilum (and mediastinum), "I" stands for infiltrates and impression

"G" stands for great vessels: assess the aortic knob and mediastinum for widening and follow the pulmonary vessels outward from each hilum.

"H" stands for hilum (and mediastinum): assess for lymphadenopathy, calcification, masses, and paratracheal thickening.

Lastly, *"I" stands for infiltrates and impression*. What are your findings and how do they fit together with the patient's presenting symptoms, medical history, and physical examination? Are you able to answer the referring clinician's clinical question?

2 The Normal Chest X-Ray

The standard frontal (posterior-anterior or PA) chest X-ray is taken when the patient stands upright between the cassette and X-ray tube spaced 6 ft apart with the patient facing the cassette. By convention with a PA chest radiograph, the "X-ray" beam enters the patient's back and exits the patient's front and then hits the film cassette or image receptor. In general, the closer an object is to the X-ray source and farther from the film cassette, the more magnified the object appears on film. When an object is closer to the image receptor and farther from the X-ray source, the more

the imaged object is closer to its actual size and less magnified. Even structures within a patient's body can be subject to magnification, most obviously the heart which resides anteriorly within the chest cavity. With PA technique because the patient is facing the film cassette, the heart is closer to the image receptor and farther from the X-ray source, capturing a truer representation of cardiac size.

More or less, the image captured on the radiograph is the result of density and a composite of structures. Density is determined by absorption and scatter of the X-ray photons as they pass through the patient. Dense structures, such as bone, absorb X-ray photon energy and cause scatter, receptor. On film, these structures appear white. On the other hand, less dense structures absorb and scatter less photons and appear as various gradations of gray with the least dense structures, specifically the lung, appearing black.

It is important to realize that images are composed of all structures that the X-ray beam passes through before reaching the film cassette, so for the most part, no structure visualized on a radiograph is a simple stand-alone and, in almost all instances, has several superimposed structures, for example, the lung with overlying anterior and posterior ribs. In practical sense, this may impact radiographic interpretation. With only a frontal radiograph, the left lung base is partly obscured by the heart and both lung bases below the domes of the diaphragms. On close observation, however, the vessels within the lung bases may be seen through the heart and liver and spleen shadows, respectively. Certainly, additional views of the chest, specifically a lateral view in this instance, may be useful to further evaluate these partially obscured areas.

When a patient is unable to be transported to the radiology department, the technologist will acquire an anterior-posterior (AP) or portable (AP and supine/semi-erect) chest X-ray. With an AP chest radiograph, the "X-ray" beam travels through the patient from front to back and then hits the film cassette. Compared to PA radiographs, AP radiographs are generally subject to more magnification, since the X-ray source to image receptor is shorter than 6 ft given space issues in hospital wards, ICUs, and operating rooms where often portable radiographs are obtained. There are many disadvantages to the AP chest X-ray that are essential to understand so abnormalities are not overcalled (in an otherwise normal patient). These "AP portable CXR overcalls" include magnification of the cardiomediastinum (making the heart or hila appear enlarged), poor inspiration crowding the vessels simulating an "infiltrate," or patient rotation (Fig. 2) [1]. How can you tell if a patient is rotated? A simple rule to follow is that the patient is not rotated if you are able to draw a vertical line down the center of the thoracic spine and the medial ends of each clavicle are equidistant to this line (Fig. 3). This will make the ribs look asymmetric as well, and by comparing the length of the anterior rib between sides can be useful to gauge rotation of the lower chest. When the patient performs an adequate inspiratory effort on PA chest X-ray, you will be able to count at least 5 anterior ribs and 8–10 posterior ribs through the lung fields (Fig. 3).

The lateral chest X-ray is taken with the patient standing with their arms (ideally) above their head and one side of their body against the detector. It is essential in helping to characterize and localize what is seen on the frontal view and can confirm or refute a frontal finding that otherwise would have remained a concern. Radiology

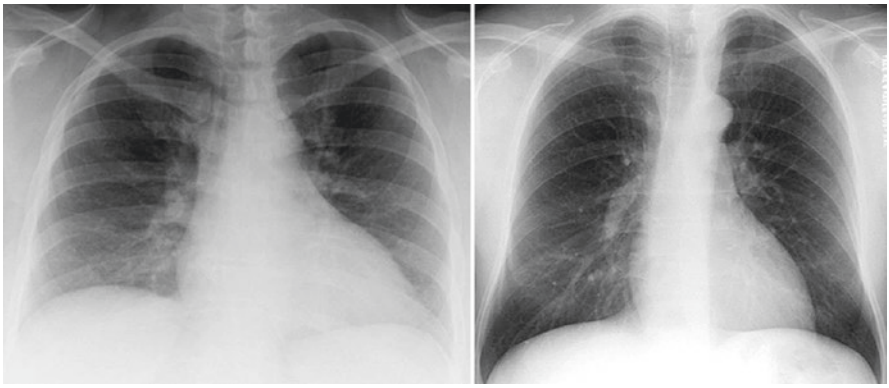

Fig. 2 The image on the left was taken with poor inspiratory effort. This makes the heart look bigger and crowds pulmonary vessels, so you would feel tempted to describe cardiomegaly with an interstitial pattern or viral pneumonia. A clue to poor inspiration is that you only see four anterior ribs. The image on the right is a repeat chest X-ray and was taken immediately after. Notice the normal heart size and lung volumes. Note the beautiful vasculature in the lung fields. It is normal to have larger lower vessels with smaller upper vessels, and this is due to gravity (more blood will flow downward with the gravitational pull). If the upper vessels become the same size as or greater than the lower vessels, this is called cephalization and is seen in congestive heart failure

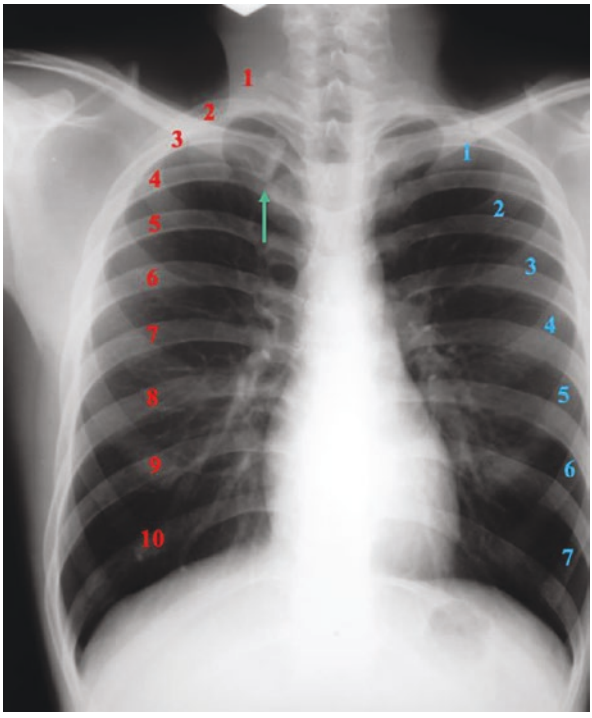

Fig. 3 In this normal PA chest X-ray, the patient demonstrates appropriate inspiratory effort. You can tell because you see more than 5 anterior ribs (blue numbering) and more than 8–10 posterior ribs (red numbering). However, this patient is slightly rotated to the left. The median right clavicle is further away from the center of the thoracic spine than the median left clavicle (green arrow)

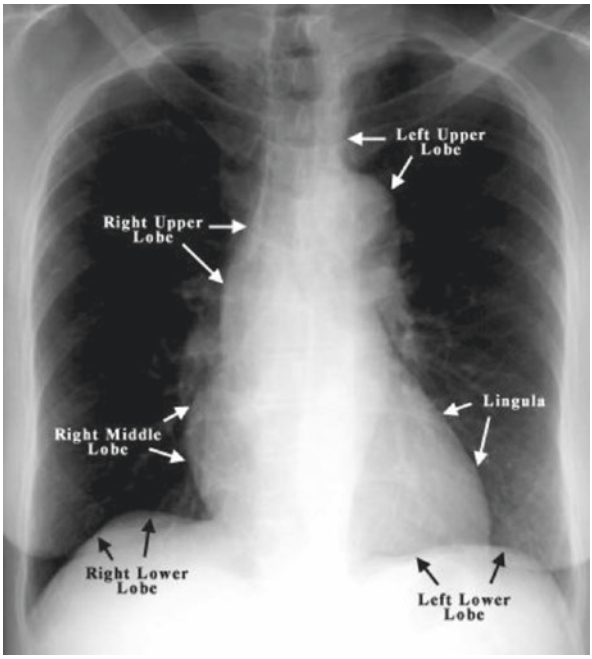

Fig. 4 The normal mediastinal silhouette with arrows (white) pointing to each specific lung lobe that borders it. Also pictured is the normal diaphragm with arrows (black) showing the lower lung lobes that make its border. When a portion of one of these sharp borders is blurred, it is a clue to the location of the airspace disease

is the imaging of anatomy. Understanding first and foremost the normal anatomy of the frontal and lateral chest X-ray will allow you to see when it is not normal. Only certain anatomy has radiographic fingerprints, or anatomic landmarks, whereas some anatomy is invisible or hard to see (Figs. 4, 5, and 6).

3 Lines and Tubes

Evaluation of all medical devices for proper placement and correct positioning is extremely important on both chest radiograph and chest CT. A false read can cause patient harm, including inadequate drainage and iatrogenic injury. Patients in the intensive care unit (ICU) may have misleading chest X-ray appearances due to a single supine radiographic technique, patient rotation, or under/overexposure of the digital image. For these reasons, in depth assessment of all lines, tubes, and catheters is extremely important.

3.1 Central Venous Catheter

A central venous catheter (also known as central line, peripherally inserted central catheter (PICC), jugular-inserted central catheter (JICC), or central venous pressure (CVP) catheter (usually in the ICU)) provides prolonged venous access, parenteral nutrition, and medication administration. A CVP catheter can also monitor right atrial pressure and provide fluid resuscitation. Its tip should reside at

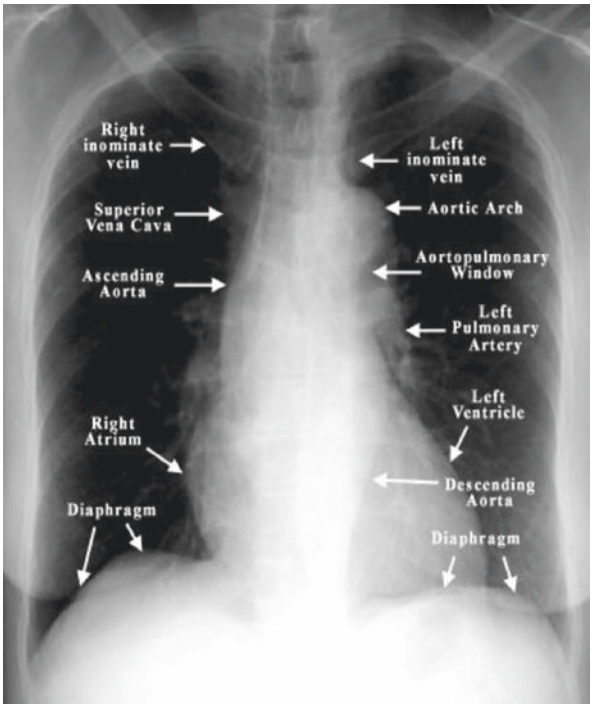

Fig. 5 The normal anatomic landmarks that compose the mediastinal silhouette (white arrows). The obscuration (or absence), enlargement, or tortuosity of any of these structures should clue you in to a pathology involving an organ in that region. Also included is the diaphragm (most inferior white arrows). If there is ever lucency between the diaphragm and inferior aspect of the mediastinal silhouette, you should consider pneumomediastinum

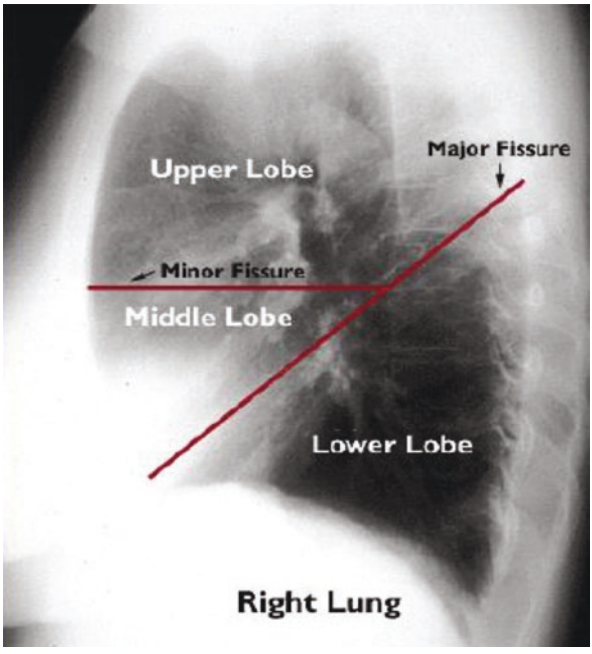

Fig. 6 The right lung has three lobes separated by the major fissure and minor fissure (red lines). The lateral chest X-ray can help to localize focal findings, such as a consolidation or pulmonary nodule, in the lungs to a specific lobe

the superior vena cava (SVC) or the junction of the SVC and right atrium (Fig. 7). Malposition can result in complications, including cardiac arrythmia or cardiac wall perforation if in the right atrium or ventricle, or inaccurate CVP measurements if the line is not advanced enough (Fig. 8). Pneumothorax is a common complication of unsuccessful subclavian or low internal jugular catheter insertion [2] but should be a rare occurrence in the era of ultrasound-guided vessel puncture. Infusion of fluid into the mediastinum or pleural space can result from extravenous catheter placement, catheter tip malposition, and catheter-related vessel injury [1].

3.2 Swan-Ganz Catheter

A Swan-Ganz (SG) catheter, also known as a pulmonary arterial catheter, measures the pulmonary capillary wedge pressure (PCWP) with balloon inflation and right hearts and pulmonary pressures without balloon inflation. This allows for an assessment of the fluid status, cardiopulmonary hemodynamics, left atrial pressure, and cardiac output. The best SG position for accurate hemodynamic evaluation and low complication risk is between the main pulmonary artery and the interlobar arteries. On the chest X-ray, the tip should generally project no more than 2 cm beyond the edge of the cardiomediastinal silhouette and should ideally reside near the lung hilum when it is not being used to measure the pulmonary arterial wedge pressure (Fig. 9) [1, 2]. Over time, these catheters may migrate peripherally with blood flow and become lodged in a small pulmonary

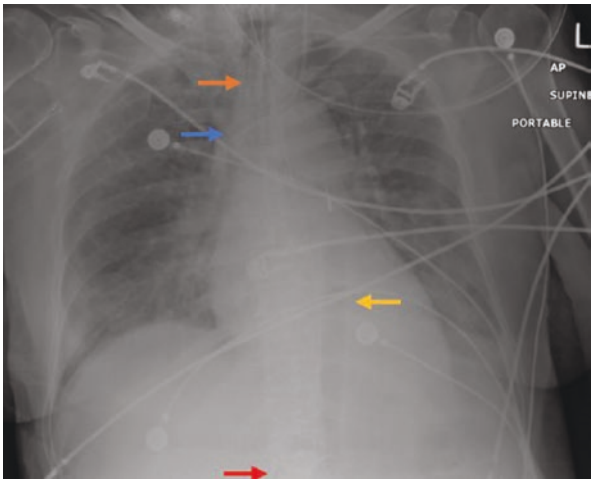

Fig. 7 This is a busy ICU portable chest X-ray that you will typically see quite often while on the wards. Can you name all of the lines and tubes? There is a left internal jugular central line in good position at the level of the SVC (orange arrow). It courses across midline through the left brachiocephalic vein to enter the SVC. Also note an ET tube in good position about 3 cm above the carina (orange arrow) and an enteric tube that courses at midline from the top to the bottom of the image (red arrow). Did you notice the tubular lucency behind the heart and just left of the spine? That is an intra-aortic balloon pump inflated in diastole (red arrow)

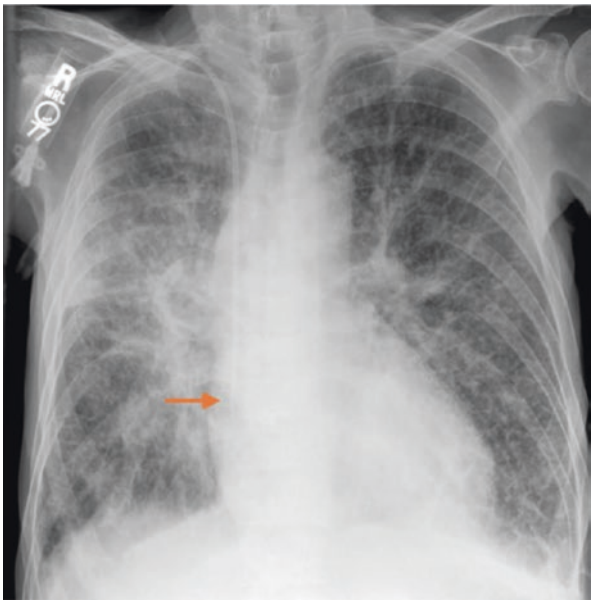

Fig. 8 Here is another very busy chest X-ray. Before getting overwhelmed with the crazy appearing lungs, take it one step at a time and begin with lines and tubes. You will notice a right subclavian dialysis catheter with its tip overlying the mid-heart near the right heart border (orange arrow). You can infer that it has surpassed the SVC and is sitting in the right atrium. Some people might advocate for leaving the tip in the RA if no ectopy or atrial arrythmia ensues. It is a balance between good flow and less likely to clot in the RA versus less likely to induce arrhythmia in SVC

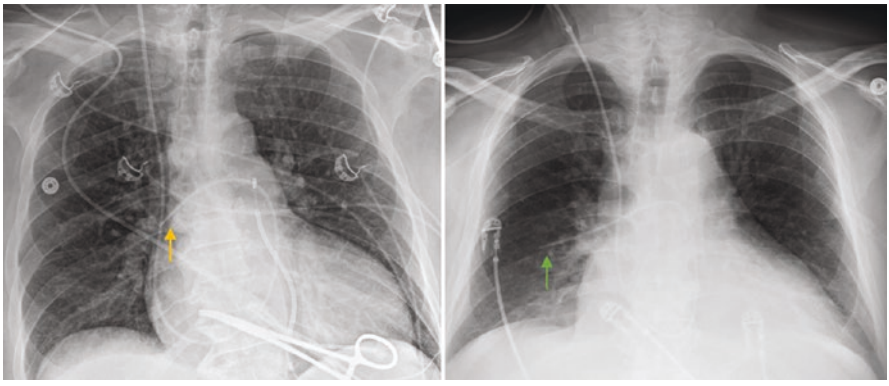

Fig. 9 The portable chest X-ray on the left demonstrates a Swan-Ganz catheter in appropriate position at the approximate level of the right main pulmonary artery (yellow arrow). Notice the Metzenbaum scissors and EKG leads that overlie the patient. It is important to be able to distinguish these from the lines that are inside the patient. The image on the right shows malposition of a Swan-Ganz catheter in a different patient, where it projects too far peripherally with the tip in the right middle lobe segmental artery (green arrow)

artery, causing vessel injury or pulmonary infarction (Fig. 9). It is prudent that the location of the SG catheter tip is known before balloon inflation to avoid pulmonary artery pseudoaneurysm and/or catastrophic rupture. Proximal migration into the right ventricle can also cause cardiac arrhythmia [1].

3.3 Enteric Tube

Enteric tubes are for either nutrition or gastric decompression. A nasogastric tube (NG), which is comprised of side holes at the distal 5–10 cm end, should extend at least 10 cm beyond the gastroesophageal junction and typically terminates in the left upper quadrant. This ensures that a side hole is not situated in the intra-abdominal portion of the esophagus. A naso-enteric tube will curve with the duodenum and reside just left lateral to the spine, anatomically beyond the gastric pylorus and in the third portion of the duodenum. G-tubes or PEG tubes enter the stomach percutaneously at the anterior abdominal wall.

3.4 Esophageal pH Probe

An esophageal pH or temperature probe will appear as a thin, linear structure that terminates in the distal esophagus, just before the gastroesophageal junction. These are used to measure core body temperature or the distal esophageal pH as part of a gastroesophageal reflux workup [2].

3.5 Chest Tube

A thoracostomy (chest) tube is commonly used for air or fluid evacuation from the pleural space. It is typically placed anterosuperiorly for pneumothorax and posteroinferiorly for fluid collections, normally positioned on the surface of the expanded lung between the visceral and parietal pleura [2]. Sometimes, a pigtail catheter is the device of choice, often for prolonged pneumothorax, pleural effusion, or empyema drainage, although chest tubes may be used for pleurodesis (gluing the pleural layers together) or for empyema thinning or dissolution for empyema treatment.

3.6 Endotracheal Tube

An endotracheal (ET) tube is a lifesaving medical device that delivers lung ventilation. In an adult, the tip of the tube should be positioned about 3–5 cm above the carina so that neck flexion, extension, and rotation can be safely accommodated, without slipping down the right mainstem bronchus (straighter pathway) with neck flexion (Fig. 10). If the tracheal carina is not well visualized, an indirect measurement of appropriate placement is if the tip is at or just above the aortic arch or aortic

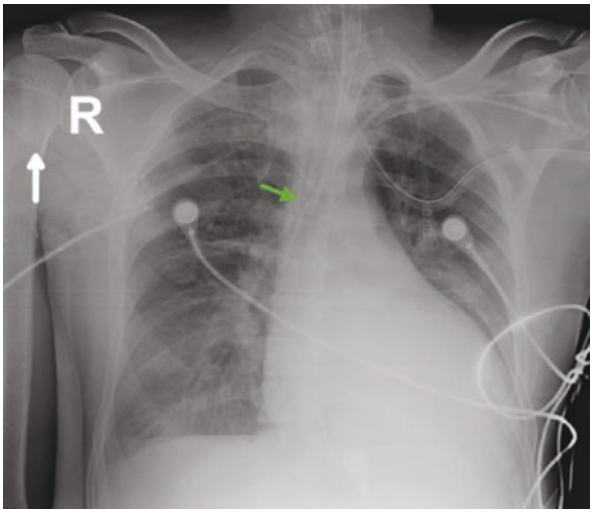

Fig. 10 This chest X-ray demonstrates a complication of ET tube placement and the importance of always localizing the tip of the tube. This ET tube is in the right mainstem bronchus (green arrow), causing the left lower lobe to collapse. This is seen by the complete obscuration of the left hemidiaphragm. Of note, there is a normal positioned left subclavian central line at the junction of the SVC and RA. There is also an NG tube coursing through the esophagus

"knob" [2]. The tip situated at the level of the clavicular heads is an additional anatomic landmark [1]. Both of these positions suggest satisfactory position of the ET tube midway between the carina and vocal cords. When advanced too far, the ET tube typically enters the right main bronchus and causes hyperinflation (lucency) of the ipsilateral (usually right) lung with atelectasis (less inflation with increased density) of the contralateral (usually left) lung (Fig. 10). In esophageal intubation, the stomach may be unintentionally distended with air.

4 Lungs and Pleura

4.1 Pneumothorax

Pneumothorax is the abnormal presence of air in the pleural space. A patient may present with acute chest pain and dyspnea but can also be asymptomatic. Pneumothorax can be organized into three groups based on the cause of the condition: primary spontaneous, secondary spontaneous, and traumatic/iatrogenic. A primary spontaneous pneumothorax typically occurs due to the rupture of a subpleural bleb in otherwise healthy, young adults. There is a male predilection (male/female, 6:1), and there are typically no signs of underlying lung disease. A secondary spontaneous pneumothorax will occur in patients with preexisting lung disease, such as chronic obstructive pulmonary or interstitial lung disease, cystic lung disease, cavitating pneumonia, or pleural metastasis [1, 3]. Spontaneous pneumothoraces occur in the absence of trauma or intervention. Traumatic

pneumothorax occurs in the setting of blunt or penetrating thoracic injury as a result of alveolar compression, pulmonary laceration, tracheobronchial disruption, or barotrauma [4]. Iatrogenic causes include complications from procedures (central line placement, lung biopsy, thoracentesis, tracheal intubation, or mechanical ventilation), although may also be associated with acute asthma and smoking tobacco or drugs [1, 4].

On upright chest X-ray, a pneumothorax will be seen as absence of lung markings between the lung edge and the chest wall, usually largest at the apex and extending laterally along the pleural space. Presence of the visceral pleura, which appears as a clearly defined line paralleling the chest wall, is a cardinal feature (Fig. 11). A skin fold can mimic a pneumothorax, but the line but may fade on one side as you would expect to see if you were looking at a folded piece of paper (sharp silhouette seen on one side) (Fig. 12). Detection of pneumothorax on supine radiographs is more challenging as air will rise toward the anterior chest wall and findings will be subtle. The "deep sulcus sign" may be present with supine pneumothorax. This is a hyperlucent deep costophrenic angle and ipsilateral upper abdomen when compared to the opposite side [5]. If there is doubt, a lateral decubitus chest X-ray (with the suspected side up) will assist in visualization, because even a small volume of intrapleural air will rise and outline the lateral margin of the lung wall. Think gravity for decubitus (lying on side) images. Air rises.

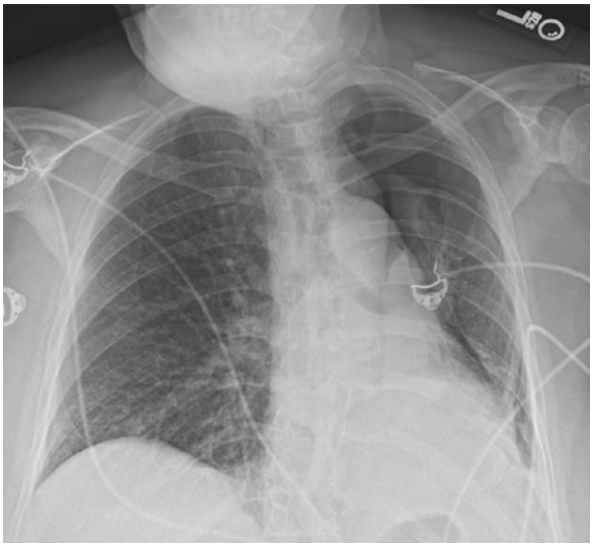

Fig. 11 In this frontal chest X-ray, there is a moderate to large left pneumothorax depicted by the vertical pleural line without lung markings in the thorax apically and peripherally. Can you name the signs for tension pneumothorax, a medical emergency (of which this patient does not have)? They are mediastinal shift away from the pressure buildup and flattening of the left diaphragm. The next step is decompression with large gauge chest tube insertion, or an even better test question answer is decompression with a needle deep into the second anterior rib interspace in the midclavicular line

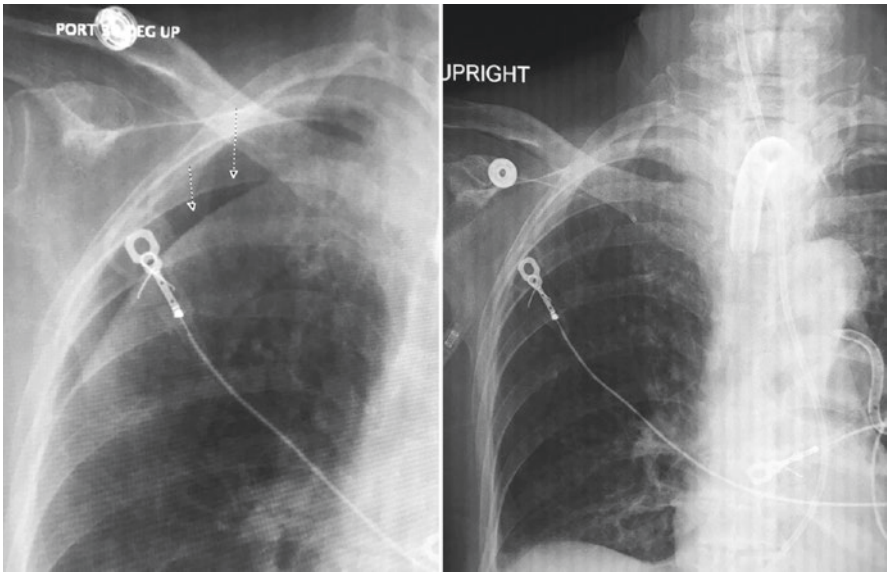

Fig. 12 The image on the left is a magnified view of the right upper lung zone. Are the arrows pointing to a pneumothorax in this ICU patient? Sometimes you can be faked out by a skin fold or bullous lung disease mimicking pneumothorax, which is what happened in this case. Luckily, the radiologist picked up on this and recommended close interval follow-up. The normal chest X-ray on the right shows the same patient 10 min later and confirmed that a skin fold caused the appearance of a small right pneumothorax. (Images Courtesy of Frederick A. Birnberg, MD)

Tension pneumothorax is a complication and is a life-threatening emergency. Radiologic findings include cardiomediastinal shift, deviation of the airway to the contralateral thorax, ipsilateral diaphragmatic flattening, and widening of the distance between the ribs [5]. Other complications of pneumothorax include re-expansion pulmonary edema (after fast evacuation or removal of large volumes of fluid or air) and pneumomediastinum.

Chest CT increases the sensitivity and specificity for many conditions, including pneumothorax, and may be indicated when there is severe underlying lung disease [3]. On CT, gas will be seen within the pleural space, between the chest wall and the lung (Fig. 13).

4.2 Atelectasis

Atelectasis means pulmonary volume loss (mini-collapse) and can be seen over a spectrum of very small areas of collapse to complete lobe or total lung collapse. When a very small portion of the lung collapses, it produces a linear, band-like opacity on both X-ray and CT and is usually referred to as linear or subsegmental atelectasis [1]. Indirect signs of atelectasis that can assist in differentiation between other opacifying pathologies (i.e., consolidating pneumonia) include displacement

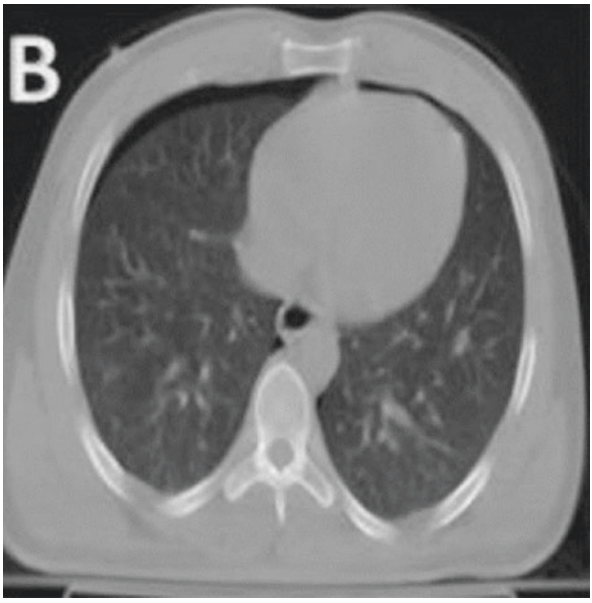

Fig. 13 This is an axial CT at the level of the mid-heart demonstrating small pneumothorax. There is air (black) between the right lung and anterior chest wall within the pleural space. IF this were without symptoms, one might watch it sequentially with CT or chest X-ray until it resolves. Chest tube may also be required. A 20 cm wall suction unless painful is a reasonable order for chest tube management. A test trial with the chest tube clamped (or evacuation chamber to "water seal") may help avoid replacement procedures after chest tube removal

of the mediastinum, hilum or a fissure, as well as elevation of the ipsilateral hemidiaphragm and ipsilateral rib crowding. It may show up as overcrowding of vessels (less splaying) and difficult to distinguish from true alveolar airspace disease.

There are three types of atelectasis: obstructive, passive (rounded, compressive), and cicatrization (fibrotic). Obstructive atelectasis most commonly occurs in the adult from lung cancer, endobronchial tumor, or mucus plug, especially in the intensive care setting [1, 6]. In children, it is often seen with foreign body aspiration [1]. Passive atelectasis occurs via external compression, such as an adjacent pleural effusion or other pleural processes that restrict the lung to fully expand, or internal compression, such as a bulla pressing on the adjacent lung. In many texts, the latter is more formally referred to as compressive atelectasis. Cicatrization atelectasis mainly occurs due to lung fibrosis, which distorts the normal lung architecture, and may be associated with tuberculosis, chronic bronchiectasis, or radiation [6]. Atelectasis is also quite common in lower lobes posteriorly during and after general anesthesia. Atelectasis is routinely confused with alveolar infiltrates since the degree of associated volume loss may be small and relatively imperceptible on chest radiographs. In this situation, the clinical scenario is extremely helpful to distinguish one from the other. A fever in the postoperative patient within 72 h of extubation is typically atelectasis and is treated with more incentive spirometry. A fever a week after surgery or interventional procedure like tumor ablation may spur a CT scan with IV contrast to search for abscess.

4.3 Right Lower Lobe Collapse

Collapse is the most extreme and obvious form of atelectasis and becomes radiographically detectable due to its increased density from loss of air and volume loss. Learning in which direction and pattern associated with each lung lobe collapse may be helpful in recognizing the appearance on chest X-ray. On frontal chest X-ray, as the right lower lobe collapses, it forms a triangular density over the right heart with effacement of the medial aspect of the right dome of the diaphragm and adjacent vertebral margin and depression of the right hilum [1]. Lateral chest X-ray can assist in confirmation of the diagnosis, which will show an opacity projecting over the lower thoracic spine with loss of the usual vertebral "fade-off" and signs of volume loss with retraction of the right major fissure posteroinferiorly and posterior displacement of the right hilum.

4.4 Left Lower Lobe Collapse

Collapse of the left lower lobe typically demonstrates similar findings as with right lower lobe on chest radiograph. Typically, there will be a triangular opacity overlying the left heart border with obscuration of the medial left dome of the diaphragm and adjacent descending thoracic aorta and vertebral margin and depression of the left hilum (Fig. 14). With inferior retraction, the left lower lobe pulmonary artery

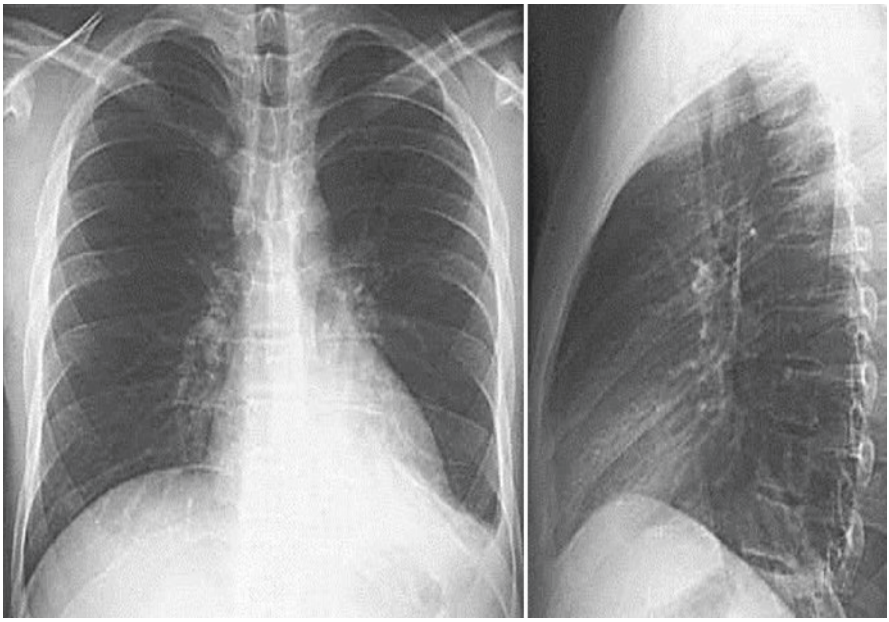

Fig. 14 On first glance, this appears to be a normal chest X-ray. However, you will notice a triangular-shaped opacity behind the heart on the frontal view. The left hemi-diaphragm is also obscured on lateral view. These are typical findings of left lower lobe atelectasis. Moral of the story: don't forget to look behind the heart!

may not be visualized [1]. CT findings of lower lobe collapse include an area of density within the posterior lower hemithorax with contact of the posterior mediastinum, posteromedial chest, and diaphragm. Rotation of the major fissure posteromedially is also seen.

4.5 Right Middle Lobe Collapse

Right middle lobe collapse may be very subtle, and often times only slight blurring of the right heart border is visualized on frontal radiograph. Other helpful findings include inferior displacement of the horizontal fissure and a density in the collapsed lobe that does not come into contact with the dome of the right diaphragm. On lateral chest X-ray, the opacified middle lobe will appear like a slice of pie overlying the cardiac shadow, making close contact with the anterior chest wall and terminating toward the hilum. The collapsed right middle lobe will assume this triangular shape on CT, with one side abutting the mediastinum. The minor fissure will rotate downward and medially, and the aerated upper and lower lobes usually separate the collapsed middle lobe from the lateral chest wall [6]. Remember location is anterior, like the heart, whose border gets blurred.

4.6 Right Upper Lobe Collapse

Collapse of the right upper lobe will demonstrate an apical opacity with superomedial displacement of the minor fissure and elevation of the right hilum. In an adult, a "golden S sign" may be present, which is associated with a right hilar mass causing obstructive right upper lobar atelectasis. The completely opacified right upper lobe creates a reverse S configuration, which is formed via superior, concave displacement of the minor fissure and an associated hilar mass producing medial convexity (Fig. 15) [7]. With collapse, chest CT will show rotation of the major fissure

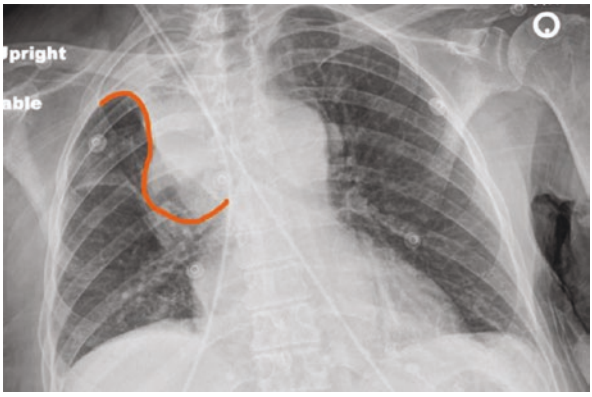

Fig. 15 The portable chest X-ray demonstrates an apical opacity with superomedial displacement of the minor fissure in the shape of a backwards "s." This is the "golden S" sign, which is a finding in right upper lobe collapse secondary to an obstructing lesion (i.e., mucus plug, tumor)

anteriorly and medially with flattening of the upper lobe against the mediastinum and anterior chest wall. The fissure may even be bowed anteriorly [6].

4.7 Left Upper Lobe Collapse

Frontal chest X-ray findings of left upper lobe collapse is very subtle and will demonstrate a veil-like density over much of the left hemithorax, which is due to lack of aeration within the collapsed and displaced upper lobe [1]. The left heart border is partially or completely obscured, and the left hilum is elevated. Sometimes, a "luftsichel sign" presents as a crescentic lucency surrounding the aortic knob (Fig. 16). This appears as an overexpanded apical segment of the left lower lobe as it positions itself between the collapsed upper lobe and the aortic arch [1]. The lateral chest X-ray will confirm the diagnosis with far displacement of the major fissure anteriorly, acting as the posterior border to the opacified, collapsed lobe (Fig. 16). Chest CT findings, as with the right upper lobe, will demonstrate anteromedial rotation of the major fissure. The overexpanded apical segment of the left lower lobe may project posteromedially to the collapsed left upper lobe, giving its posterior margin a V shape [6].

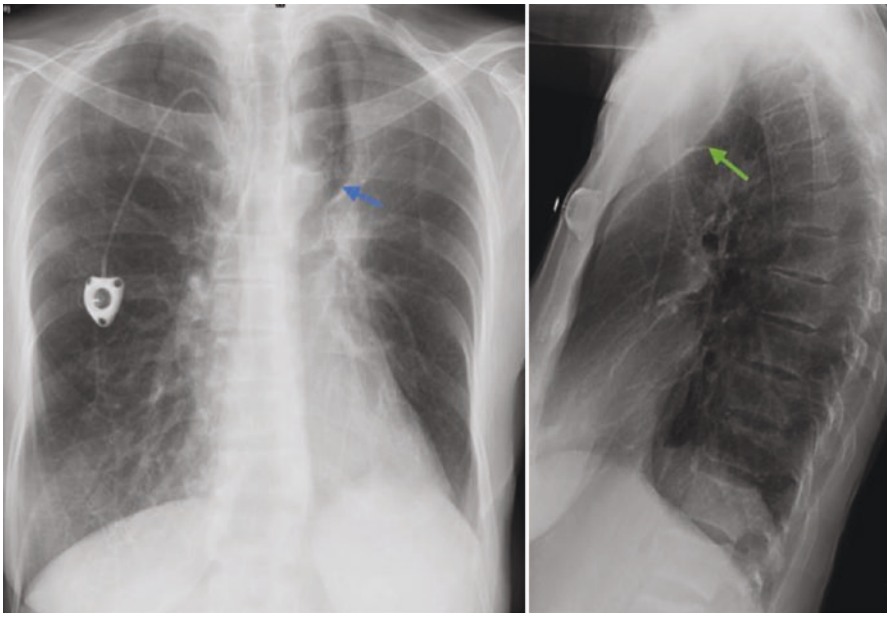

Fig. 16 The frontal chest X-ray on the left shows a veil-like density over the superior-half of the left hemithorax with elevation of the left hilum. The "luftsichel sign" is the crescentic lucency appreciated around the aortic knob; anatomically it is the overexpanded apical segment of the left lower lobe between the collapsed left upper lobe and aortic arch (blue arrow). Lateral view (right image) is diagnostic of left upper lobe collapse by demonstrating far displacement of the major fissure anteriorly (green arrow) with the left upper lobe collapsed against the anterior chest wall

4.8 Alveolar Airspace Disease

In the normal lung, the terminal bronchioles give rise to alveolar ducts that terminate into millions of alveoli or small air sacs that are bunched together like grape clusters on a stalk. Alveoli are responsible for gas exchange, or the delivery of carbon dioxide out of the body, and oxygen into the body, via the capillary beds. Alveolar airspace disease occurs when alveoli get filled with "stuff" due to a pathologic process, making them incapable of gas exchange. This stuff or abnormal material on chest X-ray has a fluffy, cloud-like appearance. On chest CT, it appears as a hazy fogginess in one or more parts of the lung parenchyma, which depicts the partial filling of alveoli while it remains partially aerated. Over time without treatment or in acutely severe cases, both imaging modalities will show consolidation or the complete filling of the alveoli. Consolidation is an area of increased pulmonary density that obscures the margins of adjacent airways and vessels on both chest X-ray and CT [8]. The abnormal material or "stuff" that replaces normal air in the alveoli may be blood, pus, water, protein, or cellular debris (or a combination of these) [1]. There is a long list of pathologic processes that can cause these findings on imaging, which we will dive into next.

4.9 Pneumonia

Pneumonia is an infection of the lung via inflamed air sacs by various bacterial, viral, and fungal pathogens and constitutes the "pus" portion of alveolar filling. A lobar pneumonia is most commonly caused by Streptococcus pneumoniae in the US in immunocompetent individuals. The "silhouette sign" is a common finding on chest X-ray in pneumonia, which describes the loss of normal lung-soft tissue interface due to the pus-filled alveoli that are anatomically contiguous with the soft tissue structure (Fig. 17) [8]. For instance, an infection in any part of the lung that touches the border of the heart, diaphragm, or aorta will obscure or obliterate that normally sharp margin. Simply, the primary reason that structures can be visualized on the chest radiograph is due to different densities of the structures imaged and their juxtaposition with very different densities, such as the air-filled lung adjacent to the blood-filled heart. When adjacent to one another, two similar densities, whether normal or not, will not be resolved as distinct. On lateral chest X-ray, the "spine sign" or loss of vertebral "fade-off" may be seen with a lower lobe pathology (including pneumonia) and manifests as a subtle increase in density in the lung superimposed on the spine on a lateral chest X-ray. Normally, the overall posterior opacity of the lateral chest X-ray decreases as your eyes march down the thoracic spine toward the diaphragm. The "spine sign" depicts a change in this normal pattern, suggesting a superimposed or coincident alveolar airspace process, if fluffy or cloudy (Fig. 18) [9].

When pneumonia is a consolidative process, the air bronchogram sign may be present on both chest X-ray and CT. This sign demonstrates the increasingly visible air-filled bronchi when surrounded by dense, consolidated lung parenchyma. However, be aware that other pulmonary processes can demonstrate this sign, including nonobstructive atelectasis, aspiration, and certain non-airway obstructing cancers like adenocarcinoma and even lymphoma [8]. For this reason,

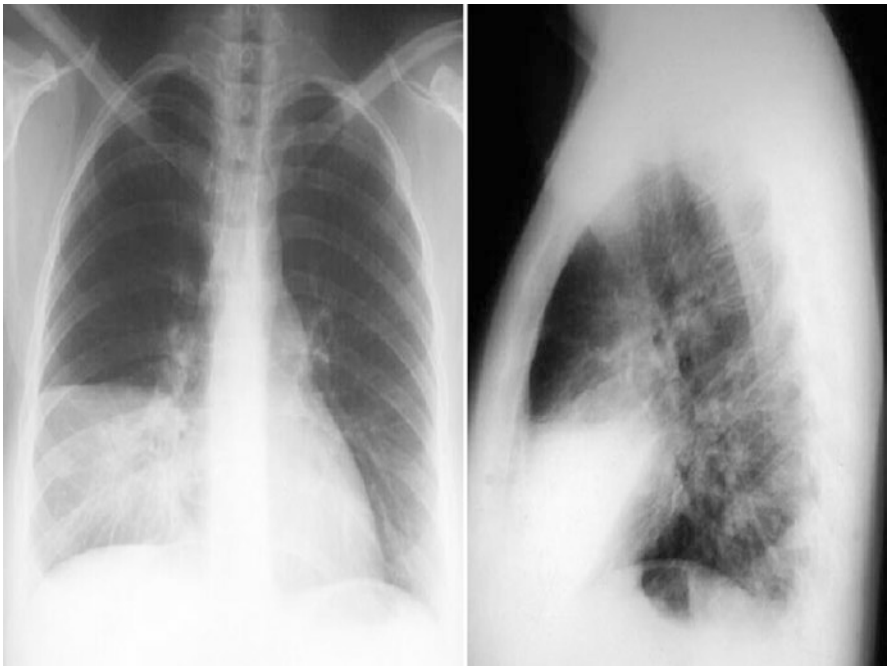

Fig. 17 On the left, the frontal chest X-ray demonstrates the "silhouette sign." This means that there is an opacity obscuring the normally sharp silhouette of the right heart border. Is this in the right middle or lower lobe? It does not obscure the right hemidiaphragm, so it is not in the right lower lobe. The accompanying lateral chest X-ray shows the opacity in the shape of a triangle (due to the intersection of the minor and major fissures), which confirms that the opacity is in the right middle lobe. This is a typical lobar pneumonia

understanding the patient's history, physical examination, and laboratory values is critical to make the correct diagnosis.

Atypical pneumonia, also known as walking pneumonia, demonstrates a pulmonary interstitial process rather than an alveolar airspace process; hence, it has a drastically different appearance on chest X-ray. The main causative organism is *Mycoplasma pneumoniae*; however, *Chlamydophila pneumoniae*, *Legionella pneumophila*, and *Coxiella burnetii* can be culprits in varying scenarios (i.e., legionella pneumonia is associated with exposure to contaminated aerosolized water, and *Coxiella* can cause Q fever pneumonia in association with exposure to livestock). Chest X-ray will show diffuse, bilateral patchy reticular or reticulonodular opacities in the lungs, more commonly in the perihilar region [10].

Discovery of an air-fluid level on chest X-ray or CT in a patient with pneumonia suggests the development of a lung abscess or empyema with bronchopleural fistula. Common organism culprits include anaerobes, Staphylococcus aureus and Klebsiella pneumoniae, as lung abscesses are most commonly associated with aspiration pneumonia and septic pulmonary emboli [8]. The air-fluid level sign is seen as an irregularly thick-walled cavity in the lung parenchyma with opacification below the "line" (depicting fluid) and lucency above it (depicting air) (Fig. 19).

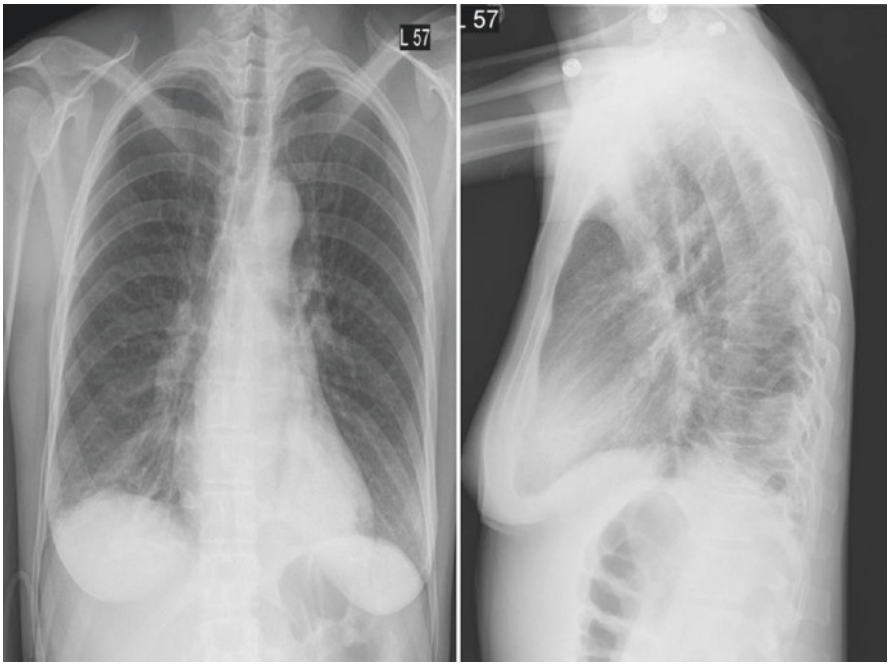

Fig. 18 The image on the left is a frontal chest X-ray. If you look closely, you will notice the retrocardiac consolidation with air bronchograms. Which lobe is this located in? The lateral chest X-ray shows a subtle increase in density in the lung superimposed on the lower thoracic spine, or the "spine sign." This confirms a left lower lobe opacity, and in this case, it was a lobar pneumonia

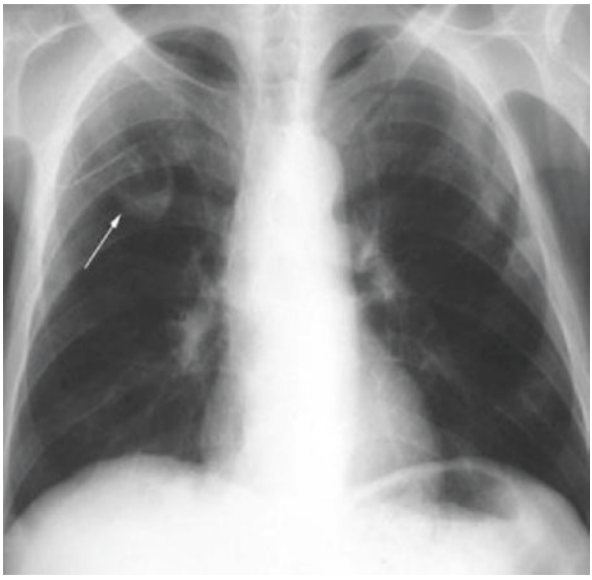

Fig. 19 The frontal chest X-ray shows a round, thin-walled lucency in the right upper lobe with an air-fluid level (white arrow). This is a lung abscess secondary to septic emboli. Lung abscesses can occur for various reasons but is most associated with aspiration pneumonia and septic emboli

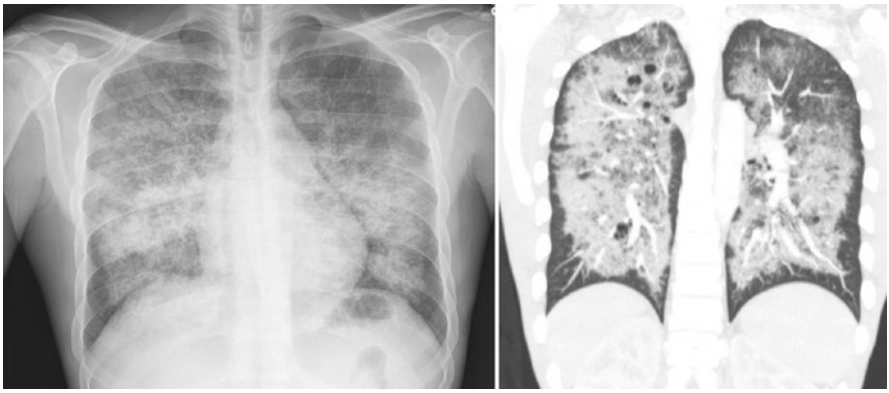

Fig. 20 The image on the right is a frontal chest X-ray demonstrating one of the classic patterns for *Pneumocystis* pneumonia, which is innumerable small pulmonary nodules (also known as miliary pattern) with areas of confluence and diffuse sparing of the periphery of the lung. If the periphery wasn't spared, you would not be able to tell the difference between miliary tuberculosis and PCP. The image on the right is a coronal CT in lung window showing these confluences of pulmonary nodules as a combination of ground glass and consolidation with small pulmonary cysts and classic sparing of the periphery

Differentiation of a peripheral lung abscess and empyema with bronchopleural fistula can be achieved by assessing the angle of their intersection with the adjacent pleural surface. An acute angle between the opacity and pleura usually implies an intraparenchymal pathology, which would be lung abscess in this case. By contrast, empyema will typically form an obtuse angle along the pleural interface and form a lenticular shape [8].

Pneumocystis pneumonia, or PCP, is seen in the immunocompromised state, including HIV and transplant patients. The causative organism is a fungus (previously classified as a protozoa) called *Pneumocystis jirovecii*, previously known *Pneumocystic carinii*. Surprisingly, chest X-ray will oftentimes appear much uglier than the patient's clinical presentation. It has varying radiographic findings, including a diffuse bilateral reticular pattern, cystic lucencies, or a miliary nodular pattern with sparing of the periphery (Fig. 20). Interestingly, there is rarely hilar lymphadenopathy or pleural effusion.

The CT findings of PCP can vary, but multiple pulmonary cysts, secondary spontaneous pneumothorax, and "crazy paving" with peripheral sparing are described. The crazy paving sign is a combination of ground glass opacity with smooth inter- and intralobular septal thickening that is thought to resemble a masonry pattern used in walkways (Fig. 20) [8].

Peripheral ground glass and airspace consolidations favoring the lower lung are the most common feature of COVID-19 from SARS-CoV-2. Other features include *crazy paving* (thick inter- and intralobular septal thickening and ground glass) and enlarged vessels within bilateral peripheral ground glass opacities (GGOs), which can rapidly progress to more consolidation patterns and even ARDS. Pleural effusions are rare with COVID-19, and presymptomatic infiltrates can exist without

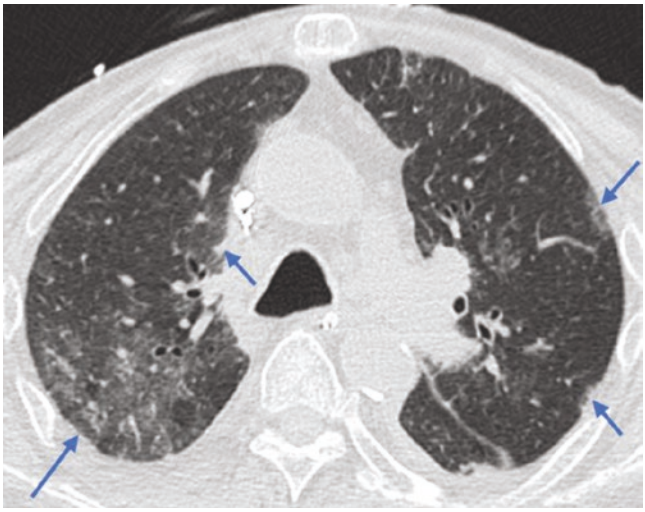

Fig. 21 The axial non-enhanced chest CT in lung window demonstrates bilateral peripheral ground glass opacities (GGO), typically seen in COVID-19 (blue arrows). The flat lines or band densities seen in the left lung are also common, as is enlargement of the vessels within the GGO. "Reversed halo" sign (dense circle of consolidation surrounding GGO) and "crazy paving" (intralobular and interlobular septal thickening, looks like a patio of irregular flat stones)

symptoms or with profound hypoxia that is worse than the remainder of the clinical appearance (Fig. 21). Although controversial, chest CT may enhance the sensitivity of RT-PCR in an outbreak setting with a high background prevalence of disease. Every single step of boards has the old sensitivity, specificity, true positive, true negative, and predictive values for both tests and disease states. Learn the two types of tables again and again and again. It gets boring, and it is easy to think you know it. But this is simply free points on exams or boards.

4.10 Pulmonary Hemorrhage

Blood is another fluid that can abnormally fill the bunches of alveoli in the lungs. It cannot be differentiated from other airspace filling processes by a single plain film or CT alone, and for this reason, the patient history is key. However, radiologists are able to infer pulmonary hemorrhage over other alveolar densities through follow-up imaging: pulmonary hemorrhage typically clears from the lungs more quickly. It is caused by trauma, pulmonary embolism, bleeding disorders, and autoimmune diseases such as Goodpasture syndrome and Wegener's granulomatosis.

4.11 Pulmonary Edema

Pulmonary edema is very common. It is the abnormal buildup of fluid in the extravascular spaces of the lungs, which can occur for many pathological reasons. For simplicity, we will discuss pulmonary edema in two categories: hydrostatic edema

and the non-hydrostatic edema. Hydrostatic edema includes cardiac, renal, and volume overload etiologies.

Hydrostatic pulmonary edema develops as the result of increased pressure in the pulmonary capillaries when forward-moving blood flow is unable to march as swiftly onward as it normally can, for example, in left-sided heart failure, a common cause for cardiogenic edema. The increased hydrostatic pressure causes the capillary walls to become leaky, allowing for fluid to seep out into the pulmonary interstitium first. The interstitium acts as scaffolding or supportive tissue in the lung, and once it is overwhelmed, the alveoli become flooded. The final step of hydrostatic pulmonary edema, when both the pulmonary interstitium and alveoli are filled, is transfer of fluid across visceral pleura into the pleural space.

Early radiographic features of mild interstitial hydrostatic pulmonary edema include apical vascular redistribution ("equalization and cephalization"). This is followed in more advancing stages by formal hydrostatic haziness about the hila, thickening of the interlobar septa, subfissural space, and peribronchial cuffing (Fig. 22). Peribronchial cuffing describes thickening +/− mistiness around the walls of a bronchus or large bronchioles. When seen end-on, it is also called the "doughnut sign" (Fig. 22). Kerley B lines are classically associated with interstitial

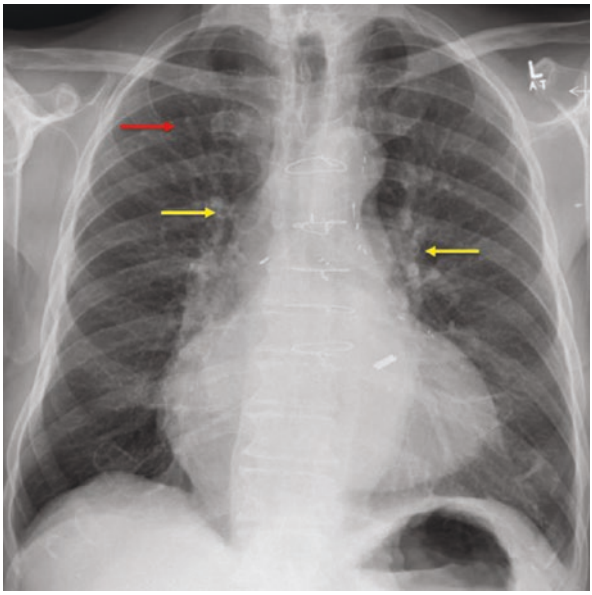

Fig. 22 This frontal chest X-ray demonstrates many classic findings in cardiogenic pulmonary edema. The first important step is to note that there are median sternotomy wires with surgical clips in the mediastinum and an enlarged cardiac silhouette. You can infer that the patient has a history of heart disease from these findings. The lungs demonstrate cephalization of the upper lobe vessels (apical vascular redistribution due to the volume overloaded state) (red arrow) and peribronchial cuffing with the "doughnut sign" (signifying thickening of the bronchioles also due to volume overload) (yellow arrows)

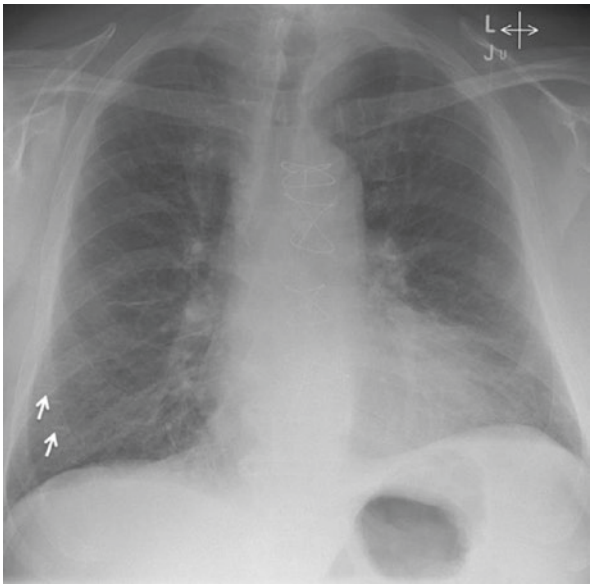

Fig. 23 Frontal chest X-ray of a patient who presented to the emergency room with shortness of breath on exertion and while laying flat, as well as bilateral lower extremity swelling. The findings include Kerley B lines (white arrows), an enlarged heart, and evidence of prior cardiac surgery (median sternotomy wires). *Kerley B lines* are enlarged lymphatics in the lower outer lung fields and are classic findings in acute decompensated heart failure and signify interstitial pulmonary edema

pulmonary edema, and it is the visualization of engorged lymphatics in the otherwise normal interlobular septa. These are thin, small lines at the periphery of the basilar lungs, extending out and perpendicular to the pleural surface (Fig. 23). Kerley A lines are longer linear densities within the upper lung, and Kerley C lines appear as short crisscrossing linear densities within the central perihilar lung resembling a mesh or a net. Even more advanced edema is alveolar fluffy edema, which is less severe than ARDS or total "whiteout." Alveolar hydrostatic edema will classically have a "bat wing" appearance, which is a fluffy haziness about the central, hilar region of the lung that extends toward the periphery in the shape of a bat or butterfly's wings (Fig. 24). An important clue to cardiogenic pulmonary edema is cardiomegaly detected by the cardiothoracic ratio, whereas this ratio may be normal in renal and volume overload causes for hydrostatic pulmonary edema. This is the ratio of the cardiac diameter to the inner edge of the ribs/pleura. An enlarged cardiac silhouette on PA chest X-ray is a ratio >0.50. As previously mentioned due to magnification, heart size cannot be relied upon in an AP chest X-ray.

On CT, interstitial pulmonary edema appears as ground glass opacities in the lungs that progress to more advanced areas of consolidation, which signifies alveolar edema or complete filling of the alveoli with the backed-up fluid. As edema worsens or especially in cases of rapidly developing severe cardiac failure, areas of airspace density will form predominantly within the central portions of lobes, sparing the periphery.

Non-hydrostatic pulmonary edema refers to a permeability edema that can occur either with or without diffuse alveolar damage (DAD). This type of edema is seen in acute respiratory distress syndrome (ARDS), transfusion reactions, renal failure, high altitude, lung transplantation, neurogenic (i.e., traumatic brain injury), reperfusion (i.e., post-thrombectomy in PE), and re-expansion (i.e., post-thoracentesis or

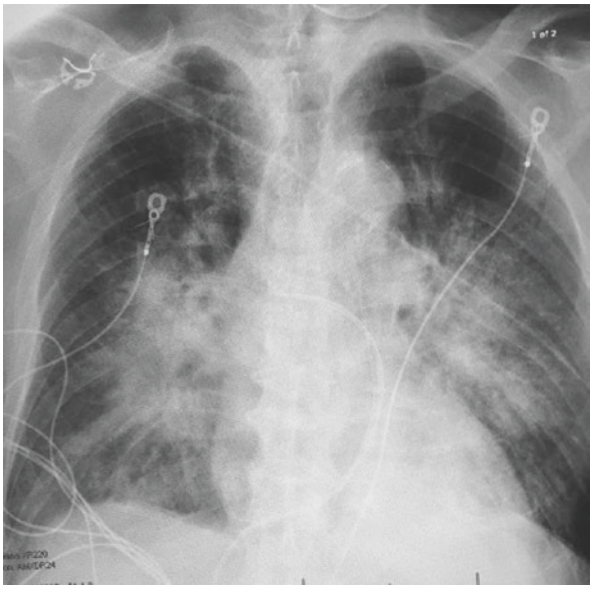

Fig. 24 Frontal chest X-ray of a patient status-post massive myocardial infarction causing acute congestive heart failure. The "bat wing" appearance can be appreciated here, which is a classic pulmonary finding for cardiogenic pulmonary edema. This appearance demonstrates filling of the alveoli with fluid, which will occur centrally and extend peripherally as the process continues without treatment. (Image Courtesy of Frederick A. Birnberg, MD)

chest-tube placement) [11]. In these processes, increased lung permeability is a result of pulmonary microvascular injury, leading to pulmonary edema [12]. DAD occurs from injury and necrosis of type II pneumocytes and alveolar endothelial cells. Because some noncardiogenic processes progress more rapidly, including ARDS, the radiographic features of hydrostatic pulmonary edema are typically absent.

Non-hydrostatic pulmonary edema on chest X-ray may demonstrate few findings of interstitial hydrostatic edema, such as interlobular septal thickening, if any, and will have a hallmark of alveolar edema as seen by hazy, fluffy opacities or consolidation (Fig. 25). In contrast to hydrostatic causes of pulmonary edema, the alveolar distribution usually affects the entire lung, not limited to the perihilar zones, or may have a more peripheral distribution [11].

4.12 Lung Cancer

Adenocarcinoma is the most common lung cancer cell type, accounting for approximately 45–50% of all lung cancers [1, 6]. The next most common is squamous cell carcinoma, followed by small cell carcinoma and then large cell carcinoma. On chest X-ray, many early lung cancers are picked up by visualization of a "solitary pulmonary nodule" or mass (Fig. 26). On CT, they have varying appearances and may be solid, ground glass, cavitary, or mixed solid and ground glass. Although definite diagnosis of lung cancer needs to be made by biopsy, the following CT findings increase the pretest probability for malignancy: irregular or spiculated margins, lobulated contour, a nodule >6 mm in size of mixed ground glass and solid components, air bronchograms, associated emphysema or fibrosis, thick-walled cavitation,

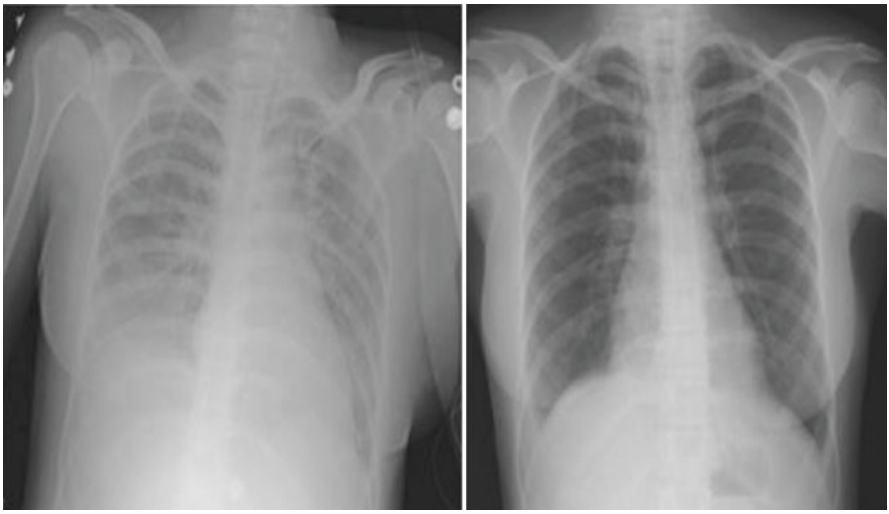

Fig. 25 The chest X-ray on the left demonstrates diffuse bilateral airspace opacities. These findings can be seen in any of the "alveolar filling" processes: this could be pulmonary "blood, pus, water, or cells" = hemorrhage, diffuse pneumonia, pulmonary edema or an infiltrative cellular process or even allergy. Note the normal heart size, which suggests a noncardiogenic cause. This patient suffered an acute transfusion reaction causing noncardiogenic pulmonary edema. The chest X-ray on the right demonstrates prompt resolution following treatment. This case shows the importance of knowing the patient's medical history and general hospital course to help narrow down your differential diagnosis for what you see. The lung fields are also big suggesting hyperinflation or COPD

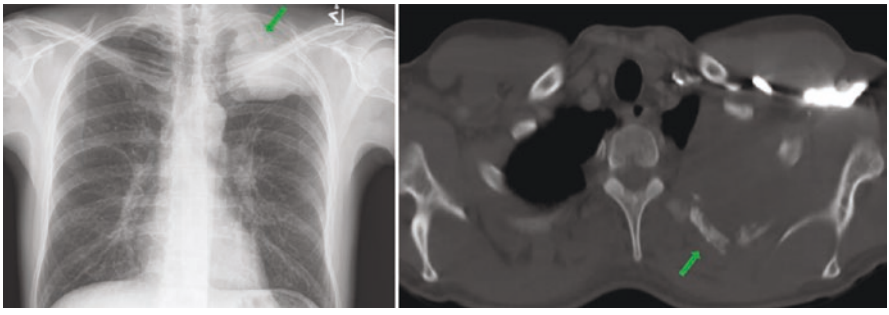

Fig. 26 The frontal chest X-ray on the left is a Pancoast tumor, which is usually squamous cell carcinoma. There is a left upper lobe mass with lucencies and irregularity to the upper left posterior ribs, representing bony erosion and destruction from the infiltrative tumor (green arrow). The axial CT on the right (in bone window) redemonstrates the apical lung mass eating into a posterior rib, causing a pathologic fracture and deformity (green arrow). The term "mass" is used because it measures greater than 3 cm. Pancoast tumor with Horner's syndrome is a favorite boards question, presenting with ptosis (small pupil), miosis (lid drop), anhydrosis (loss of sweating on face), and sinking of the eyeball into the bony eye socket cavity (enophthalmos). A classic question at multiple points of training

and a nodule diameter >2 cm [2]. Furthermore, upper lobe location is significant because it is seen in more than 70% of all primary lung cancers [6]. The solitary pulmonary nodule has a broad differential diagnosis, which includes noncancer as well. "CINTV" is a good way to make sure you hit all the possibilities: *congenital or hereditary, iatrogenic, inflammatory, infectious, neoplastic, nosocomial, traumatic, and vascular* (Table 1).

Metastasis to the lung can vary in presentation; however, they are typically round and well-defined nodules diffusely seen throughout the lung zones with a predilection for the peripheral and subpleural lung (Fig. 27) [6]. A pattern of larger size and

Table 1 Differential diagnosis for solitary pulmonary nodule using "CINTV". CINTV can also be helpful when you are thinking of general disease categories for a differential diagnosis of any imaging finding

Congenital (or hereditary)	Arteriovenous malformation, bronchogenic cyst, mucoid impaction (bronchial atresia), pulmonary sequestration
Iatrogenic	Post-lung biopsy, post-heart transplant, post-lung cancer ablation
Inflammatory	Organizing pneumonia, rheumatoid nodule, sarcoidosis, granulomatosis with polyangiitis, amyloidosis
Infectious	Angioinvasive aspergillosis, *Echinococcus*, round pneumonia, granuloma (TB, non-TB mycobacteria, fungus), lung abscess, mycetoma (aspergilloma)
Neoplastic	Carcinoma, lymphoma, lymphoproliferative disease, metastasis, lung sarcoma (chondrosarcoma, liposarcoma)
Nosocomial	Hospital-acquired pneumonia, ventilator-associated pneumonia
Traumatic	Pulmonary contusion, pulmonary hemorrhage
Vascular	Hematoma, pulmonary infarction, septic embolism

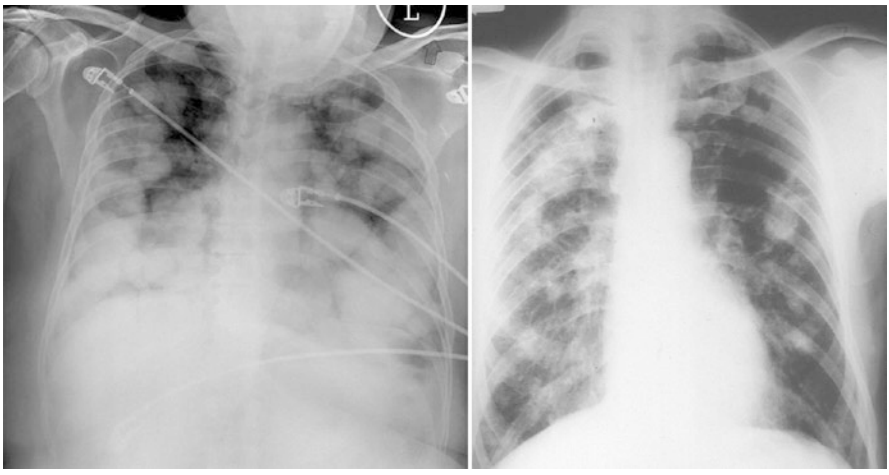

Fig. 27 The two chest X-rays are of separate patients complaining of chronic cough. Without knowing the medical history and presenting illness, you would have a very difficult time telling the disease processes apart. In both frontal chest X-rays, you see multiple bilateral solitary pulmonary nodules of various sizes. Some nodules converge on each other, appearing confluent. This is metastatic disease until proven otherwise, which is the diagnosis of the chest X-ray on the left (primary malignancy is complete hydatidiform mole). On the right, the patient has a long history of rheumatoid arthritis. These were confirmed to be pulmonary rheumatoid nodules. As is often the case, the pictures tell a thousand words, but often none of the words are the correct diagnosis

number of nodules favoring the lung bases are often associated with hematogenous metastases. Metastases are likely to range in size from small to large, which depicts the dynamic fluid process of hematogenous spread of metastatic disease. Sometimes you may even see the "feeding vessel sign," which represents the subtending artery or arteriole and illustrates their hematogenous nature [6]. Metastasis can cavitate and calcify within the nodule as well as have hemorrhage (i.e., surrounding ground glass opacity) around it.

4.13 Pulmonary Cavitation

Pulmonary cavitation is a gas-filled space that may occur within a previous consolidation, mass, or nodule that has undergone central caseous necrosis or infarction. An expansive list of pathological processes present with cavitation, including infection (i.e., septic emboli, lung abscess, necrotizing pneumonia, tuberculosis, coccidioidomycosis), pulmonary infarction, autoimmune disease (i.e., granulomatosis with polyangiitis), trauma (i.e., pneumatocele), malignancy (i.e., squamous cell carcinoma, adenocarcinoma), pulmonary metastasis (i.e., SCC of the head and neck, colon, breast, sarcoma, and cervical carcinoma), and radiation therapy. Septic emboli typically involve other solid organs too (spleen, kidney, lung, liver, brain) and, in the lungs, appear as cavitary and non-cavitary nodules with basilar lung predominance. Occasionally, septic emboli are triangular with the flat wedge on the outer surface of the lung or capsule of the organ, if associated with infarction.

On both chest X-ray and CT, pulmonary cavitation is seen as lucency within a pulmonary consolidation, mass, or nodule (Fig. 28). The "grape skin sign" is the radiographic or CT finding of a single, very thin-walled cavity with central lucency [8]. It was originally associated with chronic pulmonary coccidioidomycosis; however, it can be seen with other solitary cavitary or cystic lesions including malignancy such as metastatic sarcoma, pneumatocele, necrotizing pneumonia, and reactivation TB (especially in the upper lung zones) [8]. The "inhomogeneous enhancement sign" may allow differentiation between necrotizing pneumonia from other single cavitary lesions, and it is depicted as areas of hypoenhancing geographic regions of low attenuation. Interestingly, this is often seen before frank pulmonary abscess formation and is difficult to differentiate from adjacent pleural fluid when along the periphery [8]. In contrast, multiple pulmonary cavitations suggest processes like septic emboli, rheumatoid arthritis, granulomatosis with polyangiitis, and metastases.

The cavity wall thickness may be predictive of a benign versus malignant lesion; however, there is plenty of overlap. A recent study found that a wall thickness of less than 7 mm was highly specific for benign disease and greater than 24 mm for malignant disease [13]. These numbers are not absolute, as thin-walled carcinomas are seen.

Understanding the patient's past medical history is important because the differential diagnosis is more expansive in an immunocompromised patient and

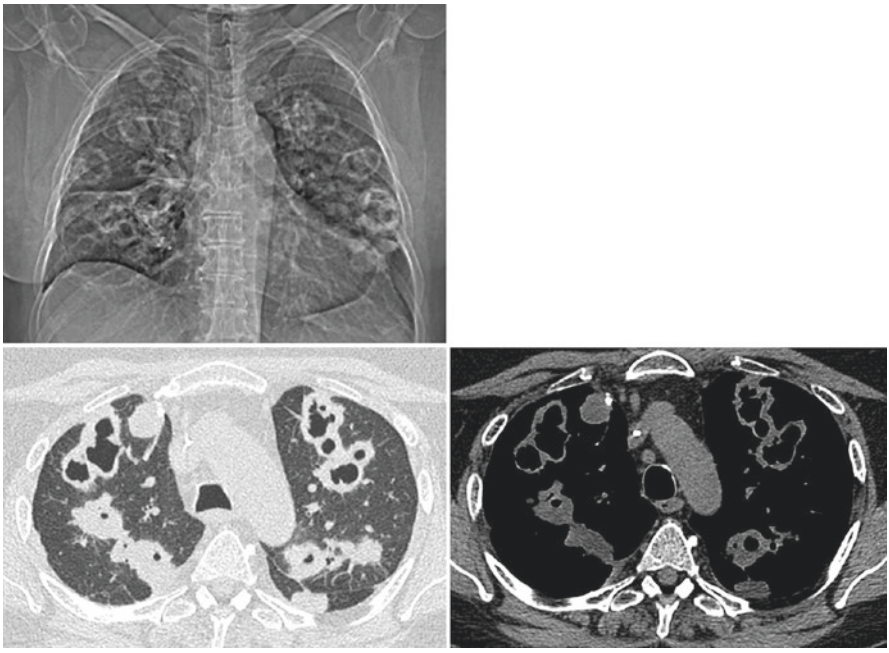

Fig. 28 This is a middle-aged patient that presented to the hospital with hemoptysis. She also admitted to bouts of sinusitis and hematuria. The scout image taken at the time of CT shows bilateral opacities with central lucencies that depict pulmonary cavitations. The CT image on the left (in lung window) shows multiple thick-walled pulmonary cavities and solid nodules. The CT image on the right is at the same level in soft tissue window. It is important to look at the lungs and mediastinum in both lung and soft tissue window to help you differentiate structures and pathologies. Notice these thick-walled pulmonary cavities are secondary to Wegener's granulomatosis (granulomatosis with polyangiitis) and are not neoplasm

includes tuberculosis, non-TB mycobacterium, endemic fungal diseases (i.e., histoplasmosis, blastomycosis), and opportunistic fungi, such as aspergillosis. Aspergillosis, caused by the fungus *Aspergillus fumigatus*, has several forms including allergic bronchopulmonary aspergillosis, aspergilloma, chronic necrotizing aspergillosis, and angio- and airway-invasive aspergillosis. Immunocompetent individuals typically present with the former two entities, although aspergilloma is often the result of local airway and parenchymal damage and depressed local immunity, and immunocompromised the latter two. The "Monod sign" on CT and chest X-ray is a finding typical for a healthy person who develops an aspergilloma or mycetoma within an underlying pulmonary cavity (Fig. 29). It describes the air that surrounds the aspergilloma/mycetoma, or fungus ball, that is sitting in a cavitation. Due to chronic inflammation and associated hyperemia, it often presents with hemoptysis. The "air crescent sign" is seen in immunocompromised individuals and describes the crescent of air that can be seen in chronic necrotizing aspergillosis and angioinvasive aspergillosis. It is important to note that this intracavitary nodule

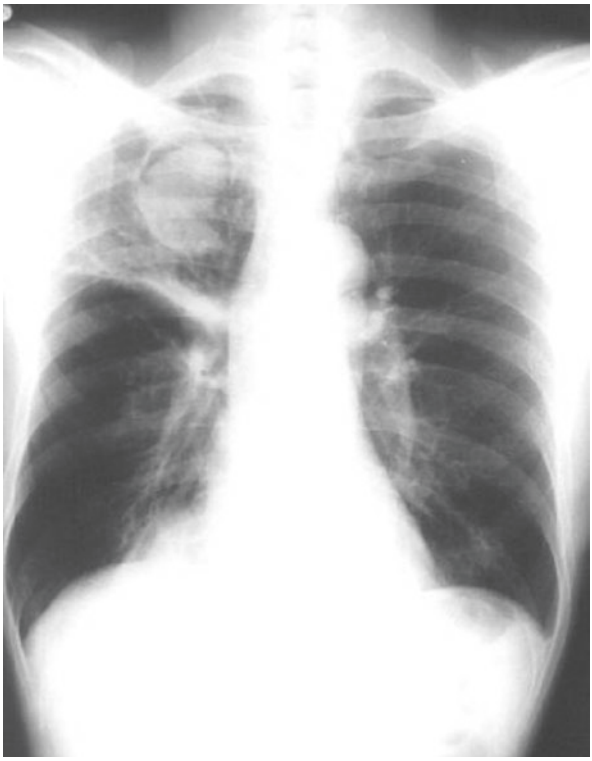

Fig. 29 This is a previously healthy patient who presented with hemoptysis. On frontal chest X-ray, there is a thin-walled cavity in the right upper lobe with the "Monod sign" surrounding a fungus ball inside the cavity. The patient was diagnosed with aspergilloma

represents necrotic, retracted lung tissue and suggests a favorable patient prognosis as it shows recovery of invasive disease [8]. The "halo sign" is a CT finding of a peripheral rim of ground glass opacity around a pulmonary nodule, mass, or cavity and represents hemorrhage. Although not specific to active angioinvasive aspergillosis, it is highly suggestive in a neutropenic patient with fever. The differential for the "halo sign" includes other infections (i.e., mucormycosis, *Candida*, *Pseudomonas*, HSV, CMV), granulomatosis with polyangiitis, hemorrhagic metastasis, and Kaposi sarcoma[8]. A "reverse halo sign" can be seen in COVID-19 as GGO surrounded by a circle of consolidation.

An immunosuppressed individual is more likely to have progression of primary TB or reactivation of the disease, both of which may result in cavitation. TB and non-TB mycobacterial cavities are typically irregularly shaped with internal septation, rarely demonstrate air-fluid levels, and are often surrounded by satellite nodules [6, 14]. However, tuberculosis has many faces and can also present in a miliary pattern from lymphohematogenous spread (innumerable tiny nodules randomly distributed throughout the lungs) in the setting of progressive primary or post-primary disease or, simply, as a solid pulmonary nodule at the lung apex (Fig. 30), representing healed disease.

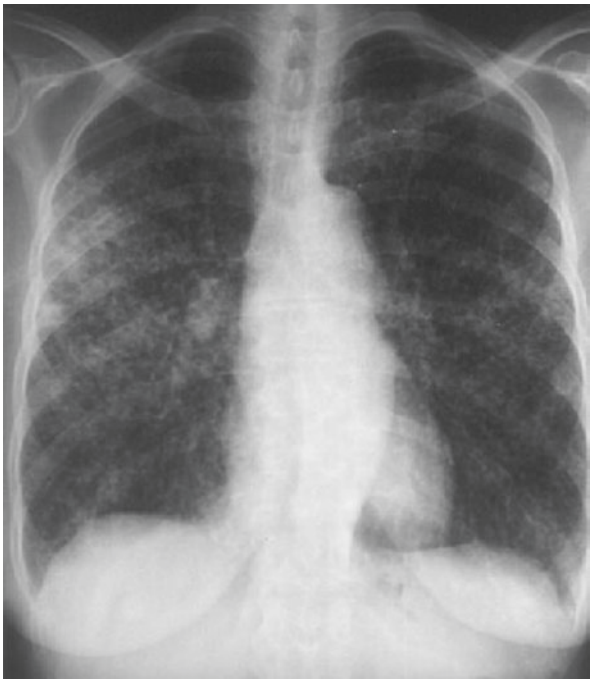

Fig. 30 Frontal chest X-ray demonstrates innumerable small solid pulmonary nodules with confluence of nodules in the mid-right lung field. This is an immuno-compromised patient that was diagnosed with miliary tuberculosis, one of the many faces of TB in the chest. Note the very small and uniform size of the nodules, as opposed to metastasis that typically demonstrates multiple pulmonary nodules of varying sizes (see Fig. 26)

4.14 Solitary Pulmonary Nodule

A solitary pulmonary nodule is a focal lung lesion (often incidentally seen) on chest X-ray or CT that is less than or equal to 3 cm in size (Fig. 31). The term "mass" refers to a lesion greater than 3 cm. The differential diagnosis is quite extensive and includes congenital lesions (i.e., arteriovenous malformation), neoplasm, metastasis, granuloma, infection, benign tumors (i.e., hamartoma), autoimmune (i.e., rheumatoid nodule, sarcoidosis, granulomatosis with polyangiitis), and round atelectasis. Non-pulmonary etiologies can trick you, some of which include healed rib fractures, nipple shadow, or even a neurofibroma on the skin (Fig. 32).

The Fleischner Society guidelines for follow-up of incidentally detected pulmonary nodules are based on nodule appearance, nodule size, and patient risk factors (history of smoking). It excludes patients with preexisting cancer, immunocompromised state, and an age of less than 35. Some sub-solid nodules may need to be followed for stability out to 5 years before excluding cancer.

4.15 Emphysema

Emphysema is the abnormal permanent enlargement of lung airspaces due to destruction of the respiratory bronchioles and ducts and sacs of alveoli dependent on emphysema type. It is a common feature in smokers consistent with chronic

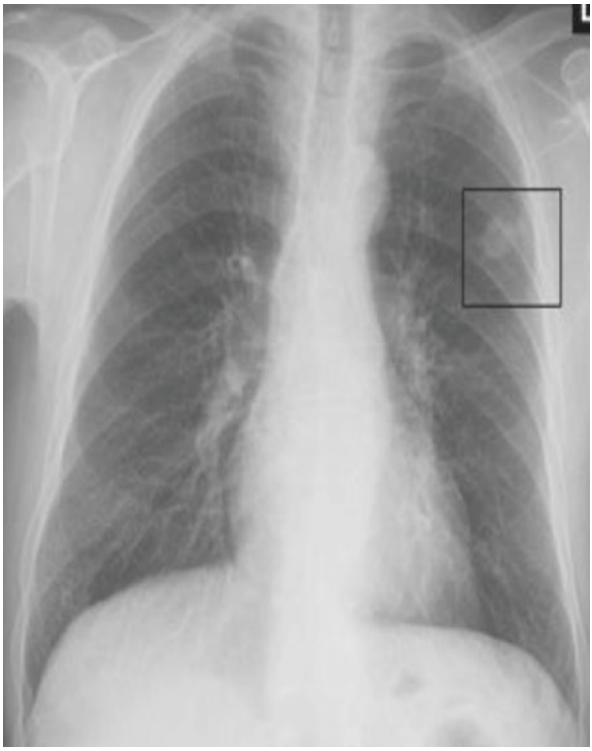

Fig. 31 Frontal chest X-ray shows a solitary pulmonary nodule in the left upper lung field with spiculated margins (black box). This lesion was biopsy-proven lung cancer

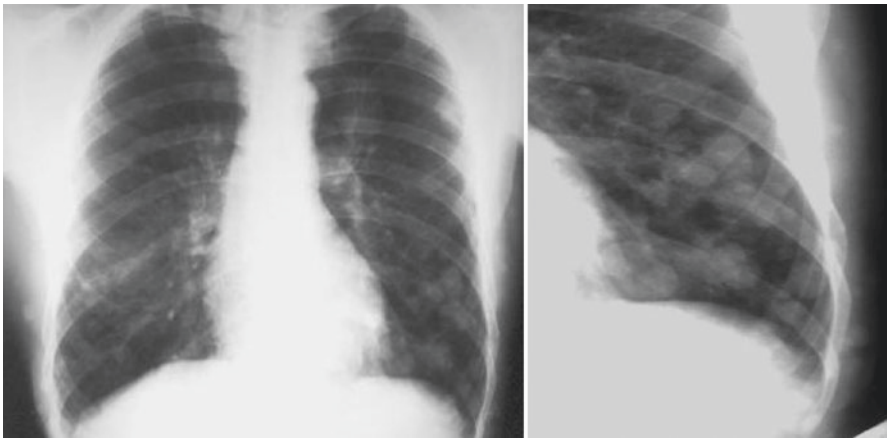

Fig. 32 Frontal chest X-ray on the left demonstrates multiple bilateral round opacities overlying the lungs, most prominently in the left lower lung field. If you look closely, you will notice that the nodules also lie over the lateral chest walls in the soft tissues. Do not mistake these for bilateral pulmonary nodules: these are neurofibromas on the skin and are associated with neurofibromatosis. Anything in the line of the X-ray can cause findings. Do not assume! Your eye and brain want to file findings into neat little familiar labels

obstructive pulmonary disease or COPD; however, this entity is typically detected clinically with confirmation of diagnosis by lung spirometry. Chest X-rays in emphysema or COPD may show lung overinflation, as seen by flattening of the diaphragm, widely spaced ribs, and a narrow, centrally situated heart. Lateral chest-x ray may demonstrate an increased anteroposterior diameter of the chest and sternal bowing. Pulmonary parenchymal abnormalities on chest X-ray are depicted by irregular areas of lucency and decreased vascular markings [6].

CT can differentiate between centrilobular, panlobular and paraseptal emphysema, which each has differing causes. The centrilobular pattern is predominantly in the upper zones of each lobe and has a patchy distribution, while the paraseptal pattern is located adjacent to the pleura and septal lines (Fig. 33). Both are strongly associated with smoking. *Panlobular emphysema* is largely located in the lower lobes and is seen particularly in alpha-1-antitypsin deficiency (and intravenous injection of methylphenidate, also known as Ritalin lung) [15]. In all three subtypes of emphysema, the lucencies are not bounded by a visible wall. Bullae may be seen with more severe disease, which are very thin-walled, air-filled structures measuring greater than 1 cm (Fig. 33). The differential diagnosis for emphysema and bullae is cystic lung disease (i.e., pulmonary Langerhans cell histiocytosis, lymphangioleiomyomatosis), all of which have visible walls.

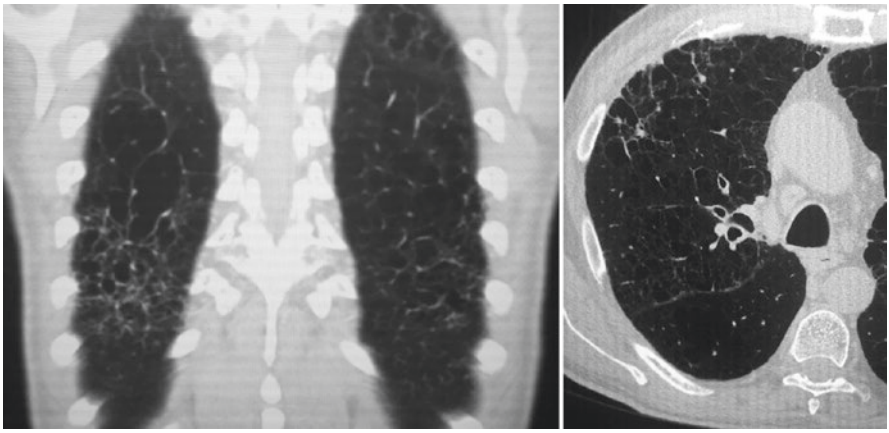

Fig. 33 The image on the left is a coronal CT (in lung window) looking at the posterior parenchyma of the lungs. Here you see mainly centrilobular emphysema with bullae predominantly in the upper zones of the lungs; however, looking closely, you will also notice evidence of paraseptal emphysema along the periphery abutting the pleura. This is better appreciated on the axial CT to the right, where you see both centrilobular and paraseptal emphysema in a patient with COPD and history of smoking. (Images Courtesy of Frederick A. Birnberg, MD)

4.16 Pleural Effusion

A pleural effusion is fluid accumulation between the visceral and parietal pleura, also known as the pleural space. On an upright chest X-ray, the most common appearance is blunting of the costophrenic angles, obscuring of the normally sharp edges of the interface between the lateral diaphragms and lower lung fields. The "meniscus sign" will eventually be seen with moderate-sized pleural effusions, which is demonstrated by a lateral opacity touching the chest wall that gently slopes medially as it reaches the diaphragm (Fig. 34). An encysted or loculated pleural effusion may show the "meniscus sign" via an oval or rounded opacity between the pleural lining in the horizontal fissure on frontal chest X-ray or horizontal and oblique fissures on lateral chest X-ray. It can be mistaken for a lung tumor or sometimes known as a "pseudotumor." A large pleural effusion (approximately 5 L) can fill the entire pleural space and be seen as complete hemithorax whiteout on chest X-ray. Typically, the mediastinal structures will shift away from the effusion since it is a space-occupying process. Hemithorax whiteout is commonly seen in other

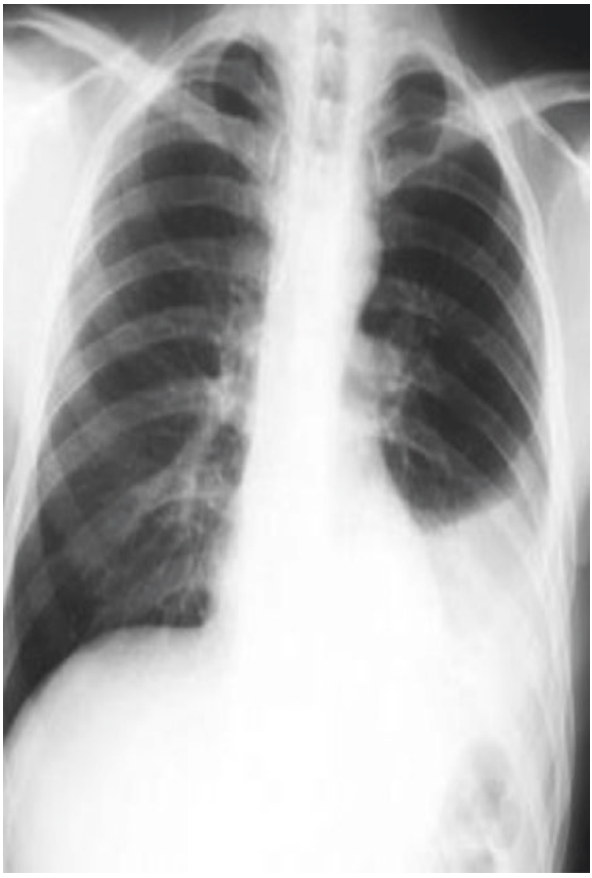

Fig. 34 The frontal chest X-ray shows blunting of the left costophrenic angle and opacity of the left lower lung field obscuring the left diaphragm and left heart border. Also seen is the "meniscus sign" with a gentle medial sloping of the pleural fluid. This is a moderate pleural effusion without evidence of mediastinal shift

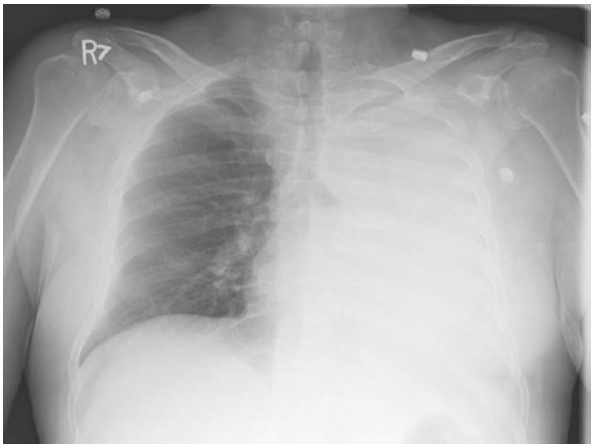

Fig. 35 Does this frontal chest X-ray show complete left lung atelectasis or large left pleural effusion? Effusions tend to occupy space and push everything to the opposite side, as seen through mediastinal shift away from the hemithorax whiteout. In this case, it appears the midline structures (i.e., trachea) are being pulled toward the opacified left hemithorax instead of away. Notice the abrupt opacification of the left mainstem bronchus, or the "bronchus cutoff sign." The pathology in this case is in the left mainstem bronchus (i.e., tumor or mucus plug) that caused complete left lung atelectasis

diseases, including pneumonectomy and total lung collapse; however, in these processes the mediastinum will be pulled toward the side of whiteout (Fig. 35).

Pleural effusion can be subtle to imperceptible on supine chest X-ray, depending on its size. If not loculated, it will layer out in the posterior or dependent pleural space and may be seen as a "mild" hemithorax density and with large effusions whiteout. For instance, the hemithorax with layering pleural fluid will appear slightly whiter or paler gray than the normal contralateral side.

There are two types of pleural effusion based on their content; however, the chest X-ray alone is unable to distinguish them. A transudative pleural effusion generally occurs when there is an increase in hydrostatic pressure or decrease in capillary oncotic pressure in the lungs. Some examples include cardiac failure, nephrotic syndrome, and cirrhosis. An exudative pleural effusion is from an increase in microvascular permeability or an alteration in the pleural space drainage to lymph nodes. Some examples are cancer, pulmonary emboli and infarction, pneumonia, mesothelioma, and systemic lupus erythematous.

An empyema, or infected pleural effusion, is most commonly secondary to pneumonia. It is essentially indistinguishable on chest X-ray from a pleural effusion. However, on contrast-enhanced CT, you will see a "split pleura sign," which represents thickening and enhancement of the inflamed visceral and parietal pleura outlining a pleural fluid collection (Fig. 36) [8]. It is not specific for an empyema and can depict a loculated fluid collection or other exudative pleural effusion, so correlation with clinical history and fluid analysis by thoracentesis would be needed to establish accurate diagnosis. Ultrasound may show overall increase in fluid echogenicity, loculations, floating septations, or compartments.

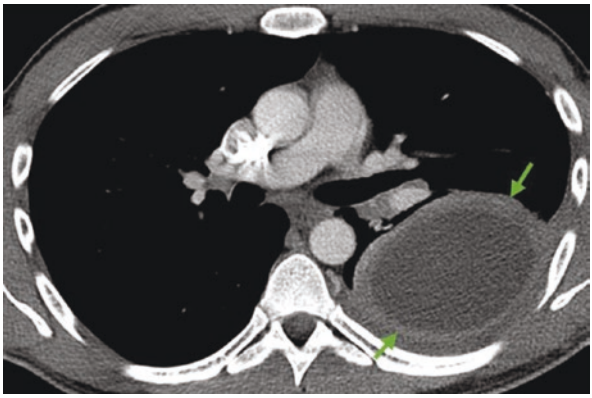

Fig. 36 Axial contrast-enhanced CT demonstrates the split pleura sign, which shows thickened and inflamed visceral and parietal pleura outlining a low-attenuating fluid collection in the pleural space, a finding seen in empyema or loculated fluid collection (green arrow)

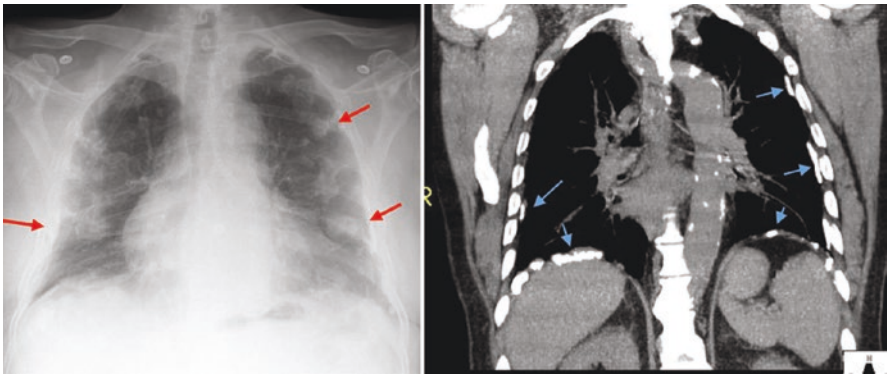

Fig. 37 The frontal chest X-ray on the left shows multiple peripherally and diaphragm-based, ill-defined opacities with the "incomplete border sign," which was found to be benign pleural plaques. It is difficult to differentiate these lesions from lung parenchymal lesions without a lateral chest X-ray or chest CT. The CT on the right (different patient) also shows benign pleural plaque, as seen by focal areas of pleural thickening with coarse calcifications (blue arrows)

4.17 Pleural Plaques

Pleural plaques are focal areas of thickening of the parietal pleura and are strongly associated with inhalation exposure to asbestos. They are characteristically in the lower 1/3 of the posterior and lateral thorax. They exhibit the "incomplete border sign" on chest X-ray, where the peripheral opacity will have an inner, well-defined border but an ill-defined outer margin (Fig. 37) [16]. Pleural plaques can calcify, and when they do, they become much more readily visible on chest X-ray. Other important etiologies to consider include pleural tumors or metastasis, which usually is accompanied by malignant effusion. Chest CT is the modality of choice for assessment of pleural plaques and will demonstrate nodular soft tissue densities without invasive features or concomitant pleural effusion (Fig. 37).

Malignant pleural mesothelioma is also strongly associated with asbestos, but unlike asbestos-related lung disease, it is not dose-dependent. On chest X-ray, it can be seen as a pleural-based opacity that may extend around or fully encase the lung. This can cause a reduced volume for the affected hemithorax, triggering an ipsilateral mediastinal shift [17]. On CT, mesothelioma will present as a pleural mass or nodular thickening of soft tissue attenuation along the pleura and fissures. Local invasion may be seen, involving the chest wall, diaphragm, and mediastinum. Calcification is seen in about 20% of cases [17]. Asbestosis plus smoking = *cancer*.

5 Pulmonary Vasculature

5.1 Pulmonary Embolism

Pulmonary embolism (PE) is the third most common acute cardiovascular disease following myocardial infarction and stroke [18]. There are few diagnostic tests for acute PE, including D-dimer, ventilation-perfusion scintigraphy, conventional pulmonary angiography and, indirectly, lower extremity venous duplex ultrasound. However, CT pulmonary angiography remains the gold standard for diagnosis due to its high sensitivity and specificity (100% when involving the main pulmonary artery branches and greater than 90% for segmental emboli) [6]. In conjunction with Well's criteria, suspected patients are risk stratified into probability of acute PE and spared the radiation exposure when deemed low pretest probability (Table 2).

The chest X-ray does not have a role in definitively diagnosing acute PE but can clue you in to the disease with the presence of the Westermark sign and Hampton's hump. However, it is important not to exclude acute PE in a patient with intermediate to high pretest probability and absence of these findings on chest X-ray. The *Westermark sign* is a hyperlucent area of the lung due to decreased lung perfusion distal to an obstructed vessel. *Hampton's hump* is a wedge-shaped density (evolving infarct) laying on its side, with its base against the lateral chest wall pleura and medial margin facing the hilum (Fig. 38) [1].

Table 2 Modified Well's criteria for clinical assessment for acute PE

Presence of:	Points
Clinical symptoms of DVT (unilateral leg swelling, pain with palpation)	3
Other diagnosis is LESS LIKELY than PE	3
Heart rate >100	1.5
Immobilization of ≥3 days (or surgery within the last 4 weeks)	1.5
History of DVT/PE	1.5
Hemoptysis	1
History of malignancy	1
PE likely	>4
PE unlikely	≤4

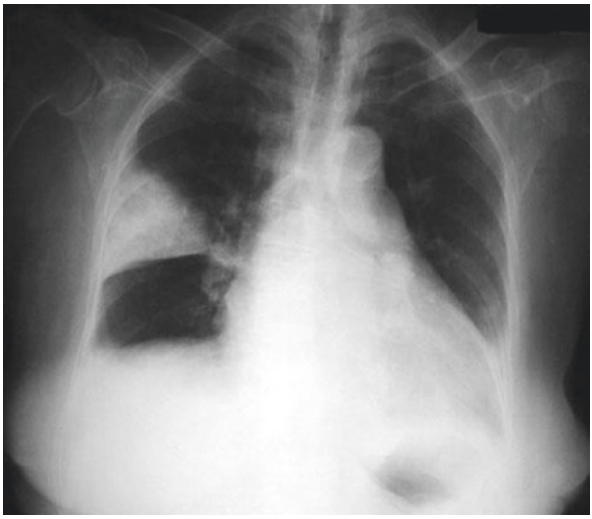

Fig. 38 Frontal chest X-ray shows a wedge-shaped opacity of the right-upper lobe bordering the minor fissure. This is Hampton's hump, or a wedge-shaped infarction of the lung parenchyma from pulmonary embolism. There is also a small right pleural effusion

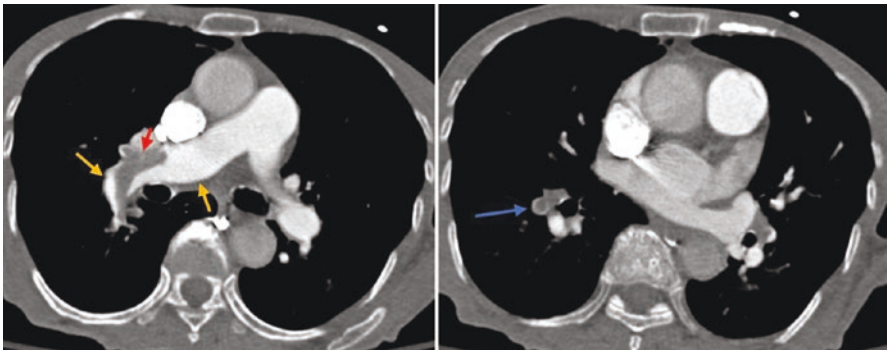

Fig. 39 CT pulmonary angiography in axial view on the left shows a large filling defect with smooth borders in the right main pulmonary artery extending into the superior trunk and the interlobar artery consistent with acute pulmonary embolism (red arrow). When a PE straddles two vessels, it is termed a "saddle embolus**.**" Notice perhaps some dilatation of the affected arteries. Acute PE can cause right heart strain, and chronic PE can show up a "pruning" or rapid tapering of the pulmonary arterial tree (yellow arrows). The image on the right shows the doughnut sign (or polo mint sign), which represents acute PE completely surrounded by contrast in the lumen (blue arrow) or an enhancing vessel wall

On CT pulmonary angiography, acute PE appears as a filling defect in the pulmonary artery with failure to enhance the entire artery lumen, with possible focal caliber increase when compared to adjacent patent arteries (Fig. 39). This large filling defect can occur anywhere in the pulmonary arterial circulation, and when it straddles a bifurcation, such as the left and right main pulmonary arteries, it is called a "saddle embolus." The "polo mint" or "doughnut sign" is a partial filling defect that is fully surrounded by contrast material when the artery is viewed end on (Fig. 39) [6, 18]. When the partially occluded vessel is viewed longitudinally, it creates the

"railway track sign." When the partial filling defect is along the periphery of the lumen, it will form acute angles with the arterial wall. The lung parenchyma may demonstrate peripheral wedge-shaped hyperattenuation and linear bands, which represent infarction or hemorrhage. A pulmonary embolus that is chronic will appear as an intraluminal filling defect or wispy, thin linear filling defects known as pulmonary artery webs[6]. Of note, the artery will appear smaller than adjacent arteries, the thrombus forming obtuse angles with the arterial wall. Occasionally, there may be extensive bronchial or systemic collateral vessels [18].

On CT, it is critical to assess the heart for findings of acute right heart strain from pulmonary arterial hypertension secondary to high burden PE due to the higher likelihood of hemodynamic decompensation or massive PE. Evidence includes right ventricular enlargement to a size larger than the left ventricle and pulmonary arterial trunk enlargement to a size larger than the aorta. The interventricular septum may be flattened or demonstrate paradoxical bowing, where the septum curves toward the left ventricle due to the elevated right heart pressures. [19] Echocardiogram (or heart ultrasound) is the more conventional way to diagnose right heart strain and may give right heart pressure estimates.

6 Heart and Pericardium

6.1 Cardiac Devices

The chest X-ray is the preferred imaging modality to evaluate cardiac conductive devices for anatomic location, lead wire integrity and complications. A pacemaker manages cardiac dysrhythmias by generating electrical impulses to stimulate coordinated atrial and ventricular contraction. It is composed of three main parts: a pulse generator, an electrode, and lead wires. [20] On frontal and lateral chest X-ray, the pulse generator will overly the anterior chest wall in the subcutaneous tissues. A dual chamber pacemaker has two leads that course from the generator intravenously (usually in the subclavian vein) to the heart. One lead's electrode will terminate in the right atrial appendage, and the second will terminate at the apex of the right ventricle. In a biventricular pacing system, a third lead will be present and terminates in the coronary sinus to pace the left ventricle [1].

On lateral chest X-ray, the atrial lead will make a smooth "U" or "J" shape overlying the heart, and the electrode tip will be angled superiorly in the appendage of the right atrium. The ventricular lead tip should be directed anteriorly and inferiorly, making about a 45-degree angle with the anterior chest wall and diaphragm (Fig. 40).

An implantable cardioverter defibrillator (ICD) contains a defibrillator unit that delivers an electrical shock to abort dysrhythmia and return the heart to sinus rhythm. Its single lead takes an identical course to the ventricular lead of a pacemaker, terminating at the right ventricular apex. However, the ICD lead has proximal and distal shock coils that appear as thick "sleeves" or patches (Fig. 41). ICDs and pacemakers can be utilized in a single device, so it is important to understand the parts of each device and their normal appearance. Device malfunction can occur

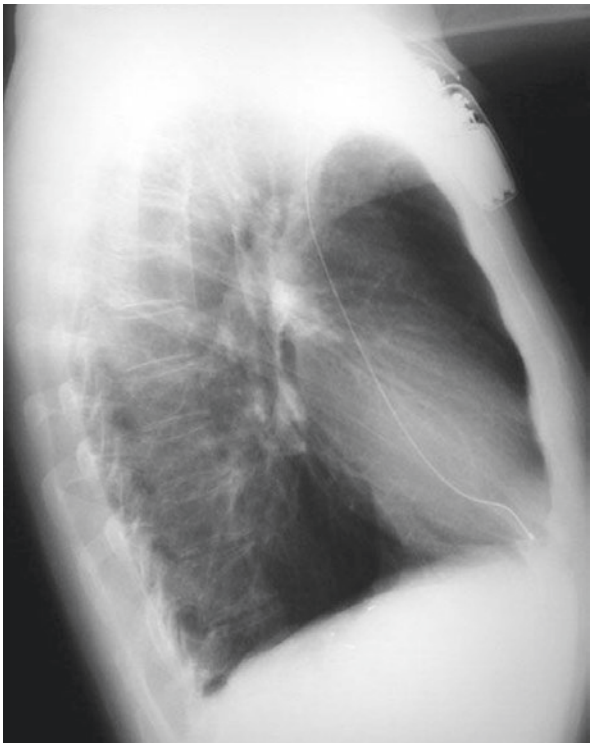

Fig. 40 The lateral chest X-ray shows a single chamber pacemaker in the anterior chest wall coursing to the correct location in the apex of the right ventricle. The ventricular lead makes an approximate 45-degree angle with the chest wall and diaphragm. When assessing cardiac devices, don't forget to look for wire fractures or kinks that imply complication

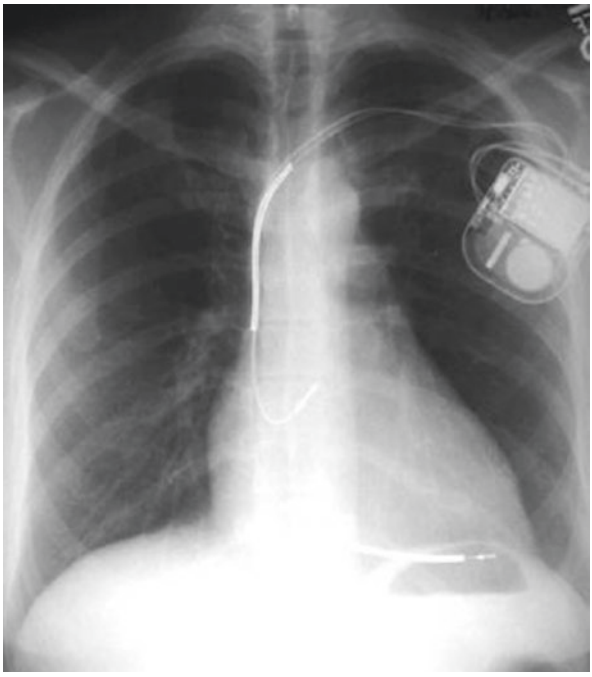

Fig. 41 Frontal chest X-ray shows a dual chamber ICD in good position. The atrial lead terminates in the right atrial appendage, and the ventricular lead terminates in the right ventricular apex. To confirm these positions, a lateral chest X-ray is necessary. Note the thick "sleeves" or patches of the ICD leads. Pacemaker leads do not have these shock coils, which is a way to tell them apart

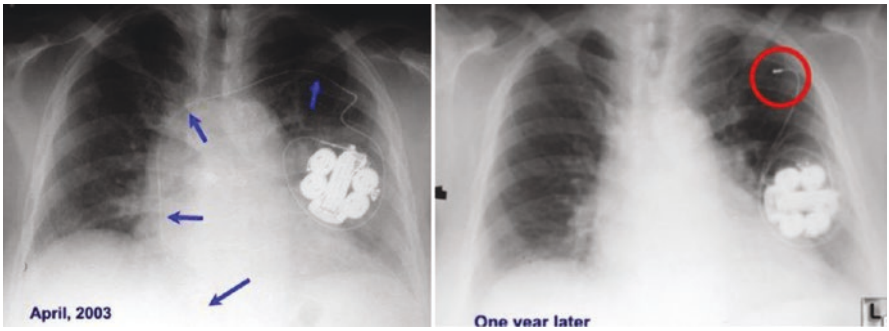

Fig. 42 These are two frontal chest X-rays taken 1 year apart. The chest X-ray on the left shows a single chamber pacemaker with the lead terminating in the right ventricle in appropriate position (blue arrows). The chest X-ray on the right shows the single lead in a new position overlying the left lung apex, likely at the distal subclavian vein (red circle). The chest wall generator has also been rotated compared to the prior image. This is Twiddler syndrome, which is a malfunction of the pacemaker due to twisting or twirling the generator in the chest wall pocket, typically done by the patient

for various reasons, and on chest X-ray you may see leads that are kinked, pinched, twisted, or fractured (Fig. 42). Complications of cardiac conductive device placement should be assessed on imaging and include pneumothorax, lead dislodgement out of proper anatomic location, and heart chamber perforation. [20]

6.2 Chamber Enlargement

There are a plethora of pathologies that cause isolated cardiac chamber enlargement and cardiomyopathy resulting in four-chamber enlargement, including valvular disease, ischemia, toxic insult, metabolic, infectious, and idiopathic processes. On frontal chest X-ray, left atrial (LA) enlargement is suggested by prominence of the left atrial appendage in the cardiac silhouette, just inferior to the main pulmonary trunk. Left atrial enlargement can result in the "ballerina sign," which is splaying of the carina to greater than 100 degrees [1], although splaying can also occur with subcarinal adenopathy. A double density inside the right heart border, astutely called the "double density sign," depicts the abnormally prominent border of the LA behind the right atrium (RA) outlined by air in the adjacent normal lung. For confirmation, one can measure the distance from the right lateral margin of the double density to the midpoint at the undersurface of the left bronchus. If this measurement exceeds 7 cm, it is likely that the LA is enlarged (Fig. 43) [21]. On lateral chest X-ray, the "walking man sign" will be seen, which is displacement of the left main bronchus posteriorly by the enlarged LA, with the bronchi creating the right and left legs [21]. As the left mainstem bronchus is posteriorly displaced, the left upper lobar bronchus can similarly be displaced and often misaligned with the right upper lobe bronchus and tracheal axis on the lateral radiograph. An additional suggestion is an opaque impression on an air- or contrast-filled esophagus anteriorly (Fig. 43). The size of the left atrium can be estimated on chest CT by measuring the left atrium (the most posterior chamber) in AP diameter (Fig. 44). Left atrial enlargement is

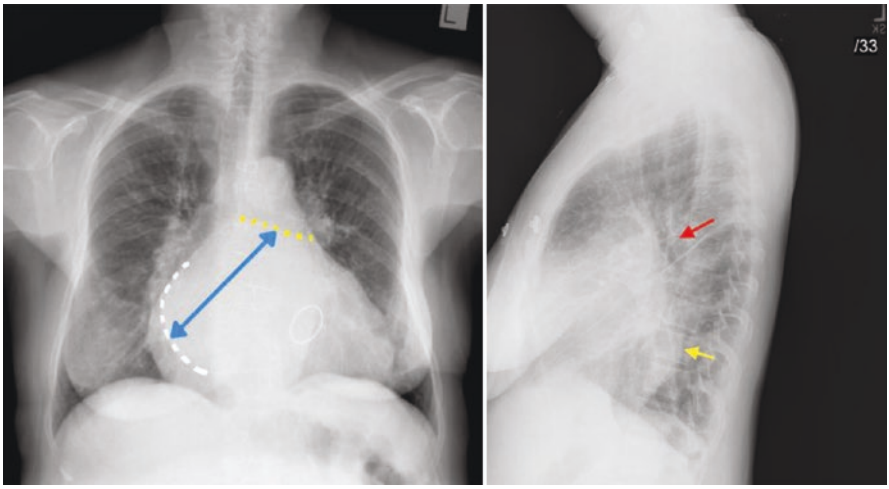

Fig. 43 Frontal chest X-ray on the left demonstrates an enlarge left atrium outlined by yellow and white dots. A left atrium that is greater than approximately 7 cm (blue arrow) suggests LA enlargement. The "double density sign" of LA enlargement is outlined by the white dots along the right heart border and signifies the abnormally prominent border of the LA behind the RA. The lateral chest X-ray on the right demonstrates the "walking man sign," which is displacement of the left main bronchus posteriorly by the enlarged LA making the trachea and its main bronchi appear as a walking man (red arrow). Notice an opaque impression on the esophagus and lower thoracic spine portraying LA enlargement

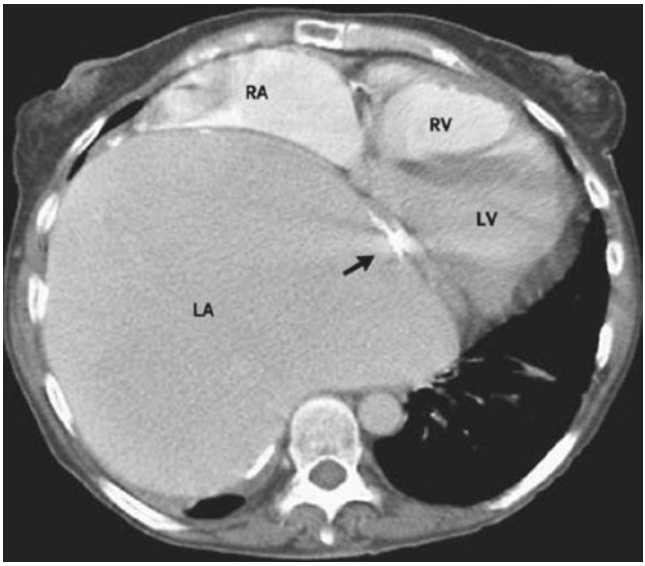

Fig. 44 Axial nonenhanced chest CT on soft tissue window shows huge LA enlargement due to failure of a calcified mitral valve. The left atrium, which is easily estimated to be much larger than 4.1 cm in AP diameter here, is compressing the remainder of the chambers. The heart takes up approximately 80% of the thorax

defined as an AP diameter greater than 4.0 cm in women and 4.1 cm in men. For more accurate measurement, a gated cardiac CT would be needed.

RA enlargement is less common than LA enlargement and much more difficult to assess. There will be prominence of the right heart border on frontal chest X-ray, measuring greater than 5.5 cm from the midline of the thoracic spine to the right [21].

Left ventricular (LV) enlargement will feature a prominent rounding of the inferior aspect of the left heart border with the addition of a downward-pointing apex on frontal chest radiographs. With LV enlargement on lateral radiograph, the posterior and inferior cardiac margin will project greater than 1.8 cm behind the inferior vena cava shadow when measured at a point 2 cm up from the diaphragm, known as the Rigler-Hoffman rule. Right ventricular (RV) enlargement often accompanies RA enlargement. It can give a similar appearance to LV enlargement, but the most prominent portion is the upper part of the left heart border with elevation of the cardiac apex [1]. On lateral view, an RV that fills too much of the retrosternal clear fatty space is suggestive of chamber enlargement [21].

6.3 Pericardial Effusion/Cardiac Tamponade

The appearance of fluid in the pericardial sac is difficult to distinguish from simple cardiac chamber enlargement on chest X-ray. Although a pericardial effusion may produce a globular cardiac outline known as the "water bottle sign" on frontal X-ray, this finding will not occur until a larger effusion is present (approximately 250–500 cc accumulation of fluid) (Fig. 45) [6]. A rapid increase in cardiac silhouette size without any changes in the lungs is a superior indicator of pericardial effusion. On lateral chest X-ray, a pericardial effusion will appear as a vertically oriented opaque line between two parallel lucent lines, one anteriorly and directly behind the sternum (i.e., paracardial fat) and the other posteriorly (i.e., epicardial fat). This is known as the "oreo cookie sign" and can be better delineated on a chest CT (Fig. 46).

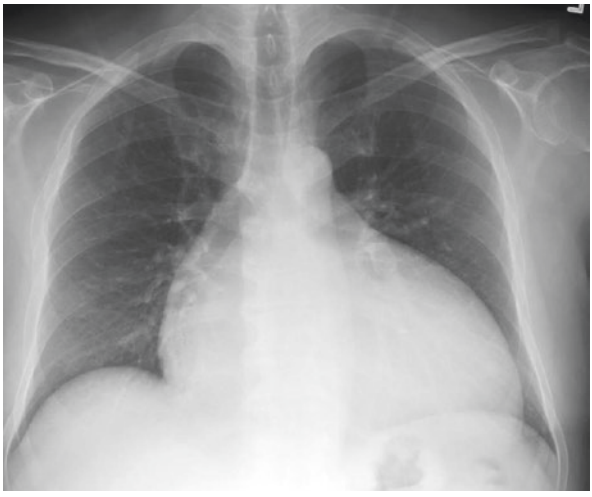

Fig. 45 Frontal chest X-ray of a patient complaining of pleuritic chest pain after a viral illness. The cardiac silhouette is enlarged in a "water bottle sign" appearance, and the remainder of the structures in the thorax appear normal. This is an example of pericardial effusion, which is nearly impossible to distinguish from cardiomegaly on frontal chest X-ray alone

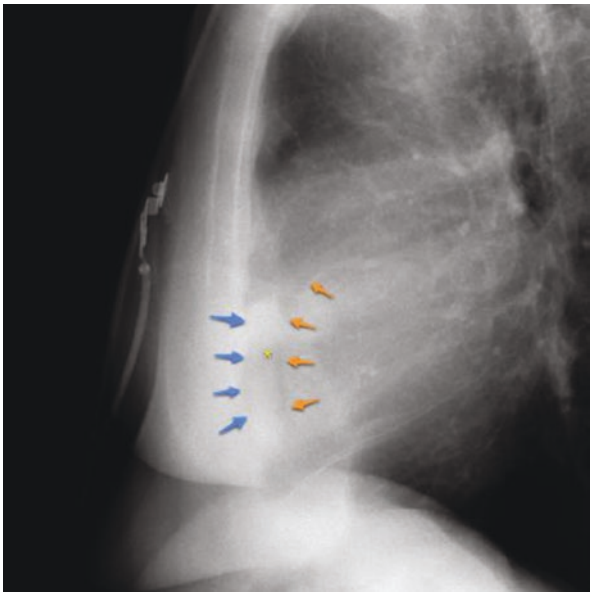

Fig. 46 The lateral chest X-ray helps to diagnose pericardial effusion when you see new onset enlargement of the cardiac silhouette. The "oreo cookie sign" of pericardial effusion is depicted here, where you will see a vertically oriented opaque line (yellow start) between two parallel lucent lines. Anatomically, the anterior lucent line is the paracardial fat (blue arrows), and the posterior lucent line is the epicardial fat (orange arrows)

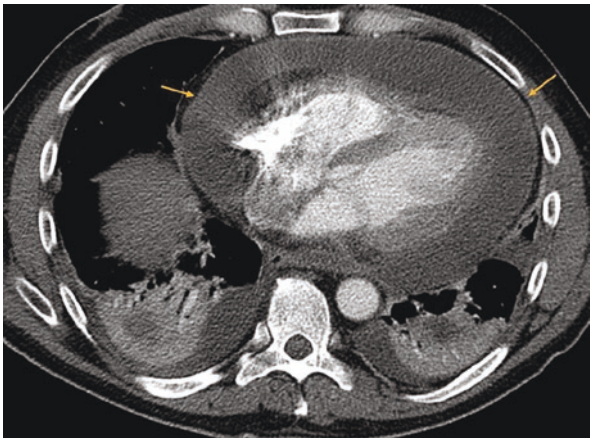

Fig. 47 This is an axial contrast-enhanced chest CT at the level of the four cardiac chambers. There is fluid attenuation surrounding the heart within the pericardial sac representing a large pericardial effusion (yellow arrows). Note the straightening of the ventricular septum, which is a clue of right heart strain and in this case, cardiac tamponade. You can suggest tamponade to the referring physician, but it is a clinical diagnosis and must be further investigated

On CT, pericardial effusion is a water-attenuating thickening of the normally 2-mm-thin pericardial stripe. It first accumulates in the dependent portion of the pericardium, which is posterior to the left ventricle, and as it enlarges it will fill the lateral and anterior portions until it is a concentric opacity around the heart (Fig. 47) [6]. Cardiac tamponade is a serious clinical diagnosis; however, CT findings to

suggest its presence include a pericardial effusion with associated dilatation of the SVC and azygos vein with contrast reflux into the IVC and, in severe cases, bowing of the interventricular septum and right heart chamber compression and collapse.

7 Systemic Vasculature

7.1 Aortic Dissection

Aortic dissection is part of the acute aortic syndromes that present with acute chest pain and can have devastating consequences. It is typically associated with hypertension and occurs when blood enters the aortic wall through a tear and dissects between the intimal and medial layers. The Stanford Classification separates the disease into two types and guides management. A Type A dissection will involve the ascending aorta and is treated with prompt surgery due to the possibility of retrograde dissection and rupture into the pericardium or occlusion of the coronary or carotid arteries. A Type B dissection arises distal to the left subclavian artery and dissects down the descending aorta. It is treated with medical management by controlling blood pressure.

The chest X-ray is useful to estimate the likelihood of a dissection being present and may be normal in up to 12% of patients with aortic dissection [1]. When abnormal, a widened mediastinum can be seen, typically with trauma or a history of sudden tearing chest pain in a hypertensive patient. A wide mediastinum is an expanded mediastinal silhouette approximately greater than 8 cm in width or mediastinal-chest ratio greater than 0.38 at the level of the aortic knob or fourth anterior intercostal space on portable AP chest X-ray (Fig. 48) [1]. Right-sided widening occurs with an ascending dissection, and left-sided widening occurs with a descending dissection. It is imperative to compare with old films as this is the most reliable way of confirming acuity. A widened mediastinum is not specific to dissection and can be a sign of multiple other etiologies: traumatic aortic injury (i.e., rupture), thoracic aortic aneurysm, aberrant right subclavian artery, double SVC, mediastinal mass, thymus, pneumomediastinum, atelectasis, and technical factors such as patient rotation. Other findings may include a widened, bumpy, or humped aortic knob, left pleural effusion, or pericardial effusion (i.e., impending cardiac tamponade) [1].

CT is highly sensitive and specific (>95%) in diagnosis and determination of location, type, and arterial branches involvement. On CT angiogram, dissection is detected when a low-density linear structure is seen within the aortic lumen surrounded by higher-density, contrast-enhanced blood (Fig. 48). This structure is the intimal flap that divides the true and false lumen of the aorta. The true lumen will typically be smaller with higher opacification depending on phase of contrast and generally favors the left in the dissected ascending aorta and right in the descending aorta. The false lumen is usually more irregularly shaped, of lower opacification due to the slower blood flow, and may contain “cobwebs” or strands of tissue [6]. False lumen generally favors the right ascending aorta, where it can involve the right coronary artery, and the left descending aorta, where it can involve the left renal artery.

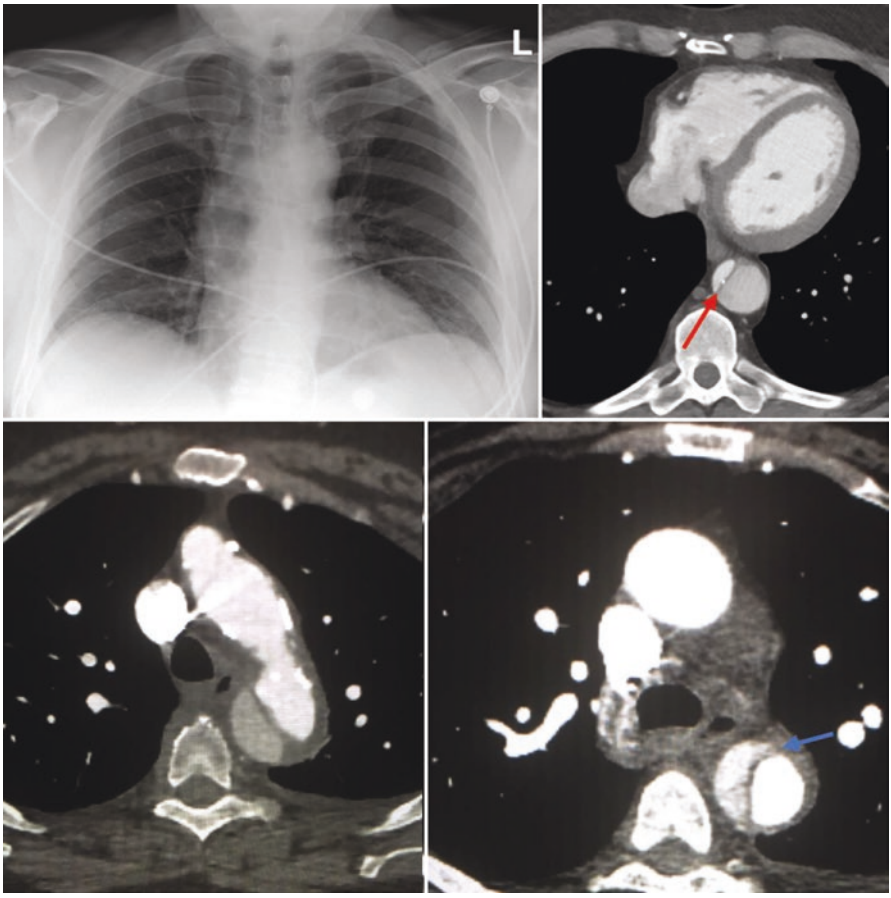

Fig. 48 The portable chest X-ray on the top left demonstrates a widened mediastinum in a patient experiencing acute "tearing" chest pain radiating to his back. Note there is right-sided widening, indicating an ascending aortic dissection. This patient had a dissection from the aortic root to the iliac bifurcation, or a Stanford Type A (**A**scending **A**orta **A**rch) and B (**B**eyond **B**rachiocephalic) dissection. The axial CT angiography on the top right shows a low-density intimal flap that divides the true, contrast-opacified lumen from the false lumen (typically darker and larger) in the descending aorta. Notice the calcification inside the descending aorta on the intimal flap: this is a clue of aortic dissection since atherosclerotic calcification buildup occurs along the peripheral arterial wall (red arrow). The bottom left and right axial CT angiography images show two additional examples of descending aortic dissection. Finding the "beak sign," which is an acute angle made by the intimomedial flap and the corners of the false lumen that resembles a bird's beak, will help you make the diagnosis (blue arrow). (Bottom Two Images Courtesy of Frederick A. Birnberg, MD)

Aortic wall calcification may become displaced inward in an abnormally central location within the lumen, which can be a clue to the diagnosis. At times, aortic dissection can rupture and cause acute blood spillage into the hemithorax or mediastinum. In cases of rupture, immediate mortality is 80–90% [1]. On CT, high-attenuation blood may be visible outside of the aortic lumen with an irregularity to a portion of

the aortic wall depicting the site of rupture. Some aneurysms or dissections may be amenable to endovascular repair with a stent graft.

7.2 Aortic Coarctation

Aortic coarctation is a congenital narrowing of the aortic arch. On chest X-ray, coarctation is suggested with the presence of rib notching, which is typically bilateral and asymmetric (Fig. 49). This finding occurs due to collateral circulatory development of the posterior intercostal arteries that cause pressure erosion of the inferior aspects of the posterior ribs. The roof of each lucent "notch" will demonstrate reactive sclerosis (bright thick lines on bottom caudal side of the rib, or high density, along the inferior rib). A direct finding is the "figure 3" configuration when looking directly at the aortic arch and descending aorta (Fig. 49) [1]. This will appear as a superior bulge followed by an inferior bulge in the mediastinal shadow making the shape of a number three, which is due to pre-stenotic enlargement of the aortic arch or left subclavian artery followed by post-stenotic dilatation of the descending aorta and a dimple in between. A prominent aortic arch, or high knuckle, with no appreciable descending aorta may be seen as well [1].

Coarctation can be readily diagnosed with CT. The narrowed segment of the aorta will be visualized as smaller than the proximal and distal portion to it. The most common site of narrowing is at the aortic isthmus distal to the origin of the left subclavian artery and near the ligamentum arteriosum [6]. Physiologically, the narrowing is not only the coarctation itself but also represents mild dilatation of the pre-stenotic and post-stenotic aorta.

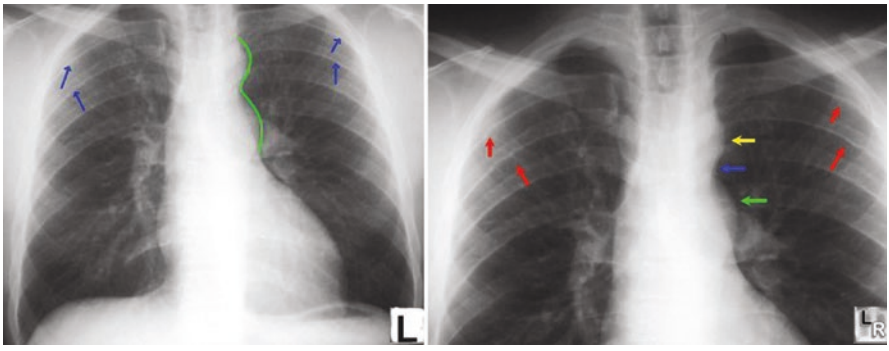

Fig. 49 Frontal chest X-ray on the left shows a *figure 3 configuration* (green line), which is a superior bulge followed by an inferior bulge in the mediastinal shadow and is a direct sign of aortic coarctation. There is bilateral "rib notching" with reactive sclerosis at the inferior aspect of the ribs, which is due to the development of collateral circulation (blue arrows). The zoomed in chest X-ray on the right shows the figure 3 configuration with anatomic landmarks: pre-stenotic enlargement of the aortic arch (yellow arrow), the coarctation (blue arrow), and the post-stenotic dilatation of the descending aorta (green arrow). Also appreciated on this image is bilateral rib notching (red arrows). This is also a test favorite

8 Hilum/Mediastinum

8.1 Pneumomediastinum and Pneumopericardium

Air can enter the mediastinum for many reasons. Pneumomediastinum is most commonly caused by rupture of an alveolus, which can happen in cases of asthma, mechanical ventilation, underwater diving, or smoking [1]. When alveoli rupture, the air dissects along the lung's vascular bundles interstitium until it reaches to hilum, which is its golden ticket to the mediastinum. Other etiologies include trauma (i.e., blunt force, penetrating object), esophageal rupture (i.e., Boerhaave syndrome), iatrogenic (i.e., traumatic intubation, endoscopy, or bronchoscopy), and mediastinitis. The mediastinum communicates with extrathoracic spaces, including vascular sheaths in the neck, retropharyngeal space, submandibular space, and retroperitoneal space, so trauma, if open or air inducing, in these regions can also be the culprit. On chest X-ray, the "continuous diaphragm sign" may be present and is the visualization of the entire surface of the diaphragm (Fig. 50). Normally, the central diaphragm is not seen because it does not make contact with the lungs. The "ring around the artery" or "tubular artery" sign describes a lucent ring that can be visualized around the aortic arch, its branching vessels, or the pulmonary artery [1]. A lucent halo may also be seen surrounding the heart as it is situated in the mediastinum.

Pneumopericardium can be difficult to differentiate from pneumomediastinum. It is extremely rare and is typically seen after thoracic surgery or from penetrating

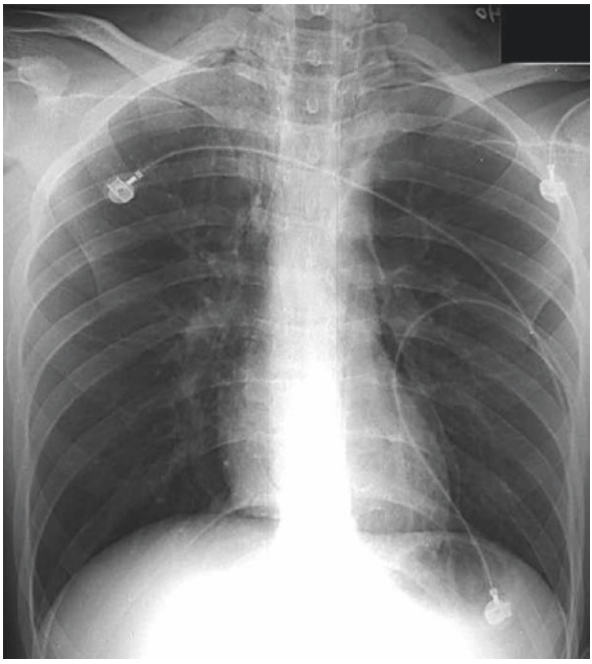

Fig. 50 Frontal chest X-ray demonstrates the "continuous diaphragm sign," which is the visualization of the entire surface of the diaphragm due to the presence of air in the mediastinum. Note the lucent halo surrounding the cardiac silhouette that is not normally present. These findings are typical for pneumomediastinum

trauma. Air in the pericardial sac will not extend above the superior pericardial borders, while air in the mediastinum will be able to extend above the ascending aorta and dissect to other spaces described above. If there is any doubt, CT can be utilized because it is sensitive to free air detection and can readily confirm location (Fig. 51). A trick to finding even the smallest amounts of air is to look at the mediastinum in the lung window, where the contrasting bright white soft tissues will allow for the black air to become easily apparent.

8.2 Mediastinal Masses

The mediastinum is a central space in the thorax, from the thoracic inlet to the diaphragm, which houses the heart, central airways, esophagus, vasculature, lymph nodes, and thymic tissue. It is divided into three anatomic compartments, which helps the radiologist differentiate pathologies that may arise there (Fig. 52). The anterior mediastinum is the pre-vascular compartment and is located posterior to the sternum and anterior to the pericardium. On frontal chest X-ray, a heart border or margin of the ascending aorta may be effaced because a mass in this space lies adjacent to these structures (Fig. 53). Importantly, it will not efface the hila nor widen a

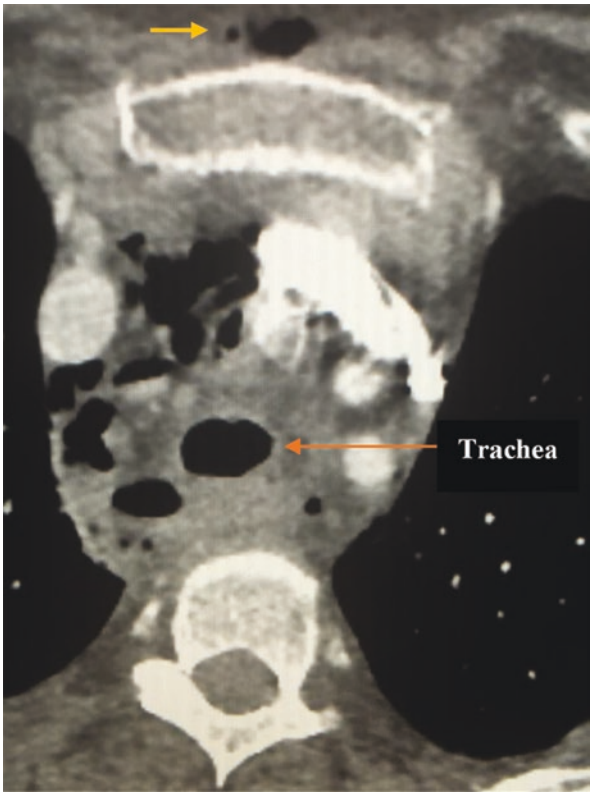

Fig. 51 Axial CT in a patient with esophageal perforation shows pneumomediastinum throughout the upper mediastinum surrounding the trachea, contrast-enhanced great vessels, innominate vein, and left brachiocephalic vein. An additional finding is subcutaneous emphysema anterior to the sternum, where the mediastinal air likely dissected to (yellow arrow). (Image Courtesy of Frederick A. Birnberg, MD)

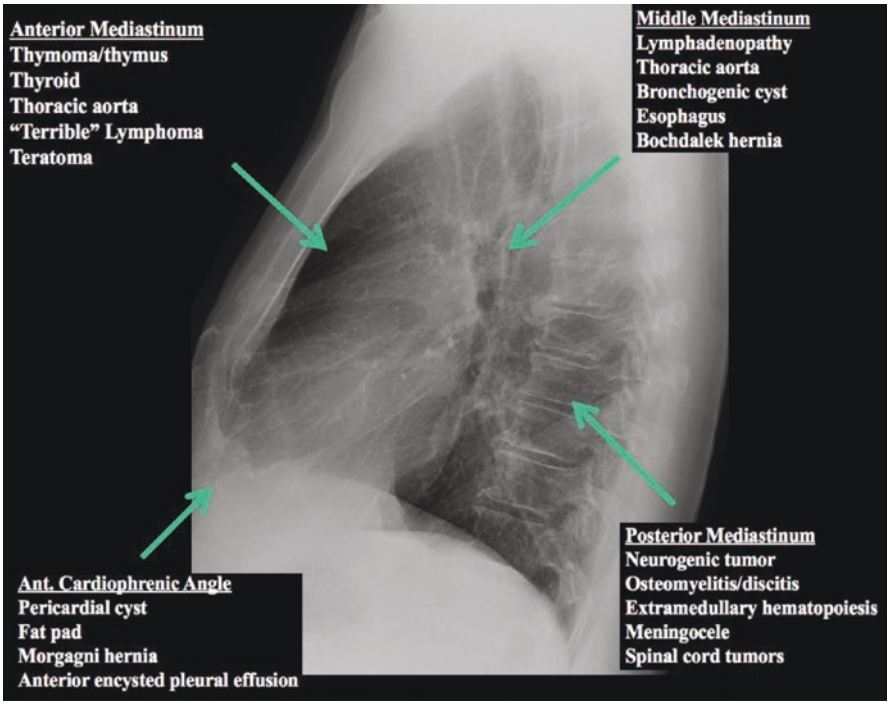

Fig. 52 Differential diagnosis of mediastinal masses by their compartments

paraspinal line since these anatomic structures lie posterior to the pre-vascular compartment [1].

The lateral chest X-ray is key to localizing and will help you place a mediastinal mass in the appropriate compartment. When a mass in this compartment is large, the aorta and great branches are posteriorly displaced without compression (due to their normally thick arterial walls) on CT. In the supra-aortic mediastinum, compression or obstruction of the brachiocephalic veins is quite common. A bit lower down, the SVC can be compressed or displaced posteriorly with right-sided masses, as with the main pulmonary artery with left-sided masses [6]. The differential diagnosis for the pre-vascular compartment includes thymic masses, germ cell tumors (i.e., teratoma, dermoid cyst), thyroid abnormalities (i.e., goiter, neoplasm), parathyroid masses, lymphoma (particularly Hodgkin's), aortic or great vessel abnormalities, cysts, fatty masses (i.e., lipoma), and lymphangioma (i.e., hygroma). Although missing a handful of diagnoses, you can remember this differential by using the mnemonic "*the five T's": thymus, thyroid, thoracic aorta, "terrible" lymphoma, and teratoma*. If a mass is located at the anterior cardiophrenic angle, think about pericardial cyst, fat pad, or Morgagni diaphragmatic hernia.

The middle mediastinum is the visceral compartment and involves the pericardium anteriorly to an "invisible line" drawn about a centimeter posterior to the anterior margin of the thoracic spine. On frontal chest X-ray, splaying of the carina or effacement of a normal hilar shadow may be seen (Fig. 54). There will be no

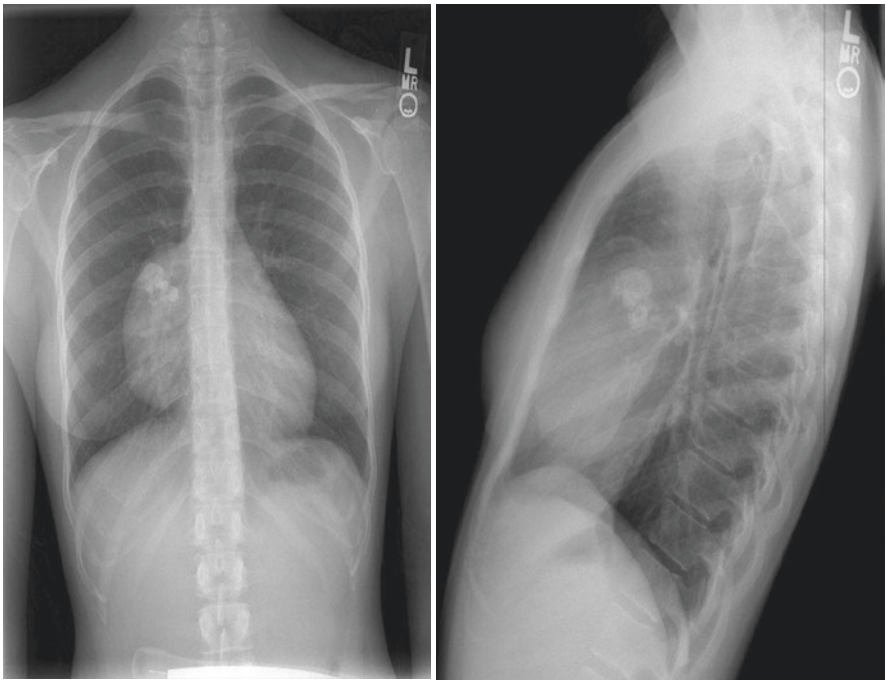

Fig. 53 Frontal chest X-ray on the left shows an anterior mediastinal mass with calcification and ectopic teeth, classic findings for a teratoma. You know it is in the anterior mediastinum because it effaces the right heart border but does not affect the normal hilum. On lateral chest X-ray (right image), the mediastinal mass appears to abut the sternum and does not distort the hilum, airway, or esophagus (middle mediastinal compartment)

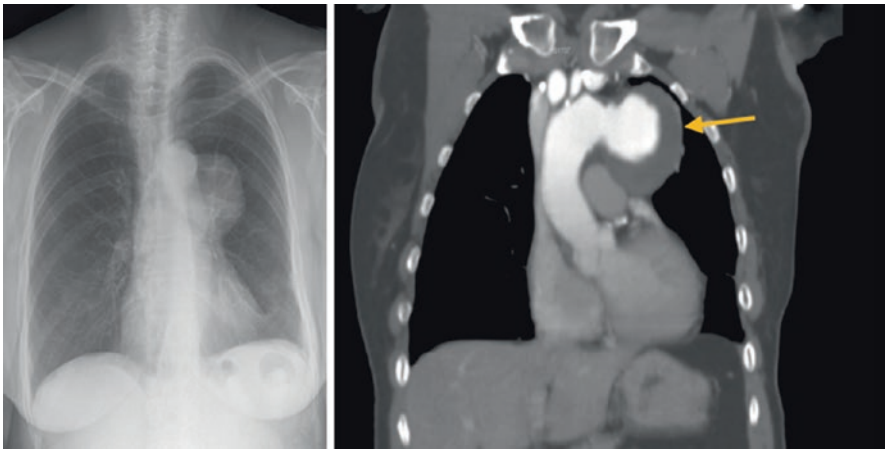

Fig. 54 Frontal chest X-ray on the left shows a rounded mediastinal mass that appears confluent with the aortic knob. It does not efface the left heart border or widen the paraspinal line, which supports a middle mediastinum location. Contrast-enhanced chest CT confirmed a leaking aortic aneurysm with a giant thrombus as the middle mediastinal mass (yellow arrow)

obscuration of a heart border or widening of a paraspinal line [1]. Masses in this compartment typically arise from enclosed structures, which include multi-station lymph nodes, trachea, aorta, or pulmonary artery and esophagus. However, these are almost always of lymph node origin [6]. Some examples of more common causes of lymphadenopathy include lung carcinoma, sarcoidosis, lymphoma, metastasis, and infections such as TB. Fatty masses (i.e., lipoma), vascular masses (i.e., hemangioma, descending aortic aneurysm), tracheal abnormalities (i.e., tumor, bronchogenic cyst), and Bochdalek diaphragmatic hernia can present in this space as well. Don't forget about the GI tract, which can present with small to large hiatal hernia, esophageal tumor, or varices (Fig. 55).

The posterior mediastinum is the paravertebral compartment and is posterior to the visceral compartment and involves both sides of the thoracic spine to the posterior chest wall. On chest X-ray, there may be widening of one or both of the paraspinal lines laterally (Fig. 56). Other findings include widened adjacent ribs with possible erosion of a vertebral body or rib. There will be no effacement of the heart borders or obscuration of the hila [1]. The differential diagnosis includes neurogenic tumors, thoracic spine abnormalities (i.e., tumors, osteomyelitis, fracture with hematoma), extramedullary hematopoiesis, vascular abnormalities (i.e., dilated azygos or hemiazygos veins), meningocele, and tumors of the spinal cord. Neurogenic tumors comprise of approximately 75% of posterior mediastinal masses, and some

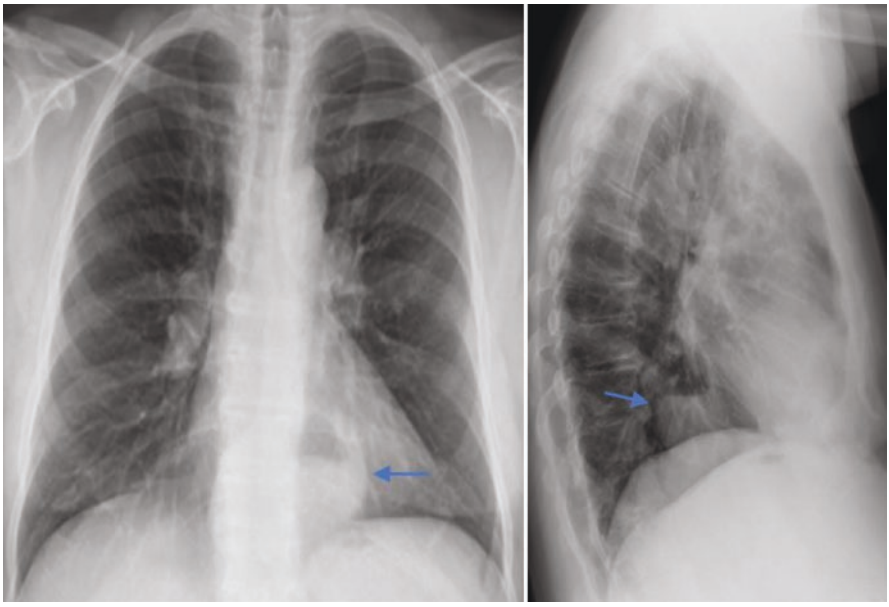

Fig. 55 Frontal and lateral chest X-ray show a round retrocardiac opacity with an air-fluid level in the mid-chest above the diaphragm (blue arrows). This is the classic appearance of a hiatal hernia in the middle mediastinum

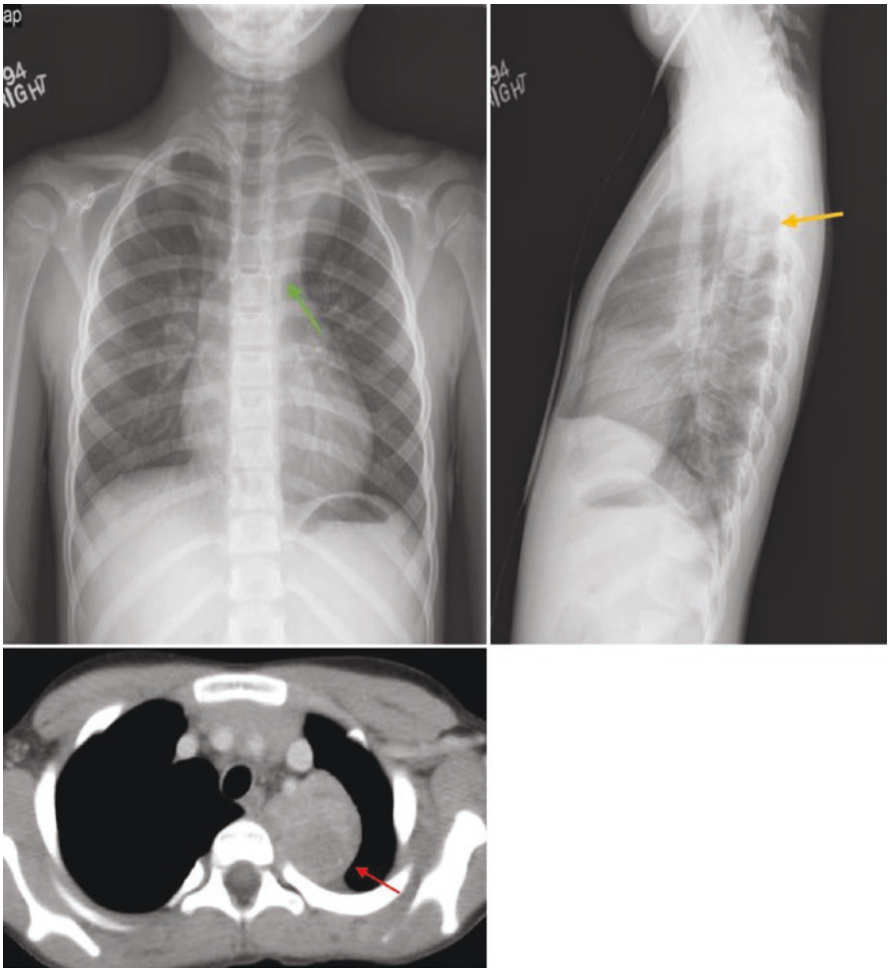

Fig. 56 Frontal chest X-ray shows an enlarged mediastinal silhouette at the superior left border. It is continuous with the left paraspinal line, causing slight widening (green arrow) and suggesting a posterior mediastinal mass. The lateral chest X-ray shows a large opacity overlying the upper thoracic spine, which confirms a posterior mediastinal location (yellow arrow). Axial contrast-enhanced chest CT shows a heterogeneously enhancing posterior mediastinal mass consistent with a neurogenic tumor (red arrow)

more common examples include schwannoma, neurofibroma, ganglioneuroma, and paraganglioma [6]. Pay particular attention to the figures and examples of these mediastinal masses because they may appear similar on chest X-ray, but the CT attenuation characteristics vary based on their content (air, fat, water, tissue, or calcification).

8.3 Hilar Lymphadenopathy

The lung hila are the "roots" of the lungs. They are at the medial aspect of each lung and are similar in composition, containing major structures such as the pulmonary arteries, bronchi, pulmonary veins, and lymph nodes but are not identical (Fig. 57). The differential diagnosis for a lung nodule or mass with ipsilateral hilar lymphadenopathy includes lung cancer, metastasis, lymphoma, inflammatory disease (i.e., sarcoidosis, silicosis, coal workers' pneumoconiosis), and infection (i.e., primary TB, fungal diseases).

The most common cause of a hilar mass or lymph node enlargement is lung cancer [6]. Other typical carcinomas that metastasize to the hila include head and neck squamous cell, melanoma, thyroid, renal cell, testicular, and breast carcinoma. Approximately 25% of patients with Hodgkin's lymphoma and 10% with non-Hodgkin's lymphoma will have multiple nodal stations in an asymmetric pattern [6].

Primary TB will usually demonstrate unilateral hilar adenopathy in additional to a "Ghon focus," which is a tuberculous calcified granuloma in the ipsilateral lung**.** These findings make up the "Ghon complex" (Fig. 58). Imaging of sarcoidosis will show bilateral and symmetric lymphadenopathy [6]. Fungal diseases, such as histoplasmosis and coccidioidomycosis, can also cause both unilateral and bilateral lymphadenopathy and should remain on the differential diagnosis in these cases. Sarcoidosis, TB, and silicosis may all demonstrate calcified hilar lymph nodes, deemed eggshell calcification, due to its peripheral location making the node appear as an egg within its thin bright shell.

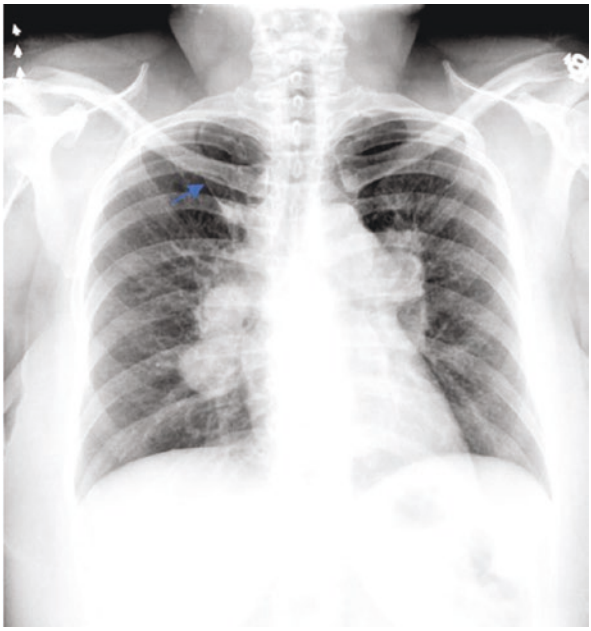

Fig. 57 Frontal chest X-ray shows prominent, well-rounded bilateral hilar opacities that represent bilateral hilar lymphadenopathy. Note an accessory azygous fissure and lobe, which is present in approximately 1 in 200 chest X-rays (blue arrow) as a normal anomaly

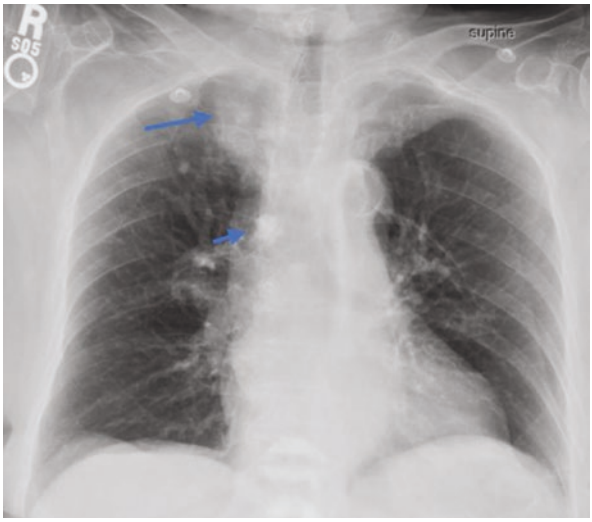

Fig. 58 This is a frontal chest X-ray of a patient with a history of tuberculosis. Here you can see a calcified granuloma near the right lung apex with ipsilateral calcified hilar adenopathy (blue arrows). These findings make up the "Ghon complex" of primary tuberculosis

9 Do Not Miss Findings on CXR Outside of the Thorax

9.1 Pneumoperitoneum

Although the abdomen is not completely assessed on a chest X-ray, the upper quadrants are visible and are very important to inspect. Pneumoperitoneum, or air in the abdominal cavity from viscus perforation (or normal postoperative air), can be seen as free air under the diaphragm on an upright X-ray (Fig. 59). As with pneumomediastinum, the "continuous diaphragm" sign may be present; however, it will be seen as smooth and continuous below the diaphragm. Detection of pneumoperitoneum can be difficult on a supine radiograph. However, nondependent gas will rise anteriorly and accumulate underneath the central tendon of the diaphragm at midline. If this occurs, you may see the "cupola" sign, which is a lucency overlying this region at the lower thoracic vertebral bodies. As with pneumomediastinum or pneumothorax, CT of the abdomen can readily demonstrate free air and, importantly, can lead to an identifiable source. Utilizing the lung window in addition to your routine soft tissue window in the abdomen will make pneumoperitoneum more obvious in a majority of cases (Fig. 60).

9.2 Thoracic Musculoskeletal Trauma

In any trauma case, it is important to closely look at the ribs and other musculoskeletal structures for integrity. Rib fractures can be difficult to detect, especially in a particularly poor or busy chest X-ray. Flail chest is a segment of fractured ribs that paradoxically moves opposite the chest wall with inspiration and expiration. It can

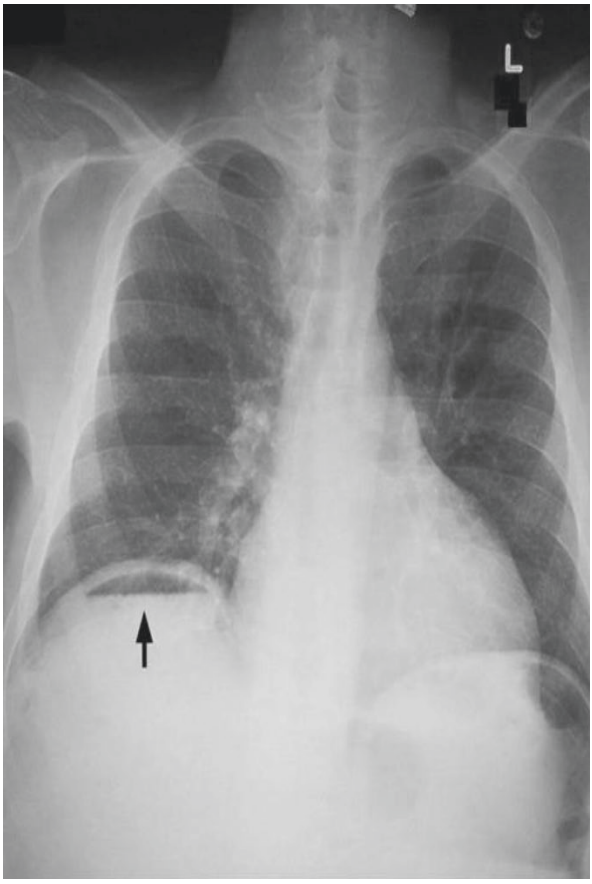

Fig. 59 Frontal chest X-ray shows a concave lucency under the right hemidiaphragm in the typical location of the liver (black arrow). This is free air = pneumoperitoneum

have devastating gas exchange consequences for the underlying lung and necessitate mechanical ventilation in some cases. Flail chest can be detected on chest X-ray by visualization of two fractures in two or more adjacent ribs (Fig. 61). Chest CT can easily detect acute fractures by demonstration of sharp discontinuity of bone at the fracture site. Do not forget to look at the "edges" of each chest X-ray and CT, as you may be the first person to diagnose a shoulder dislocation, clavicular fracture, or lower cervical spine abnormality (Fig. 62). Chest wall soft tissue injury is difficult to detect on chest radiographs but may present as outer chest wall asymmetry and increased density sometimes overlying the hemithorax. The type and extent of soft tissue injury is better identified and characterized on chest CT. Acute blood appears hyperdense and can be irregular or focally collected, and on contrast-enhanced chest CT, survey for active contrast extravasation is critical, particularly in early triage and management of vasculature injury. The vast majority of rib fractures require no chest tube and no procedural treatment other than pain relief and instructions on the effects of splinting. However, it may explain chronic or severe pain and can be associated with other injury such as splenic laceration.

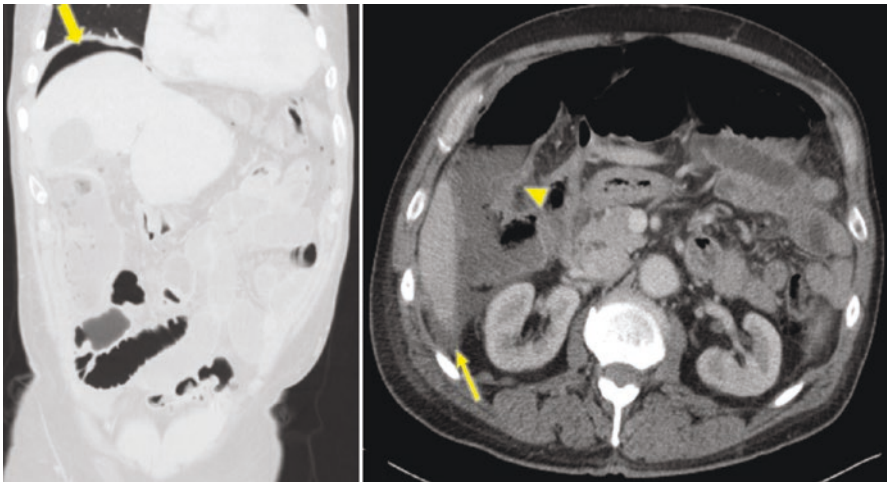

Fig. 60 Coronal CT of the abdomen (in lung window) on the left shows free air under the diaphragm above the liver (yellow arrow). Axial contrast-enhanced CT in soft tissue window shows stranding in the area of the duodenum (yellow arrowhead) and free fluid (yellow arrow) secondary to duodenal ulcer perforation. Don't forget to utilize your different windows as they can assist in detecting even the smallest amount of free air and free fluid

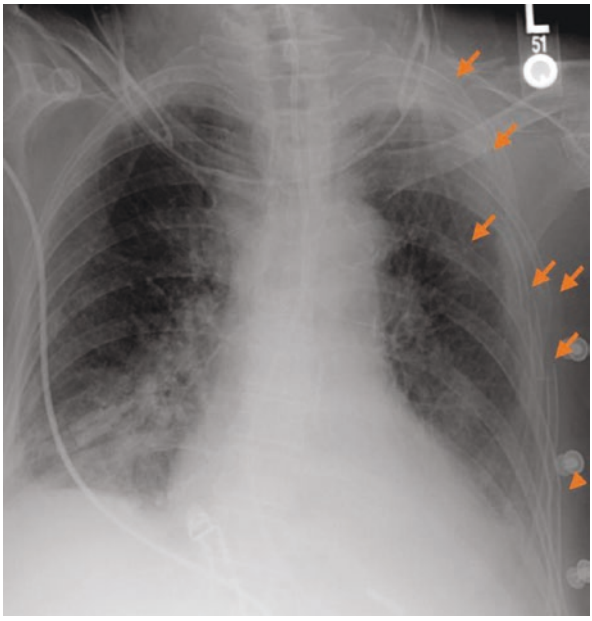

Fig. 61 This patient presented to the emergency room with severe injuries after a motor-vehicle collision. There are multiple, contiguous displaced left rib fractures causing rib segmentation and flail chest. Notice an enteric tube terminating in the left upper quadrant and cervical collar in place

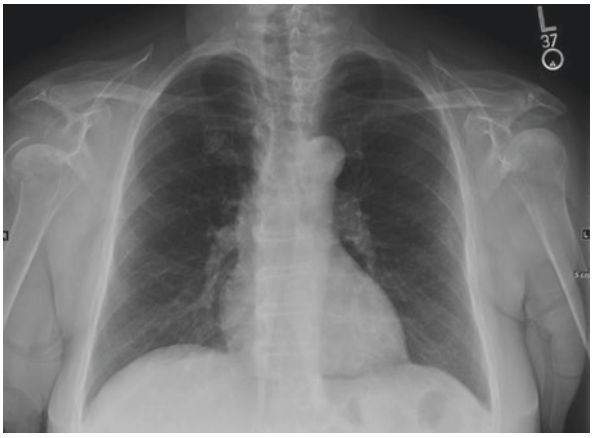

Fig. 62 This frontal chest X-ray is seemingly normal at first glance. However, do not forget to look at the film's "edges" where things can easily go unnoticed. For example, here we see an acute right proximal humerus fracture with inferior subluxation of the humeral head. If your systematic approach doesn't include the bones or the periphery of films, your eyes will never be trained to look in these easily missed places. *Don't spend too long staring at an X-ray* in a boards scenario! Time is precious – Don't waste it!

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Abdominal Imaging

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1 Liver

1.1 Liver Disease

Ultrasonography (US) is often a first-level instrumental investigation for the study of hepatic parenchyma with suspicion of diffuse liver disease (Table 1).

1.1.1 Hepatic Steatosis and Hemosiderosis

Although occasional, the most frequent finding in asymptomatic patients is focal or diffuse hepatic steatosis, which is an accumulation of intracellular fat. More often idiopathic than alcohol abuse, nonalcoholic fatty liver disease (NAFLD) associated with insulin resistance and dyslipidemia, acute fatty liver of pregnancy, steroid intake, and drugs can lead to hepatic steatosis [1].

Radiographic features include increased echogenicity without mass effect, and even if islands of normal hepatic tissue are present, associated with mild hepatomegaly [2].

On non-contrast computed tomography (CT), steatosis is seen as liver hypoattenuation of at least 10 HU lower than that of spleen [3] or absolute liver attenuation lower than 40 HU [4] (Fig. 1).

Table 1 Differential appearances for focal liver lesions on US and CT

	Focal liver lesion					
	Cyst	Typical hemangioma	FNH	Abscess	HCC	Metastases
US	Well-defined Thin wall Simple or lobulated Anechoic No internal vascularity on ECD	Well defined Hyperechoic Possible peripheral vessels on ECD	Difficult to detect	Poorly defined Hypoechoic No central perfusion on ECD Internal gas bubbles	Hypoechoic or inhomogeneous echotexture	Rounded and well defined Infiltrative Hypoechoic Hypoechoic halo Hyperechoic
CT	Homogeneous hypoattenuation (0–10 HU) No contrast enhancement	Hypoattenuation in non-contrast scan Peripheral enhancement on arterial phase Progressive central fill-in enhancement No washout on delayed scan	Peripheral arterial phase enhancement Isodense to liver on portal venous phase Central scar enhancement on delayed scan	Hypodense on basal scan Double target sign on contrast-enhanced CT Internal gas bubbles	Lively arterial enhancement Portal venous washout	Peripheral enhancement with possible central filling on portal venous phase and washout on delayed scan Arterial phase hyperenhancement and delayed washout Calcification or cystic component

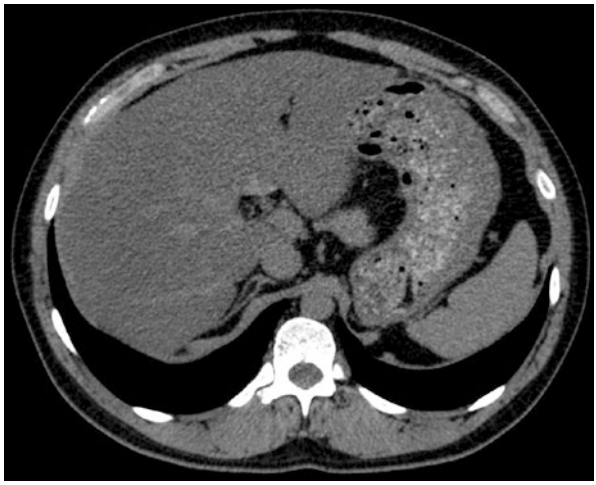

Fig. 1 Hepatic steatosis, non-contrast CT. In this patient, the liver demonstrates diffuse hypoattenuation (10 HU), compared to the spleen (50 HU)

In contrast, hyperattenuation of the hepatic parenchyma is seen in overload of hepatic iron, caused by genetic disorders, hemochromatosis, or ineffective erythropoiesis (as with repeated blood transfusions in β-thalassemia major) [5]. Magnetic resonance imaging (MRI) represents the imaging of choice for the quantification of iron; however, hemosiderosis may appear on CT as diffuse high-density liver parenchyma or high attenuating nodules [6].

1.1.2 Hepatitis

The symptoms of hepatitis are often nonspecific. The patient may present with fever, abdominal pain, and jaundice. Laboratory serum values may include an increase of hepatocellular injury levels (AST, ALT) and stasis values (ALP, GGT), plus or minus reduction of liver function (reduction of albumin and coagulation factors). The most common etiologies must be confirmed by labs and include viral infections (HAV, HCV, HBV), drug- and toxin-induced hepatitis, and autoimmune and metabolic diseases. Diagnosis of acute hepatitis remains a clinical diagnosis, even when a normal imaging appearance of the liver is present [7].

US often represents the first instrumental imaging for the suspicion of acute hepatitis. Even if it has been found to have poor sensitivity and specificity [8], a "starry sky appearance" (bright hyperechoic dots throughout hepatitis a hypoechoic liver parenchyma) is often associated with acute hepatitis [9]. Hepatitis findings include an accentuated brightness of portal vein walls, periportal edema (decreased attenuation or hypoechogenicity around the portal system and at the hepatic hilum), a diffusely low parenchymal echogenicity, diffusely low hepatitis attenuation edema on non-contrast CT, and gallbladder wall thickening. Hepatomegaly is considered the most sensitive sign of hepatitis. Hepatomegaly is defined when the liver measures greater than 15.5 cm, when measured at the midclavicular line [2].

1.1.3 Cirrhosis

Chronic liver diseases can develop into cirrhosis. Symptoms are variable depending on the degree of compensation. The clinician may suspect cirrhosis from a history of hepatic infections or biliary disease, metabolic or autoimmune disorders, vascular disease, or laboratory tests. Complications of cirrhosis include liver failure, ascites, and portal hypertension.

Even if imaging is not helpful to discover the underlying etiology, in advanced cirrhosis the liver takes on a typical morphology which involves hypertrophy of the caudate lobe and the left lobe with concomitant atrophy of the posterior segments of the right lobe. Regenerative nodules, siderotic nodules, and dysplastic nodules can be present.

US is useful for assessing surface nodularity and estimating segmental hypertrophy and atrophy [10], as well as the heterogeneous echotexture with signs of portal hypertension. As a dynamic instrument, ultrasound not only allows assessment of the caliber of the portal vein (>13 mm diameter = portal hypertension) but quantification of the portal flow (<15 cm/sec) and identification of reversed portal flow (hepatoFugal, your F'ed). By convention, doppler blue flows away from the transducer, and red is toward the transducer. Normal hepatopetal flow in the main portal vein is red (portal vein flows toward the transducer on the skin). Other findings of portal hypertension captured by ultrasound include an enlarged superior mesenteric vein and splenic vein (>10 mm), portal venous thrombosis, recanalization of paraumbilical venous flow, and portalization of hepatic vein waveform, a "corkscrew" appearance of the hepatic artery, a cluster of porta hepatis collaterals that look like a spider ("cavernous transformation" of the portal vein"). Splenomegaly and ascites may also be seen.

CT is helpful to evaluate the liver surface and parenchyma to identify regenerative nodules that are isodense to the rest of the liver, as well as siderotic nodules that are hyperdense due to accumulation of iron. In addition to assessing liver morphology and the indirect signs of portal hypertension (portal vein enlargement and portal venous thrombosis or cavernous transformation), CT allows evaluation of the liver parenchyma both on the pre- and post-contrast scans, through a dynamic study.

In this heterogeneous parenchyma, the radiologist must recognize the conditions that modify hepatic vasculature and that require an interventional procedure (e.g., TIPS - a portal to systemic shunt) and, certainly, a possible dysplastic nodule suspected for hepatocellular carcinoma (HCC or hepatoma).

1.2 Liver Focal Lesion

Focal hepatic lesions may be benign or malignant. Although some are congenital, most arise in both healthy and sick livers. Often the clinical history integrated with imaging allows good diagnostic accuracy, using the LI-RADS scale and criteria (hyperenhancement, size, rapid washout, and capsule).

1.2.1 Hepatocellular Carcinoma (*HCC)/Hepatoma*

Chronic liver diseases that evolve into cirrhosis, biliary cirrhosis, and metabolic disorders are the leading causes of HCC [11].

The symptoms are variable (constitutional symptoms, jaundice, tumor invasion of the portal vein resulting in portal hypertension, mass symptoms, tumor hemorrhage, or even upper GI bleeding varices from associated portal hypertension). The diagnosis generally is an incidental finding in the screening program for patients with risk factors. Among lab tests, alpha-fetoprotein (AFP) levels may be elevated.

On US, the small focal nodule of HCC is hypoechoic compared to normal liver, while large nodules have an inhomogeneous echotexture due to fibrosis, fat, or necrosis and calcification; a peripheral hypoechogenic halo may be present, like focal fatty sparing.

Usually, the typical HCC nodule has lively and robust arterial enhancement with subsequent washout, so it becomes iso- or hypoattenuating in the portal venous phase compared to the rest of the liver (Fig. 2). Typically, wedge-shaped perfusion anomalies may be associated. In cases of concurrent portal vein thrombosis, it is important to diagnose portal vein tumor thrombus when the thrombus itself demonstrates enhancement. "Bland thrombus" without tumor invasion of the clot does not demonstrate enhancement [12].

1.2.2 Metastases

Liver metastases are generally asymptomatic but may become symptomatic when the lesion burden compromises liver function or affects the capsule [13].

Metastases are generally rounded and well-defined and have a mass effect with distortion of neighboring vessels.

In most cases, metastases from lung cancer, breast cancer, pancreatic adenocarcinoma, and lymphoma are hypoechoic and may present with a hypoechoic halo due to fat sparing (Fig. 3).

Similarly, they are generally hypoattenuating on non-contrast CT and present with typically peripheral enhancement and sometimes with central filling on portal venous phase or washout on delayed scan. (Remember that benign hemangiomas will fill in over time from a nodular outer part to finally looking like normal liver on delayed scans.)

Metastases from melanoma, colorectal carcinoma, renal cell carcinoma, and neuroendocrine tumors are usually hyperechoic due to hypervascularization. On contrast-enhanced CT, arterial phase demonstrates corresponding hyperenhancement that later washes out on delayed phase (Fig. 4). Colorectal carcinoma metastases can have variable echogenicity on US and similarly variable attenuation and enhancement on CT, and this may change following therapies.

Calcification can occur with metastases of various mucinous adenocarcinomas, and cysts can be associated with squamous and colorectal cell carcinoma, ovarian carcinoma, and pancreatic adenocarcinoma.

On occasion, melanoma, breast cancer, and lung cancer can diffusely infiltrate the hepatic parenchyma [14].

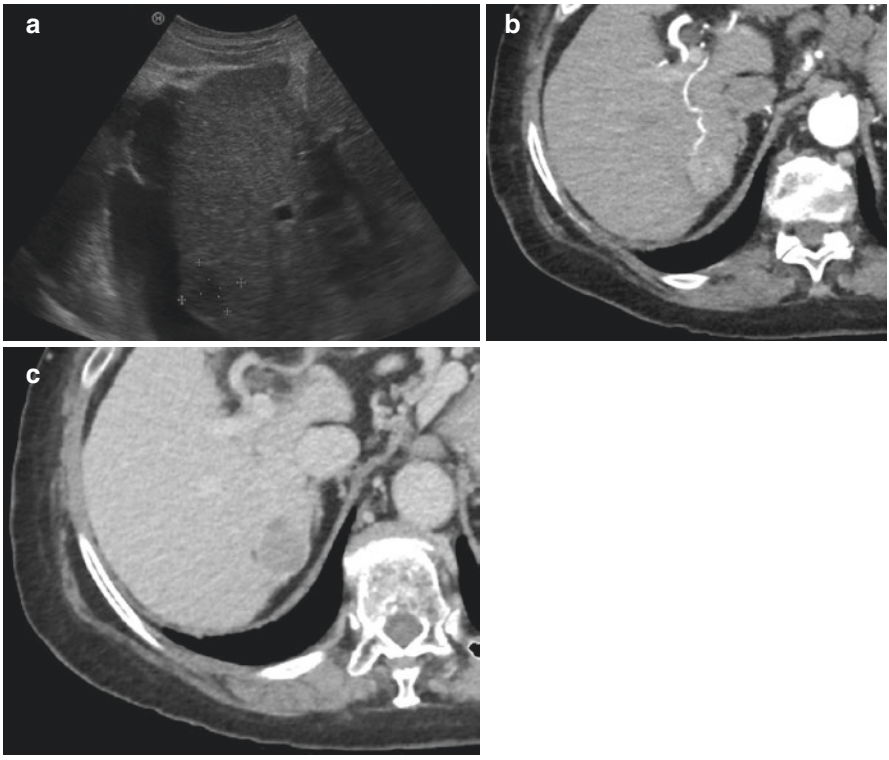

Fig. 2 HCC. US, the nodule has an inhomogeneous echotexture, hypoechoic compared to normal liver (**a**). On contrast-enhanced CT, the typical HCC nodule has lively arterial enhancement (**b**) with subsequent rapid washout, and becomes iso- or hypo-attenuating in the portal venous phase (**c**) compared to the rest of the liver. The presence of a pseudocapsule is seen (**c**). This is likely a LIRADS 4-5, which is very likely an HCC

1.2.3 Hepatic Abscess

Typically, patients with a hepatic abscess present with fever and jaundice associated with right upper quadrant pain. Other nonspecific symptoms such as anorexia, malaise, and weight loss may be seen.

Regardless of the etiology (bacterial, parasitic, or fungal), radiographic features are similar. Bacterial and fungal abscesses are often multiple, while the amebic abscesses are usually single and located in the subdiaphragmatic liver [15].

On US, hepatic abscesses are typically poorly defined with inhomogeneous predominantly hypoechoic echotexture, without central perfusion on color Doppler; gas bubbles may be seen.

On contrast-enhanced CT scan, the "double target sign" is a typical imaging feature: a central fluid-filled hypoattenuation surrounded by a hyperattenuation inner rim (that is the abscess membrane that takes early contrast enhancement which persists on delayed phase) with a hypoattenuation outer ring that is hepatic edema which enhances on delayed phase (Fig. 5). Recurrent hepatic abscesses may be

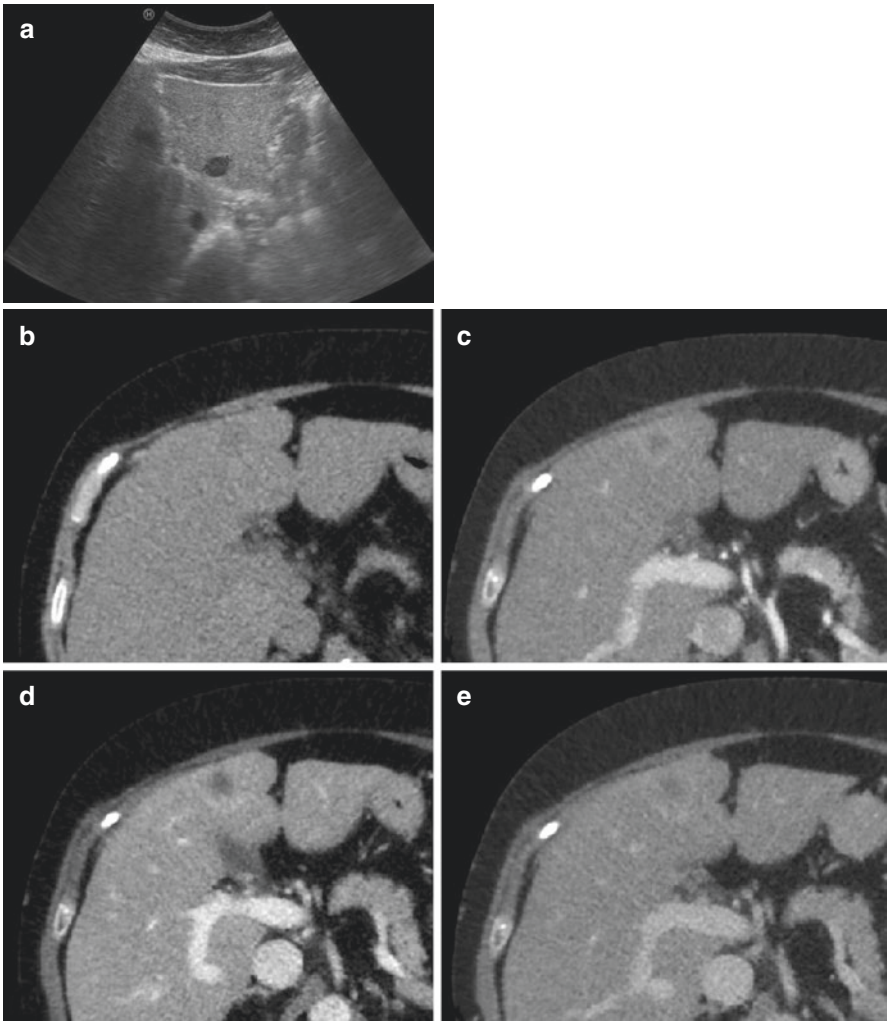

Fig. 3 Metastases from breast cancer. US (**a**), they are often defined hypoechoic lesions. On CT, metastases are generally hypoattenuating on non-contrast CT (**b**) and present typically peripheral enhancement (**c**) with possible central filling on portal venous phase (**d**) and washout on delayed scan (**e**)

associated with chronic granulomatous disease, where the white blood cells have impaired oxidative burst.

1.2.4 Other Benign Lesions: Cyst, Hemangioma, Focal Nodular Dysplasia (FNH)

Hepatic cysts are typically asymptomatic and are often incidentally discovered.

They are a round or ovoid shape with well-defined margins due to the presence of a thin wall. On US (Fig. 6), a cyst is anechoic and may be lobulated, without any

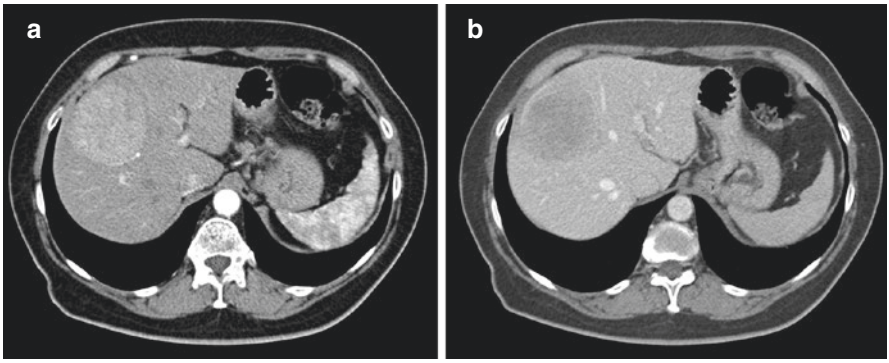

Fig. 4 Metastases from melanoma, contrast-enhanced CT. The right hepatic lobe presents a voluminous hypervascular lesion, i.e., with intense enhancement on arterial phase (**a**), which washes out on venous phase (**b**)

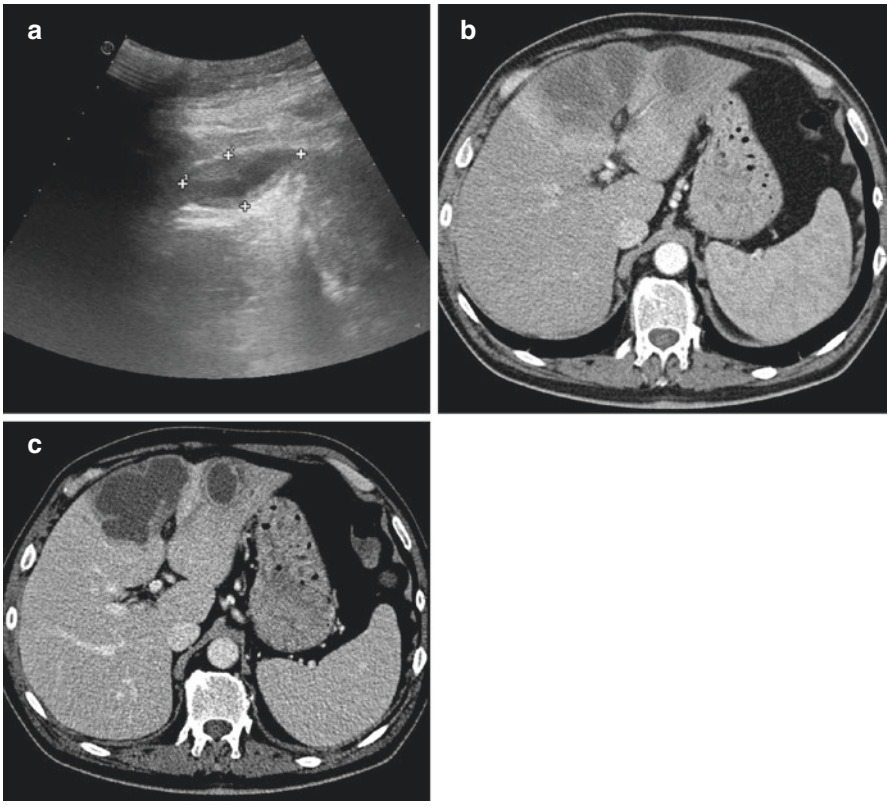

Fig. 5 Hepatic abscess. On US, the lesion has inhomogeneous and predominantly hypoechoic echotexture with corpuscular and fluid component in the context (**a**). On contrast-enhanced CT scan, the abscess presents the typical "double target sign", more evident on venous phase (**c**); an alteration of the perfusion of the neighboring parenchyma is visible on arterial phase (**b**)

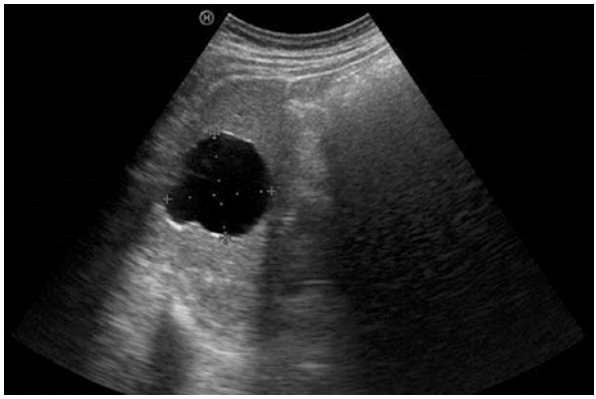

Fig. 6 Hepatic cyst, US. The hepatic cyst is anechoic and has a round or ovoid shape with well-defined margins due to the presence of a thin wall. Increased "through-transmission" or sonographic enhancement is seen deep to the cyst as brighter echoes

internal vascularity on color Doppler. On CT, the cyst has a homogeneous hypoattenuation (0–10 HU) without contrast enhancement.

Benign vascular lesions of the liver may be distinctive; the most common is hepatic hemangioma. Often incidentally found, hemangiomas are well-defined hyperechoic lesions with possible peripheral vessels on color Doppler (but in a minority of cases it is hypoechoic in a relative background of diffuse hepatic steatosis). Contrast-enhanced CT is helpful to distinguish hemangioma from other malignant lesions. Typical lesions may be seen as hypoattenuating on non-contrast CT scans and demonstrate peripheral nodular-shaped enhancement on arterial phase with progressive central fill-in/enhancement on portal venous phase that does not wash out on delayed phase (Fig. 7) or becomes identical to normal liver on delayed phase.

Focal nodular hyperplasia (FNH) is generally asymptomatic and is found in young to middle-aged women who take exogenous estrogens or BCP, which may increase the size of the mass.

On US, FNH and its "central scar" are difficult to detect, while on contrast-enhanced CT, the nodule has bright arterial phase enhancement except its central scar, becomes isodense to liver on portal venous phase, and shows enhancement of the central scar on delayed phase.

1.3 Liver Traumatic Injury

In abdominal trauma, when a patient presents with right upper quadrant pain, right shoulder pain, hypotension, and shock, traumatic injury of the liver must be excluded.

Polytrauma represents a common on-call situation in which a whole-body contrast-enhanced CT is mandatory.

The radiologist must identify the presence of blood, active bleeding, and organ laceration, and the interventional radiologist may embolize for hemostasis.

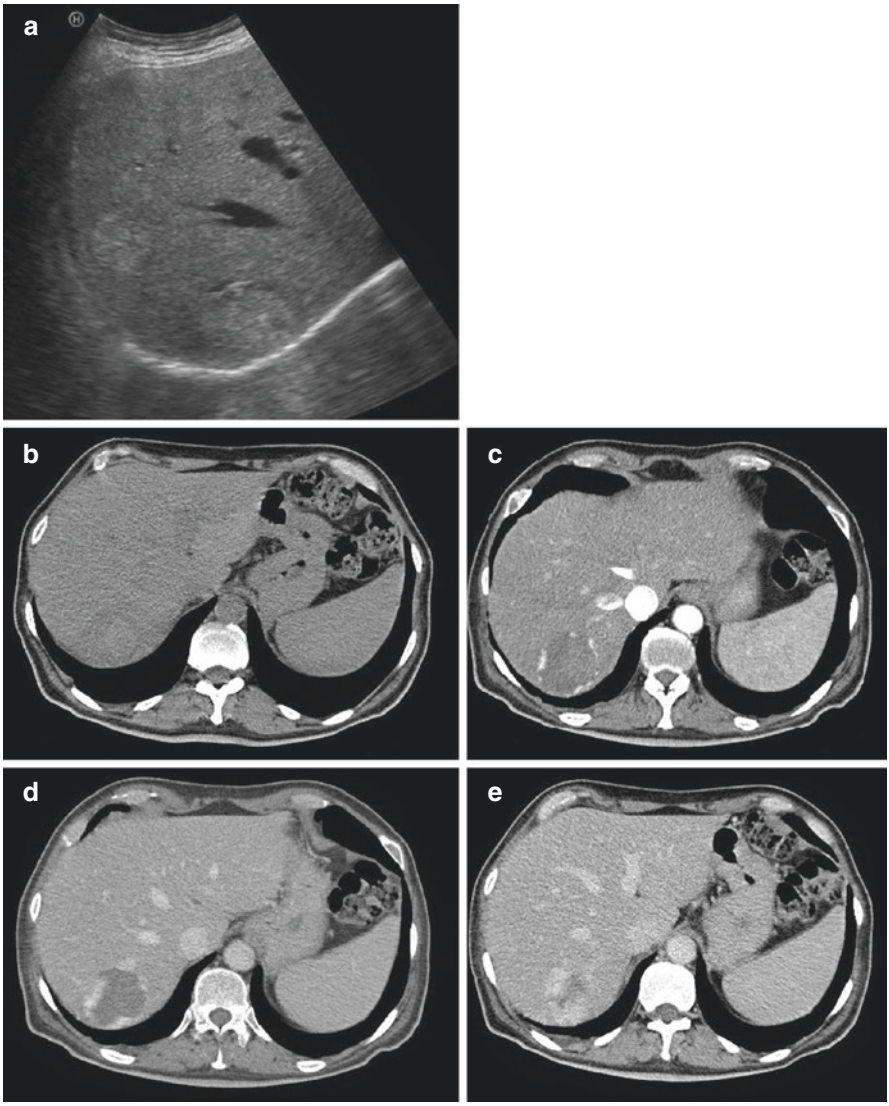

Fig. 7 Hepatic hemangioma. On US (**a**), the typical hepatic hemangioma is seen as a well-defined hyperechoic lesion. On non-contrast CT, it is seen as hypoattenuation (**b**), while on contrast-enhanced CT, it has peripheral enhancement on arterial phase (**c**) with progressive centripetal fill-in enhancement on portal venous phase (**d**) that does not readily wash out on delayed phase (**e**)

In particular, liver lacerations appear as irregular linear or branching regions of hypoattenuation. Hematomas can be subcapsular or intraparenchymal and appear as a hypodensity between the liver and its capsule extending into the hepatic parenchyma.

The acute hemorrhage is typically hyperdense (40–60 HU) compared to normal parenchyma, and the presence of blushing on dynamic phases indicates an active bleed [16] or pseudoaneurysm.

2 Gallbladder and Biliary Tract

2.1 Gallbladder Cholecystosis

Whether accumulation of cholesterol esters and triglycerides (cholesterolosis) or hyperplasia of the gallbladder wall (adenomyomatosis), US is the best test with which to assess the gallbladder.

Cholecystosis may occur as localized or, as a diffuse form, is known as "strawberry gallbladder."

Clinically silent, this condition presents like a diffuse, focal, or annular wall thickening that may be characterized by the "comet-tail" artifact, that is, the V-shaped comet tail reverberation artifact emanating from the hyperechoic adenomyoma [17].

2.2 Gallstone Disease

This condition may present as a chronic disease, in which the patient may be asymptomatic or complain of abdominal discomfort after meals with bloating. Alternatively, acute disease is characterized by pain, nausea, vomiting, and (in case of infection) fever [15].

The radiologist assesses for the presence of gallstones, correlates localized symptoms with imaging findings, and surveys for complications. The sonographic Murphy sign is a painful gallbladder upon inspiration, with the ultrasound transducer (camera) over a sensitive gallbladder below the rib cage.

In order for the gallbladder to be bile-filled, therefore assessable, the patient must be fasting.

Some radiopaque gallstones may be seen on the plain radiograph (Fig. 8a), but ultrasound is the best single initial test for seeing gallstones and their complications [18], although a nuclear medicine HIDA scan can be used to diagnose a blocked common bile duct.

Regardless of their composition, gallstones are hyperechoic structures that move according to gravity with the patient position changes (the rolling stone sign), characterized by prominent posterior acoustic shadowing. On color Doppler, the stone may demonstrate the twinkling artifact, a focus of posterior alternating colors which simulates turbulent blood flow [18].

When the gallstone is wedged in the common bile duct (choledocholithiasis), a dilatation of the biliary tree upstream plumbing is seen (Fig. 8b).

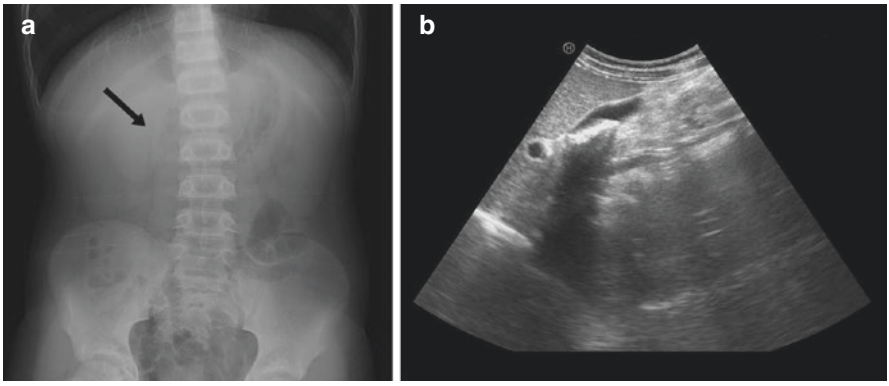

Fig. 8 Gallstone disease. On the plain radiograph (**a**), many small radiopaque densities are projected adjacent to the right lumbar spine (black arrow). On US (**b**), gallstones are seen as hyperechoic structures; prominent posterior acoustic shadowing is present

Depending on their composition on CT, gallstones may be hypoattenuating to bile (pure cholesterol stones), hyperattenuating (calcified gallstones), or isodense to bile. The latter are not clearly identified on CT [19] but might be easier seen on MRCP.

Acute cholecystitis as complication of cholelithiasis is characterized by intermittent right upper quadrant pain of biliary colic, and gallstone obstruction is often in the gallbladder neck or in the cystic duct. A minority of cases of cholecystitis are acalculous (ICU, burns, or vasculopathy). In addition to sonographic evaluation of the gallstone and gallbladder distention, the US demonstrates the presence of acute cholecystitis when gallbladder wall thickening (>3 mm) and pericholecystic fluid are seen.

The same findings can be seen on CT scan, which can also demonstrate mural or mucosal gallbladder hyperenhancement and possible enhancement of the adjacent liver parenchyma, even if stones isodense to bile are usually missed [20].

Although rare, emphysematous and suppurative cholecystitis must be excluded because they represent a potential surgical emergency. A boards question associates *E. coli* in diabetics with emphysematous cholecystitis (gas in gallbladder wall).

In the first case, the radiologist may see the gallbladder wall necrosis through the presence of gas in the lumen or wall, best detected on contrast-enhanced CT [21]. A possible perforation is demonstrated by the presence of pneumoperitoneum.

In the suppurative cholecystitis, the sonographic and densitometric findings are identical to cholecystitis with the exception of hyperechoic/hyperattenuating content often seen within the gallbladder lumen [20]. Another boards question might associate acalculous cholecystitis (no stones but dilated gallbladder or hydropic gallbladder and gallbladder wall thickening) in a patient who is a vasculopath, in ICU, burn unit, or posttrauma. The treatment is a cholecystostomy tube (gallbladder tube) placed at the bedside by IR.

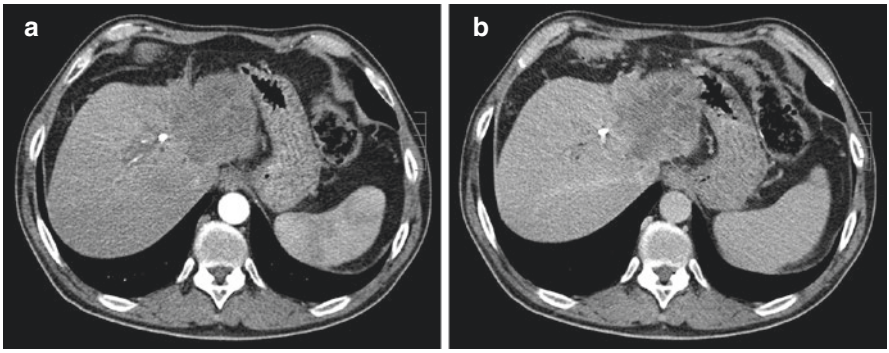

Fig. 9 Mass-forming cholangiocarcinoma, CT. The tumor demonstrates minor peripheral enhancement (**a**) with gradual centripetal enhancement (**b**) which depends on the degree of central fibrosis

2.3 Cholangiocarcinoma

Often the only clinical presentation of cholangiocarcinoma is insidious jaundice, the primary reason the patient undergoes investigations. Even if MRI is the imaging modality of choice, it is important to suspect a cholangiocarcinoma to direct the patient toward the correct diagnostic procedure.

Cholangiocarcinoma is often extrahepatic and, less often, intrahepatic, in the mass-forming, periductal infiltrating, or intraductal variant. It is usually central near the porta hepatis.

The mass-forming tumor variant presents as a homogeneous isoechoic mass with a hypoechoic periphery; capsular retraction may be present. On CT, it demonstrates minor peripheral enhancement with gradual centripetal enhancement which depends on the degree of central fibrosis (Fig. 9).

Periductal infiltrating and intraductal tumors are characterized by altered caliber bile duct, typically at the hepatic hilum, with peripheral dilation of the biliary tree, without the presence of a well-defined mass. When seen, the polypoid mass is usually hyperechoic in the intraductal tumor and demonstrates contrast enhancement on CT [22].

3 Spleen

Most primitive splenic lesions are detected incidentally. Conventional ultrasonography allows confident detection of cystic lesions but is less accurate for characterizing lesions with solid components, which should be evaluated with contrast-enhanced CT. Contrast-enhanced ultrasonography (CEUS) may be an optimal alternative to CT, when available [23].

3.1 Cysts

Splenic cysts can be either congenital (true cysts) or acquired (secondary/false cysts or pseudocysts). These may be indistinguishable on imaging.

Congenital cysts may be multiple or isolated and present as intraparenchymal, unilocular fluid-filled structures with thin epithelial walls, hypoattenuating on CT without contrast enhancement. Congenital desmoid cyst is an exception since it may show a thickened wall with linear enhancement. Splenic lymphangioma is a relatively rare congenital malformation of lymphatic ducts and usually appears as a complex multiloculated cyst with septa. It is usually subcapsular and does not enhance after contrast, although fine internal vessels may be seen [24, 25]. All the congenital cysts may be complicated by superinfection (visible as solid intralesional debris and calcification) or hemorrhage (fluid-fluid levels and hyperattenuation on CT) [24].

Secondary cysts occur after traumatic splenic injury or infection and are more common than simple congenital cysts. These appear as fluid collections with no epithelial lining and possible mural calcifications [23]. Splenic parasitic echinococcal cysts (hydatid cysts) are rare infective cysts with a complex structure characterized by septa and wall calcifications; their complexity is related to the stage of the infection [23, 25].

3.2 Hemangioma

On ultrasound, splenic hemangiomas are hyperechoic with irregular but defined margins. CT typically shows an irregular enhancing pattern (unlike hemangiomas in the liver) which is usually nodular and peripheral and may progress heterogeneously during the venous and late phases. Cavernous variants show internal cystic areas and often incomplete enhancement due to thrombosis and/or fibrosis [23].

3.3 Hamartoma

Hamartoma is a benign malformation originating from the red pulp, containing disorganized vascular channels in a fibrous stroma. On ultrasonography, it presents as an inhomogeneous hypoechoic structure, with internal anechoic foci and septations, and hypervascularity on color Doppler. On CT, hamartomas are usually isoattenuating compared to splenic parenchyma, with fatty components and internal calcifications, which may be hardly detectable after contrast administration. Multiphase contrast evaluation shows centripetal or centrifugal enhancement progression and may be difficult to distinguish from a splenic hemangioma [23].

3.4 Abscess

Splenic abscess is an uncommon finding, which usually presents as a complication of a penetrating injury or during infectious or septic conditions, such as endocarditis often related to intravenous drug abuse. Suggestive symptoms and findings include fever, left upper quadrant pain, and leukocytosis. In the suspicion of abscess, ultrasonography-guided drainage is required as the first step of therapeutic management. Different patterns are observed according to the underlying pathogen [24]. Septic emboli are wedge-shaped triangles, with the base along the capsule.

3.4.1 Pyogenic Abscess

On ultrasonography, a pyogenic abscess presents as an isolated hypoechoic lesion with irregular wall. When large (2–3 cm) it may show an anechoic core with septa or reverberation artifacts due to gas bubbles. On CT, findings suggestive for an abscess include a focal area with irregular margins and hypoattenuation reflecting the presence of necrosis or possibly containing gas bubbles. Enhancement may be present in large, encapsulated, or organized abscesses and involve the external margin (ring enhancement) and internal septations. Late contrast washout is usually seen. Wedge-shaped regions of low attenuation and reduced vascularization in the surrounding parenchyma indicate subsequent infarctions, often due to septic emboli [24]. Septic emboli and resulting ischemia may appear as triangular lesions with broadly based at the outer capsule, especially in organs with one blood supply (spleen, brain, kidney, lung). For example, in the lung a pulmonary thromboembolus may produce a "Hampton's hump," which is a wedge-shaped area of lung infarction.

3.5 Fungal or Parasitic Abscess

Fungal and parasitic splenic abscesses present usually as multiple lesions, less than 2 cm in size. On ultrasound, they show a hypoechoic structure with a target-like or "wheel within a wheel" morphology. Usual pathogens associated with this pattern are fungi and *Pneumocystis carinii*, mostly observed in immunocompromised patients.

3.6 Mycobacterial Abscess

Mycobacterial abscesses present as multiple small lesions of 10–20 mm in size with a hypoechoic structure [25]. Mycobacterial lymph nodes can have low attenuation (dark).

3.7 Infarction

Splenic infarction (SI) is either caused by occlusion of the splenic artery, venous thrombosis of the sinusoids, or by arterial embolism. Other possible causes include

pancreatic diseases and portal hypertension, leading to thrombosis of the splenic vein [24]. Isolated gastric varices may suggest splenic vein thrombosis, based on vascular plumbing.

Ultrasonography is not useful in the acute phase, with false negatives rising up to 50% of cases. When evident, SI are displayed as hypoechoic triangular lesions. Organ swelling may occur due to the development of edema. Color Doppler may reveal acute splenic infarcts, especially if large, as an area of reduced vascularity compared to the normal parenchyma. If available, CEUS drastically improves the detection of SI. In the late healing phase (due to fibrosis or scarring), the infarctions become more evident and become hyperechoic [25].

CT shows a higher sensitivity for SI, especially in the acute phase. The infarcted area is seen as triangular reduced attenuation with indistinct margins; the base of the triangle is always directed toward the splenic capsule (Fig. 10). Organ swelling may

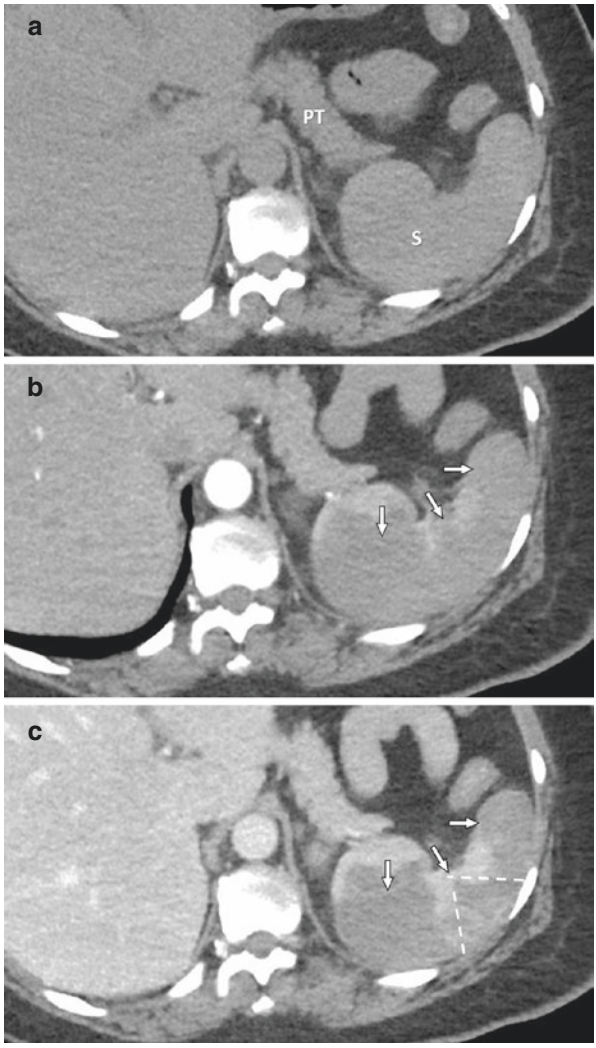

Fig. 10 Infarction of the spleen, CT. Contrast-enhanced CT scan performed in a 55 year old woman after an episode of arterial embolism caused by atrial fibrillation. Images acquired before contrast media injection (Fig. a) show no abnormality. Images acquired during arterial phase (Fig. b) and venous phase (Fig. c) reveal multiple large infarctions in the spleen (arrows) which show no enhancement when compared to pre-contrast images. The infarcted areas show a vaguely triangular shape, with the base of the triangle coinciding with the splenic capsule (dotted lines in Fig. c). Abbreviations: pancreatic tail (PT), spleen (S)

also be evident. After contrast administration, CT reveals characteristic triangular areas of reduced perfusion. In the subacute phase, the lesions experience volume loss due to necrosis, associated with further reduced attenuation. By the late phase, infarcts evolve into areas of fibrosis with bright calcifications [16, 23–25].

4 Pancreas

4.1 Acute Pancreatitis

Diagnosis is obtained when at least two of the following criteria are satisfied: [1] abdominal pain, [2] amylase/lipase >3 times the upper normal value, and [3] characteristic imaging findings. Since severity might be underestimated in the early phase disease, it is best to perform a second assessment at imaging in the advanced phase (from the second week since onset of symptoms), in order to optimize treatment and identify complications (Fig. 11) [26].

Ultrasonography has no role in acute diagnosis but may be performed for detecting stones or biliary dilatation and for monitoring fluid collections.

CT should be performed roughly 72 h after the onset of symptoms. Indications for CT include the confirmation of diagnosis, the evaluation of clinically severe AP, reassessment due to lack of improvement following initial treatment, assessment for pseudocyst, or ruling out pancreatic adenocarcinoma as an underlying cause of AP in patients >40 years of age [27]. If complications are not present at the first imaging (72 h), a reevaluation in the late phase is recommended.

Two types of AP include interstitial edematous pancreatitis (IEP) and necrotizing pancreatitis (NP).

4.1.1 Interstitial Edematous Pancreatitis

Focal or diffuse enlargement without necrosis and mild peripancreatic fat stranding due to fluid [26]. Complications of IEP include:

- Acute peripancreatic fluid collection: <4 weeks since onset of AP, fluid collections surrounding the pancreas, no discreet walls, spontaneous resolution.
- Pseudocyst: ≥ 4–6 weeks, fluid collection confined within a wall, possible communication with main pancreatic duct (MPD), spontaneous resolution. Takes 4–6 weeks to develop and organize. Endoscopic ultrasound can also drain into the stomach.

4.1.2 Necrotizing Pancreatitis

Areas of low density and reduced enhancement in ≥30% of the gland indicating necrosis (Fig. 12) [26]. Complications of NP include:

- Acute necrotic collection: <4 weeks, dense fluid collections (necrotic debris) within or near the gland, possible communication with MPD.

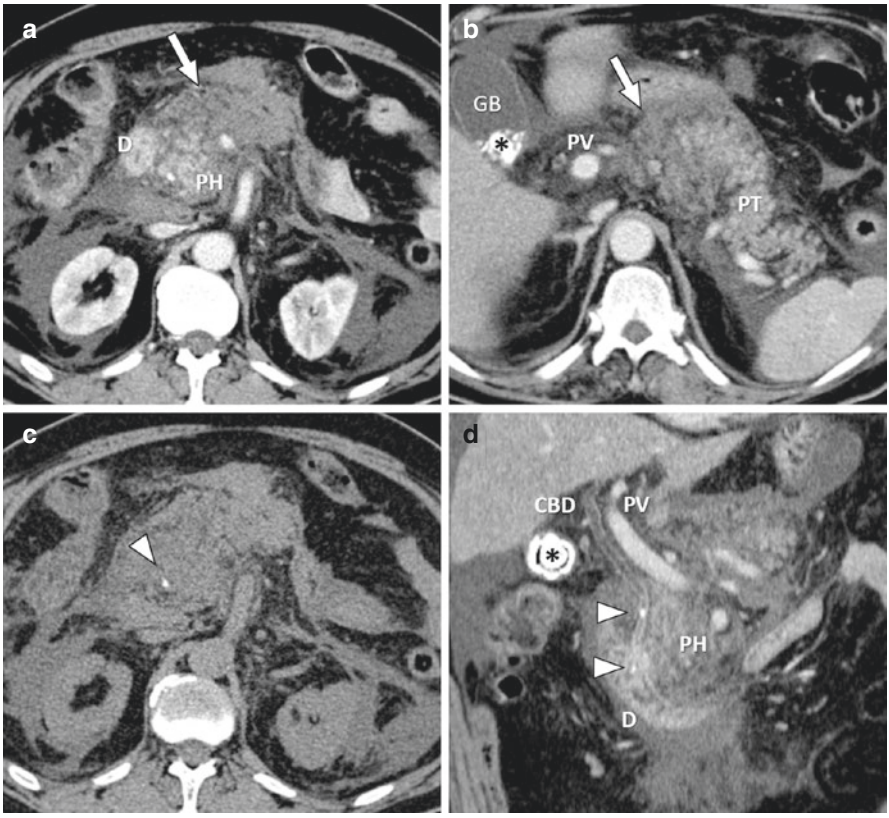

Fig. 11 Acute pancreatitis in early phase, CT. Contrast-enhanced CT obtained in a 53 year old man for confirmation of acute pancreatitis at the time of admission. Figures a and b obtained after intravenous contrast administration show possible initial necrosis in the pancreatic body (arrow) seen as a faint area of reduced enhancement, compared to the normal parenchyma of the head and tail of the gland. Acute peripancreatic fluid collections (APFC) can be seen surrounding the pancreas and in the intraperitoneal space anterior to the kidneys. Figure c acquired before contrast administration shows a focal hyperattenuating object in the pancreatic head, representing a calculus (arrowhead) causing biliary obstruction and pancreatitis. A coronal reformation in Fig. d shows actually two calculi causing obstruction of the common bile duct (arrowheads) and a larger one in the gallbladder (asterisk in figures b and d). Abbreviations: duodenum (D), portal vein (PV), pancreatic head (PH), pancreatic tail (PT), common bile duct (CBD)

- Walled-off necrosis: ≥4 weeks, collection of non-fluid debris within a thickened wall of granulation tissue, requires surgical resection. This is similar to infected solid tumors that may also be difficult to treat with small IR tube drainage, due to solid phlegmonous material or solid tumor debris.

Necrotic collections may develop superimposed infection as a further complication. This is usually evident from the presence of free extraluminal gas in the

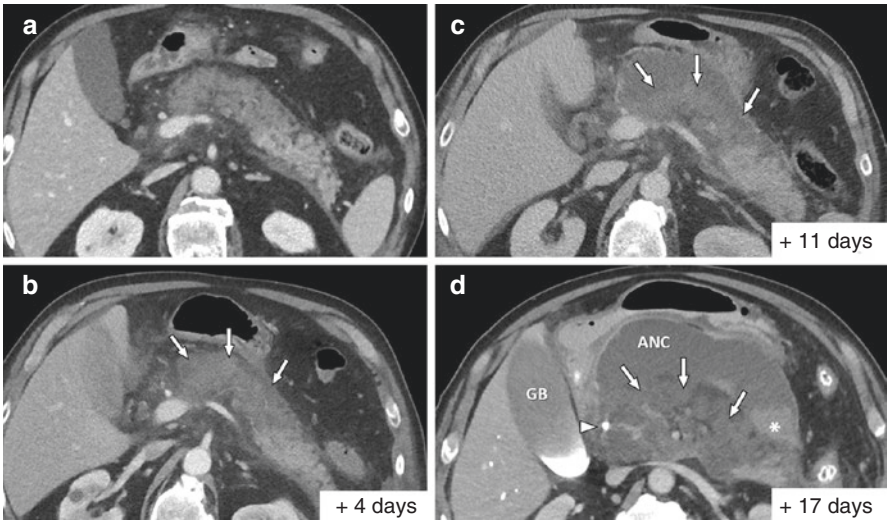

Fig. 12 Development of acute necrotic collection in necrotizing pancreatitis, CT. Contrast-enhanced CT scan was performed for the characterization of early acute, revealing a faint area of reduced enhancement in the pancreas as seen in Fig. a. A second CT performed four days later (Fig. b) clearly shows further increase in the hypoattenuating area in the pancreatic body, strongly suggestive for necrosis (arrows). Two other CT scans performed respectively 11 and 17 days after the first (Fig. c and d) show persistence of the necrotic area (arrows) and progressive development of a fluid collection anterior to the pancreas. Figure d shows a large acute necrotic collection (ANC) with a dense anterior wall and fluid-debris level (asterisk). Due to mass effect on the common biliary duct causing biliary obstruction and icterus, percutaneous drainage of the common bile duct was performed, resulting in the collection of hyperattenuating contrast media in the duct (arrowhead) and in the gallbladder (GB)

abdomen. Other possible signs consistent with necrosis are air bubbles or gas-fluid levels in the collection [27].

Reassessment should be performed if the clinical picture suggests complication, in cases of severe AP (7–10 days after admission and before discharge) and to assess efficacy of surgical treatment. All that is dark on CT may not be fluid. Ultrasound is the best method to tell solid from cystic. High interstitial pressures in tumors can also preclude enhancement and look like necrosis.

4.1.3 Chronic Pancreatitis

Imaging is utilized to diagnose chronic pancreatitis and to grade its severity. Early CP should be evaluated with MRI +/− MRCP, as CT shows low sensitivity. In late CP, typical CT findings include dilation of the main pancreatic duct (MPD) and side branches, with irregular contour of the ducts. Parenchymal atrophy may be present, although not specific for CP, as atrophy (with fatty replacement) is common and normal in older ages. Parenchymal or intraductal calcification may be seen in those cases of CP that are related to alcohol use. Occasionally, enlargement of the pancreatic head may mimic tumor [27].

Complications include pseudocyst, pseudoaneurysm, splenic vein thrombosis, and compression on biliary ducts due to mass effect.

4.2 Cystic Pancreatic Lesions

Most common lesions in this category include *intrapancreatic mucinous tumors (IPMN), mucinous cystic neoplasms (MCN), serous cystic adenoma (SCA), and pseudocysts* [28] (Table 2).

Ultrasonography may be used as a surveillance tool but not for diagnosis.

CT detects malignant features when present, although correct classification is not always possible. Fig. 13 shows a main duct IPMN on contrast-enhanced CT.

Small, unilocular, and asymptomatic pancreatic cystic lesions for which classification is not possible should be reassessed at 6 and 12 months after detection [27, 28]

4.3 Pancreatic Adenocarcinoma (PDA)

Ultrasonography has very low accuracy for the detection of PDA, and its use is limited to assessment for concomitant biliary obstruction or choledocholithiasis. Due to overlying bowel gas, the pancreas may be hard to visualize on US.

Contrast-enhanced CT with arterial and venous phases, multi-planar reconstruction and super thin slices is required for staging. Contrast is mandatory for detection, as PDA is usually hypovascular compared to the normal gland. Typical signs include mass effect with interruption and dilation of the MPD and of the common bile duct, as well as atrophy of the distal parenchyma (Figs. 14 and 15). CT shows moderate sensitivity for hepatic and regional lymph node metastases [27].

Local staging is performed with either contrast-enhanced CT or MRI. The National Comprehensive Cancer Network guidelines for local staging classify tumors as either resectable, borderline resectable, or unresectable, with regard to the possibility of a curative resection with disease-negative margins. Resectability

Table 2 Pancreatic lesions

Lesion type	Sex, age, site	Characteristic CT findings	Malignant features
SCA	F 75%, M 25% 60–70y Head, body, tail	Microcystic (<2 cm) Sponge-like, lobulated Calcifications (central) Central scar in 1/3 No communication with MPD	None (usually *benign*)
IPMN side branch	F 40%, M 60% 60–70y Head, body, tail	Macrocystic “Bunch of grapes” shape Communicates with MPD	>6 mm MPD dilation Size >3 cm Mural nodules (*Surveillance possible*)
IPMN main duct	F 40%, M 60% 60–70y Head, body, tail	Diffuse MPD duct dilatation	>10 mm MPD dilatation Mural nodules (*Resection recommended*)
MCN	F 40–50y Body, tail	Macrocystic Oval with thick walls Calcifications (peripheral) No communication with MPD	Solid component Peripheral calcification (*Resection advised*)

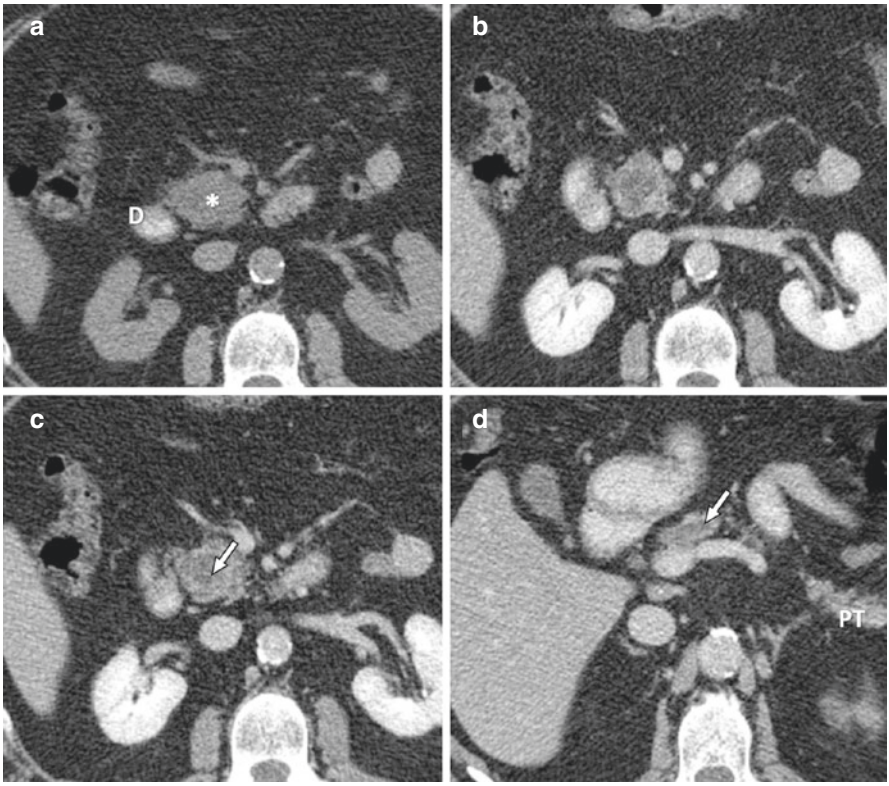

Fig. 13 Main duct IPMN, CT. Contrast-enhanced CT of a 70 year old man show a hypodense cystic lesion in the pancreatic head of fluid density (asterisk in **a**) without significant enhancement compared to the surrounding parenchyma after contrast enhancement (**b**. The lesion measures 22 mm suggestive for macrocystic type and shows growth inside the main pancreatic duct (arrow in **c** and **d**). Location, MPD involvement and macrocystic features suggest main duct intrapapillary mucinous neoplasia (IPMN) as the most likely diagnosis. Abbreviations: duodenum (**d**), pancreatic tail (PT)

criteria are based upon the involvement of nearby arteries (superior mesenteric artery, common hepatic artery, celiac trunk) and veins (superior mesenteric vein, portal vein) [29].

Distant staging must be performed once the patient has been classified as having a resectable or borderline resectable PDA. A contrast-enhanced MRI of the liver and a contrast-enhanced CT of the chest are then required to evaluate for distant metastases [29].

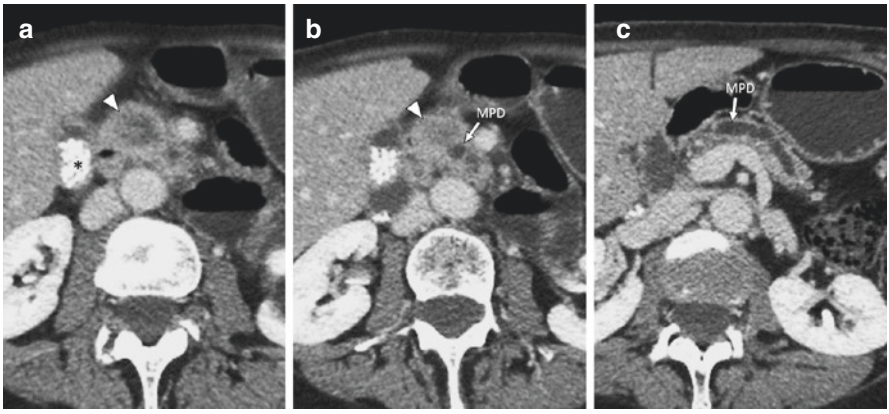

Fig. 14 Pancreatic adenocarcinoma, CT. Contrast-enhanced CT performed in a 62 year old woman shows a 30 mm lesion located in the pancreatic head with indistinct margins (arrowhead in **a**) and reduced enhancement compared to normal parenchyma, . The lesion's size causes mass effect on surrounding structures, with retro-dilation of the main pancreatic duct (arrow in **b** and **c**). The parenchyma of the pancreatic tail shows moderate atrophy. Innumerable hyperattenuating biliary stones can be seen in the gallbladder (asterisk in **a**)

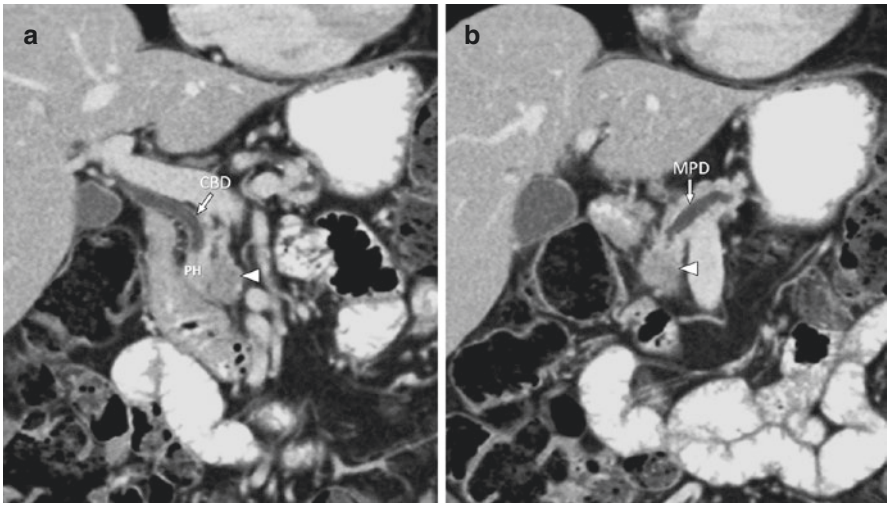

Fig. 15 Pancreatic adenocarcinoma, CT. Coronal reformations of a contrast-enhanced CT scan performed in a 70 year old woman with adenocarcinoma of the uncinate process of the pancreas. The lesion is again visualized as an area of reduced attenuation (arrowhead in Fig. a and b). Figure a shows how the lesion causes compression over the pancreatic head (PH) with biliary obstruction and retro-dilation of the common bile duct (CBD). Figure b shows retro-dilation of the main pancreatic duct (MPD), proximal to and extending immediately adjacent to the mass

5 Bowel

5.1 Appendicitis

Appendicitis is an inflammation of the vermiform appendix. It is a very common condition in general radiology practice and is one of the main causes for abdominal surgery in young patients. Clinical exam is key, but imaging may provide useful information for surgical decisions.

Acute appendicitis is most common between the ages of 10 and 20 years but may occur at any age, with a slight male predilection (male to female ratio of 1.4:1) [30].

The triggering factor in the development of acute appendicitis is the obstruction of the appendiceal lumen from any cause, commonly due to a fecalith (appendicolith).

Appendiceal luminal obstruction leads to distention, tissue ischemia, bacterial overgrowth, and inflammation. The inflammatory process can extend from the appendix to the adjacent peri-appendiceal fat and may also involve the cecum and terminal ileum.

The classical presentation of acute appendicitis consists of referred periumbilical pain, which within a day or later migrates to right iliac fossa, with associated loss of appetite, fever, nausea, and vomiting. However, not all patients present in a typical manner.

Imaging techniques for the evaluation of suspected appendicitis include plain radiographs and more commonly ultrasound and CT (Figs. 16 and 17).

Radiography has very low sensitivity and specificity for appendicitis. In perforated but contained appendicitis, the finding of free intraperitoneal air is infrequent because the inflammatory process is usually blocked off by the adjacent mesentery. Plain radiographs may show an appendicolith in a small number of cases. Rarely, dilated segments of small bowel may be recognized in the right lower quadrant as a result of obstruction from the inflammatory process (sentinel loop).

Ultrasound is recommended as an initial imaging study in children, thin young women, and pregnant women [31]. The sensitivity of US ranges between 44% and 98%, and its specificity ranges between 47% and 95% [32]. The primary ultrasound finding for the diagnosis of acute appendicitis is the presence of aperistaltic, non-compressible, blind-ended tubular structure with a diameter $\geq$ 6 mm cross section in the right iliac fossa with thickened wall (>3 mm) and a targetoid or laminated appearance. The inflamed appendix may demonstrate wall hyperemia on color Doppler ultrasound; an appendicolith may be identified as a rounded intraluminal calcified deposit with associated distal shadowing.

The fat surrounding the appendix may appear echogenic due to the inflammation and focal fluid collections or reactive enlarged lymph nodes may be present.

The sensitivity and specificity of CT examination of appendicitis range between 87% and 100% and 89% and 99%, respectively [32], and CT involves a relatively small dose of ionizing radiation.

CT features of acute appendicitis are comparable to US findings: a distended fluid-filled appendix with a thickened, hyperenhancing wall, peri-appendiceal fat

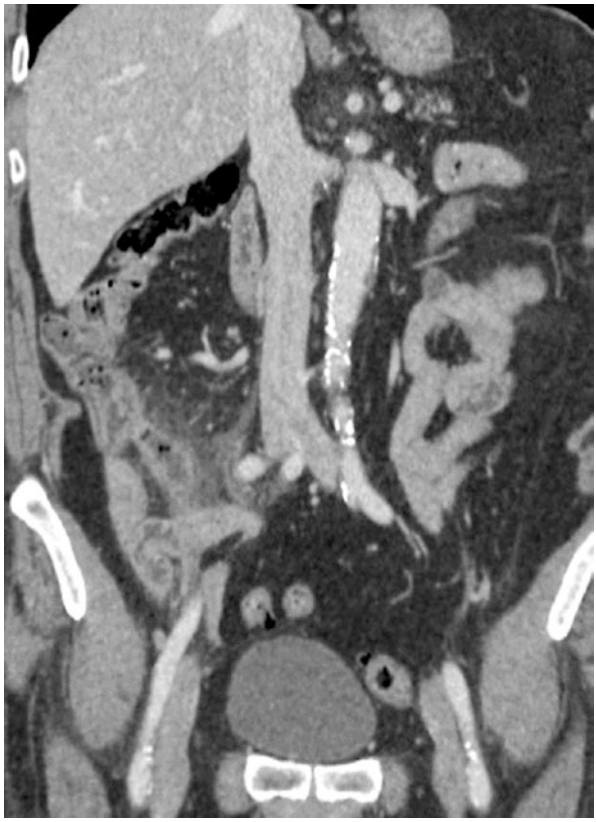

Fig. 16 Acute appendicitis, CT. Reformatted coronal intravenous contrast-enhanced CT image shows a thick walled, distended, fluid-filled appendix (arrow) with adjacent peri-appendiceal inflammatory stranding or dirty fat

stranding, and extraluminal fluid. An appendiceal diameter greater than 6 mm is indicative of appendicitis (Table 3).

A calcified appendicolith may be present in the appendiceal lumen. An indistinct wall or focal areas of absent enhancement may indicate ischemia and infarction, while signs of perforation include phlegmon, abscess, and extraluminal air [33].

The evolution of the inflammatory process can lead to appendicular perforation and consequent complications, which most commonly include abscess and peritonitis. Abscess is the most frequent complication of perforation [34]. CT shows a loculated, rim-enhancing fluid collection that may have mass effect on adjacent bowel loops (Figs. 18 and 19).

Bacterial peritonitis is a dangerous complication more common in young children, due to appendiceal rupture before formation of inflammatory adhesions.

CT and sonography can show between-loop fluid and free fluid following the peritoneal reflections, even far from the appendix. Frequent sites are the pelvis, the paracolic gutters, and the subhepatic and subphrenic spaces.

Uncommon complications include gangrenous appendicitis (CT findings of wall pneumatosis, shaggy appendiceal wall, and patchy areas of mural nonperfusion)

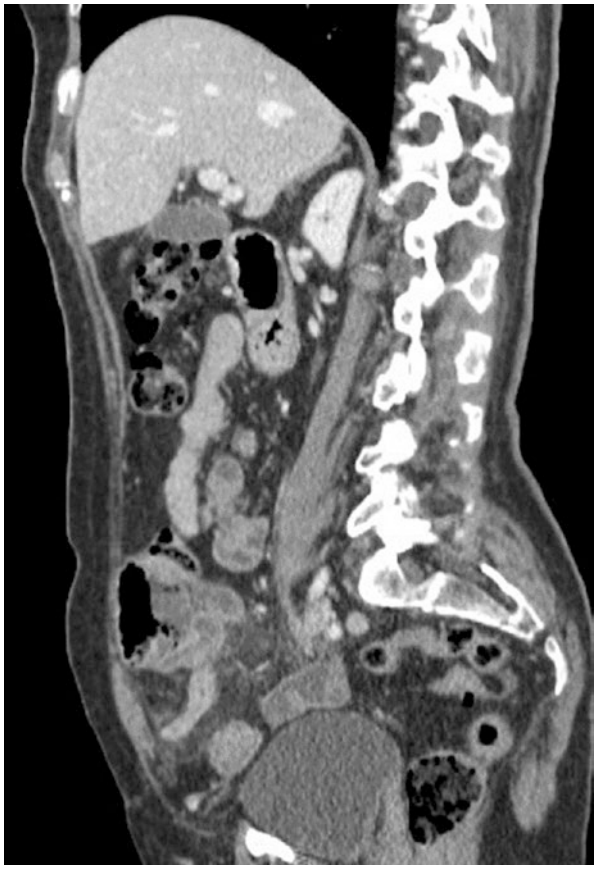

Fig. 17 Acute appendicitis, sagittal CT. Acute appendicitis shows a fluid-filled dilated appendix (arrow) with thickened wall, fat stranding and small amount of free fluid in the right paracolic gutter and in the Douglas pouch

Table 3 CT findings of appendicitis in symptomatic patients

CT findings	Interpretation
<6 mm appendix or >6-mm-thin walled appendix, completely gas-filled	Excludes appendicitis
6–10 mm appendix + wall thickening + wall hyperenhancement (no fat stranding)	Probable appendicitis
>10 mm appendix or >6 mm appendix + wall thickening + wall hyperenhancement + fat stranding	Definite appendicitis

and bowel obstruction secondary to entrapment of the distal ileum in a periappendiceal phlegmon.

Several alternative conditions may mimic appendicitis, and distinguishing between each is important because many are self-limited and respond to conservative treatment. Differential diagnosis include mesenteric adenitis, cecal diverticulitis, omental infarction, infectious terminal ileitis, IBD, perforated appendiceal carcinoma, and mucocele. *Yersinia enterocolitica* is the most common cause of mesenteric adenitis in North America, temperate Europe, and Australia.

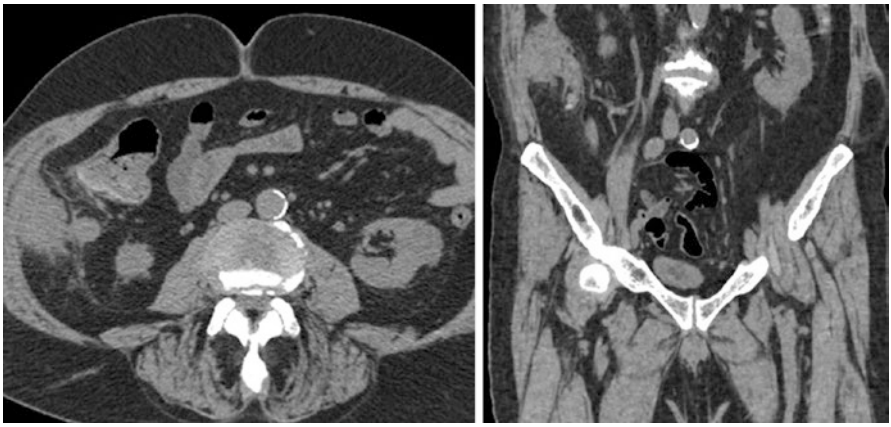

Fig. 18 Acute appendicitis with peri-appendiceal abscess, CT. Non-contrast axial and coronal CT scans show a fluid-filled blind-ending tubular structure on the right flank (arrow) with a subhepatic abscess (lateral arrow). The appendix is distended due to an obstructing hyperdense appendicolith (arrow, right image)

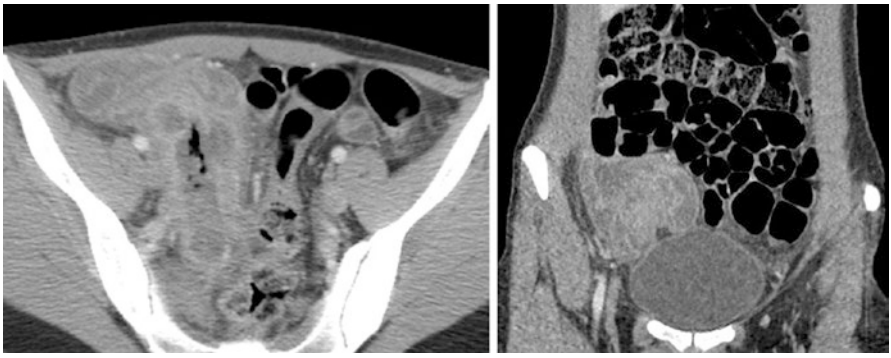

Fig. 19 Acute appendicitis with peri-appendiceal abscess, CT. Contrast-enhanced axial and coronal CT scan of an inhomogeneous fluid collection with enhancing rim, consistent with appendiceal abscess, within the right iliac fossa involving terminal ileum from a perforated appendix, confirmed by surgery

5.2 Diverticulitis

Diverticulosis (presence of diverticula) is a common condition in Western society, affecting 5–10% of the population over 45 years of age and approximately 80% of those over 85 years of age [35]. Diverticula can occur anywhere throughout the colon but are most common in the sigmoid colon. They represent acquired herniations of the mucosa and portions of the submucosa through the muscularis propria.

Diverticula occur mostly where the vessels penetrate the muscularis. Diverticula vary in size but usually range from 2–3 mm up to 2 cm.

Diverticular disease of the colon represents a continuum from an initial, pre-diverticular phase of marked muscular thickening of the colon wall, to frank outpouching (diverticulosis) and finally to frank diverticular inflammation (diverticulitis).

The clinical management of patients with acute diverticulitis depends on the severity, type, and extent of the pericolic inflammatory changes. For mild forms of diverticulitis, medical management with antibiotic therapy and supportive care is adequate.

Surgical resection is indicated for complicated forms, either at the time of the diagnosis or after an interval of antibiotic therapy and percutaneous abscess drainage [36]. Tube drainage of diverticular abscess may cool it down and allow for a single stage resection procedure (without a colostomy phase).

Approximately 15% of patients will require surgery for diverticular disease [37].

Complicated diverticulitis includes a broad spectrum of disease presentation, ranging from small pericolic abscesses to perforation with generalized peritonitis and sepsis, as well as late complications, including fistula and stricture formation.

The most commonly used grading system to describe the severity of complicated diverticulitis is the Hinchey classification (Table 4).

In most patients with acute diverticulitis, abdominal radiographs are of limited value and do not contribute to the diagnosis. Radiographs can be diagnostic only in the most severe forms of diverticulitis, in which the patient presents with free abdominal air.

CT is now considered the gold standard for assessing diverticulitis and its complications (Figs. 20, 21, and 22).

At CT, diverticulosis appears as small, air-filled outpouchings of the colonic wall, more numerous in the sigmoid colon. The wall of the involved colonic segment may appear thickened due to muscular hypertrophy.

In mild diverticulitis, the most frequent finding is a slight increase in the attenuation of fat adjacent to the involved colon, with engorgement of the vasa recta. The degree of fat stranding may vary from minimal "dirty fat" to severe inflammation. Dirty, inflamed fat appears as hyperattenuation relative to the hypoattenuation of normal fat on CT.

Fine linear strands, small fluid collections, and several bubbles of extraluminal air may be present. In more severe cases, pericolic heterogeneous fluid collections (phlegmons) or intramural/ extraintestinal abscess can occur. On CT, abscesses appear as fluid collections that may contain bubbles of air or air-fluid levels, with

Table 4 Classification of acute diverticulitis (Hinchey)

1a	Pericolic phlegmon and inflammation
1b	Diverticulitis with pericolic or mesenteric abscess
2	Diverticulitis with walled off pelvic abscess
3	Diverticulitis with generalized purulent peritonitis
4	Diverticulitis with generalized fecal peritonitis

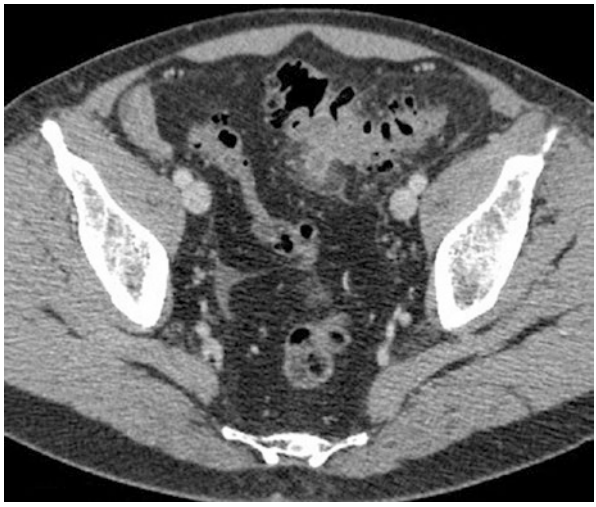

Fig. 20 An example of uncomplicated diverticulitis, CT. Contrast-enhanced axial CT demonstrates edema and thickening of the sigmoid colon wall with multiple diverticular outpouchings (arrow) and fat stranding involving the sigmoid mesocolon around the diverticula, representing mild diverticulitis

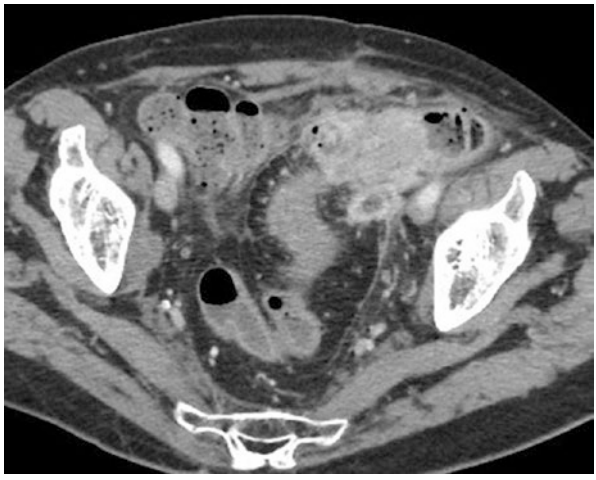

Fig. 21 Diverticulitis with confined peri-sigmoid inflammation, CT. Axial contrast-enhanced CT image shows diverticulitis with edematous sigmoid colon, multiple diverticula and perisigmoid abscess with enhancing wall (arrow) and fat stranding

central necrotic component and peripheral ring enhancement. These collections can appear near to the involved segment of the colon, or they can form at a distance: in the psoas muscle, flank, groin, thigh, subphrenic space, or liver. Other complications such as bowel obstruction, hepatic abscess, and fistula can often be demonstrated with CT. Fistulas frequently communicate with an abscess or other hollow viscus (more commonly the bladder). Severe complications necessitate intensive management. This is not to be confused with epiploic appendagitis, which can look like dirty fat benzene rings on CT in the pericolonic fat (from loss of blood flow to the small fatty appendages on the antimesenteric side of the colon, due to twisting or thrombosis in the small central draining vein).

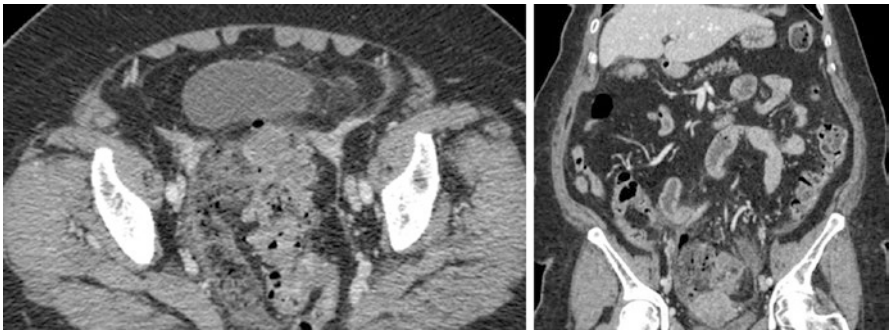

Fig. 22 Free perforation of sigmoid diverticulitis with the development of peritonitis, CT. Contrast-enhanced axial and coronal CT images of the sigmoid colon shows segmental mural thickening and multiple diverticula surrounded by inflammatory fat stranding and a perisigmoid abscess with free air bubbles. These findings suggest perforated diverticulitis and combined peritonitis, confirmed by surgery

5.3 Idiopathic Inflammatory Bowel Disease

Inflammatory bowel disease (IBD) is a chronic idiopathic disease affecting the gastrointestinal tract that include two related intestinal disorders, ulcerative colitis (UC) and Crohn's disease (CD). The etiology of IBD is still unknown; currently, the prevalent theory is an inappropriate immune response to gut luminal microbes in genetically susceptible people who are exposed to environmental risk factors. There is no gender predominance in IBD, with peak age of onset between 15 and 30 years of age but affecting people of all ages. Up to 20% of people with IBD are diagnosed during infancy, and the prevalence and incidence of pediatric IBD are growing worldwide [38].

5.3.1 Crohn's Disease

The pathologic findings in Crohn's disease are highly dependent on the duration of disease. Early disease presents superficial aphthoid ulcers of the mucosa that may become confluent. There is progression from limited mucosal disease to transmural inflammation of the bowel with collagen deposition eventually. Collagen deposition leads to obliteration of the submucosa, which results in stricture formation that may lead to obstruction.

Crohn's disease can involve any portion of the gastrointestinal tract from the mouth to the anus, although the small bowel is the most commonly affected portion of the bowel, particularly the RLQ distal and *terminal ileum* [39].

The earliest phases of small bowel inflammation may be characterized only by subtle mucosal hyperenhancement on the arterial phase images, with little or no wall thickening.

As the degree of inflammation progresses, the typical feature is bowel wall thickening of 1–2 cm. During the acute phase, the colon preserves mural stratification and often has a "target" or "double halo" appearance, due to submucosal edema among inner (mucosa) and outer (muscularis propria) rings of high attenuation [40].

In patients with long-standing Crohn's disease, mural stratification is lost, so that the affected bowel wall typically has a homogeneous attenuation on CT, suggestive of fibrosis or intramural deposition of fat.

Extraluminal, locoregional manifestations of Crohn's disease include fibro-fatty proliferation, the loss of the sharp interface between the involved bowel segment and mesentery, small mesenteric lymph nodes (from 3 to 8 mm), hypervascularization, and dilatation of the involved mesentery ("comb" sign).

CT evaluation may be useful to assess extraluminal disease complications of CD (e.g., fistula formation between bowel loops and between bowel and other visceral organs, abdominopelvic abscesses, or perforations) or extraintestinal manifestations of disease (e.g., renal lithiasis and sacroiliitis) [41] (Fig. 23). Air in the bladder in a young patient without bladder catheterization is from a fistula in Crohn's disease until proven otherwise.

Ultrasound findings of CD include mural wall thickening, loss of mural stratification, and increased blood flow at color Doppler imaging and often show that the colon has less peristalsis and less compressibility than normal.

5.3.2 Ulcerative Colitis

Ulcerative colitis is a diffuse inflammatory disease that primarily involves the colorectal mucosa but later extends to other layers of the bowel wall. The disease begins in the rectum and extends proximally in a continuous pattern to involve either part of the colon or its entirety.

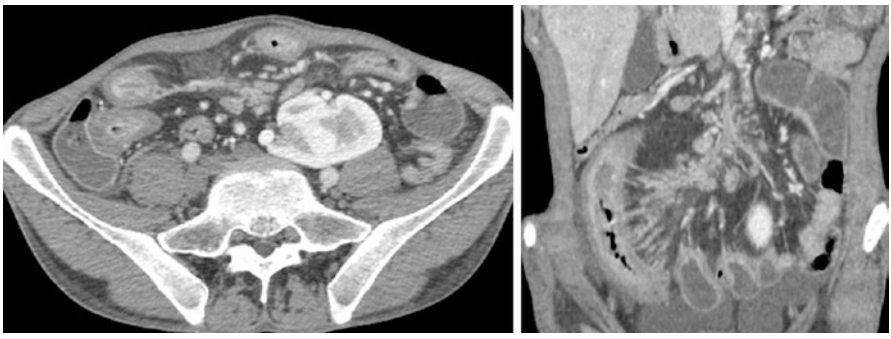

Fig. 23 Active Crohn's disease, CT. Contrast-enhanced axial and coronal CT images show marked wall thickening and hyperenhancement of the terminal ileum (arrow), near the ileocecal valve, with free peritoneal fluid and enlarged mesenterial lymph nodes. These findings suggest active inflammatory Crohn's disease, confirmed by endoscopy

Clinically, patients have chronic diarrhea, sometimes bloody, associated with tenesmus, pain, and fever. Some patients may have extraintestinal manifestations.

Abdominal X-rays can provide information in the acute setting, assessing for bowel perforation and toxic megacolon.

The radiological hallmark of active UC is the presence of colonic mural thickening and enhancement with luminal narrowing. A mean wall thickness of 8 mm has been reported in UC patients with active disease [42].

Radiological features of chronic UC may also include deposition of fat in the colonic wall, rectal narrowing, and consequent widening of presacral space and stranding of perirectal fat.

CT has an important role in the evaluation and detection of complications in patients with UC (Fig. 24). Toxic megacolon is a rare but very serious complication of UC. CT findings of toxic megacolon include luminal distension with thinning of the colonic wall and pneumatosis. Severe cases can lead to perforation and free air.

Beyond radiological imaging, colonoscopy is the principal modality for diagnosing and determining extent of disease in UC. In fact, mucosal abnormalities that characterize the early stages of the disease are beneath the spatial resolution of CT.

5.4 Small Bowel Obstruction

Small bowel obstruction (SBO) is a common clinical condition representing 20% of surgical admission for acute abdominal pain [43].

Since clinical findings are neither sensitive nor specific enough to determine the severity of SBO, the radiological investigation is fundamental in confirming the diagnosis, identifying its underlying cause, and detecting complications that require prompt surgery.

Plain abdominal radiography is the initial examination due to its wide availability and low cost (Fig. 25). However, radiographs are diagnostic in only 50–60% of cases and have high sensitivity only for high-grade obstruction [44].

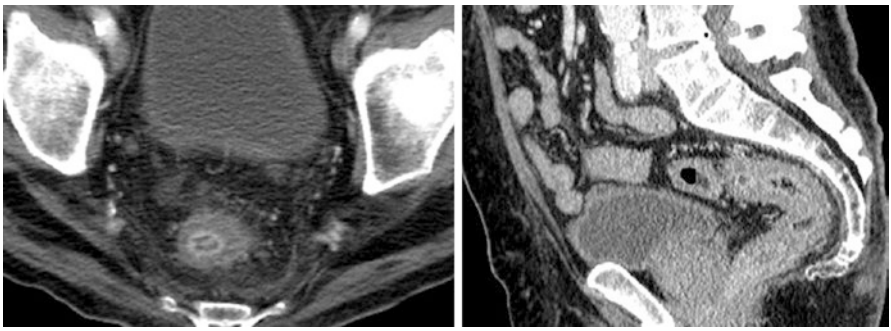

Fig. 24 Active ulcerative colitis, CT. Contrast-enhanced axial and sagittal CT scans show thickened rectal and sigmoid walls with target sign and inflammatory fat stranding (arrow). Active ulcerative colitis was confirmed by endoscopy

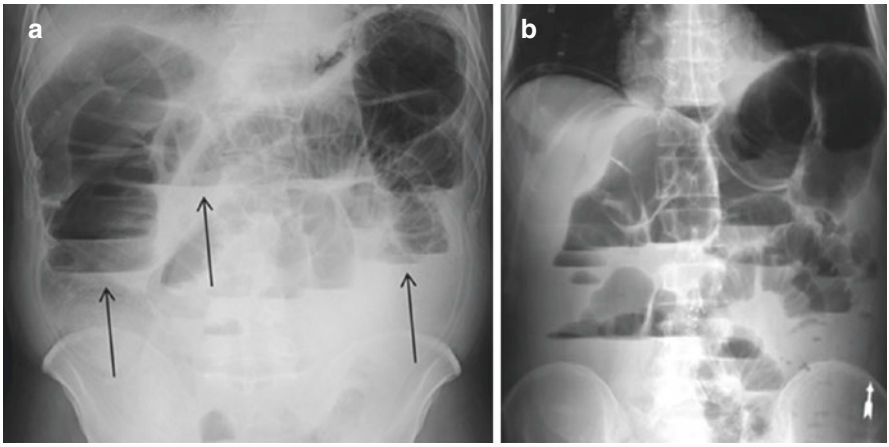

Fig. 25 High-grade SBO, Radiograph. Plain abdominal radiographs show multiple air-fluid levels within the small bowel. Active bowel peristalsis (**a**) working against the blockage results in air-fluid levels (black arrows) of varying heights in different locations (craniocaudal).In contrast, because of minimal peristalsis, an adynamic ileus (**b**) demonstrates air-fluid levels of the same or similar height (red and blue arrows). Radiograph *b* shows a paralityc ileus after abdominal surgery

Table 5 Diagnosis of SBO

Criteria	Specific criteria for diagnosis of SBO
Major	Small bowel dilated to 2.5 cm or greater and colon not dilated. Transition point from dilated to nondilated small bowel.
Minor	Air fluid levels. Colon decompressed.

The key radiographic signs of significant SBO is the presence of small bowel distention (greater than 3 cm), the absence of colonic dilatation, and the presence of multiple air-fluid levels (at different levels vs ileus which has air-fluid levels at the same horizontal level) (Table 5).

The cause of obstruction is generally not detectable on simple radiography.

US has a limited role in the assessment of SBO because of poor visualization of gas-filled structures.

CT is considered the best imaging modality for the assessment of bowel obstruction with a high accuracy. The typical features on CT include dilated proximal small bowel (>2.5 cm) with collapsed distal small bowel and colon. Air-fluid levels are also present [45].

In high-grade or chronic obstruction, the presence of particulate feculent material mixed with gas bubbles in the small bowel creates an appearance analogous to feces in the colon, the "small bowel feces" sign [46].

The main importance of this sign is that it is usually seen just proximal to the transition point (Fig. 26) [47].

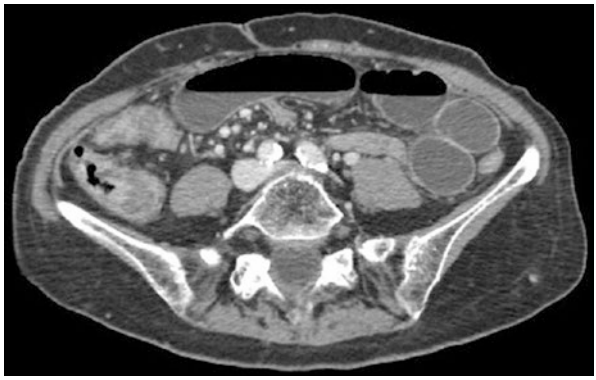

Fig. 26 Identification of the transition point in an SBO secondary to post-surgery adhesions, CT. Contrast-enhanced CT scan shows dilated small bowel loops. There is an abrupt change in caliber between the proximal dilated bowel loops and collapsed distal bowel loops. The change in caliber was due to adhesions

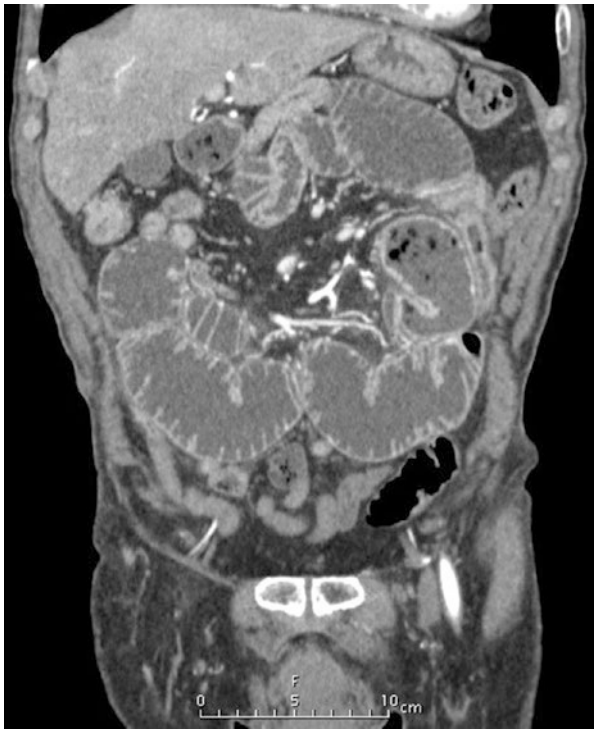

Fig. 27 SBO, CT. Small bowel feces sign in a patient with high-grade SBO secondary to postoperative adhesions. Contrast-enhanced oronal CT scan shows the presence of feculent material mingled with gas bubbles in the lumen of dilated loops of the small intestine. Small bowel can be identified with recognition of the valvulae conniventes (plica circulares, arrow)

Further, CT provides important information about the bowel wall, mesenteric vessels, and adjacent mesenteric fat, allowing the identification of coexistent ischemia or bowel perforation and free extraluminal gas (Fig. 27).

Causes of SBO can be divided into intrinsic to the bowel, extrinsic, and intraluminal. Adhesion is the most common cause of SBO, accounting for approximately 70% of all SBOs [48], but may show nothing on imaging, other than a transition point (Table 6).

Table 6 Causes of SBO

Causes of small bowel obstruction		
Intrinsic causes	Extrinsic causes	Intraluminal causes
Inflammatory diseases (Crohn's, tuberculosis, eosinophilic gastroenteritis) **Neoplasias** (primary or secondary) **Vascular lesions** (radiation enteropathy, ischemia) **Hematoma** **Intussusception**	**Adhesions** **Hernias** (external, internal) **Hematomas**	**Gallstones** **Bezoars** **Foreign bodies**

Approximately 80% of patients with SBO due to adhesions have a history of prior intra-abdominal surgery; the remainder have prior peritonitis or no precipitating causes.

Adhesions represent bands of fibrous tissue that obstruct the lumen, as a consequence of an inflammatory process. They are infrequently seen on CT, and the diagnosis is made by exclusion.

An abrupt change in the caliber of the small bowel with kinking or tethering at the transition zone, without any other identifiable cause, is highly suggestive of adhesion.

External hernias are the second most frequent cause of SBO [48]. They involve most frequently the inguinal canal or the anterior abdominal wall.

In cases of hernias, bowel obstruction occurs if there is incarceration (Fig. 28).

SBO due to hernia manifests with dilated bowel up to the hernia sac followed by decompressed bowel exiting from the sac. Internal hernias occur though acquired or congenital defects in the mesentery, through which bowel may traverse.

In patients with known primary tumors, the most likely cause of SBO is metastatic involvement of the bowel or the peritoneum, in the form of peritoneal carcinomatosis [49]. Tumors with a tendency to cause extensive diffuse peritoneal metastases include ovarian, colonic, pancreatic, and gastric neoplasms.

Primary neoplastic causes of SBO are rare. Intrinsic small bowel neoplasms constitute less than 2% of gastrointestinal malignancies, and they usually manifest at an advanced state as an irregular mural thickening at the transition point [50].

Malignancies that involve the cecum and colon can also result in SBO when there is involvement of the ileocecal valve.

SBO may occur in Crohn disease with an acute presentation or as the manifestation of a long-standing disease, which usually results in stenosis of affected segments.

Intussusception refers to telescoping of a proximal segment of the gastrointestinal tract within the lumen of the downstream segment. Adult intussusception represents 5% of all cases of intussusception and accounts for only 1–5% of intestinal obstructions in adults, whether idiopathic or secondary to any pathologic lesion of the bowel wall [51].

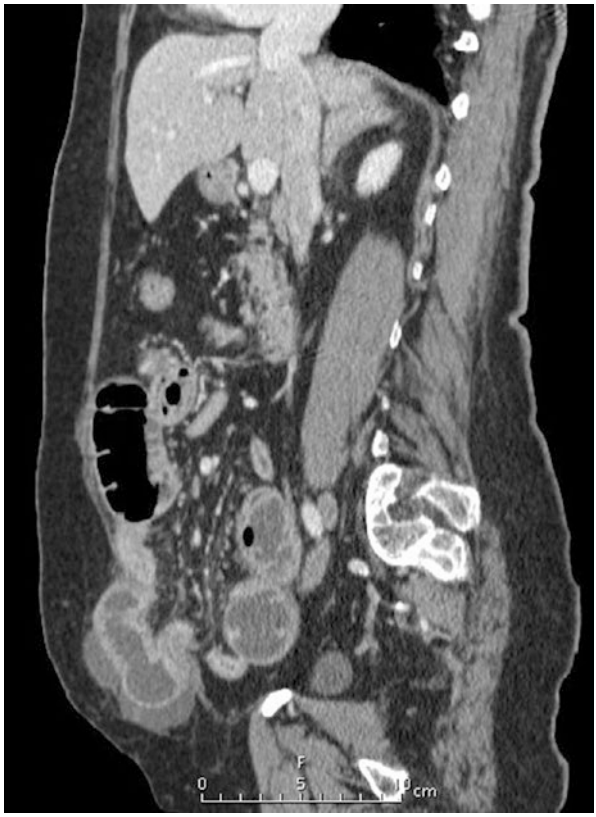

Fig. 28 SBO from incarcerated hernia, CT. Reformatted sagittal contrast-enhanced CT scan shows herniation of small bowel loops through an abdominal wall defect (arrow). The sac of the hernia contains extraluminal fluid and fluid-filled, mildly thickened bowel loops and causes SBO. Incarceration with small bowel obstruction was confirmed at surgery

The typical features of CT include a heterogeneous "target" shaped soft tissue mass with a layering effect; mesenteric vessels within the bowel lumen are also characteristic.

SBO is rarely caused by intraluminal material. The site of obstruction is usually at the ileocecal valve. Gallstone ileus is a mechanical obstruction due to impaction of one or more gallstones within the gastrointestinal tract that typically manifests with the Rigler triad, SBO, pneumobilia, and ectopic gallstone, usually in the right iliac fossa.

Closed-loop obstruction occurs when a segment of bowel is obstructed at two points along its course, resulting in progressive accumulation of fluid in gas within the isolated loop, placing it at risk for volvulus and subsequent ischemia.

Ischemia is the complication that increases the morbidity and mortality in patients who undergo surgery for SBO; when ischemia is suspected, immediate surgery is required to avoid transmural necrosis and perforation.

The CT findings of ischemia include bowel wall thickening, fluid in the adjacent mesentery, mesenteric edema, decreased bowel wall enhancement, and intestinal pneumatosis with or without associated mesenteric or portal venous gas [52].

5.5 Colorectal Cancer

Colorectal carcinoma (CRC) comprises approximately 90% of all large bowel tumors and is a major cause of morbidity and mortality in the western world. Worldwide, CRC is the third most commonly diagnosed cancer in both men and women. CRC incidence patterns are generally similar in men and women. The lifetime risk for developing colorectal cancer is 5–6% and increases with age; the typical age of diagnosis is during the sixth and seventh decades of life.

The CRC mortality has declined over the past two decades, due to advances in early detection and treatment [53]. The majority of CRC cases are sporadic (80%), and the rest are the result of genetic mutations [54]. The adenoma-carcinoma sequence accounts for approximately 70% of CRC pathogenesis, which takes 7–10 years to progress from benign adenoma to malignant tumor, which is why screening colonoscopy or virtual colonoscopy is typically recommended every 5 years. Sessile serrated polyps are associated with mutations and do not follow the usual rules of slow predictable transition from adenomas; therefore more frequent colonoscopy may be indicated.

The signs and symptoms of CRC vary and include abdominal pain, hematochezia, melena, unexplained iron deficiency anemia, diarrhea, obstruction, and bowel habit changes. However, there are no specific symptoms of early-stage colon cancer.

The initial diagnosis of CRC is usually made with colonoscopy. Colonoscopy allows biopsy samples to be taken for definitive diagnosis with a simultaneous opportunity for a therapeutic polypectomy.

Imaging, however, plays a fundamental role in determining the stage of disease at diagnosis.

In some cases, the radiologist may be the first to suggest the diagnosis of colon cancer in patients who undergo abdominal CT as the initial imaging modality for a variety of gastrointestinal symptoms.

The sensitivity of CT in detection of primary colon cancer is variable and depends on the size of the tumor [55].

The TNM classification is commonly used for staging of colorectal cancer and is based on the extent of tumoral, nodal, and metastatic involvement (Table 7).

On CT (Fig. 29), CRC commonly presents as asymmetric, short-segment wall thickening, or soft tissue mass that narrows the colonic lumen, with or without an irregular surface. Early-stage CRC can be seen as a polypoid or fungating mass without extracolonic tumor extension. Large tumors may appear as a mass with a central area of low attenuation due to necrosis.

Bulky fungating cancers most often arise in the cecum and ascending colon. Adenocarcinomas in the transverse and descending colon frequently become infiltrative and ulcerating, forming annular constricting tumors that narrow the lumen, referred to as eaten "apple-core" lesions.

Sometimes, it can be difficult to differentiate between benign and malignant colon wall thickening. Imaging features such as asymmetry, loss of haustral pattern, destruction of wall layer pattern, and "shouldered" edges favor malignant colon wall thickening. Classic boards case is iron deficiency anemia and a circumferential

Table 7 TNM staging colorectal adenocarcinoma

The AJCC TNM Staging (eighth Edition) classification for colon and rectal cancer	
Classification	Definition of TNM classification
Primary tumor (T)	Primary tumor cannot be assessed
TX	No evidence of primary tumor
T0	Carcinoma in situ
Tis	Tumor invades submucosa
T1	Tumor invades muscularis propria
T2	Tumor invades through muscularis propria into the pericolorectal tissues
T3	Tumor invades through the visceral peritoneum
T4	Tumor directly invades or adheres to other adjacent organs or structures
T4a	
T4b	
Lymph nodes (N)	Regional lymph nodes cannot be assessed
NX	No regional lymph node metastasis
N0	Metastasis in 1–3 regional lymph nodes
N1	Metastasis in four or more regional lymph nodes
N2	No distant metastasis by imaging
Distant metastasis (M)	Distant metastasis
M0	
M1	

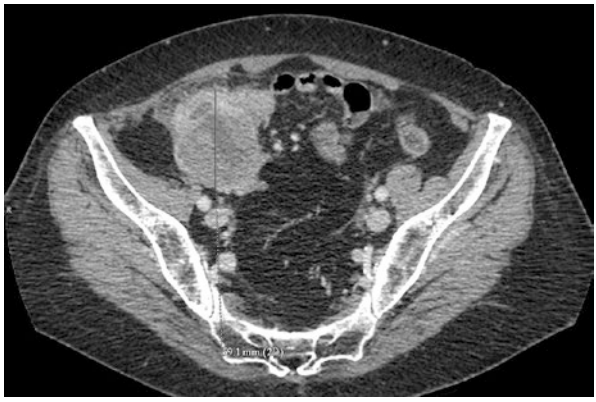

Fig. 29 Cecal adenocarcinoma, CT. Axial contrast-enhanced CT scan show marked circumferential thickening of the cecum. Low-attenuation may represent fluid or necrosis. Adenocarcinoma was confirmed at endoscopy.

"apple-core" cancer lesion on barium enema or CT. Conversely, the presence of fluid in the root of the sigmoid mesentery and engorgement of adjacent sigmoid mesenteric vasculature favor the diagnosis of benign conditions, more often diverticulitis [56].

Owing to its ability to demonstrate the colon and surrounding structures, CT allows detection of regional extension of tumor. Extra-colic spread of tumor is demonstrated by thickening and infiltration of pericolic fat and loss of fat planes between the colon and adjacent organs [57]. Regional lymph node involvement may be seen.

The liver is the most common site of metastasis in patients with colorectal cancer due to its anatomical situation with regard to portal circulation [58]; thus, accurate imaging of the liver is essential.

Hepatic metastases can vary widely in size and usually appear as hypoattenuating masses, which are best visualized during the portal venous phase of liver enhancement. Liver metastases may be amenable to surgical resection, percutaneous thermal ablation, or radioembolization in interventional radiology/interventional oncology.

Other common sites of metastases from colon cancer include the lungs, adrenal glands, and bones.

CT is fundamental for postoperative surveillance for recurrence, both local and distant, and to document "normal" postoperative anatomy. Recurrent tumor after surgery usually appears as a soft tissue mass in or near the surgical anastomosis or in the liver.

CRC can be associated with diverse complications such as obstruction, intussusception, ischemia, perforation, and fistula formation with adjacent organs (Figs. 30 and 31).

Obstruction is the most common complication and is more frequent with left-sided colon cancers because of the smaller diameter of the left colon. The incidence rate of bowel obstruction associated with colon cancer is 8–29% [59]. Right-sided lesions more commonly present with bleeding and anemia, whereas left colon lesions often present with obstructive symptoms. Left may have a better prognosis than right CRC, and left is more likely to respond to the addition of an EGFR-targeted agent.

Perforation can occur either at the site of primary tumor, due to necrosis, or proximal portion of the tumor due to increased luminal pressure. Reported frequency of perforation and abscess formation associated with colon cancer is 2.5–10% and 0.3–4%, respectively [59].

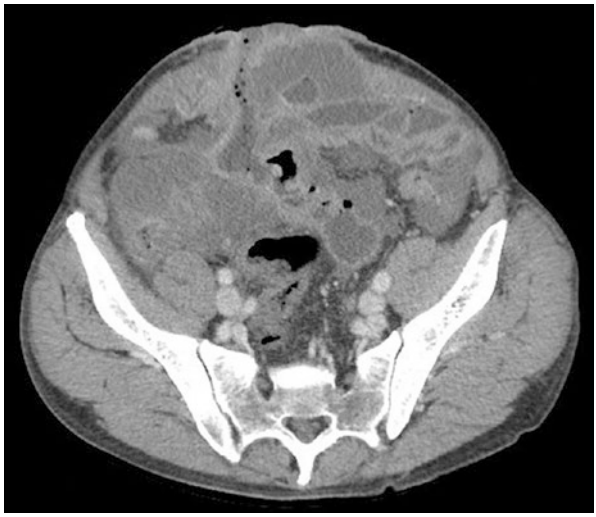

Fig. 30 Cutaneous fistulization of colonic carcinoma in a patient with peritoneal carcinomatosis, CT. Contrast-enhanced axial CT scan shows widespread peritoneal metastases and free abdominal fluid with a colonic loop opened into an abscess and a fistula (arrow) communicating to the skin

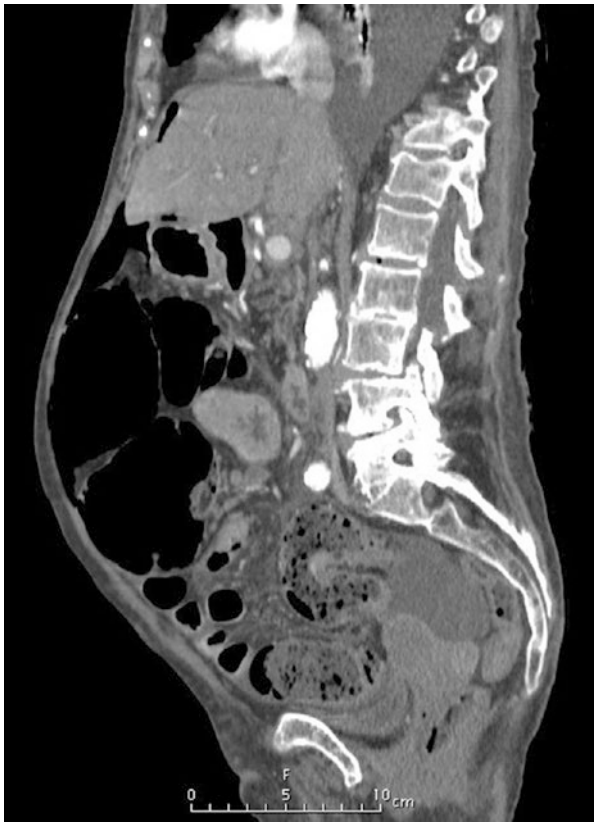

Fig. 31 Colonic perforation in a patient with a history of sigmoid adenocarcinoma who presented with pain and sepsis, CT. Contrast-enhanced sagittal reformatted CT scan shows extensive extraluminal air and free fluid in the abdomen. Focal scleroses involve the lower lumbar and sacral vertebrae, consistent with blastic metastases. Note the metastatic bone involvement of the iliac wing in the axial scan. Surgery confirmed sigmoid cancer perforation.

5.6 Carcinoid Tumor

Carcinoid tumors are a type of neuroendocrine tumor typically found throughout the gastrointestinal tract, with distal predilection in the small bowel. A smaller percentage of them may involve other organs such as the lung, liver, ovary, and thymus. They are slow-growing tumors capable of metastasizing commonly to the mesentery, liver, and lymph nodes.

Clinical presentation of gastrointestinal tract carcinoid depends on the location. More often asymptomatic and usually found incidentally, they can sometimes present as abdominal pain, weight loss, fatigue, and diarrhea.

Chromogranin A is a valuable diagnostic biomarker since elevated serum values are associated with different types of neuroendocrine tumors. It may be found alone or in combination with a 5-hydroxyindoleacetic acid (5-HIAA) test, which suggests a functioning carcinoid tumor.

Carcinoid tumors involving the jejunum and ileum can be large at presentation. On CT, they appear as a well-defined hyperenhancing solid mass associated with desmoplastic reaction and retraction of adjacent small bowel loops. Central calcifications are often described, most frequently in the primary mesenteric masses [60].

5.7 Intestinal Vascular Disorders

5.7.1 Mesenteric Ischemia

Mesenteric ischemia is caused by reduced or interrupted splanchnic perfusion due to arterial or venous occlusion or hypotension. It may be acute or chronic.

Acute mesenteric ischemia (AMI) can be categorized into four specific types based on its cause: arterial embolism (most frequent cause of AMI, responsible for approximately half of cases), arterial thrombosis, nonocclusive mesenteric ischemia, and mesenteric venous thrombosis [61].

The final common pathway of all the specific causes of mesenteric ischemia is bowel infarction.

Clinical presentation is often nonspecific and in most cases can be characterized by a discrepancy between severe abdominal pain and minimal clinical findings. In general, patients with superior mesenteric artery (SMA) embolism or thrombosis have an acute onset of symptoms and a rapid deterioration in their clinical condition.

The mortality rate in patients with acute mesenteric ischemia exceeds 60% [62].

Chronic mesenteric ischemia accounts for less than 5% of intestinal ischemia. Atherosclerotic occlusion or severe stenosis is the most common cause. Symptoms occur when at least two of the three main splanchnic vessels (celiac trunk, superior mesenteric artery, or inferior mesenteric artery) are affected [63] (Fig. 32).

Abdominal radiographs are usually nondiagnostic in the setting of mesenteric ischemia and either are normal or show nonspecific signs.

Although historically, catheter angiography was the gold standard for imaging of suspected intestinal ischemia, CT/CTA is now the investigation of choice. CT allows the visualization of the mesenteric vasculature, bowel, and the entire abdomen for other processes.

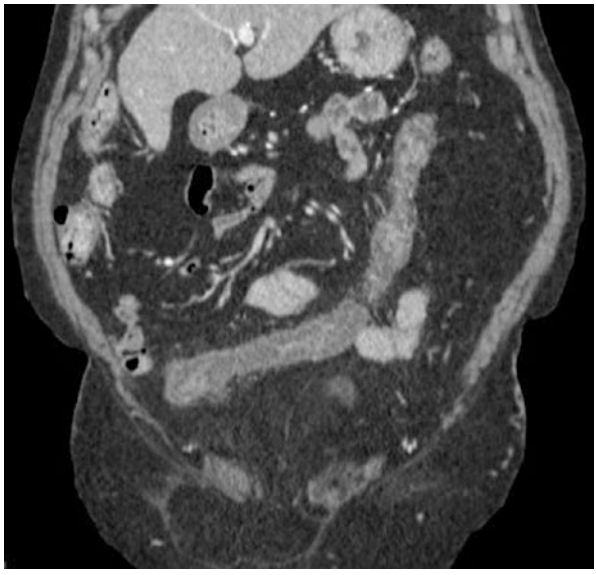

Fig. 32 Ischemic colitis, CT. Contrast-enhanced coronal CT scan shows fat stranding and wall thickening involving the transverse colon through the proximal left colon (arrow). Endoscopic findings confirmed the diagnosis of segmental ischemic colitis

The mesenteric vessels should be evaluated on unenhanced images for arterial calcifications and on intravenous contrast-enhanced CT images for the presence of thrombus or embolus. Acute arterial thrombi and emboli appear as low-attenuation filling defects in the SMA, its branches, or other major mesenteric arteries.

While large emboli and thrombosis may occlude the proximal SMA and major mesenteric vessels causing extensive small bowel and colon ischemia, smaller emboli can obstruct the distal portions of the vessel and lead to smaller regions of segmental ischemia [64]. Filling defects may have high attenuation in the vessels on non-enhanced CT images.

The bowel wall should be assessed on unenhanced images for the presence of increased attenuation, a specific sign of mural hemorrhage.

The presence or lack of contrast enhancement is evaluated on contrast-enhanced images. The absence of bowel wall enhancement is the most specific sign of acute mesenteric ischemia that suggests cessation of arterial flow. It usually indicates transmural infarction [65].

Pneumatosis intestinalis (the presence of gas within the wall of the gastrointestinal tract) is a typical finding of transmural bowel ischemia. The coexistence of pneumatosis intestinalis and porto-mesenteric gas has a very high specificity for ischemic bowel [66]. However, pneumatosis intestinalis is not a specific finding of intestinal ischemia and may occur in a wide range of benign conditions such as COPD or steroid use; however, when found, bowel ischemia must be first excluded [67].

In cases of venous occlusion, circumferential bowel wall thickening, typically under 1.5 cm, is the most common finding. The degree of wall thickening, however, does not correlate with the severity of mesenteric ischemia.

CT findings of venous ischemia include decreased mural enhancement, segmental mesenteric fat stranding, and free fluid interleaved between folds of the mesentery (Table 8).

Thrombosis within the mesenteric veins may appear as a low-attenuation filling defect on contrast-enhanced CT.

In cases of chronic mesenteric ischemia, the small bowel usually appears normal but may have a malabsorption pattern with fluid-filled loops, bowel wall thickening, and free peritoneal fluid. The diagnosis is supported by the presence of calcified atheromatous plaque at the origin of the mesenteric arteries and demonstration of arterial occlusions or severe stenoses in at least two of the three main splanchnic arteries.

5.7.2 Ischemic Colitis

Ischemic colitis refers to inflammation of the colon secondary to decrease in blood flow in the small arterioles of the colon. Most patients with colonic ischemia are elderly and present with mild lower abdominal pain and rectal bleeding. The clinical course and the outcome are highly variable.

Table 8 CT findings in intestinal ischemia

Features	Arterial ischemia	Venous ischemia	Nonocclusive ischemia
Incidence	60–70%	5–10%	20–30%
Acuity	Acute or chronic	Acute or chronic	Acute or chronic
Clinical risk factors	Cardiovascular disease. Septic emboli. Systemic vasculitis	Bowel strangulation, hypercoagulable state, portal hypertension, infection	Hypotension, heart failure, recent surgery or trauma, medications
Vasculature	Arterial filling defect, severe arterial narrowing, dissection, aneurysm	Venous filling defect	Nonspecific
Bowel wall thickness	Thin (acutely) or thickened; may be involved with hematoma, edema, or inflammation	Thickened and edematous	Generally thickened
Bowel wall enhancement	Variable	Diminished enhancement of mucosa and serosa, target appearance	Diminished enhancement
Mesentery/fat	Mesenteric fat stranding with free fluid associated with the territory of ischemia	Mesenteric fat stranding with free fluid associated with the territory of ischemia	Mesenteric fat stranding with free fluid associated with the territory of ischemia

The underlying pathophysiology of colonic ischemia is insufficient blood supply to the bowel with consequent injury. This leads to mucosal ulceration, inflammation, and hemorrhage. With disease progression, necrosis of the muscle layer can lead to the fibrotic stricture or transmural infarction.

Any portion of the colon and rectum can be affected, but the splenic flexure, descending colon, and sigmoid colon are the most involved segments. The splenic flexure is the most susceptible as a "watershed zone," furthest from the IMA and SMA interconnected flow (but supplied by the "arc of Riolan" and the more peripheral or outer "marginal artery of Drummond," which anastomose the IMA and SMA in the colon mesentery).

Most patients with colonic ischemia have no major vascular occlusion, so the condition is attributed to low blood flow states, small vessel disease, or sepsis.

CT and colonoscopy are the primary means of establishing the diagnosis of ischemic colitis.

The most common CT findings in ischemic colitis are bowel wall thickening, mesenteric fat stranding, and abnormal wall enhancement. The distribution of findings is segmental in most cases [68].

Pneumatosis coli and porto-mesenteric venous gas are infrequent but threatening findings of complicated ischemic colitis, together indicating transmural infarction.

6 Adrenal Glands

The adrenal glands are paired retroperitoneal endocrine organs, located above the upper poles of the kidneys. The adrenal glands usually display a linear, comma, and V or Y shape.

On ultrasound, the adrenal glands are studied at the same time as the kidneys. Low frequency allows more depth of penetration and deep images. In the adult, the glands are often not detectable on US unless enlarged.

CT is the primary modality for detection and characterization of adrenal masses [69]. Fatty thickening of adrenal tissue is usually adenoma. Fat has negative CT numbers on CT and has fatty characteristics on MRI as well (signal drop out with fat suppression sequences) also indicating adenoma.

6.1 Functioning Adrenal Masses

This category includes adrenal enlargement (hyperplasia) and adrenal masses (adenoma and pheochromocytoma).

Diffuse adrenal hyperplasia is a nonmalignant growth of the gland, seen on non-contrast CT scan as an enlarged adrenal gland with preserved morphology.

Functioning adrenal adenoma is the minority of adrenal adenomas. The non-contrast CT evaluation displays a hypodense mass characterized by attenuating values less than 10 HU due to fat contents. If it is unilateral, there may be atrophy of the contralateral gland [70].

Pheochromocytoma is an uncommon, more often sporadic adrenal tumor. It is called the "ten percent tumor" or "rule of tens" because in 10% of the cases it is malignant, 10% familial, 10% extra-adrenal, and 10% bilateral. In a non-contrast CT scan it appears as a large mass, sometimes with calcifications. After intravenous contrast media injection, it shows an early, strong, and inhomogeneous enhancement; the malignant form infiltrates contiguous structures and may develop hepatic metastases [69].

6.2 Nonfunctioning Adrenal Masses

Nonfunctioning adrenal masses include benign (hematoma and adenoma) and malignant lesions (carcinoma).

Hematoma can result from traumatic or nontraumatic causes. On non-contrast CT, hematoma appears as hyperdense circular mass (acute phase) or with pseudocystic "water-like" density (chronic phase). After contrast media, no enhancement is observed.

Nonfunctioning adenoma is generally detected incidentally (incidentaloma). It appears as a well-circumscribed, hypodense formation (−10/+20 HU) with possible outbreaks of cystic degeneration and calcifications. After contrast, enhancement is appreciated in the parenchymal phase and strong washout in the late phase [69].

Carcinomas are rare non-secreting malignant tumors that occur as large masses with calcifications in 35% of cases. In CT after contrast medium administration, they show inhomogeneous enhancement due to areas of necrosis [70]. Survival is improved with aggressive application of surgical resection and local percutaneous thermal ablation therapy.

7 Kidneys

The ultrasound examination is the first level of examination in the study of the urogenital system and can identify hydronephrosis or stone disease.

CT has a relevant role in the diagnosis of urothelial obstruction, neoplasm, trauma, and vascular pathology [71].

The administration of intravenous contrast medium shows the whole excretory system with different phases: cortico-medullary (25–40 s postinjection) with bright outer kidney, nephrographic (80–100 s) with equally enhanced cortex and medulla, and excretory (~3–10 min) when the renal collecting system is opacified and full.

7.1 Renal Stones

The first level exams are abdomen X-ray or KUB (kidneys, ureter, and bladder) and ultrasound. X-ray is useful primarily to identify radiopaque stones. Ultrasound reveals radiopaque and radiolucent kidney stones that appear as hyperechoic nodules with a posterior shadow or cone. Renal ultrasound can easily detect dilation of the proximal collecting system, but evaluation of the lumbar and pelvic segments of the ureter is limited (even when very dilated) by bowel gas obscuration. Air is no friend of US; think why dolphins can speak over miles of waterways, since sound waves travel best in water. This is also why US is so good at differentiating between cystic and solid structures.

CT is the gold standard in the study of nephrolithiasis: the stones are characterized by high attenuation values (Fig. 33) [71].

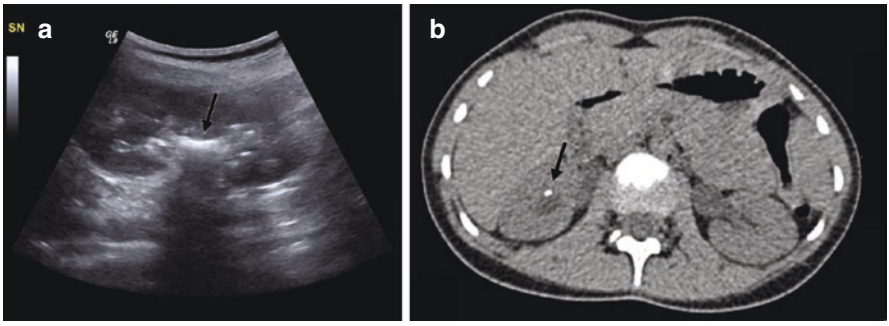

Fig. 33 Kidney stones. (**a**) At US, kidney stones are hyperechoic structures (arrow) with posterior shadow cone. (**b**) Non-contrast CT-scan is the gold standard in the study of renal lithiasis: the stones are characterized by high attenuation values (arrow)

The use of the contrast medium is indicated in the case of failed or uncertain identification of stones, in the suspicion of other obstructive pathology (clots, neoplasms), suspected complications (pyelonephritis, abscesses), or treatment planning.

7.2 Pyelonephritis

Acute pyelonephritis is generally of bacterial origin. At ultrasound, the edematous kidney is large and hyperechoic. On contrast-enhanced CT, pyelonephritis occurs as a non-enhancing hypodense focal area. Diffuse pyelonephritis manifests as an enlarged and hypodense kidney due to interstitial edema. Contrast administration shows lack of cortico-medullary differentiation and delay of the nephrographic phase (Fig. 34) [71].

Chronic pyelonephritis on CT is characterized by a small kidney, often associated with compensatory hypertrophy of the contralateral kidney. With contrast, thinning of the renal cortex, deformed calva calyxes, and a compensatory columnar hypertrophy can be identified [71].

7.3 Kidney Infarction

Occlusion of the renal artery or its branches may be total or regional/district.

In the acute phase, total infarction of the kidney appears hypodense due to edema without parenchymal enhancement or excretion of contrast media. It may be associated with subcapsular fluid collections and reactive thickening of the fascial planes.

In regional infarction during the nephrographic phase, a wedge-shaped hypodense area may be apparent with a cortical base and apex toward the renal hilum. Chronic renal infarction may show a small non-enhancing kidney (total infarction) or hypodense cuneiform foci with cortical retraction (district or regional infarction).

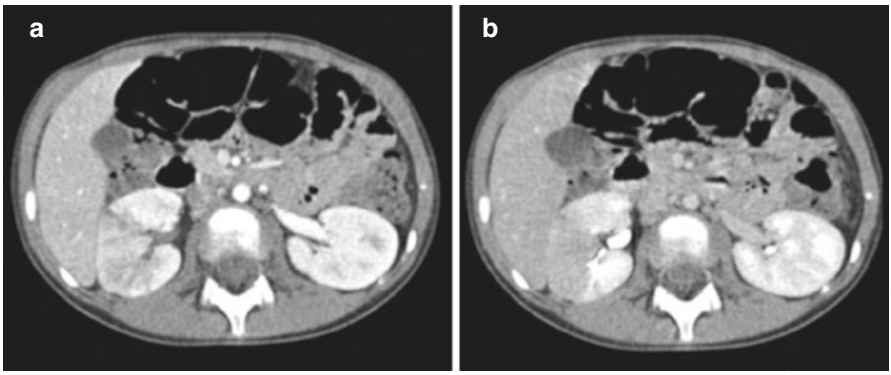

Fig. 34 Pyelonephritis, CT. Pyelonephritis manifests as a hypodense focal area: no enhancement after contrast medium injection is seen. Contrast medium administration shows the lack of cortico-medullary differentiation (**a**) and delay of the nephrographic (**b**)

7.4 Renal Masses

In most cases, renal masses are incidentally detected during abdominal imaging performed for other reasons.

At ultrasound, two types of kidney lesions, cystic or solid, can be recognized and should be characterized by a CT scan if previously unknown [71].

In general, renal masses fall into three categories by their appearance on contrast-enhanced CT: those without enhancement, those with enhancement, and those with macroscopic fat [71].

7.4.1 Cysts

Cysts are fluid-filled masses of varying dimensions characterized by thin and sharp epithelial wall.

At ultrasound, cysts are anechoic with posterior acoustic enhancement, although this may not be seen with smaller cysts (Fig. 35).

On a non-contrast CT scan, uncomplicated cysts are uniformly hypodense (less than 20 HU), the wall is not visible, and its attenuation value does not increase after intravenous contrast media injection (less than 10 HU).

Cysts are complicated, if the CT attenuation values increase after contrast media administration or if they have thickened septa or calcification [71].

Complicated cysts could result from infection or intracystic hemorrhage, but in most cases, they are identical to cystic tumors. The Bosniak classification has been created to assess the likelihood of malignancy in cysts and differentiate between benign and potentially malignant cystic lesions.

7.4.2 Angiomyolipoma

Angiomyolipoma (AML) is the most common benign solid renal mass, and it is made up of fat, vessels, and atypical muscle fibers.

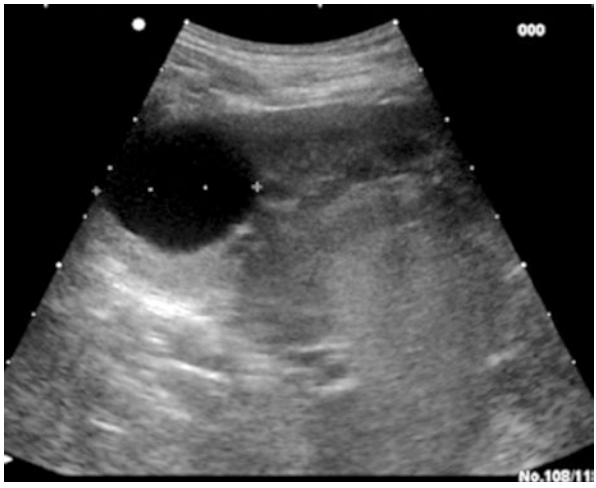

Fig. 35 Renal Cyst, US. Cysts in most cases are incidental findings during ultrasound examinations performed for other reasons. Cysts are anechoic (arrow) with posterior acoustic enhancement (arrows) but this finding may not be evident with smaller cysts

At ultrasound, AMLs are hyperechoic relative to renal parenchyma and often an incidental finding in asymptomatic patients (Fig. 36) [72], unless they present with bleeding or pain.

If the hyperechoic lesion is small (<3 cm), the diagnosis of AML is more likely [72].

The detection of fat (attenuation values less than −20 HU) on a CT scan confirms the diagnosis of AML. Rarely (5%) AMLs do not contain macroscopic fat, and these lesions are indistinguishable from RCC on CT. If calcifications and fat are detected in the same lesion, the diagnosis of renal cell carcinoma is more probable [71, 72]. AML >4 cm diameter may require prophylactic embolization due to bleeding risk, and the presence of pseudoaneurysms >5 mm further implies risk for subsequent spontaneous bleeding, which can be induced by trauma. Fatty masses ("lipo") will shrink less over time than more solid or vascular masses ("angio + myo") following embolization. Repeated embolizations may be required over time, depending upon growth rates, symptoms, and risk.

7.4.3 Oncocytoma

Oncocytoma is the second most common benign solid renal mass.

On US, it appears like an iso-hypoechoic solid lesion with well-defined margins.

On CT, oncocytomas are characterized by uniform contrast enhancement indistinguishable from a renal cell carcinoma or other malignant tumor (Fig. 37). Sometimes in larger lesions, a central scar may be detectable, which is a characteristic of oncocytomas, but it is seen only in a third of cases.

7.4.4 Renal Cell Carcinoma

Renal cell carcinoma (RCC) is the most common adult renal epithelial cancer representing more than 85% of all renal malignancies. Imaging plays a significant role in the early diagnosis of this tumor before clinical signs are observed (macroscopic hematuria, flank pain, palpable flank mass) [72].

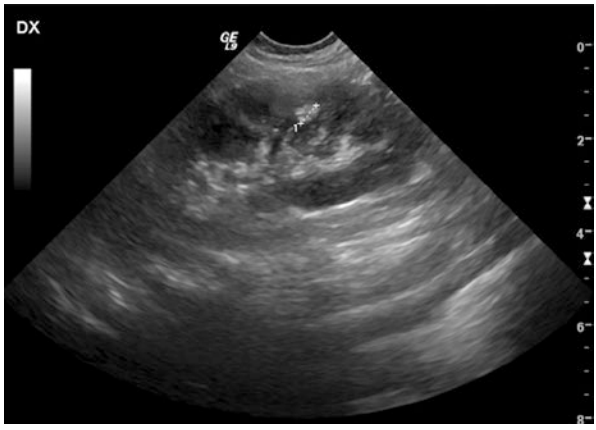

Fig. 36 Angiomyolipoma, US. On ultrasound, angiomyolipomas are hyperechoic to renal parenchyma (arrow) and often incidental finding in asymptomatic patients. If the hyperechogenicities are small (<3 cm), the diagnosis of AML is more probable

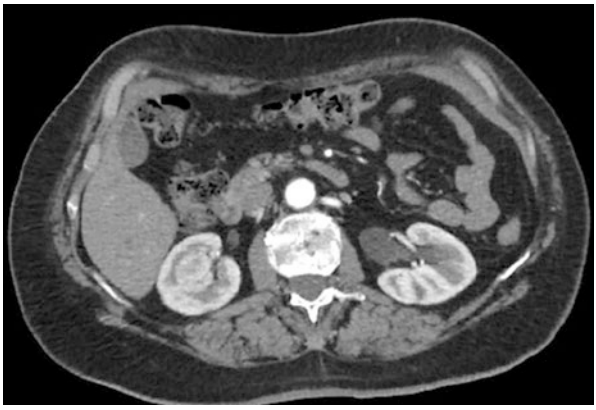

Fig. 37 Oncocytoma, CT. On contrast CT, oncocytomas are characterized by uniform enhancement (arrow) similar to renal cell carcinoma or other malignant tumor. Occasionally in the large lesions, a central scar may be detectable

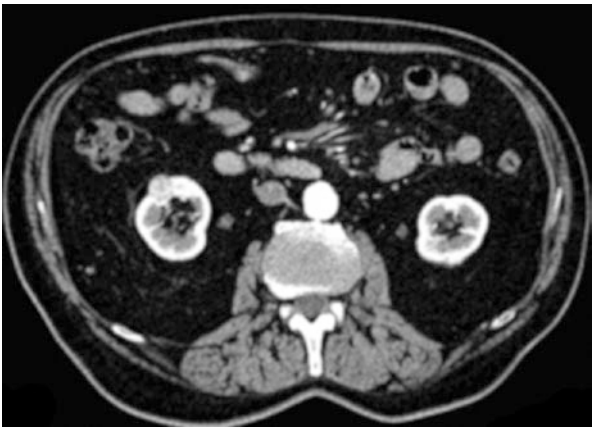

Fig. 38 Renal Cell Carcinoma, CT. On contrast-enhanced CT, renal cell carcinoma has strong enhancement (arrow) in the early arterial phase (corticomedullary phase) due to its hypervascularity

At ultrasound examination, it is a solid lesion, with irregular margins and varying sonographic appearance.

On non-contrast CT scan, RCC appears as inhomogeneous solid mass, with blurred margins, and in larger lesions, hypodense areas of necrosis can be present. Approximately 30% of cases demonstrate some calcifications [72].

After contrast media administration, RCCs strongly enhance in the early arterial phase (cortico-medullary phase). The parenchymal (nephrographic) phase is the most sensitive phase for the detection of RCC: the lesion is hypoattenuating to the normal parenchyma which has a homogeneous marked enhancement (Fig. 38) [72]. Hereditary RCC is seen with Von Hippel-Lindau disease which is associated with cerebellar hemangioblastomas and multiple RCC over a lifetime. About 5–10% of RCC are bilateral. RCC typically grow slowly at a rate of under 5 mm per year. They rarely metastasize when under 3 cm diameter. Under 3 cm diameter may be removed by partial nephrectomy or percutaneous ablation with RFA, microwave, or cryoablation in interventional radiology or interventional oncology. Interventional

radiology is the newest independent clinical residency in the USA, and IR has clinics, has admission privileges, and takes longitudinal care of patients, often as part of a multidisciplinary team. See chapter on minimally invasive image guided therapy ("video-game surgery").

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Neuroradiology

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Contents

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1 Vascular Pathology

1.1 Epidural Hematoma

An epidural hematoma is located between the inner table of the calvarium and the outer (periosteal) side of the dura. Classically, a temporal bone fracture lacerates the middle meningeal artery which lies in this potential space (Fig. 1a). The arterial bleed forms a hematoma and rapidly expands this potential space. The hematoma strips the dura from the calvarium, resulting in a biconvex shape of the hematoma. Tight dural adherence to the calvarial sutures does not allow the collection to cross suture lines [1].

Epidural hematomas are far less common than subdural hematomas. The reason epidural hematomas are important is that 90% have an arterial source which can result in rapid expansion of the hematoma, midline shift, and herniation [1]. Neurosurgical teams may take these patients to the operating room for decompressive craniotomy for source control of the lacerated artery and prevention of herniation. Venous epidural collections are less common and less morbid and are less likely to be tested.

On a non-contrast exam, active bleeding can be indicated by a "swirl sign" which is a hypodense swirl of dark blood within the hyperdense bloody collection (Fig. 1b). On a CT angiogram (CTA), active bleeding is indicated by a "spot sign." The classic CTA study is triphasic (a pre-contrast study, followed by an early arterial phase and then a delayed phase). On the early phase, contrast extravasation is identified as bright contrast outside the boundary of a vessel (Fig. 1c). On the delayed images, contrast remains or increases in volume in the exact same location, indicating that this is not intraluminal blood (which would be washed away by intact vasculature). The remaining extraluminal contrast is called the "spot sign" (Fig. 1d). The swirl sign and spot sign raise concern for rapid expansion of the hematoma.

The testable scenario usually involves a head injury, implying a temporal bone fracture, with a "lucid interval" after the trauma followed by somnolence, though this is present only 50% of the time [1].

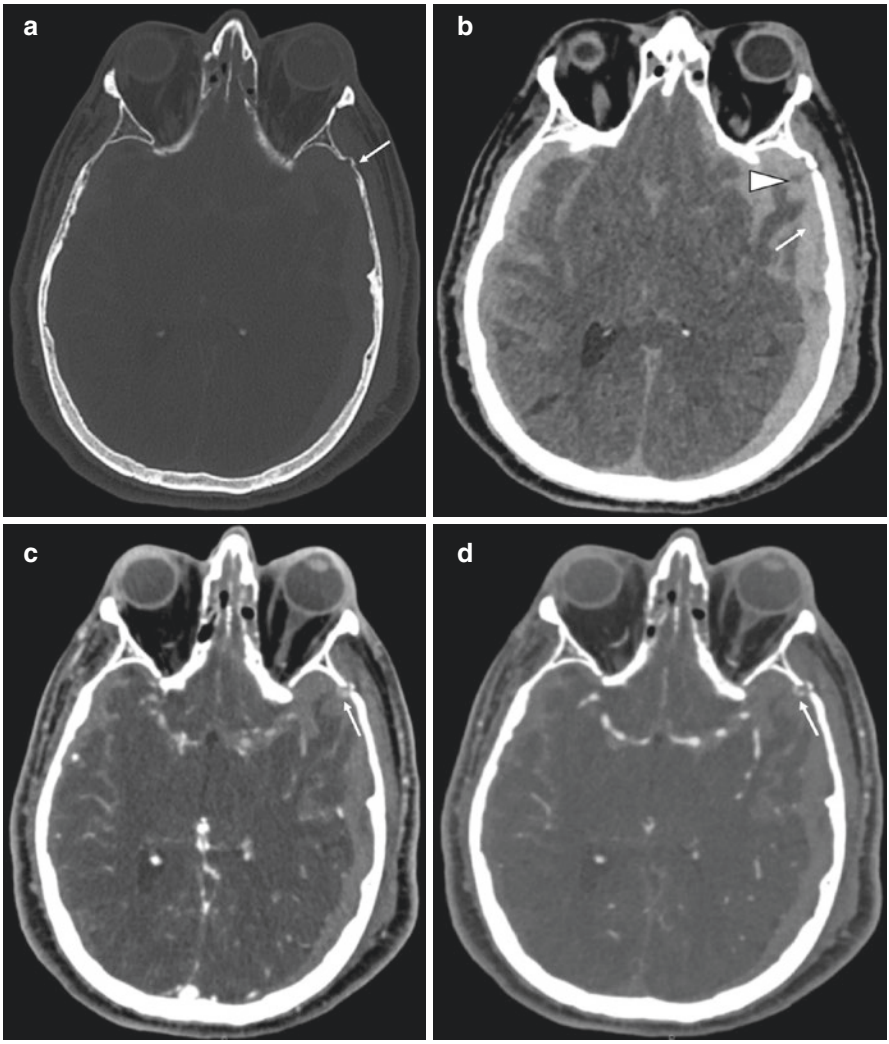

Fig. 1 (**a**) The bone window of the CT shows the temporal bone fracture (arrow) responsible for lacerating the middle meningeal artery. (**b**) The non-contrast head CT demonstrates a hyperdense (acute) extra-axial hematoma (arrow). Within the acute hematoma, there is a hypodensity (arrowhead) called a "swirl sign" which demonstrates active bleeding or plasma which will cause rapid expansion of hematoma. (**c**) There is bright contrast outside the lumen of a vessel on the arterial phase (arrow). (**d**) The contrast does not wash away on the delayed phase (arrow), indicating a "spot sign" or active extravasation from the vessel

1.2 Subdural Hematoma

Acute subdural hematomas are more common than epidural hematomas and are responsible for greater overall morbidity and mortality, given their higher prevalence [1].

A subdural hematoma is located between the inner layer of dura and the arachnoid. Draining cortical veins bridge this potential subdural space as they enter a dural venous sinus and can be torn from head trauma such as a fall with a head strike. The venous bleed is crescentic and can spread over the affected hemisphere, the falx, and the tentorium. Unlike epidural hematomas, these collections cannot cross the falx or the tentorium and do not require a skull fracture. Clinical deterioration may be delayed or chronic, when compared to epidural hematomas [1].

Chronic subdural hematomas contain membranes of granulation tissue as the collections evolve. These membranes are delicate and can rebleed even in the absence of major trauma. This rebleeding can cause an acute on chronic hematoma and appear as hyperdensity (bright new blood) on a background of hypodensity (dark old blood) (Fig. 2a and b) [1].

Acute on chronic subdural hematomas are most common in older individuals. If a child presents with an acute on chronic subdural hematoma, child abuse should be considered and discussed with the ordering clinician or social services.

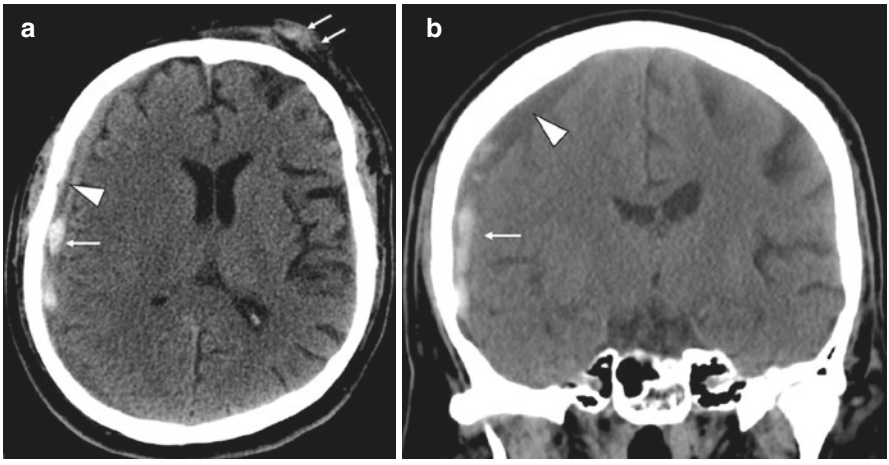

Fig. 2 (**a** and **b**) A subdural hemorrhage is usually related to a venous bleed from bridging veins. Because the collection is under less pressure than an arterial epidural hematoma, the hematoma is crescent shaped. A subdural hematoma with both hyperdense (arrow) and hypodense (arrowhead) regions indicates bleeding of different ages and is called an "acute on chronic" hematoma. (**a**) The left frontal laceration (double arrow) is a clue that shows the location of head strike from the fall

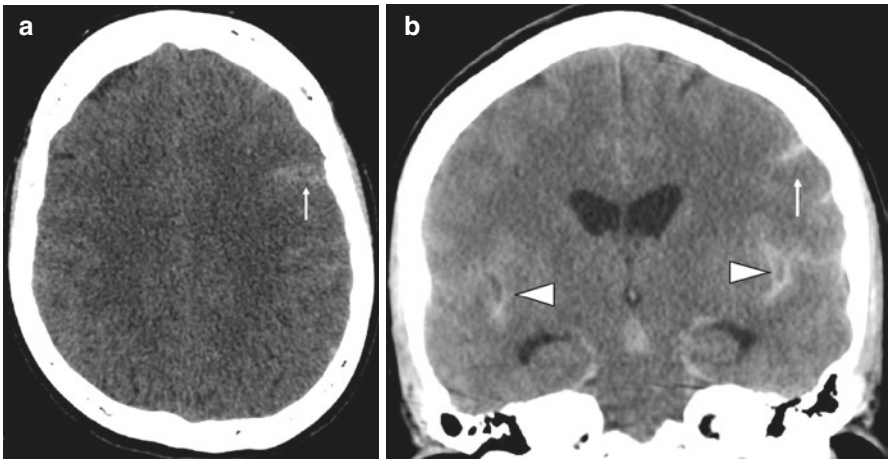

Fig. 3 (**a** and **b**) A subarachnoid hemorrhage appears as hyperdensity within a sulcus or cistern as seen in the left frontal sulci (arrows) and the Sylvian fissures (arrowhead)

1.3 Subarachnoid Hemorrhage

Subarachnoid hemorrhage (SAH) is located within cisterns and sulci, in the space between arachnoid and the pia mater that covers the parenchyma (Fig. 3). Trauma is the most common cause of SAH [1]. SAH, epidural hematomas, and subdural hematomas can be found alone or in combination.

SAH can also be secondary to aneurysm rupture in the classic scenario of a "thunderclap headache" or "worse headache of life." After subarachnoid hemorrhage is identified, a CTA head should be ordered to exclude an aneurysm or vascular malformation with intermittent bleeding.

1.4 Pseudo-Subarachnoid Hemorrhage

A classic clinical and testable mimic of subarachnoid hemorrhage is the "pseudo-subarachnoid sign." In the setting of diffuse cerebral edema, the brain parenchyma appears more hypodense (dark) than normal. Increased pressure from the swollen parenchymal also pushes venous blood into the dural venous sinuses. The combination of parenchymal hypodensity and increased venous return make the falx and tentorium appear hyperdense (bright) and mimic subarachnoid hemorrhage (Fig. 4). A CTA is not required in this scenario [1].

1.5 Posterior Communicating Artery Aneurysm

A new dilated pupil with cranial nerve III (oculomotor nerve) palsy is an important clinical and testworthy scenario that requires an emergent CTA head to rule out a posterior communicating artery aneurysm.

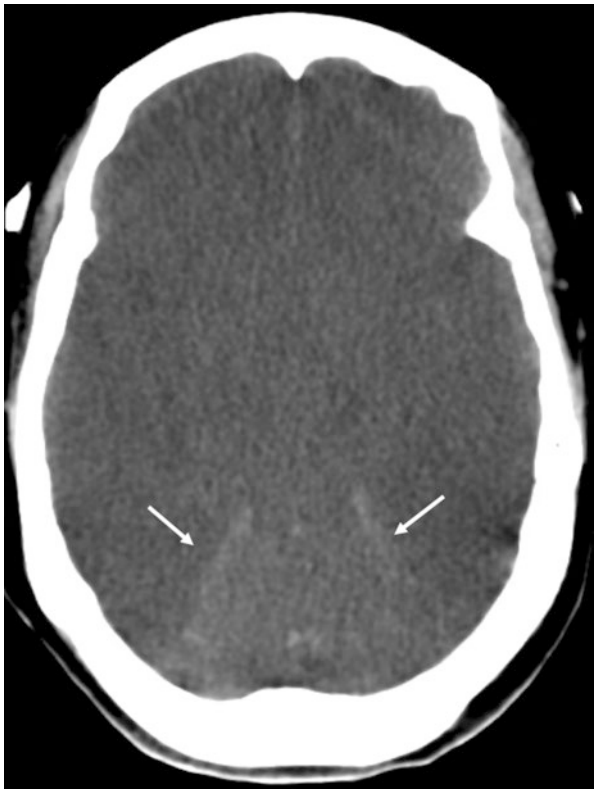

Fig. 4 An axial non-contrast CT head shows the sulci, gyri, ventricles, and gray-white differentiation are absent. The tentorial leaflets appear hypertense(arrows). In the setting of a normal head CT, this hyperdensity could be subarachnoid hemorrhage lining the tentorial leaflets. On this CT head with global cerebral edema, the tentorial leaflets appear hyperdense relative to the edematous brain parenchyma

Parasympathetic fibers contract the iris to shrink the pupils. These fibers run along the periphery of the oculomotor nerve (cranial nerve III) on their way to the pupil. When an oculomotor palsy involves the pupil, pathology is localized to the outer layers of the nerve where the parasympathetic nerves lie, susceptible to compression. The classic compressive lesion is a posterior communicating artery aneurysm (Fig. 5a–c). In a patient with new unilateral pupillary dilation, CTA or MRA should be ordered emergently to exclude a posterior communicating artery aneurysm.

In contradistinction, a patient with a history of diabetes or hypertension (vascular origin, not compression) may present with a pupil-sparing CN III palsy because the penetrating arteries supply the nerve approach from outside-in and are unable to reach the inner most layers of the nerve bundle. In this scenario, the outer layers, including the parasympathetic fibers, maintain blood supply and function.

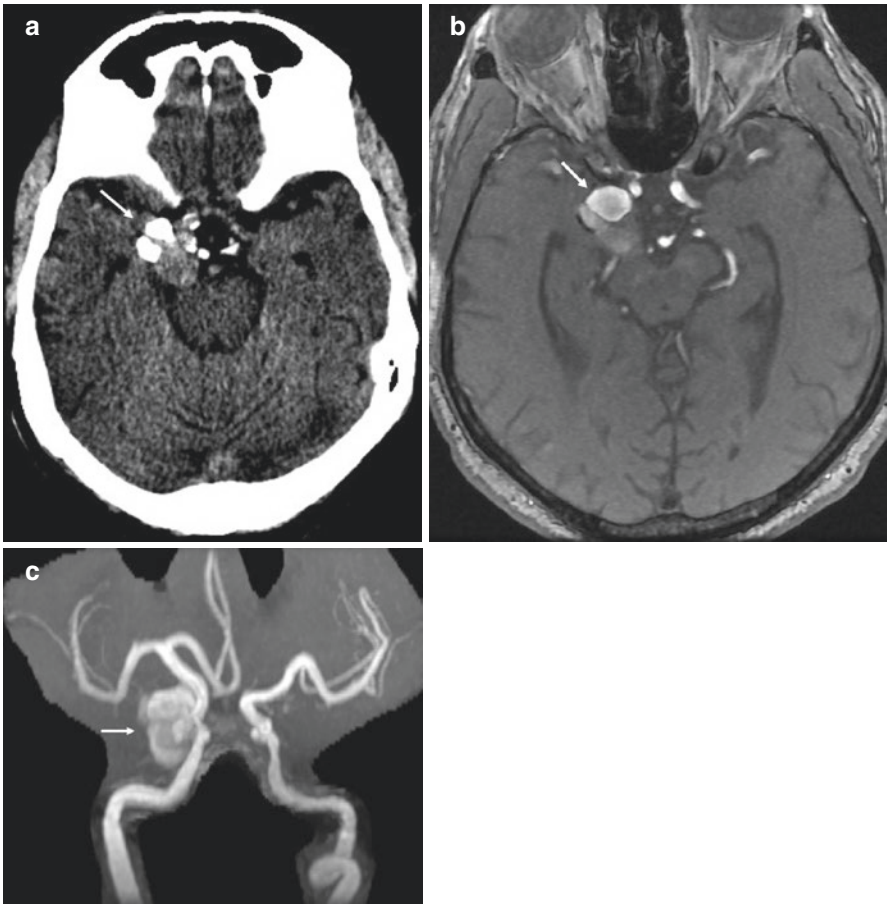

Fig. 5 (**a**) A multi-lobulated, partially calcified hyperdense lesion (arrow) is located in the middle cranial fossa on a non-contrast CT head. (**b**) On an MRA of the head, the same lesion demonstrates flow (arrow) and is a posterior communicating artery aneurysm. (**c**) A reconstruction of the intracranial arteries gives an additional perspective of the relationship of the large saccular aneurysm (arrow) and the vasculature

2 Infection

2.1 Brain Abscess

A brain abscess is a walled-off collection of pus within the parenchyma. Brain abscesses are rare, most commonly secondary to hematogenous seeding and most common in immunocompromised patients. An abscess is best evaluated by MRI and often requires an urgent consult for neurosurgical evacuation. An abscess has an enhancing rim (Fig. 6a) and central diffusion restriction or hyper-intensity

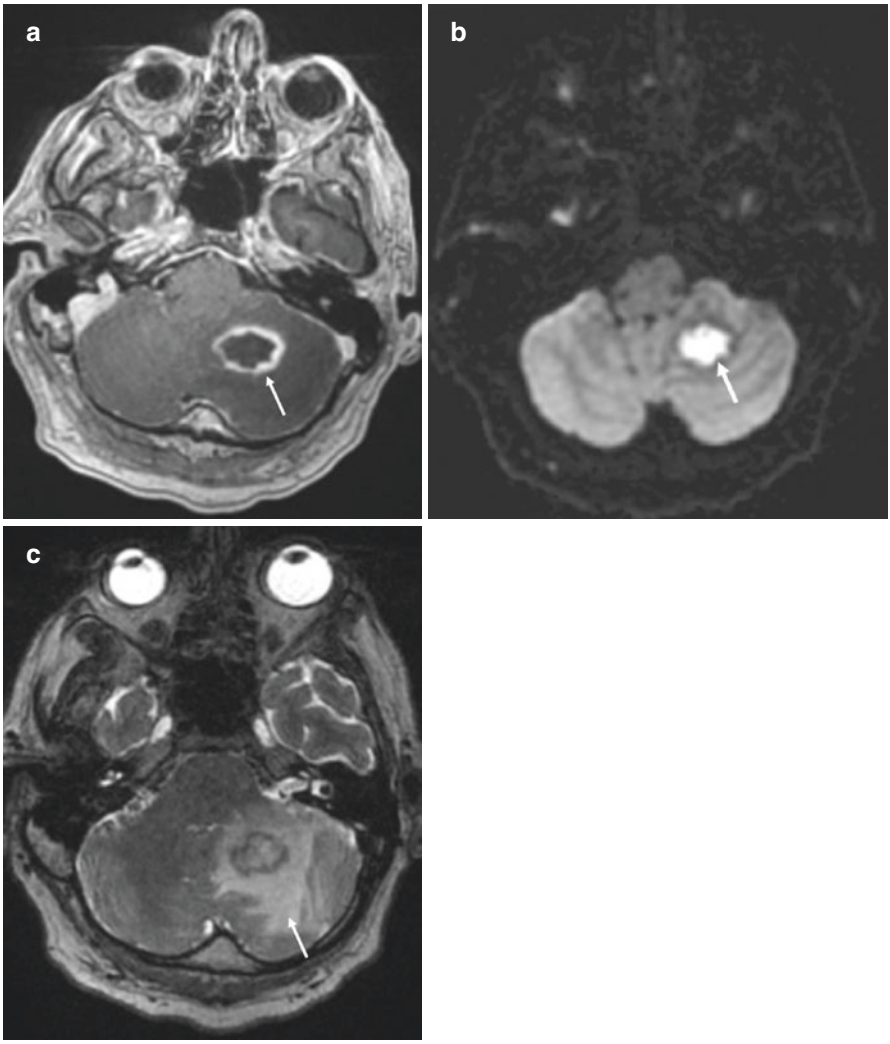

Fig. 6 (**a**) A lesion in the left cerebellum demonstrates irregular, complete rim enhancement (arrow). (**b**) The center of the lesion restricts diffusion which is classic for an abscess (arrow). (**c**) There is extensive vasogenic edema (arrow) surrounding the lesion with secondary mass effect on the T2-weighted sequence. Remember T2 = H2O (fluid or edema are easier seen on T2)

(brightness) on diffusion-weighted imaging (DWI) (Fig. 6b) because the center is composed of pus. There is often surrounding vasogenic edema (Fig. 6c), mass effect, and possibly encephalitis. Ventriculitis occurs if infection extends into the ventricle which portents a much worse prognosis for the patient. Empyemas (extra-axial collections of pus) have similar imaging characteristics.

A very succinct differential diagnosis of a rim-enhancing lesion includes a primary brain tumor, a metastatic lesion, or an inflammatory lesion. A high-grade

primary brain tumor such as a glioblastoma usually restricts diffusion around the periphery of the lesion in the region of viable hypercellular tumor. These tumors often have central necrosis, so there is no central diffusion restriction. Metastases often come in multiples and are more likely in the setting of a known primary cancer. An inflammatory lesion such as a demyelinating lesion classically has a "leading edge" of eccentric diffusion restriction and enhancement in the region of active demyelination rather than a complete rim of diffusion restriction and enhancement.

2.2 Osteomyelitis-Discitis and Epidural Abscess

Osteomyelitis-discitis is clinically considered a single entity because spread is contiguous between structures (Fig. 7a–c). Even early subtle findings deserve a phone call to the ordering clinician because destruction from infection can progress rapidly.

Staphylococcus aureus is the most common pathogen. Tuberculous osteomyelitis-discitis or "Pott's disease" is an important atypical infection in endemic regions which characteristically results in an acute kyphotic "gibbus" deformity from vertebral body collapse (Fig. 7d).

An epidural abscess is an important pertinent positive or negative finding in a patient with osteomyelitis-discitis because this may change management from medical treatment with intravenous antibiotics alone to surgical treatment with a washout by neurosurgery or orthopedic surgery. The management shift is twofold:

1. Antibiotics cannot penetrate an abscess because it is avascular, and an "I&D" or incision and drainage are required to clean out the infection.
2. The spinal canal is a fixed space. Mass effect from an epidural abscess can compress the spinal cord or cauda equina, causing "cord compression" and serious neurological consequences.

2.3 Prion Disease

Prion disease (also known as transmissible spongiform encephalopathy) is grouping of neurodegenerative disorders which include Creutzfeldt-Jakob disease (CJD) and bovine spongiform encephalopathy or "mad cow disease" in the animal kingdom. CJD is both an infectious and neurodegenerative disorder driven by abnormally folded prions that result in rapidly progressive, fatal dementia. The diagnosis is often first suggested on imaging due to its characteristic MRI appearance. Even suggestion of this diagnosis requires careful biopsy and autopsy technique with special autoclaving of tools to keep medical workers and other patients safe [1].

On MRI, CJD primarily affects gray matter structures. Classically, the basal ganglia, thalami, cortex, hippocampus, and cerebellum restrict diffusion (hyperintensity on DWI) and are FLAIR hyperintense. "Cortical ribboning" describes cortical diffusion restriction (Fig. 8). The "hockey stick sign" of describes the FLAIR hyperintensity in the pulvinar (in the posteromedial thalamus) [1].

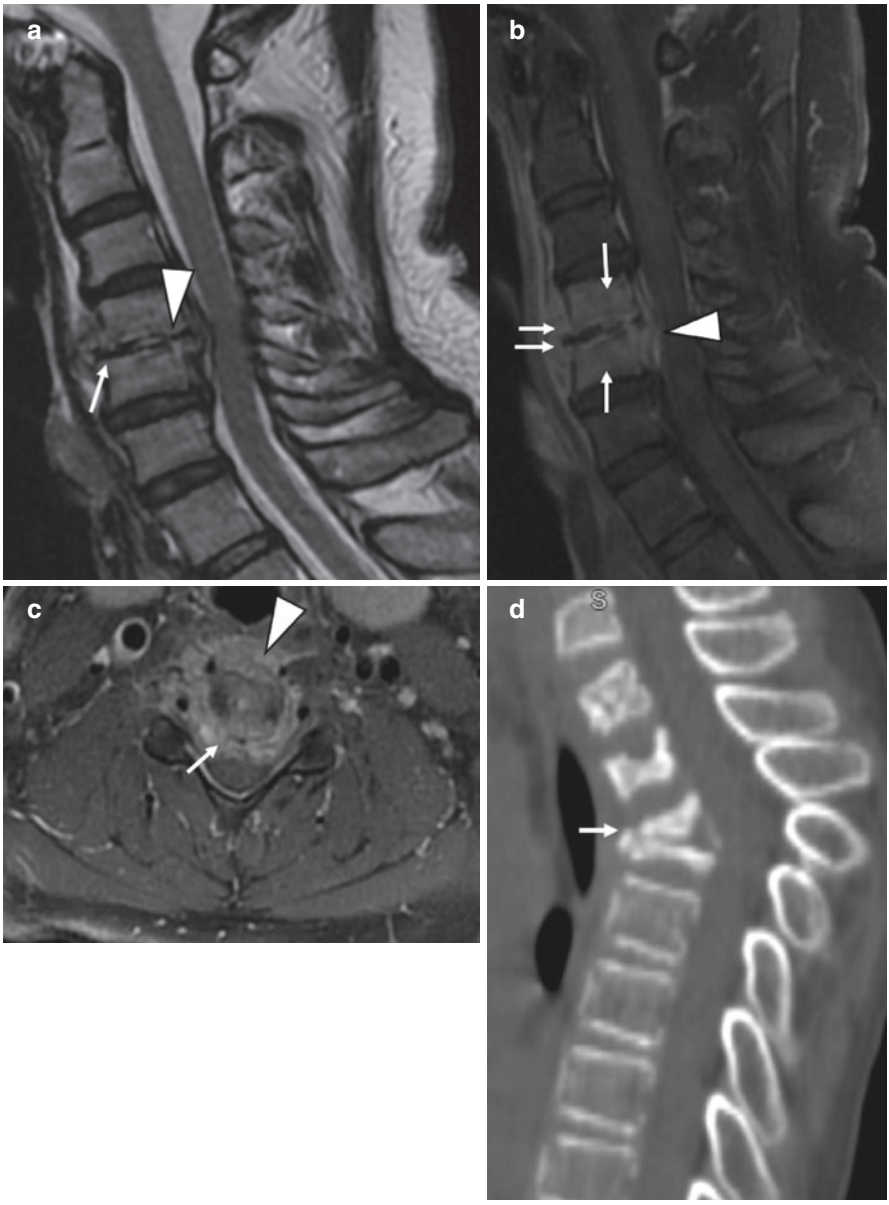

Fig. 7 (**a**) On this T2-weighted sagittal image of the cervical spine, the C4–C5 intervertebral disc (arrow) is significantly smaller than the other discs. The vertebral body endplates adjacent to the disc appear ragged and irregular (arrowhead). (**b**) On sagittal T1-weighted post-contrast imaging, there is avid enhancement of the vertebral bodies (arrow). Enhancement extends beyond the posterior cortex of the vertebral bodies into the epidural space, consistent with an epidural abscess (arrowhead). Enhancement extends beyond the anterior cortex consistent with a pre-vertebral abscess (double arrow). (**c**) Axial T1-weighted post-contrast imaging also shows the epidural enhancement narrows the spinal canal (arrow). (**d**) This sagittal CT of the thoracic spine of a different patient shows sclerosis of the infected vertebral bodies. Anterior wedging of the one of the vertebral bodies from collapse is classic in tuberculous osteomyelitis-discitis and is called a "gibbus deformity" (arrow) which causes acute kyphosis or spinal angulation secondary to the collapse

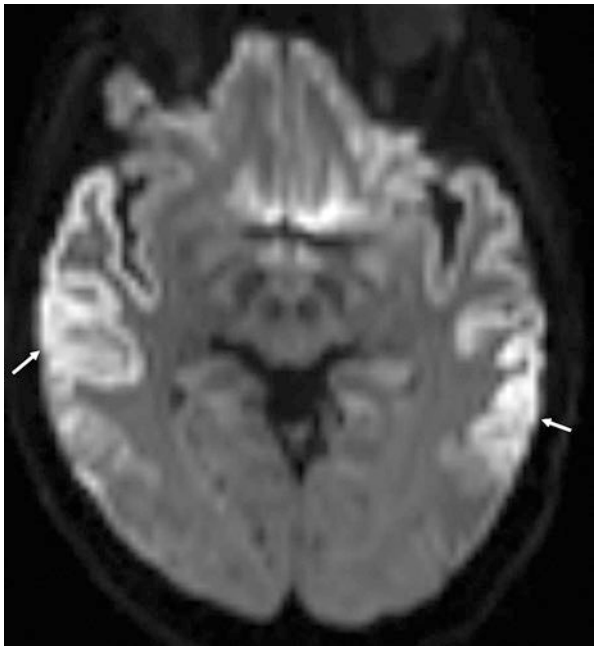

Fig. 8 DWI sequence on MRI shows the classical cortical ribboning of CJD (arrow)

2.4 Neurocysticercosis

Neurocysticercosis is the most common CNS parasite which results from ingestion of the tapeworm *Taenia solium* in undercooked pork. More than half of people infected with the tapeworm will have CNS disease [1]. The life cycle of the tape worm has four stages. On imaging, there are often multiple larva at various stages. The second stage in the lifecycle, the colloidal vesicular stage, brings the patient to imaging. In this stage, the larva degrades, and the immune system identifies antigens and mounts an aggressive inflammatory response, classically resulting in a seizure [1, 2].

On imaging, the scolex (head) of the tapeworm is surrounded by a cyst which looks like a circle with an eccentric dot on the side (Fig. 9a–c). During the colloidal vesicular stage, the fluid within the cyst thickens (becomes colloid). There is enhancement of the cyst wall and eccentric scolex (Fig. 9b) with surrounding edema (Fig. 9c) and mass effect. At the end of the lifecycle, the final lesion is calcified and quiescent or incidental [1, 2].

Clinical history and test questions will include a history of travel or immigration from an endemic region such as Central or South America.

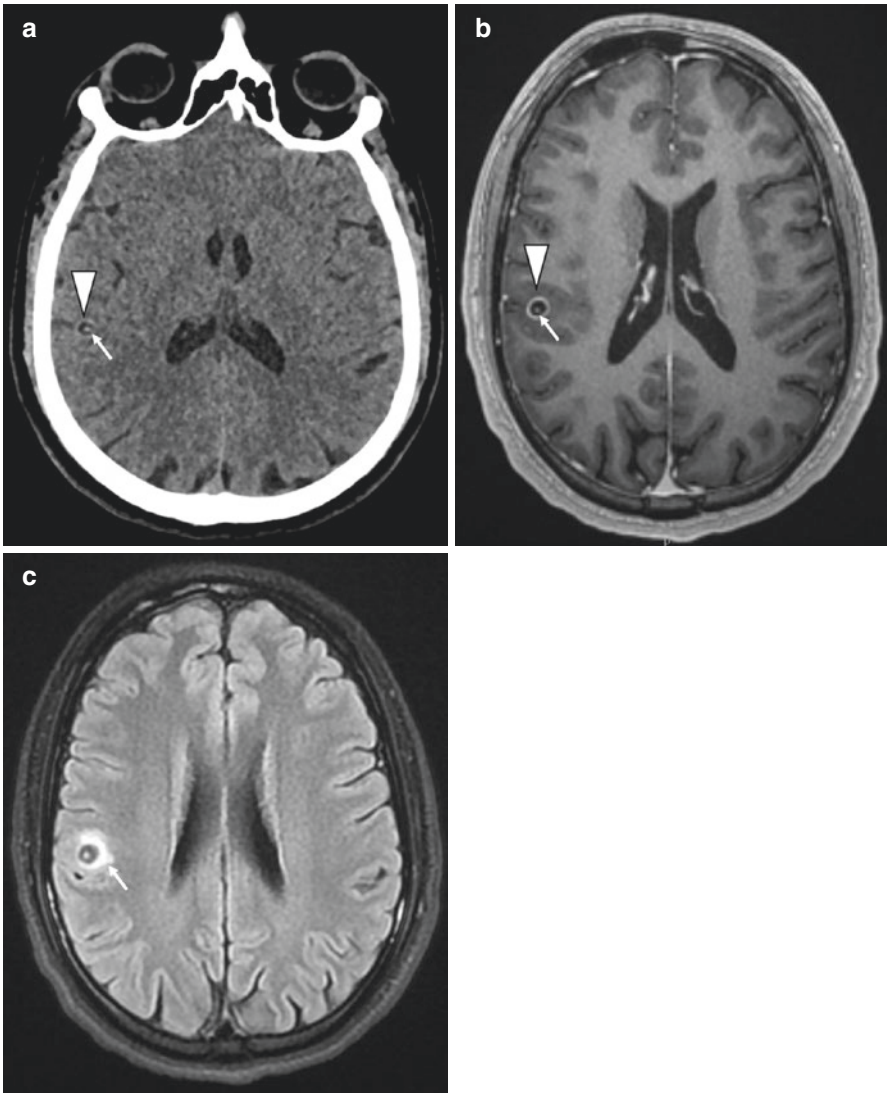

Fig. 9 Classic neurocysticercosis: (**a**) On CT, the *Taenia solium* scolex (arrow) and rim of the cyst are hyperdense (arrowhead). Within the cyst, fluid is hypodense fluid. (**b**) On the T1-weighted post-contrast MRI sequence, the scolex is a tiny enhancing focus (arrow), and the rim of the cyst smoothly enhances (arrowhead). (**c**) On the FLAIR MRI sequence, vasogenic edema surrounds the lesion (arrow)

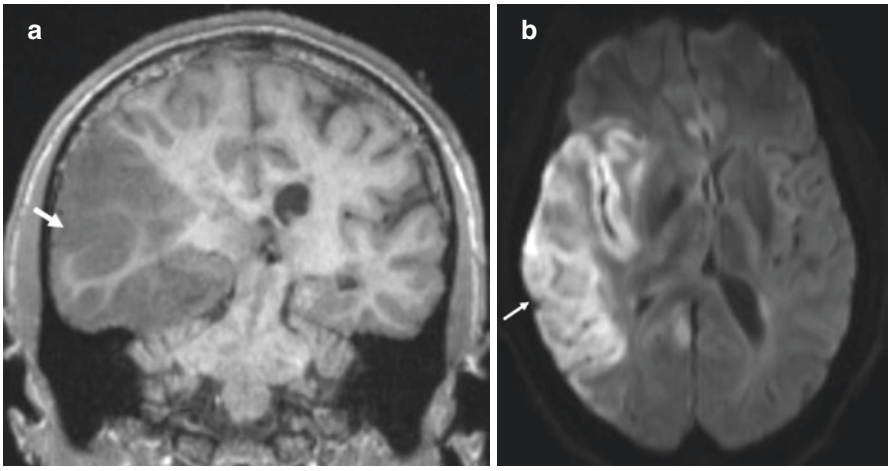

Fig. 10 Herpes simplex encephalitis: (**a**) The T1-weighted pre-contrast image shows severe gyral swelling (arrow) of the right temporal lobe and right inferior frontal gyrus. (**b**) The temporal lobe (arrow) and insula (arrowhead) restrict diffusion on DWI. Temporal lobe is the classic location of herpes encephalitis

2.5 Herpes Simplex Encephalitis

Herpes simplex encephalitis (HSE) is a devastating, rapidly destructive disease that is treatable. Recognizing or even suggesting the possibility of HSV infection can dramatically change mortality, which can be as high as 50–70% [1]. If any of the classic imaging findings are present, the primary team must be notified immediately to start prophylactic antivirals (acyclovir) until HSV is confirmed or ruled out by serologic or cerebrospinal fluid PCR testing [1].

Reactivation of herpes simplex virus (HSV-1) is responsible for the viral encephalitis. HSE characteristically affects the limbic system, including the anterior and medial temporal lobes and cingulate gyrus. Findings are often bilateral, though asymmetric. MRI demonstrates swollen gyri of the involved temporal (Fig. 10a) and limbic lobes. The susceptibility-weighted imaging sequence demonstrates characteristic findings of hemorrhagic necrosis. Restricted diffusion may also be present (Fig. 10b) [1].

3 Demyelination

3.1 Multiple Sclerosis

Multiple sclerosis (MS) is an autoimmune disease of the central nervous system white matter which has specific and readily recognizable MRI findings, making it highly testable. Though the disease has been extensively studied, the primary etiology of the disease is still not well understood [1].

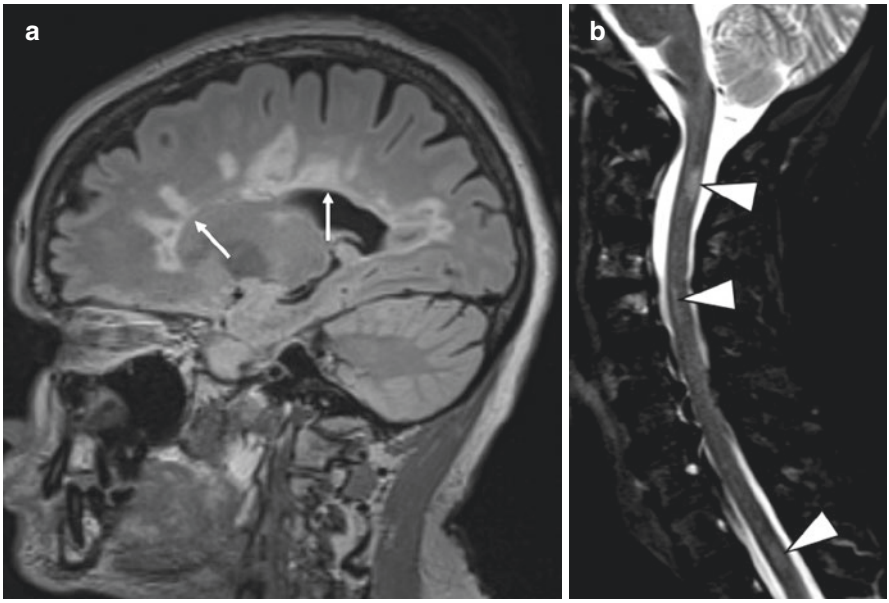

Fig. 11 Multiple sclerosis: (**a**) This sagittal FLAIR image demonstrates the classic "Dawson fingers" of MS white matter plaques (arrow) that extend from the corpus callosum into the periventricular white matter. (**b**) This sagittal STIR (short tau inversion recovery, i.e., fluid-sensitive sequence) shows multiple hyperintense MS plaques (arrow) in the cervical and thoracic spine. Note that STIR sequence on MRI suppresses fat and water and is insensitive to magnetic field inhomogeneities (beyond the scope of USMLE)

The stereotypical patient is a young Caucasian woman who presents with vague neurologic complaints such as numbness, tingling, weakness, or change in proprioception. Changes in vision from optic neuritis often accompany an MS flare. Dissemination in time and space are key parts of the modified McDonald criteria used to diagnose MS, which is easy to define comparing multiple MRIs. However, the diagnosis can and is often made on a single MRI based on white matter lesions that restrict diffusion and enhance, indicating active demyelination, and white matter lesions that do not restrict diffusion or enhance, indicating old lesions from a previous episode of demyelination [1].

MS white matter lesions are classically found in the corpus callosum and periventricular white matter, that is, the white matter around the ventricles. The typical appearance is called "Dawson fingers" for the radiating pattern of the lesions that are perpendicular to the ventricle (Fig. 11a). Cord lesions are typically in the cervical cord and are short segment lesions that span no greater than two vertebrae (Fig. 11b).

4 Toxicity

The basal ganglia are the target of many toxic injuries due to the high metabolic rate. If you see signal abnormalities of both basal ganglia, a toxic or metabolic process should be high on the differential diagnosis.

4.1 Methanol Poisoning

Methanol is an alcohol that is metabolized into formic and lactic acid and inhibits the electron transport chain within mitochondria. Ingestion of even a small volume can result in hemorrhagic necrosis of the putamen (Fig. 12a, b, and c) and possibly death.

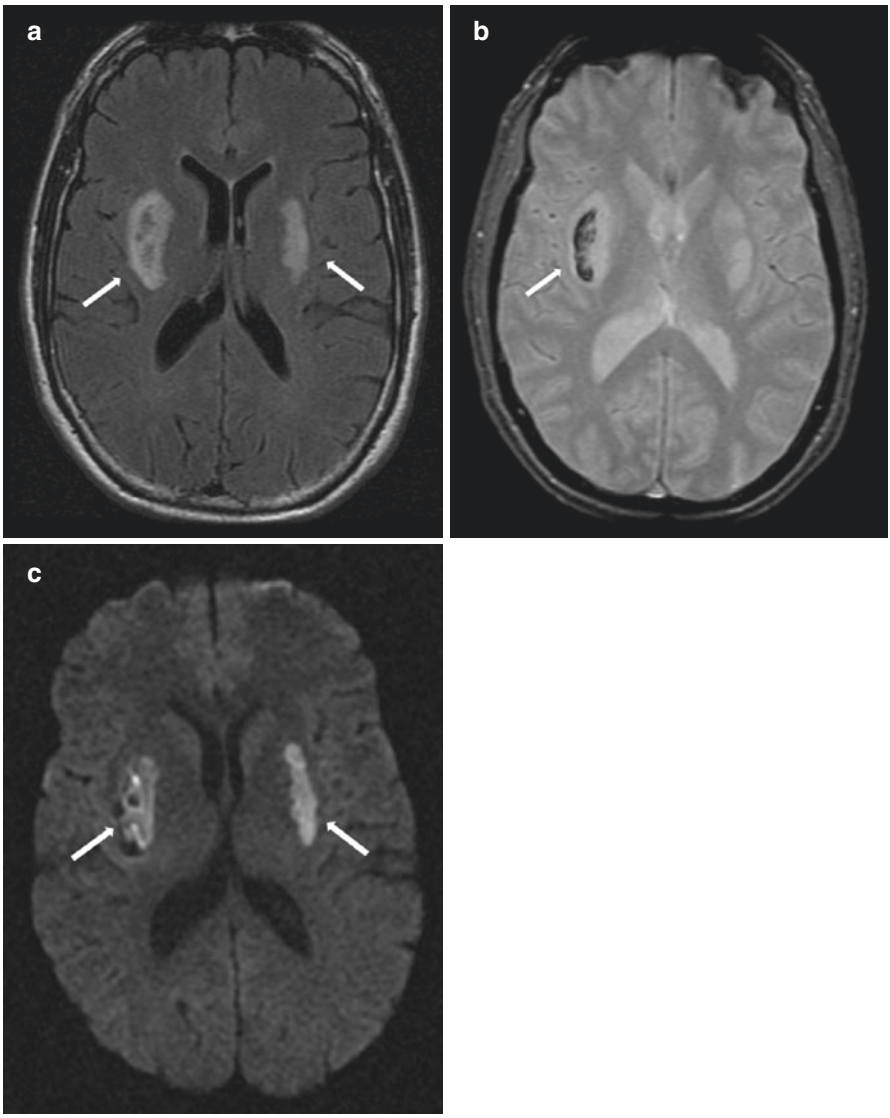

Fig. 12 Methanol poisoning: (**a**) FLAIR hyperintensity in both putamina is classic for putaminal necrosis in methanol poisoning (arrows). (**b**) This gradient echo sequence shows susceptibility from hemorrhage within the right putaminal necrosis. (**c**) The region of putaminal injury restricts diffusion (arrows)

Methanol is common in many industrial solvents, antifreeze, and paint removers as well as an impurity in homemade alcohol such as moonshine. A question stem may involve accidental ingestion of windshield wiper fluid, imbibing in homemade alcohol or an alcoholic patient who has run out of the usual ethanol drinking alcohol. Methanol ingestion is followed by a variable latent period before nausea, vision loss, and possible death. If ingestion is recognized early, fomepizole or ethanol is given to the patient to block the metabolism of methanol, and hemodialysis can remove methanol and its byproducts from the serum.

4.2 Carbon Monoxide Poisoning

Carbon monoxide (CO) binds irreversibly to hemoglobin and with greater affinity than oxygen. Though blood is delivered, the lack of oxygen within that blood results in tissue hypoxia. Globus pallidus necrosis is the hallmark of the CO poisoning (Fig. 13), followed by delayed diffuse white matter demyelination [1].

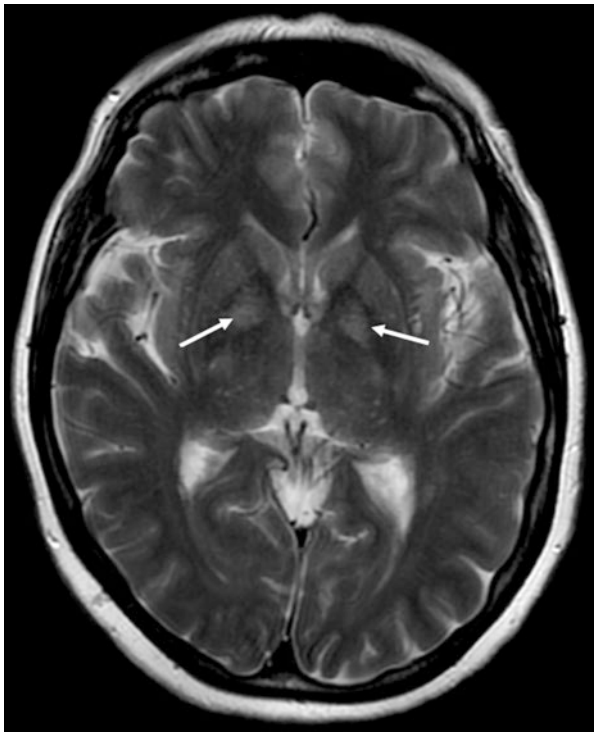

Fig. 13 Carbon monoxide poisoning: Symmetric T2 hyperintensity in the globus pallidus is classic for bilateral globus pallidus necrosis in carbon monoxide poisoning (arrows)

5 Metabolism

5.1 Wernicke Encephalopathy

Wernicke encephalopathy is the dreaded complication of thiamine deficiency most frequently seen in alcoholics with poor nutrition. Because the clinical triad of ophthalmoplegia, ataxia, and encephalopathy becomes irreversible if the thiamine deficiency is not corrected, the ordering clinician must be notified emergently. Imaging findings include T2 hyperintense signal in the medial thalamus lining the third ventricle (Fig. 14a), the mammillary bodies (Fig. 14b), and the tectal plate and the periaqueductal gray matter lining the cerebral aqueduct (Fig. 14b and c) [2].

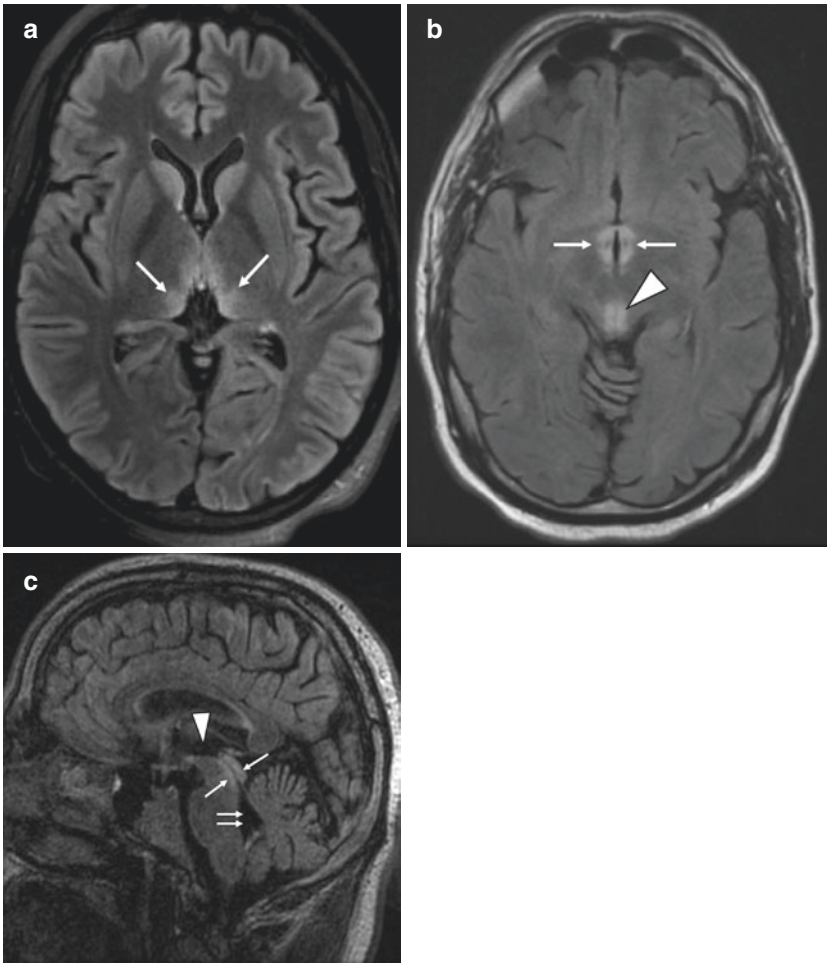

Fig. 14 Thiamine deficiency (Wernicke encephalopathy): (**a**) This axial FLAIR sequence demonstrates hyperintensity within the medial thalami (arrow). (**b**) A different axial FLAIR sequence shows hyperintensity in the mamillary bodies (arrow) and the periaqueductal gray matter (arrowhead). (**c**) This sagittal sequence demonstrates a different perspective of the hyperintense signal surrounding the cerebral aqueduct (arrow) which connects the third ventricle (arrowhead) to the fourth ventricle (double arrow)

5.2 Subacute Combined Degeneration

Subacute combined degeneration is caused by vitamin B12 deficiency usually secondary to malnutrition. The question stem for these patients may include a history of alcoholism or recent loss of a spouse (implying poor nutrition, if the deceased spouse was the cook). The dorsal columns of the spinal cord are the white matter tracts involved. These will appear hyperintense on T2-weighted images or STIR images (Fig. 15a and b) [1].

5.3 Idiopathic Intracranial Hypertension

Idiopathic intracranial hypertension (also known as benign intracranial hypertension or pseudotumor cerebri) is a complex and poorly understood entity associated with obesity. The classical presentation of idiopathic intracranial hypertension is a

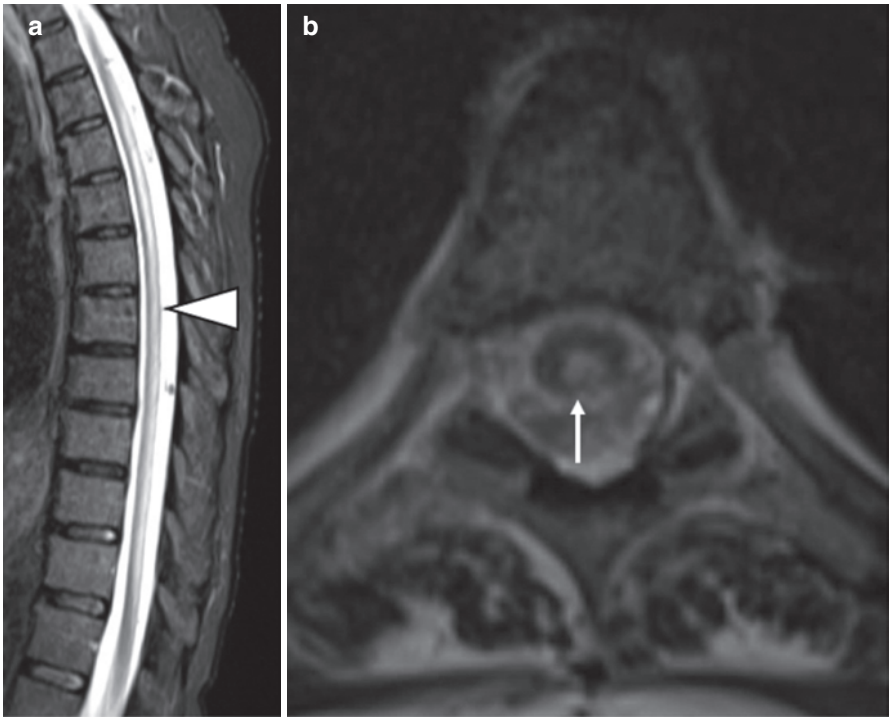

Fig. 15 B12 deficiency: (**a**) The sagittal STIR image shows hyperintense signal the entire length of the thoracic (arrowhead). (**b**) On this axial T2-weighted image, the hyperintense signal is localized to the posterior columns (arrows) of the spinal cord which is the classic imaging finding of vitamin B12 deficiency on imaging

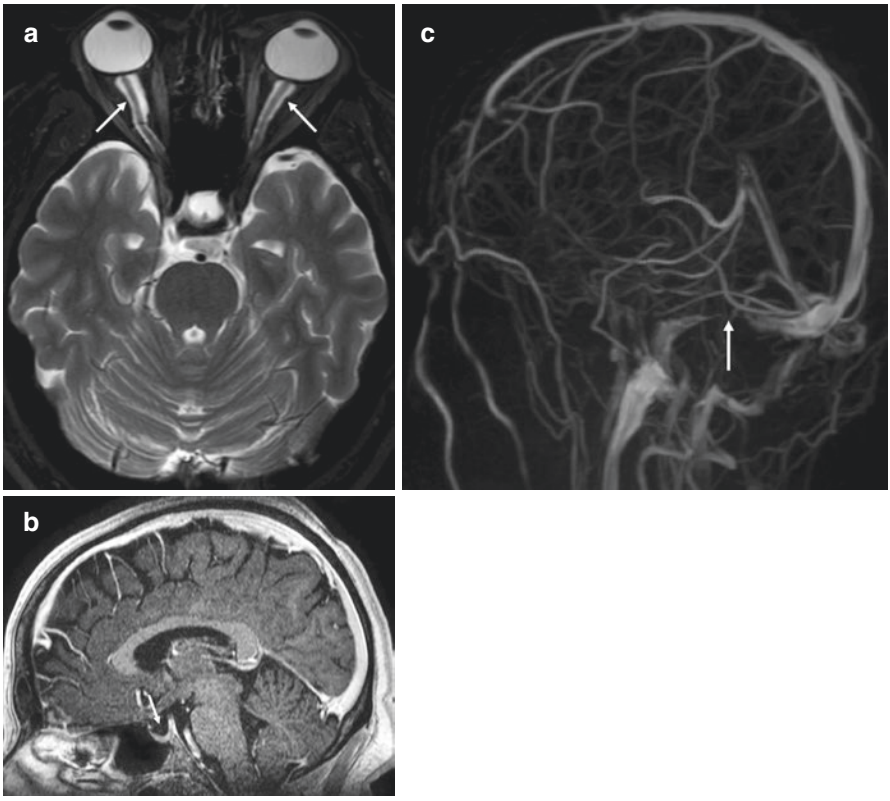

Fig. 16 Pseudotumor cerebri: (**a**) The axial T2-weighted sequence shows dilation of the optic nerve sheaths from increased intracranial pressure. (**b**) The sagittal post-contrast image shows the partially "empty sella." Both images A and B are secondary signs of intracranial pressure, though these findings are not specific to IIH. (**c**) This MRV shows narrowing of the sinodural angles (arrow) at the transition from the transverse sinus to the sigmoid sinus which may be the cause of "idiopathic" intracranial hypertension in some patients

20–45-year-old overweight woman presenting with headaches with or without vision loss and tinnitus. On physical exam, these patients classically have papilledema and often abducens nerve (cranial nerve VI) palsies [1].

Imaging is performed to rule out any structural cause of increased intracranial pressure (such as a tumor) and to identify secondary findings of intracranial hypertension, including flattening of the posterior globes, protrusion of the optic nerve head into the globe, increased CSF within the optic nerve sheaths (Fig. 16a), an empty sella (Fig. 16b), low lying cerebellar tonsils, and narrowing of the transverse sinus or sinodural angle (the sigmoid sinus as it transitions into the jugular bulb) (Fig. 16c). The diagnosis can be suggested on imaging but requires a lumbar puncture for opening pressure for diagnosis. Draining a large volume of CSF to lower the intracranial pressure gives short-term headache relief [1].

5.4 Pontine Myelinolysis

Pontine myelinolysis is a devastating complication of rapidly corrected hyponatremia (low serum sodium). If serum sodium is raised too quickly by hyperosmolar fluids, the axons of white matter are demyelinated and are T2/FLAIR hyperintense (Fig. 17a) with or without diffusion restriction on DWI (Fig. 17b). Given the vital functions of the brain stem, this demyelination can be lethal [1]. Extra-pontine myelinolysis is also possible but not classical and therefore not testable.

6 Neoplasm

6.1 Glioblastoma

Glioblastoma is the most common type of adult glial neoplasm and, unfortunately, is also the most aggressive. All treatments are palliative, and though there are several immunotherapies improving the short-term survival, long-term prognosis is grim. No treatments available are curative. Physical exam findings are driven by the location and mass effect of the tumor [1].

Glioblastoma neoplasms spread along white matter tracks. These tumors are dramatic and usually demonstrate all the features of a high-grade neoplasm, including irregular enhancement with central necrosis (Fig. 18a), diffusion restriction

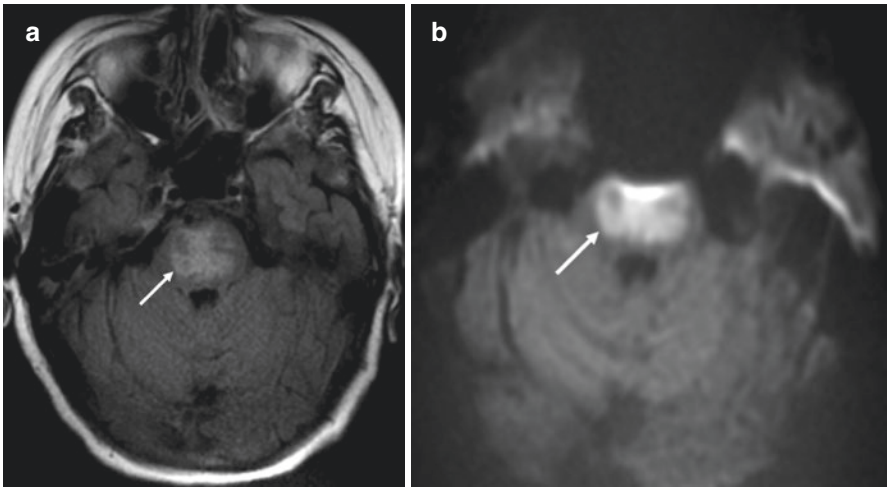

Fig. 17 Pontine myelinolysis from rapid correction of hyponatremia: (**a**) The pons is FLAIR hyperintense (arrow). (**b**) The region also restricts diffusion

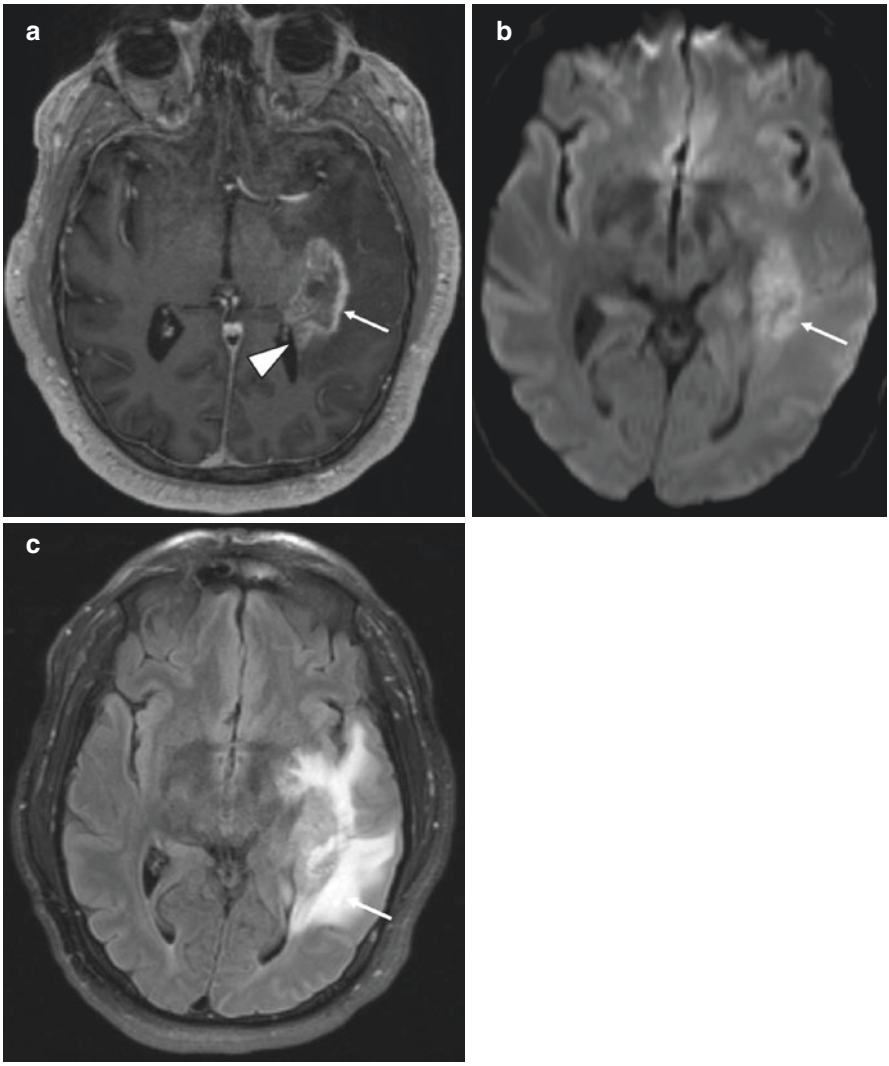

Fig. 18 Glioblastoma: (**a**) The heterogeneous lesion in the posterior left temporal lobe demonstrates irregular rim enhancement (arrow) and extends to the lining of the ventricle (arrowhead). (**b**) The lesion demonstrates heterogeneous diffusion restriction (arrow), unlike the central restriction of an abscess or the leading-edge restriction of a demyelinating lesion. (**c**) There is extensive FLAIR signal abnormality (arrow) in the white matter surrounding the lesion with mass effect and partial effacement of the adjacent ventricle (arrowhead)

(Fig. 18b), and extensive mass effect (Fig. 18c) often with midline shift or herniation. If the tumor spreads from one hemisphere to the other via the corpus callosum, it is called a "butterfly" glioma; lymphoma is the other pathology that can have this butterfly appearance.

6.2 Cord Compression from Metastatic Disease

When a patient with known malignancy presents with new neurologic symptoms such as extremity weakness or bowel and/or bladder incontinence, an emergent MRI is warranted for evaluation of "cord compression." Compression of the cord or caudal equina nerve roots in the setting of vertebral metastatic disease is secondary to extension of metastatic disease through the cortex of the vertebral body into the epidural space (Fig. 19a–c). Cord compression is one of the few oncologic emergencies and requires emergent surgical decompression to relieve the mass effect on the cord and restore the neurologic deficits, although some soft tumors such as lymphoma may be treated with radiation and steroid rather than structural decompression.

Metastatic disease can also present as leptomeningeal disease within the thecal sac (as opposed to epidural disease outside the thecal sac) and can also lead to similar symptoms. Leptomeningeal disease should also be considered in the differential diagnosis in the setting of known metastatic disease [3].

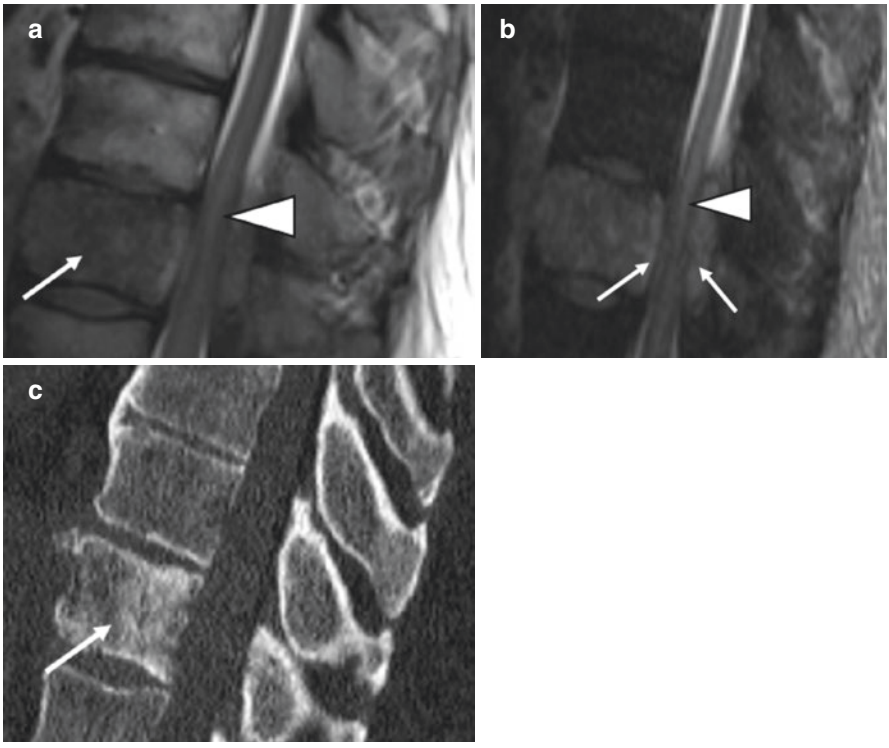

Fig. 19 Cord compression: (**a**) The sagittal T2-weighted MRI sequence shows the vertebral body is hypointense (arrow) relative to the other vertebral bodies. The tumor extends into epidural space of the spinal canal, compressing the spinal cord. There is increased T2 hyperintense signal within the cord (arrowhead) due to the compression. (**b**) The sagittal STIR sequence also demonstrates the tumor extending into the spinal canal (arrow) and highlights the increased signal within the cord (arrowhead) from compression. (**c**) The sagittal CT or the thoracic spine shows a sclerotic metastasis (arrow). The epidural soft tissue is not seen as well on CT

6.3 Schwannomas

Schwannomas are benign Schwann cells tumors which arise from neural crest precursors. Histology shows Antoni A and Antoni B subtypes on staining. Both sporadic and genetic (neurofibromatosis type 2) schwannomas have been linked to the NF2 suppressor gene on chromosome 22 [1].

Schwannomas are usually identified along cranial nerves III–XII at the skull base. They homogeneously enhance (Fig. 20), become cystic when large, and smoothly remodel foramen and bone. The most common location for a schwannoma is the vestibular nerve within the internal auditory canal (also known as acoustic schwannomas or acoustic neuromas) (Fig. 20) [1].

These lesions can enlarge and extend into the cerebellopontine angle, resulting in the "ice cream on a cone" and causes mass effect upon the adjacent pons and/or cerebellum (Fig. 20). Patients usually present with hearing loss or tinnitus [1].

6.4 Neurofibromatosis Type 2

Neurofibromatosis type 2 (NF2) will be discussed before neurofibromatosis type 1 to emphasize this unrelated syndrome is more simple than neurofibromatosis 1. There are only three similarities: they both have schwannomas, an autosomal dominant inheritance pattern, and the common diagnostic criterion of an affected first-degree relative [1].

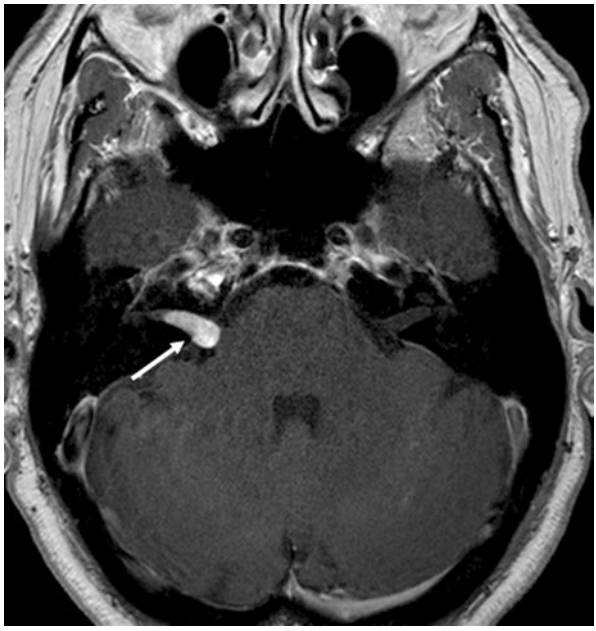

Fig. 20 Schwannoma in cerebellopontine angle: The axial T1-weighted post-contrast sequence demonstrates the classical "ice cream (cerebellopontine angle component) on a cone (intercanalicular component) sign" of a vestibular schwannoma

NF2 (also known as "central neurofibromatosis") is caused by a mutation on **chromosome 22**. Findings yields the popular acronym "MISME" = multiple inherited schwannomas, meningiomas, and ependymomas [1].

Diagnostic criteria include:

- Two vestibular schwannomas are diagnostic for NF2 (test worthy) (Fig. 21a and b).
- A single vestibular schwannoma and a history of a first-degree relative with NF2.
- A history of a first-degree relative with NF2 and a schwannoma (Fig. 21a–c), meningioma (Fig. 21B), glioma, or subcapsular lenticular opacities or cataracts [1].

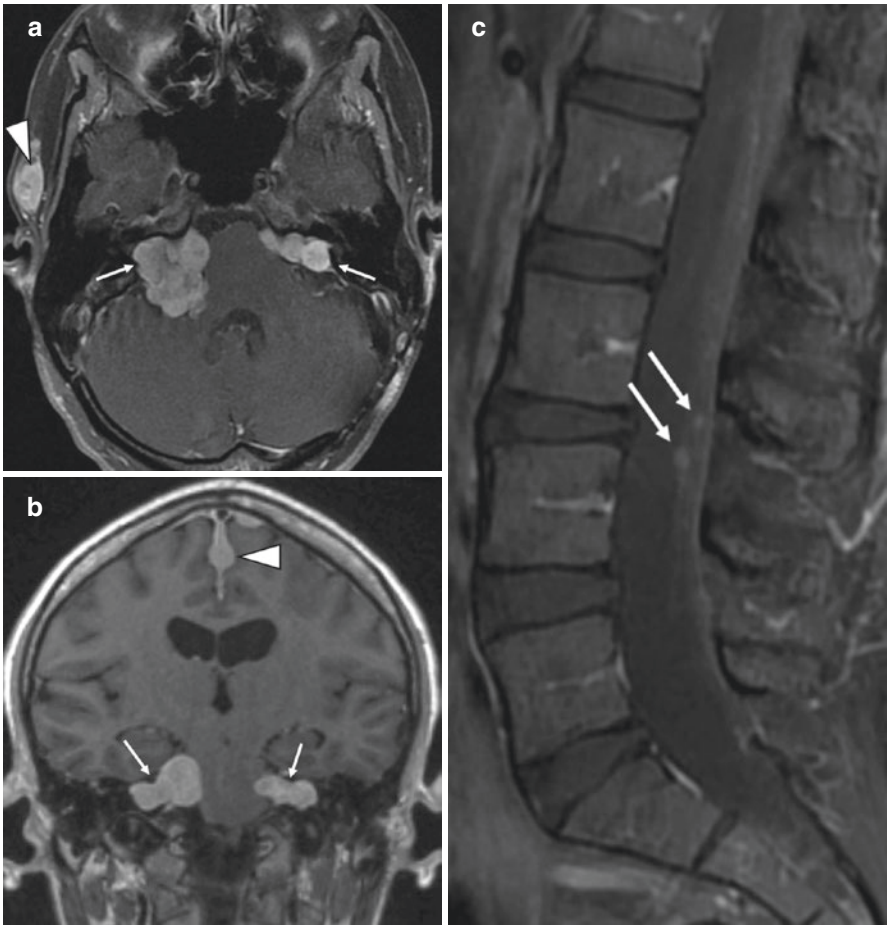

Fig. 21 Neurofibromatosis Type 2: (**a**) The axial T1-weighted post-contrast sequence demonstrates enhancing lesions (arrows) in both cerebellopontine angles consistent with vestibular schwannomas which is diagnostic for NF2. A third schwannoma is present in the right temporal fossa (arrowhead). (**b**) A coronal T1-weighted post-contrast sequence of the same patient shows a different perspective of the vestibular schwannomas as well as a homogeneously enhancing dural-based lesion along the falx, consistent with a meningioma. (**c**) A sagittal post-contrast image of the lumbar spine shows multiple enhancing nodules along the cauda equina nerve roots, consistent with schwannomas (arrows)

6.5 Neurofibromatosis Type 1

Neurofibromatosis type 1 (NF1) is inherited in an autosomal dominant pattern with variable expression. It is also known as peripheral neurofibromatosis or von Recklinghausen disease which is conveniently 17 letters—a clever way to remember the NF1 gene is located on chromosome 17. Mutations affect the Ras tumor suppressor gene and regulation of neural stem cell proliferation and differentiation [1].

Diagnostic criteria require two of the following findings:

- History of a first-degree relative with NF1 (same as NF2).

Skin:

- >/= 6 cafe au lait spots
- Armpit or groin freckling.
- >/= 2 neurofibromas (Fig. 22a)
- 1 plexiform neurofibroma (Fig. 22b)

Eye:

- Optic pathway pilocytic astrocytoma (Fig. 22c).
- >/= 2 Lisch nodules.

Bone:

- Sphenoid bone dysplasia.
- Long bong cortical dysplasia or thinning [1].

Notice how different this is from NF2 despite the similar name! Other NF1 tumors include T2/FLAIR focal areas of signal intensity (FASI) in the basal ganglia (Fig. 22d) and white matter of the brain (which usually resolve by adulthood), spinal neurofibromas, and rarely malignant peripheral nerve sheath tumors. Narrowing of the internal carotid arteries may result in a "moyamoya" pattern of the intracranial arteries. Dural ectasia with scalloping of posterior vertebral bodies (Fig. 22e), optic nerve sheath, and/or Meckel's cave expansion is not included in the diagnostic criteria but is a classical NF1 finding [1]. **Reflecting the greater number of letters in the name von Recklinghausen than the acronym MISME, there are more potential findings in NF1 than NF2!**

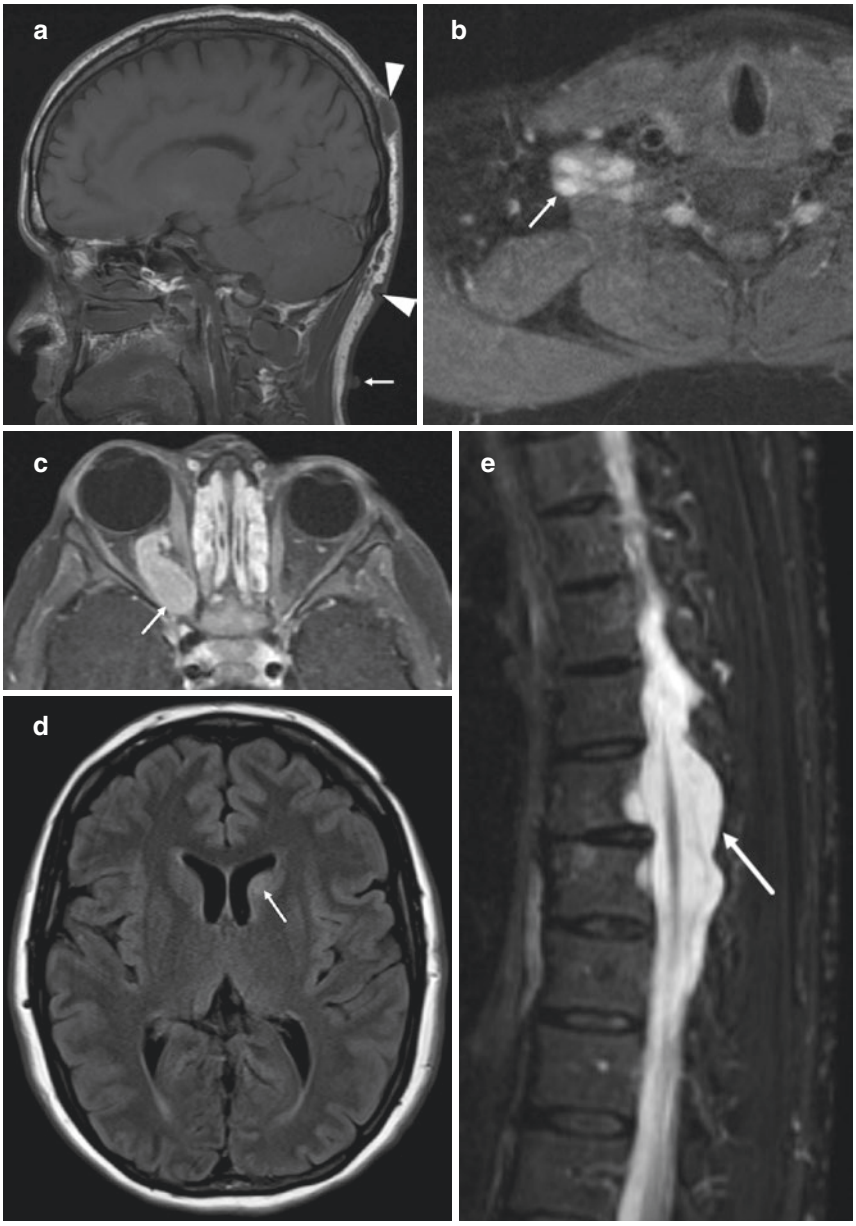

Fig. 22 Neurofibromatosis Type 1: A) A sagittal T1-weighted pre-contrast image shows a cutaneous (arrow) and subcutaneous (arrowhead) neurofibromas. B) An axial STIR image of the neck shows a hyperintense plexiform neurofibroma of the brachial plexus (arrow). C) An axial T1-weighted post-contrast image through the orbits shows the right optic nerve is enlarged and enhancing, consistent with an optic pathway glioma (arrow). The mass effect results in bulging or proptosis of the right globe. D) An axial FLAIR image demonstrates a focal area of signal intensity (FASI) in the head of the left caudate (arrow). E) A sagittal STIR sequence through the thoracic spine shows dura ectasia (arrow) and scalloping of the vertebrae

7 Congenital Malformations

7.1 Chiari 1 Malformation

Chiari 1 malformation describes herniation of the cerebellar tonsils through the foramen magnum, "peg-shaped" tonsillar deformity, and CSF effacement (Fig. 23a and b). CSF flow dynamics are altered which sometimes results in syrinx formation. Children present with oropharyngeal dysfunction and headaches less than 2 years of age and scoliosis after 2 years of age. Adults present with headaches and neck pain which resolve after a decompressive suboccipital craniectomy [1]. Many patients are asymptomatic, and the malformation is identified incidentally.

7.2 Chiari II Malformation with Myelomeningocele

Chiari II malformation is a hindbrain malformation syndrome describing multiple findings caused by a myelomeningocele (Fig. 24a and b) during prenatal development. Maternal folate deficiency increases the risk of this neural tube defect [1].

These findings include a small posterior fossa with extension of the cerebellar vermis into the cervical canal (Fig. 24c), kinking of the medulla, and elongation of the fourth ventricle, pons, and medulla into the cervical canal (Fig. 24c). Most patients have supratentorial abnormalities, including hydrocephalus (Fig. 24c), tectal beaking (Fig. 24c), and interdigitating gyri [1].

On ultrasound, Chiari II yields testable fruit signs: the lemon sign shows symmetric flattening of the frontal calvarium (seen in other neural tube defects as well) and the banana sign which shows the cerebellum surrounding the brain stem in the small posterior fossa [1].

For test day, the myelomeningocele (Fig. 24a and b) is the finding that must be remembered. Think of the myelomeningocele "pulling" the structures of the posterior fossa inferiorly (Fig. 24c) toward the neural tube defect to remember these findings.

8 Neurodegenerative Disease

8.1 Alzheimer's Disease

Alzheimer's disease is a progressive neurodegenerative disease. It is the most common form of dementia which leads to cognitive impairment and inability to perform activities of daily living. Amyloid plaques **and** tau neurofibrillary tangles are identified on histology before structural changes are present. Though there are classical imaging findings, imaging is often far from black and white. The findings can be subtle, and there are multiple subdivisions of Alzheimer's disease which have different regions of volume loss and corresponding functional decline which are beyond the scope of this text. The classical and therefore testable appearance of

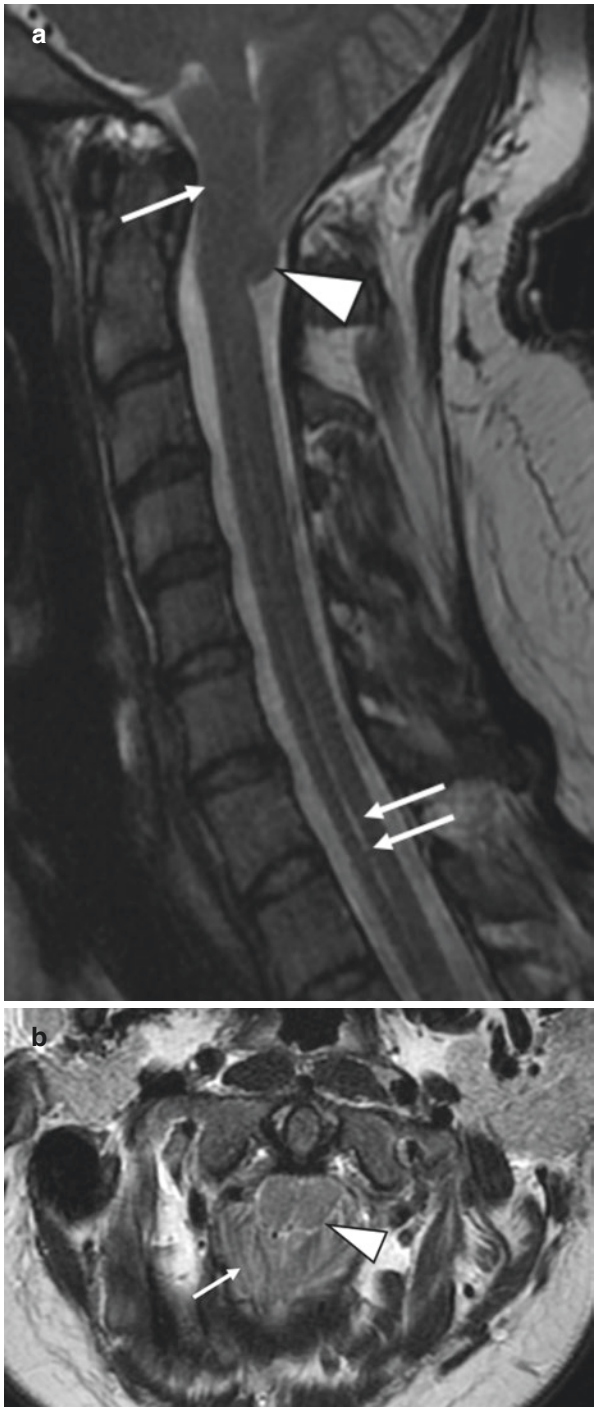

Fig. 23 Chiari 1 malformation: (**a**) A sagittal T2-weighted image of the cervical spine shows one of the cerebellar tonsils terminates below the foramen magnum and has a "peg-shaped" configuration (arrowhead). There is mass effect upon the medulla (arrow) and cord. The central canal is prominent though not yet a syrinx (double arrow). (**b**) An axial T2 sequence through the foramen magnum shows the tonsillar effacement of CSF (arrows) and mass effect upon the cord (arrowhead)

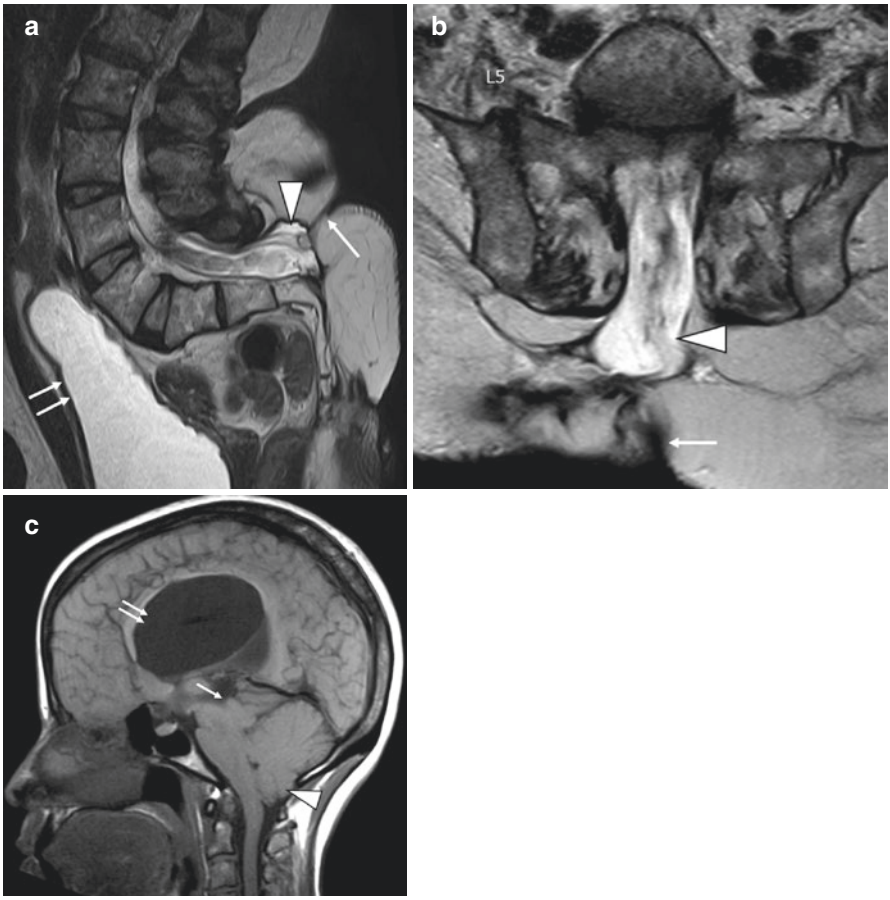

Fig. 24 Chiari II malformation: (**a**) A sagittal T2-weighted image of the lumbar spine shows an open spinal dysraphism with deficiency of the sacral posterior elements and a myelomeningocele (arrowhead) that extend to a sacral dimple (arrow). Notice the bladder (double arrowhead) is extremely large and fluid-filled, likely secondary to a neurogenic bladder. (**b**) An axial T2-weighted image shows a different view of the myelomeningocele (arrowhead) extending through the open spinal dysraphism towards a sacral dimple (arrow). (**c**) An axial T1-weighted pre-contrast image shows the contents of the posterior fossa appear pulled inferiorly. There is elongation of the brain stem, tectal beaking (arrow) with aqueductal stenosis and hydrocephalus (double arrow), and extension of the cerebellum through the foramen magnum (arrowhead)

Alzheimer's disease on MRI or CT is parietal and temporal lobe volume loss, particularly involving the hippocampus (Fig. 25a). The sulci, Sylvian fissures, and ventricles adjacent to the areas of volume loss are enlarged (Fig. 25a) [1].

Nuclear medicine scans may provide valuable metabolic information. An 18-fluorine FDG positron emission tomography (PET) scan shows decreased metabolic uptake of the radiotracer (Fig. 25b) in the region of volume loss. Amyloid PET

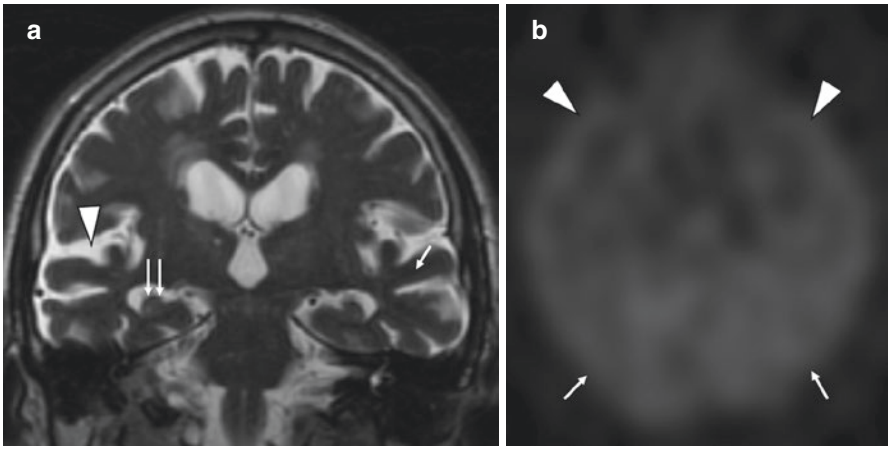

Fig. 25 Alzheimer's disease: (**a**) A coronal T2-weighted image shows symmetric volume loss that is greatest in the temporal lobes (arrow), particularly the hippocampi (double arrows) with enlargement of the Sylvian fissures (arrowhead). (**b**) An axial F^{18}FDG scan demonstrates decreased metabolic activity in the temporal lobes (arrowheads) compared to the occipital lobe (arrows)

images 11-carbon labeled radiotracer Pittsburg Compound B (PiB) or 18-F radiotracer bound to amyloid plaques. The radiotracer clears from the normal brain. Increased radiotracer uptake on an amyloid PET is pathologic [1].

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Emergencies/Common On-Call Scenarios/Specific Cases Imaging

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1 Strategies for Multiple-Choice Questions

1.1 What You See Is All There Is (WYSIATI)

In behavioral psychology, the term *what you see is all there is* (WYSIATI) refers to jumping to conclusions based on limited evidence [1]. To avoid this cognitive bias, you are supposed to take a systematic approach to evidence by seeking information that you don't have.

But on multiple-choice exams, often what you see *is* all there is. There is likely one conspicuous key finding on the image shown, and that finding is often corroborated by the clinical data presented in the question. Do not get stuck staring at an image looking for small hidden findings. Look for the obvious free air, pneumothorax, small bowel obstruction, heart failure, or cancer. The image presented is not an eye test. After you see a main finding, if you see something else that is very subtle and you question whether it is real, then it is not real. Therefore, avoid the temptation to look at an image too long. Search the image for the single obvious finding that might be suggested by the history, and move on quickly.

In your studying, focus on familiarity with the basic images that result in major bad outcomes if missed. This will be the likely goal of any question writer who decides to add an image; it is added for a reason. Think of big findings. For example, in a patient with shortness of breath and tachypnea, try to differentiate general fluid overload due to heart failure (fluffy white airspaces all over the lung + big heart and pleural effusions) from a focal lobar community-acquired strep pneumonia (consolidation of a lobe). A right middle lobe *Klebsiella* pneumonia in an alcoholic patient who aspirates down the straighter and gravity-dependent right bronchus intermedius is a classic image that blurs the right heart border. Look quickly for free air and pneumothorax on every image; it only takes a second.

1.2 The Negative Image

Beware also of the general image that is not pathognomonic for any one specific diagnosis. Totally negative images are possible and are often purposeful, particularly in the setting of an obvious history or labs suggestive of a specific diagnosis.

For example, a normal abdominal radiograph showing no kidney stones in the setting of hematuria and weight loss in an elderly patient may spur ordering a computed tomography (CT) scan. If ordering a CT is not one of the answers, find the BEST answer once you have ruled out major and obvious imaging findings. Likewise, a normal chest radiograph without obvious findings in the setting of substernal chest pain and shortness of breath might exclude pneumonia or heart failure and lead you toward a cardiac or gastrointestinal workup.

1.3 History Is King

Always use the history from the question. Avoid looking at the image until you read the text of the question. As you gain more experience answering practice questions, try to formulate in your mind a list of common findings based on the history. For example, on a chest radiograph:

- History of trauma: look for free air, pneumothorax, widened mediastinum (aortic dissection), and rib fractures.
- History of leukocytosis: look for lobar consolidation from community-acquired bacterial pneumonia (right shift), scattered pulmonary opacities from atypical pneumonia (such as mycoplasma or legionella), or peribronchial opacities from viral pneumonia (left shift).
- History of aspiration, alcohol abuse, or postanesthesia: look for patchy lower lobe opacities from aspiration pneumonia, especially in the right middle lobe or lower lobes.
- History of acquired immunodeficiency syndrome with CD4 count <200: look for bilateral perihilar reticular opacities from *Pneumocystis jirovecii* pneumonia.

1.4 More Exam-Taking Tips

- Emergencies are a very common boards scenarios that include associated imaging.
- Collect a "basket of relevant findings" from each question. Filter through normal (or slightly abnormal but irrelevant) information. Highlight and make a mental note of the major abnormalities, and cluster them into unified diagnoses or decision tree. Imaging will only be one thing in your basket, most often with corroborating evidence for a specific diagnosis, especially in the emergency setting.
- Common things are common. See how the image may be corroborated by the text of the question.
- Never miss the killers: always search for free air and pneumothorax, but expect the more common gallstones, kidney stones, stroke, fractures, CHF, obstruction, cancer, pneumonia, and ectopic pregnancy.

2 Chest Case 1

An 82-year-old man with esophageal cancer and recurrent malignant left pleural effusions underwent thoracentesis a few hours ago. He is now feeling increasingly short of breath with left shoulder pain worse on inspiration. Radiograph of the chest was performed (Fig. 1).

The tension pneumothorax was precipitated by the thoracentesis. The subcutaneous emphysema is due to air leak through the puncture track into the subcutaneous space.

The patient underwent chest tube placement. A chest radiograph was performed to confirm chest tube position (Fig. 2).

An alternative correct answer for treatment might be emergent placement of a needle in the second intercostal space at the midclavicular line anteriorly. This could be followed by a chest tube. Either answer is correct depending upon how the question is written. With acute decompensation, place a needle fast!

The patient's pneumothorax and subcutaneous emphysema resolved 8 days after chest tube placement.

In general, chest tubes are connected to "Pleur-evac" devices (sealed suction devices with chambers for suction and sealing and another for drainage) to help manage and protect the pleural space and provide a one-way air flow from the pleural space. The chest tube is connected to suction through the Pleur-evac. For large air leaks from the lung, low continuous suction is applied to the chest tube to promote complete and ongoing evacuation of air from the pleural space. This helps ensure constant contact between the visceral and parietal pleura to help seal the hole in the visceral pleura. At any point, a "water seal" trial can be performed at the bedside to evaluate for air leak by turning off the suction and asking the patient to

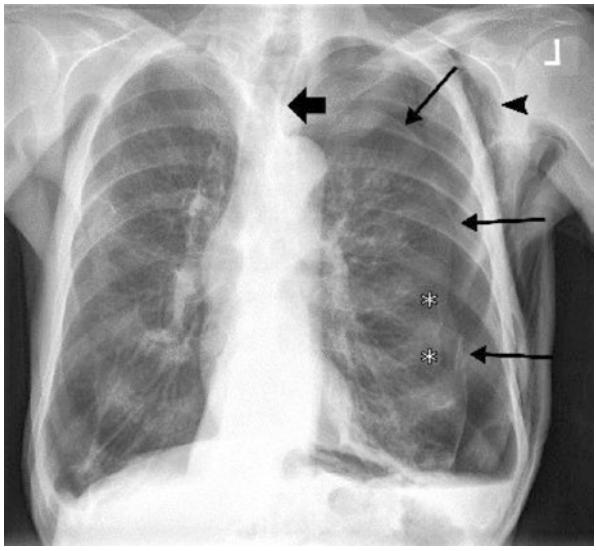

Fig. 1 *Technique:* PA chest radiograph. *Findings:* A large left pneumothorax is present (arrows). There are signs of positive pressure on adjacent structures, including mild rightward tracheal shift, depressed left hemidiaphragm, and subtle widening of left intercostal spaces (*). Gas is present in the left chest wall (arrowhead). *Diagnosis:* Left tension pneumothorax with left subcutaneous emphysema

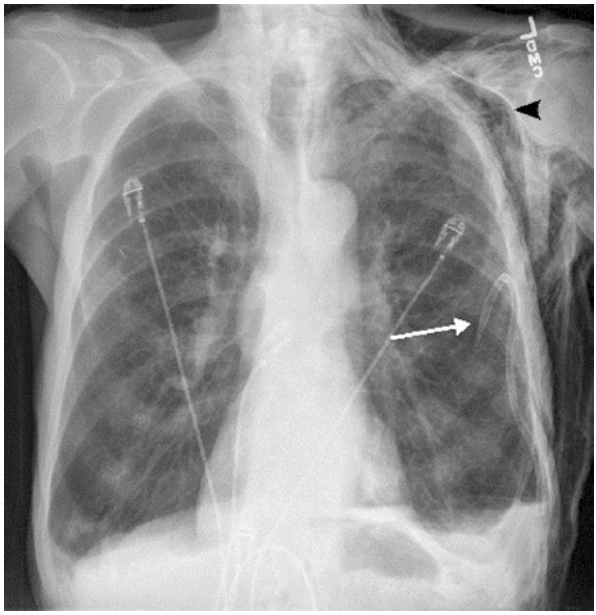

Fig. 2 *Technique:* PA chest radiograph. *Findings:* A left-sided "pigtail" chest tube has been placed (arrow). Compared to Fig. 1, the left pneumothorax is no longer present, and the left hemidiaphragm appears more elevated than before. There remains gas in the left chest wall (arrowhead). *Diagnosis:* Status post chest tube placement with resolution of pneumothorax. Persistent subcutaneous emphysema

cough. If an air leak is present, air bubbles will be seen passing through the water seal chamber in the Pleur-evac. The degree of air leak is reflected by the rate and amount of air bubbles seen. If a lot of air bubbles are seen, suction is restarted. As the air leak slows, the chest tube can be taken off suction and be managed by water seal, which acts as a check valve, allowing air to leave the pleural space but not reenter. Typically, a prolonged period of water seal is desired, allowing further healing and closure of the site of air leak. Serial chest radiographs can be used to monitor the presence, degree, and stability of pneumothorax, if any, and lung reinflation. If there is no pneumothorax or a small pneumothorax is stable over time, no evidence of air bubbles at bedside, and intrapleural pressure is restored on the Pleur-evac, some operators will then remove the chest tube. Other operators, however, will perform a "clamp trial" during which time the chest tube is clamped for several hours. Clamping the chest tube allows for detection of very small, transient residual air leaks that may be missed on water seal, since air can still leave the pleural space. If no pneumothorax is seen on chest radiographs and intrapleural pressure has been restored on the Pleur-evac, the chest tube can be safely removed.

In this case, this patient passed a water seal trial. The chest tube was removed, and the pneumothorax did not return on post-removal follow-up chest radiograph.

2.1 Learning Points: Tension Pneumothorax

- Tension pneumothorax occurs when the hole in the lung behaves as a "check valve," allowing air to leave the lung into the pleural space but preventing it from

reaching equilibrium across the hole or be released from the pleural space via another route. Progressive accumulation of intrapleural air exerts increasing pressure on surrounding structures, resulting in the following findings:
  - Ipsilateral hemidiaphragm depression
  - Ipsilateral intercostal space widening
  - Mediastinal shift away from the side of tension pneumothorax
  - Compression of large systemic veins and right heart chambers leading to obstructive cardiopulmonary collapse
- Depending on the answer choices given, the next step in treatment is either needle thoracostomy or chest tube placement.

3 Chest Case 2

A 61-year-old active smoker man who has not seen a doctor in 5 years presented with worsening acute-onset tearing chest and back pain. Blood pressure was 178/110, otherwise hemodynamically stable. Physical exam and laboratory studies were unrevealing. Echocardiogram and chest radiograph (not shown) were unremarkable. CT angiogram of the chest, abdomen, and pelvis was performed (Fig. 3).

The patient was initially monitored in the ICU with medical management of his hypertension; however, he subsequently developed urinary retention and right leg weakness, which are concerning symptoms for spinal cord ischemia. As a result, thoracic endovascular aneurysm repair (TEVAR) was performed.

3.1 Learning Points: Aortic Dissection

- According to the Stanford classification, aortic dissections are divided into Type *A* (involving the ascending thoracic aorta) and Type B (involving the descending

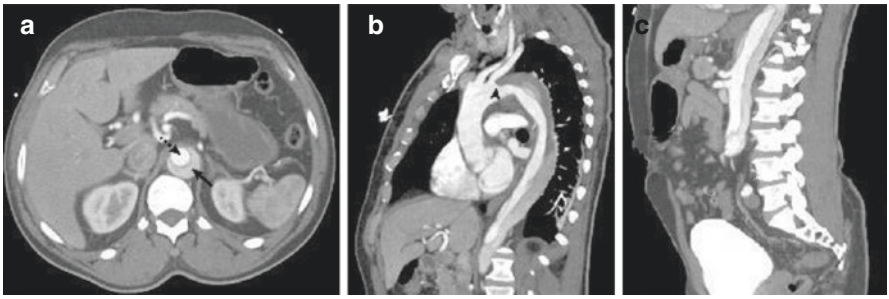

Fig. 3 *Technique:* CT angiogram of chest, abdomen, and pelvis. Axial (**a**), sagittal oblique (**b**), and sagittal (**c**) images. *Findings*: A curvilinear hypodense intimal flap within the aortic lumen separates the large and less well opacified false lumen (solid arrow) from the smaller true lumen (dashed arrow). The aortic dissection extends from just distal to the takeoff of the left subclavian artery (arrowhead) to the distal abdominal aorta. *Diagnosis*: Stanford type B acute aortic dissection

thoracic aorta distal to the left subclavian artery). Type A dissections need to be surgically repaired to prevent fatal complications such as coronary artery occlusions and cardiac tamponade. Type B dissections can be managed medically with blood pressure control, unless underperfusion symptoms or complications occur.
- It is important to look for imaging findings of potential complications resulting from aortic branch occlusion, such as end-organ ischemia and distal emboli.
- Chest X-ray may be normal in 10–40% of aortic dissections [2].

4 Chest Case 3

A 49-year-old man with no significant past medical history presented with 5 days of exertional dyspnea. Oxygen saturation was 94% on room air. Electrocardiogram (ECG) showed sinus tachycardia. Serum D-dimer was markedly elevated at >5000 ng/mL (normal 0–500 ng/mL). CT pulmonary angiogram was performed (Fig. 4).

The patient was treated with anticoagulation, although TPA and mechanical thrombectomy may be considered with hemodynamic collapse. A (removable) IVC filter might be placed in settings of contraindications to anticoagulation.

4.1 Learning Points: Pulmonary Embolism ("PE")

- Due to its speed and high sensitivity, CT pulmonary angiogram is the imaging modality of choice for PE in the emergent setting.
- *A normal D-dimer is nearly 100% predictive in ruling out PE*. An elevated D-dimer, however, is nonspecific and is seen in many other conditions.

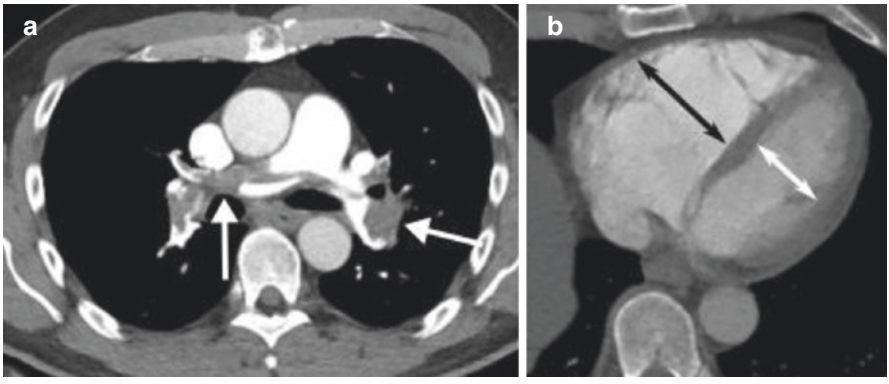

Fig. 4 *Technique:* CT pulmonary angiogram. Axial images. *Findings:* (**a**) There are contrast filling defects in the main pulmonary artery and the left and right pulmonary arteries (arrows). (**b**) The right ventricle (double black arrows) is dilated relative to the left ventricle (double white arrows). *Diagnosis:* Saddle pulmonary embolism with right ventricular strain

- Always look at the ventricular size on CT. Right ventricular strain, a predictor of mortality related to PE, is suggested on CT when the intracavitary diameter of the right ventricle relative to that of the left ventricle (RV/LV ratio) is greater than 0.9 [3]. Echocardiography is a more common method for documenting right heart strain or signs of elevated right heart pressure. Physical exam will show jugular venous distension. On ECG, PE with right heart strain classically shows an *"S1Q3T3" pattern: presence of an S wave in lead I, a Q wave in lead III, and an inverted T wave in lead III*. This can be recalled with the mnemonic "SIQETE PE."
- Aside from the usual risk factors for PE such as malignancy, immobility, and thrombotic disorders, a young female smoker on birth control pills is a classic patient demographic for an exam question (as well as anti-phospholipids, cardiolipin antibodies, lupus anticoagulant, or anti-beta-2 glycoprotein 1, as well as deficiency of protein C, protein S, or antithrombin III).

5 Brain Cases

Patient 1 A 60-year-old woman with hypertension and hyperlipidemia presented with inability to talk and progressive right upper extremity weakness and numbness, onset approximately 1 h prior. Physical examination revealed left gaze palsy, right-sided hemiplegia and facial droop, and aphasia, consistent with left middle cerebral artery (MCA) syndrome. She scored 26 on the NIH stroke scale, indicative of severe stroke. Head CT and CT angiogram were performed emergently (Fig. 5).

The patient was given systemic tissue plasminogen activator (tPA) and taken emergently for endovascular thrombectomy (Fig. 6).

Patient 2 An 85-year-old woman with hypertension, hyperlipidemia, and coronary artery disease presented with aphasia followed by neurologic decline. She was treated with systemic tPA followed by endovascular thrombectomy for a distal left MCA occlusion. Head CT was performed 8 h after thrombectomy (Fig. 7).

Over the next several days, her head imaging remained stable with gradual decrease in subarachnoid hemorrhage. Her head CT 7 days following thrombectomy is shown (Fig. 8).

On day 17 following thrombectomy, the patient was found unresponsive with gaze deviation. Head CT was urgently performed (Fig. 9).

The patient's family was informed of the poor neurologic prognosis to allow for advanced directives and spiritual care. The patient expired the following day.

5.1 Learning Points: Ischemic and Hemorrhagic Stroke

- Non-contrast head CT is the first-line imaging modality for suspected stroke.
- In patients with large ischemic strokes, the risk of hemorrhagic transformation is high, particularly following thrombolytic and/or endovascular therapies.
- Intracranial hemorrhage is conspicuous on non-contrast head CT.

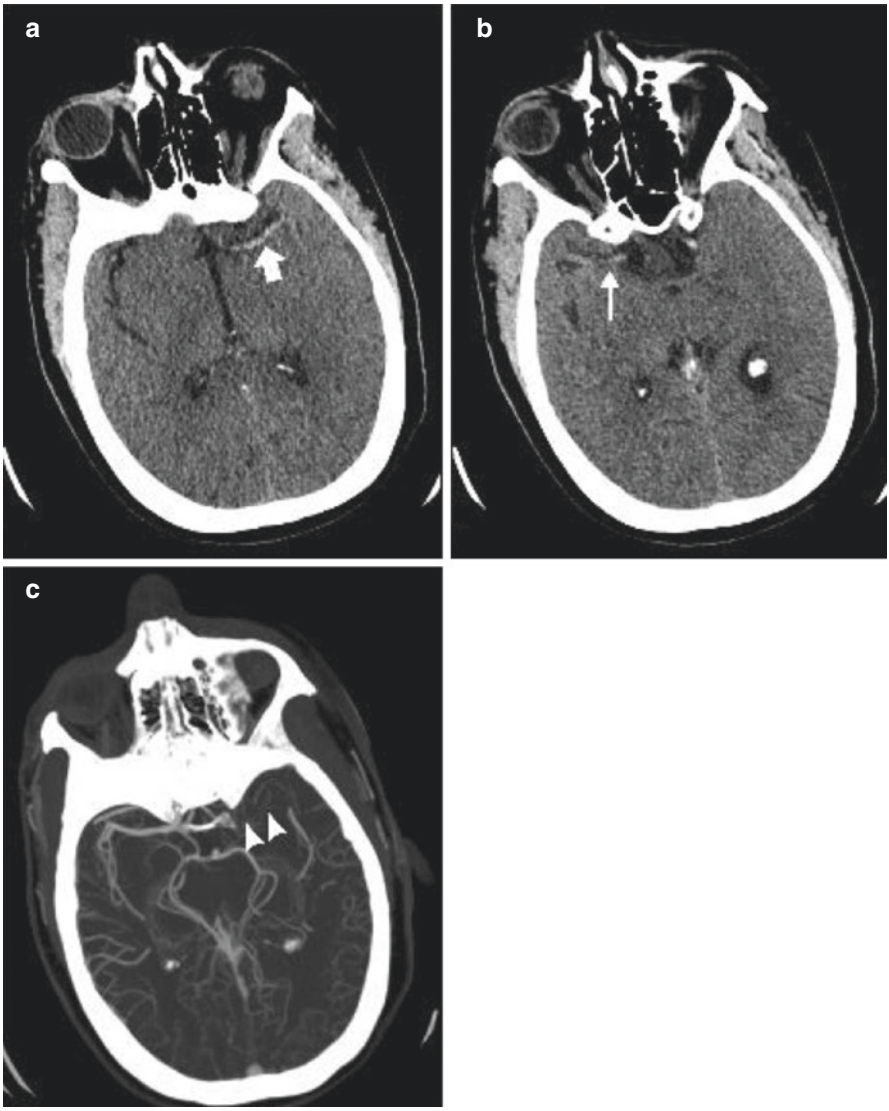

Fig. 5 *Technique:* (**a** and **b**) Head CT without intravenous contrast. Axial images. (**c**) Head CT angiogram with intravenous contrast. Axial image. *Findings:* On head CT without intravenous contrast, the distal left internal carotid artery and left MCA M1 segment are hyperdense (block arrow) compared to the contralateral MCA (arrow), suggestive of acute intraluminal thrombus. This finding is known as the dense vessel sign. The left distal ICA and MCA M1 segment are not opacified on CT angiogram (arrowheads), confirming the occlusion. *Diagnosis:* Acute ischemic stroke with left ICA and M1 occlusion

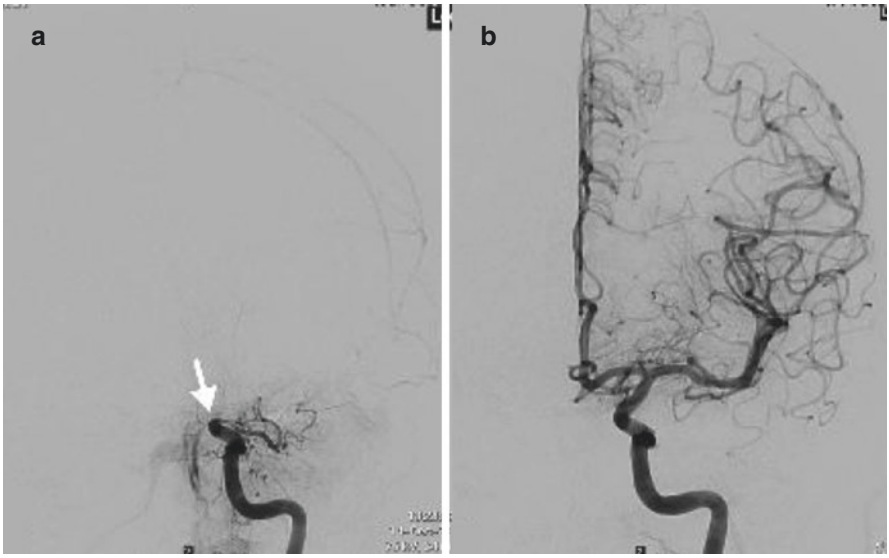

Fig. 6 *Technique:* Digital subtraction angiogram with left ICA injection. Anterior-posterior views before (**a**) and after (**b**) endovascular thrombectomy. *Findings:* The distal left ICA is occluded (arrow) on initial injection. Following successful endovascular thrombectomy to remove the intraluminal thrombus, flow was restored to the left MCA and anterior cerebral artery branches

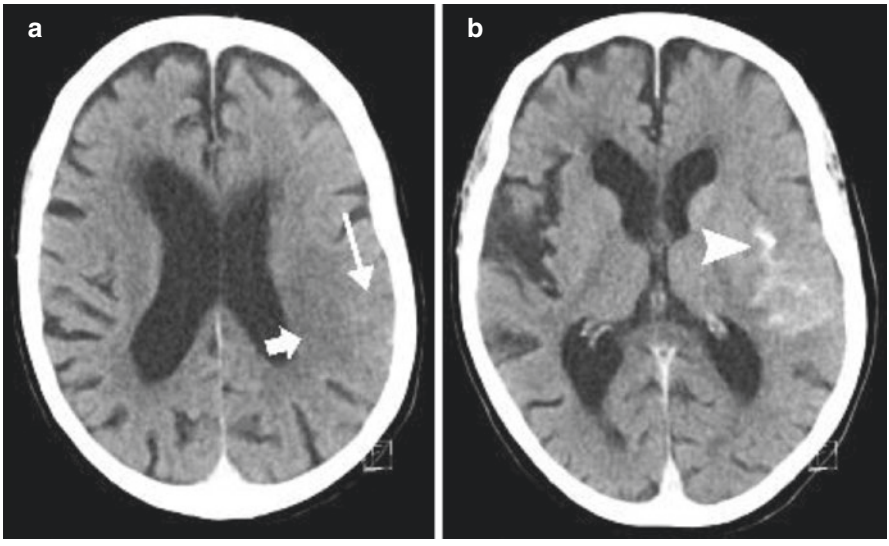

Fig. 7 *Technique:* Head CT without intravenous contrast. Axial images. *Findings:* (**a**) Subtle frontoparietal white matter hypodensity (block arrow) and sulcal effacement (arrow) are present. (**b**) There is hyperdensity in the Sylvian fissure (arrowhead), which was not present before thrombectomy. *Diagnosis:* Left MCA infarction. New subarachnoid hemorrhage in the Sylvian fissure

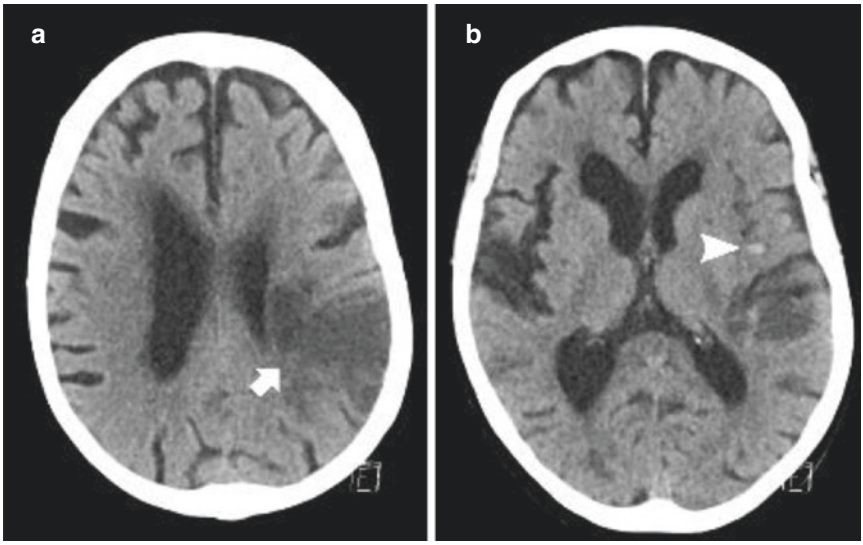

Fig. 8 *Technique:* Head CT without intravenous contrast. Axial images. *Findings:* (**a**) Frontoparietal hypodensity is now more apparent (block arrow). (**b**) Hyperdensity in the Sylvian fissure has decreased (arrowhead). *Diagnosis:* Expected evolution of left MCA territory infarction. Resolving subarachnoid hemorrhage

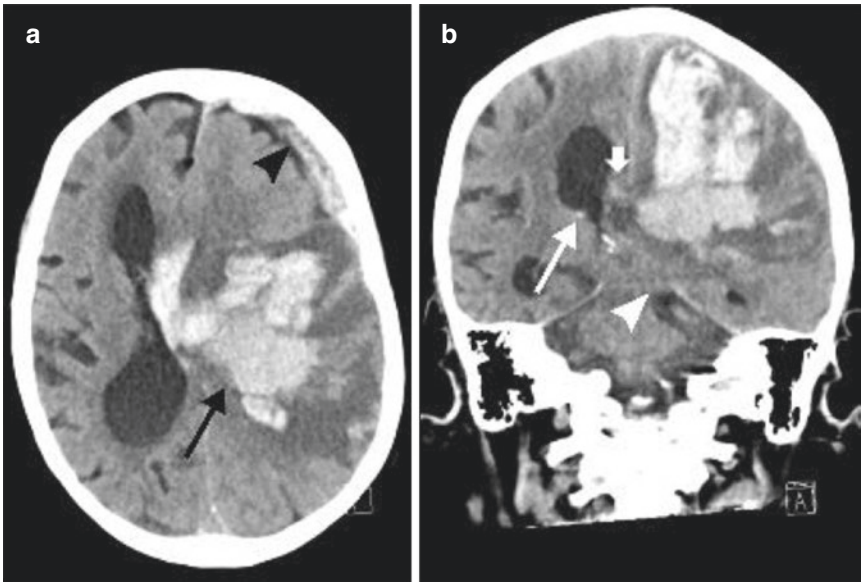

Fig. 9 *Technique:* Head CT without intravenous contrast. Axial (**a**) and coronal (**b**) images. *Findings:* A large left cerebral intraparenchymal hemorrhage (black arrow) and a left frontal subdural hemorrhage (black arrowhead) are present. There is mass effect with subfalcine herniation (block arrow) and uncal herniation (white arrowhead). The left lateral ventricle is effaced; the right lateral ventricle is dilated and contains hyperdense blood (white arrows). *Diagnosis:* Hemorrhagic transformation of left MCA territory infarction with intraventricular rupture, resulting in mass effect, subfalcine herniation, uncal herniation, and obstructive hydrocephalus

- In acute stroke, local swelling may be seen as gyral flattening or sulcal effacement, the location of which depends on the vascular territory involved.
- Brain parenchyma changes in ischemic stroke can be subtle on non-contrast head CT; MRI is much more sensitive. Therefore, images on test questions will show you either an evolved large-vessel territory stroke or a conspicuous secondary sign such a dense vessel sign.

6 Spine Case

An 89-year-old man fell forward while getting out of bed and presented with neck pain. He was immediately placed into a cervical collar. CT was performed (Fig. 10).

The patient went on to receive an occiput to C4 fusion to stabilize the cervical spine. Post-procedure radiographs were obtained to confirm hardware position (Fig. 11).

6.1 Learning Points: Cervical Spine Fracture

- The Anderson and D'Alonzo classification (Fig. 12) describes three types of odontoid (dens) fractures [4]:
  - Type I: fracture at upper part of odontoid peg; usually considered stable

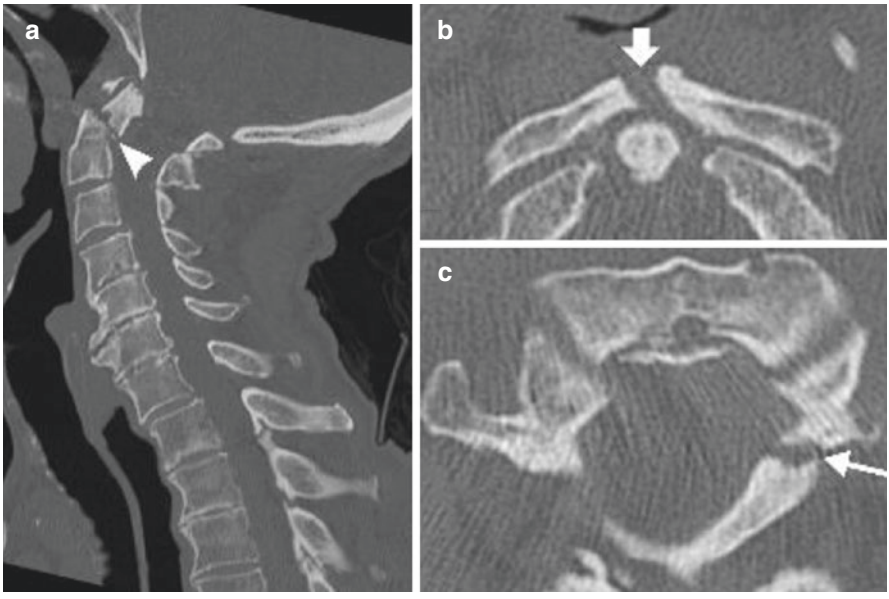

Fig. 10 *Technique:* Cervical spine CT without intravenous contrast. Sagittal (**a**) and axial (**b**, **c**) images. *Findings:* There is a displaced fracture through the base of the odontoid process of C2 (arrowhead), as well as fractures through the anterior arch (arrow) and posterior arch (arrow) of C1. *Diagnosis:* Type II odontoid fracture (unstable). C1 fractures

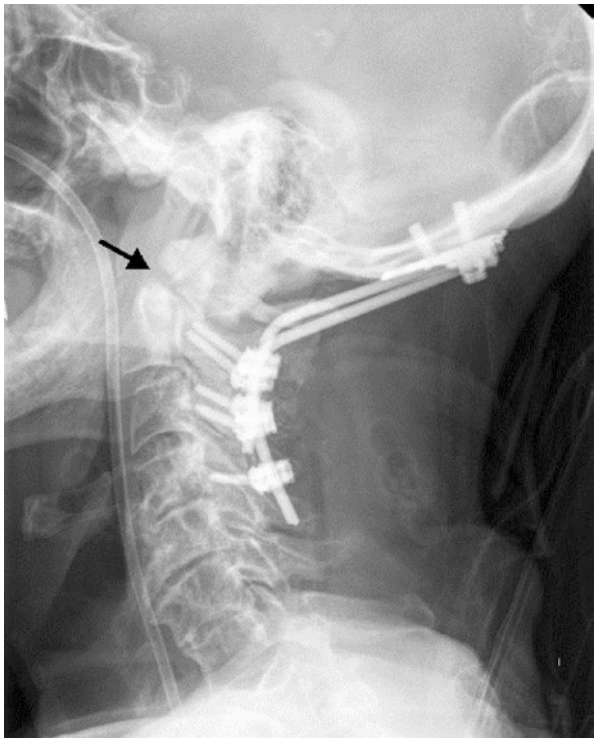

Fig. 11 *Technique:* Lateral cervical spine radiograph. *Findings:* Expected appearance of occiput to C4 posterior instrumented fusion. Displaced odontoid fracture is re-demonstrated (arrow)

  - Type II: fracture at base of odontoid; considered unstable and has high risk of nonunion
  - Type III: fracture through odontoid and into lateral masses of C2; considered stable if not excessively displaced
- For purposes of the test, type II odontoid fracture is surgical. Types I and III can be managed nonoperatively.
- A normal stable variant called *os odontoideum* (Fig. 13) may look like a type II odontoid fracture but has a sclerotic border that indicates cortical bone. In an acute fracture, osteoclasts have not had time to lay down cortical bone; thus, the sclerotic border is absent.
- When subjected to excessive force, ring-shaped bony structures such as the C1 vertebra and the pelvic ring almost always break in two or more places. If you identify one fracture of a ring, always look for additional fractures (it is hard to break a pretzel in just one place).

7 Abdominal Case 1

A 70-year-old active smoker man with hypertension and hyperlipidemia presented with 2 weeks of intermittent lower abdominal pain radiating to his back. Physical exam revealed a non-tender pulsatile midline abdominal mass. Laboratory studies were largely normal. CT angiogram of the chest, abdomen, and pelvis was performed (Fig. 14).

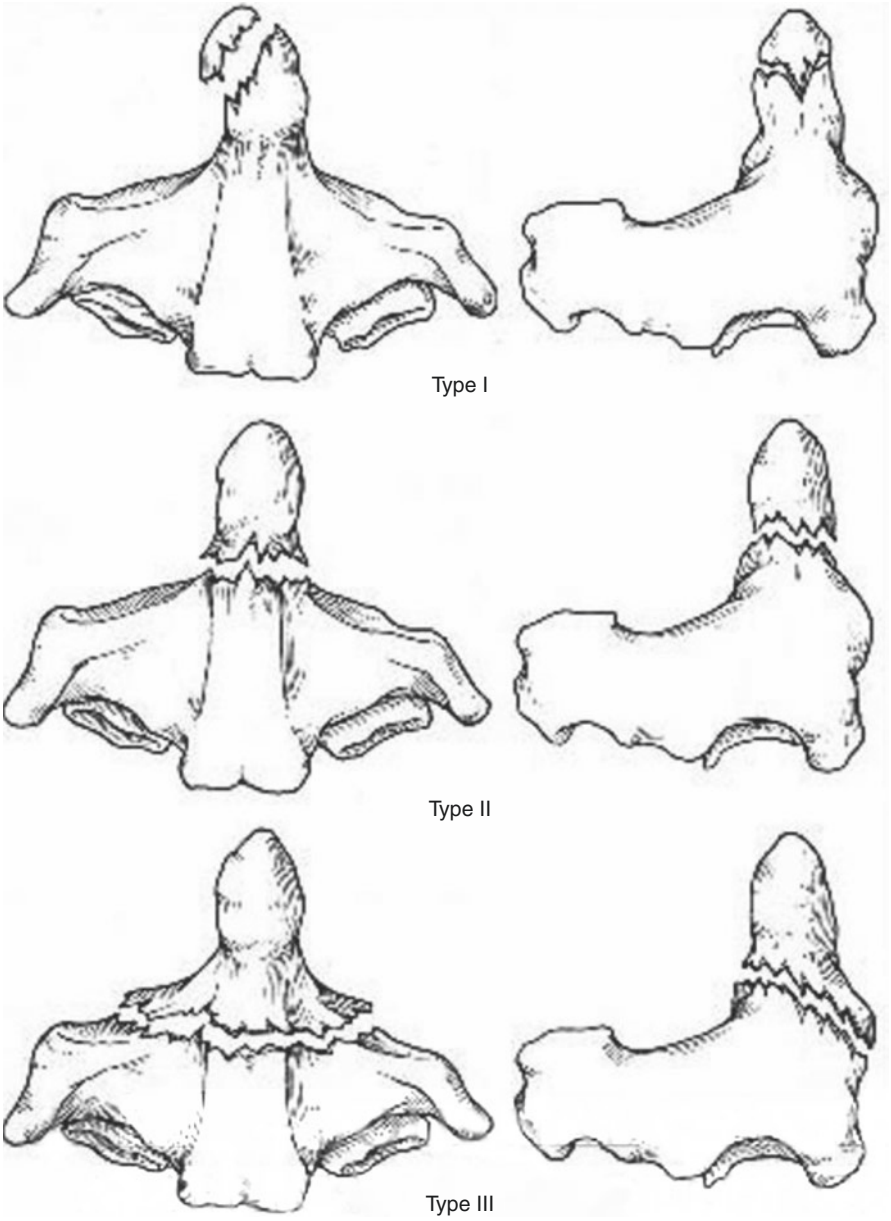

Fig. 12 The Anderson and D'Alonzo classification of odontoid fractures. (Reprinted with permission from Hsu and Anderson [4])

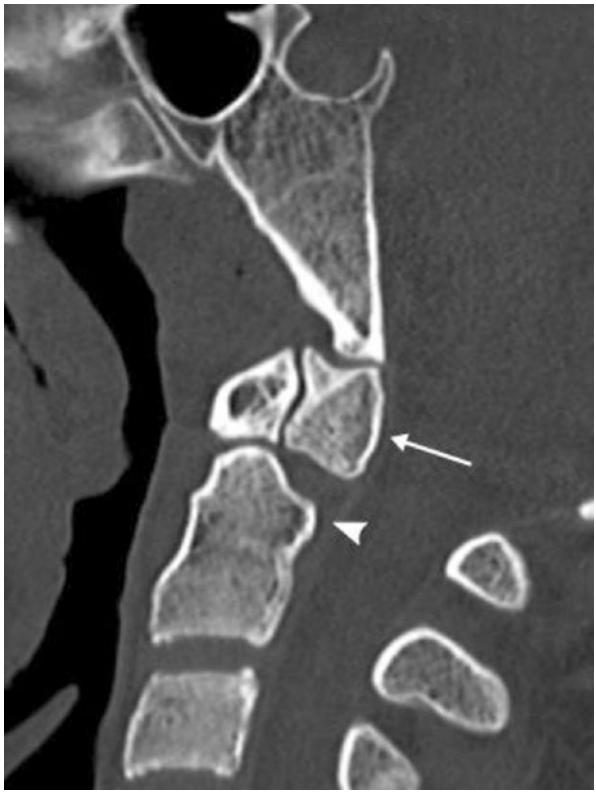

Fig. 13 Os odontoideum. Sagittal CT image of the cervical spine demonstrates an os odontoideum (arrow), located superior to a small odontoid process (arrowhead) and separated by a gap. Note the smooth, sclerotic borders of the os odontoideum

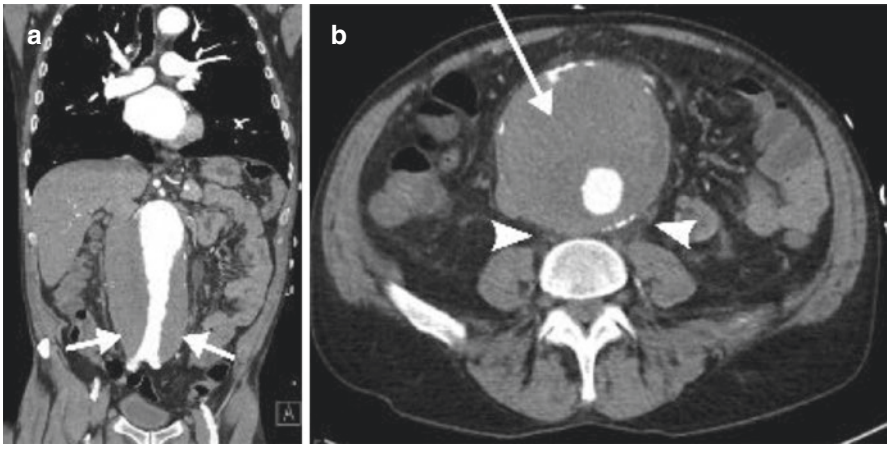

Fig. 14 *Technique:* CT angiogram of chest, abdomen, and pelvis. Coronal (**a**) and axial (**b**) images. *Findings:* The infrarenal abdominal aorta is markedly dilated, measuring 11 cm in diameter. Along the aortic wall, there is extensive non-opacification, representing mural thrombus (arrow). The aorta abuts the vertebral body with loss of normal fat plane between the posterior wall of the aorta and the vertebral body (arrowheads). *Diagnosis:* Contained rupture of fusiform abdominal aneurysm

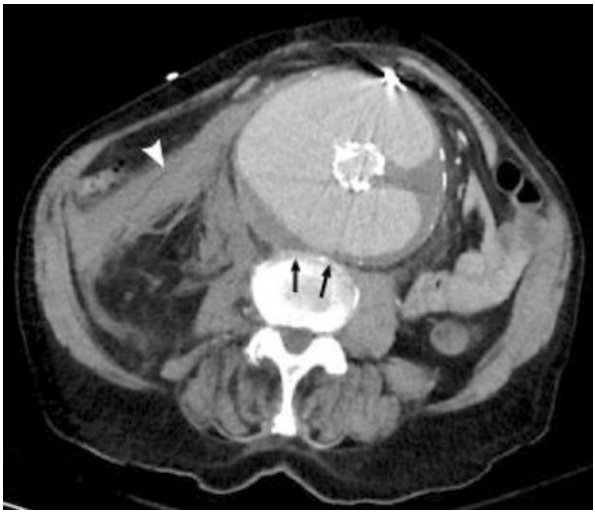

Fig. 15 Draped aorta sign. Axial CT angiogram image through the abdomen demonstrates a massive abdominal aortic aneurysm in this patient who had previously undergone endovascular aneurysm repair but whose aneurysm sac continued to enlarge due to "endoleak." The posterior wall of the aneurysm appears to drape over the anterior surface of the vertebral body (arrows); the fat plane in between the aneurysm and the vertebral body has been lost. This is a specific sign of contained rupture of abdominal aortic aneurysm. The finding of retroperitoneal hemorrhage (arrowhead) further supports the diagnosis

The patient underwent an emergent open surgical aneurysm repair using a Dacron (synthetic polyester) graft.

7.1 Learning Points: Ruptured Abdominal Aortic Aneurysm

- Frank retroperitoneal hemorrhage and contrast extravasation are highly specific for aneurysm rupture. Note that these findings are rarely seen in real life, as these patients often die before arrival to the hospital.
- The draped aorta sign, in which the fat plane in between the posterior wall of the abdominal aortic aneurysm and the vertebral body is lost such that the aneurysm appears to drape over the vertebral body, is a specific sign of contained aneurysm rupture. A suggestion of the draped aorta sign is present in Fig. 14, although a more dramatic example is shown in Fig. 15.

8 Abdominal Case 2

A 62-year-old man fell with right 8th through 11th rib fractures, which resulted in a pneumothorax that resolved after chest tube placement during a brief hospital admission. Five days after discharge (9 days after the fall), he re-presented to the emergency department with progressively worsening right upper quadrant

abdominal pain and new fever. Laboratory studies showed a mild leukocytosis. Chest radiographs were performed (Fig. 16).

CT was performed for better assessment of free air and to localize the perforation (Fig. 17).

The patient underwent an exploratory laparotomy, which revealed a 1-mm-sized perforation in the ascending colon just proximal to the hepatic flexure and purulent

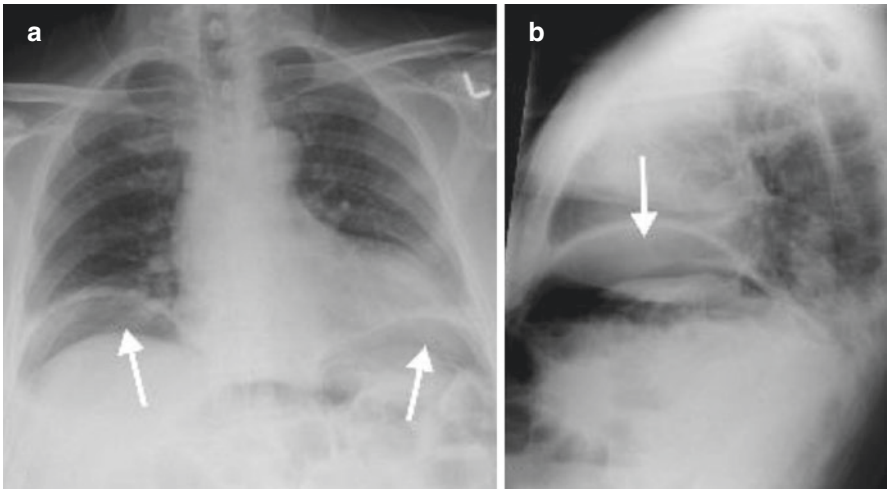

Fig. 16 *Technique:* Upright AP (**a**) and lateral (**b**) chest radiographs. *Findings:* Crescent-shaped lucency is present under both hemidiaphragms (arrows). *Diagnosis:* Large pneumoperitoneum = free air!

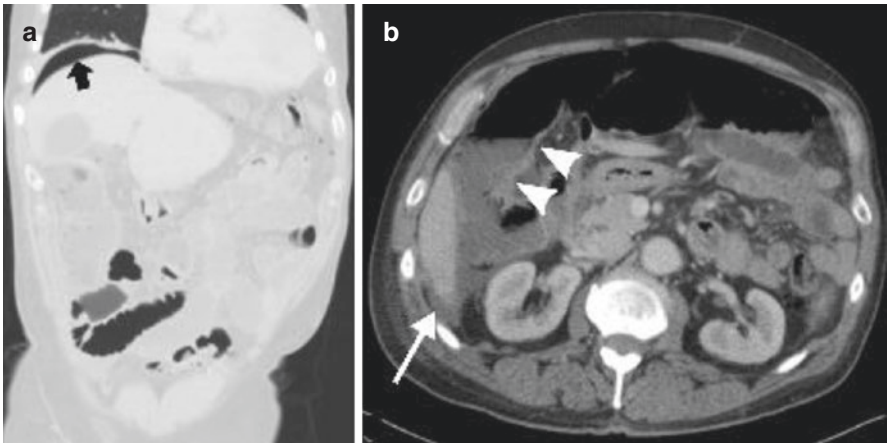

Fig. 17 *Technique:* CT of abdomen and pelvis. Coronal image on lung window (**a**) and axial image on soft tissue window (b). *Findings:* There is air under the right hemidiaphragm (block arrow). The left hemidiaphragm is not included on this slice. Perihepatic fluid is present (arrow). There is mild thickening of the nearby hepatic flexure of the colon (arrowhead). *Diagnosis:* Pneumoperitoneum. Inflammatory changes in the right upper quadrant and perihepatic fluid, suggestive of a hollow viscus perforation

fluid throughout the abdomen. The findings were thought to be due to (initially missed) colonic injury from displaced rib fracture in the fall. The patient underwent a right hemicolectomy and diverting loop ileostomy that was subsequently reversed 3 months later.

8.1 Learning Points: Pneumoperitoneum

- Although free intra-abdominal air is an abdominal finding, question writers love to show subdiaphragmatic air on chest radiographs. Always look for free air on every chest radiograph with a patient history that supports bowel injury, bowel ischemia, or ulcer perforation.
- Subdiaphragmatic free air is only reliably seen on radiographs taken in the upright position, not supine. In situations where a patient cannot sit up, a cross-table lateral radiograph of the abdomen can be used.
- In cases of trauma with lower rib fractures, workup to exclude intra-abdominal injury should be performed.

9 Abdominal Cases 3 and 4

Patient 1 A 64-year-old woman with history of polysubstance abuse was found down with multiple unidentified bottles at her side. She was intubated in the field and brought into the emergency department. Serum laboratory studies showed creatinine 1.9 mg/dL (baseline 0.9 mg/dL), lactic acid 12 mmol/L, and WBC 26,700/μL. CT of the abdomen and pelvis was performed (Fig. 18).

Given extensive bowel ischemia, surgical intervention was thought to be futile. The patient expired 1 day later. Pneumatosis may be a harbinger of catastrophic bowel ischemia, but benign pneumatosis may also be seen with COPD or steroid use (where it may be meaningless).

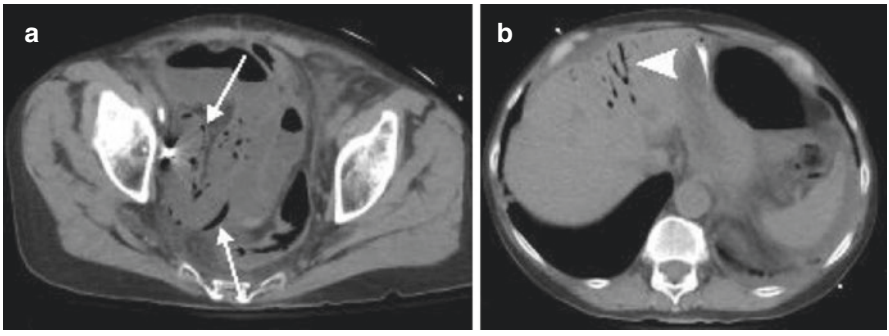

Fig. 18 *Technique:* CT of abdomen and pelvis without intravenous contrast. Axial images. *Findings:* (**a**) There is ileocolic pneumatosis intestinalis (arrows). (**b**) Portal venous gas is present (arrowhead). *Diagnosis:* Bowel ischemia in the setting of global hypoperfusion

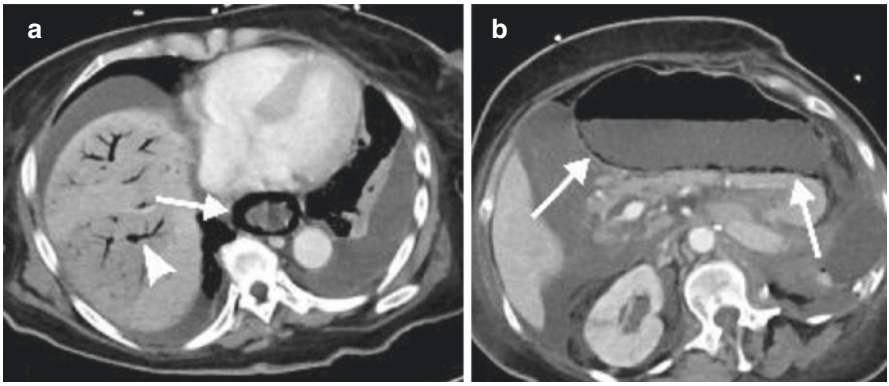

Fig. 19 *Technique:* CT of abdomen and pelvis with intravenous contrast. Axial images. *Findings:* Pneumatosis intestinalis is present in the walls of the esophagus (**a**, arrow) and stomach (**b**, arrows). Portal venous gas is present (arrowhead). *Diagnosis:* Esophagogastric ischemia

Patient 2 A 66-year-old woman underwent a left nephrectomy for a renal sarcoma. Postoperative course was complicated by intra-abdominal infection leading to septic shock. CT of the abdomen and pelvis was performed (Fig. 19).

The patient gradually improved during her 6-week stay in the surgical intensive care unit.

9.1 Learning Points: Pneumatosis Intestinalis

- On test questions, if there appears to be gas inside the bowel wall, there is pneumatosis intestinalis. In real life, there can often be (intraluminal) gas trapped in between stool and the bowel wall.
- Not all pneumatosis intestinalis is due to bowel ischemia. Other relatively common causes include drugs (chemotherapy, steroids), instrumentation (surgery, endoscopy), COPD, and mechanical ventilation.
- The hepatic gas pattern is *p*eripheral with *p*ortal venous gas and *c*entral with *c*ommon bile duct gas (pneumobilia). Bile duct gas can be from ERCP, choledocho-duodenal anastomosis, sphincterotomy, or biliary infection. Think *E. coli* emphysematous cholecystitis in diabetics or vasculopathy with gas in the gallbladder or gallbladder wall.

10 Abdominal Cases 5, 6, and 7

Patient 1 A 28-year-old woman presented with several hours of nausea, vomiting, diarrhea, and abdominal pain that began periumbilical and subsequently localized to the right lower quadrant. She had a low-grade fever. Physical exam showed right

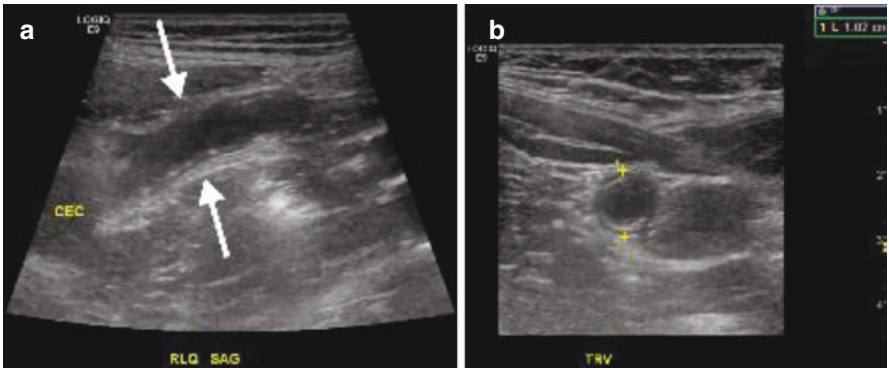

Fig. 20 *Technique:* Right lower quadrant ultrasound. Sagittal (**a**) and transverse (**b**) B-mode images. *Findings:* In the right lower quadrant, there is a thick-walled, dilated (> 6 mm), non-compressible, blind-ending tubular structure (arrows). (Cecum labeled CEC). No fluid collection. *Diagnosis:* Acute appendicitis, uncomplicated

lower quadrant tenderness with guarding. Laboratory studies were significant for leukocytosis. Ultrasound of the right lower quadrant of the abdomen was performed (Fig. 20).

The patient underwent a laparoscopic appendectomy, which confirmed acute uncomplicated appendicitis.

Patient 2 A 63-year-old man presented with 3 hours of right abdominal pain radiating to the right flank. He was afebrile. Laboratory studies showed a mild leukocytosis (WBC 10,130/μL). CT of the abdomen and pelvis was performed (Fig. 21).

The patient underwent a laparoscopic appendectomy. The surgeons saw a small amount of purulent fluid adjacent to the inflamed appendix, but there was no clear perforation.

Patient 3 A 60-year-old woman presented with 2 days of periumbilical pain radiating to the left lower quadrant, as well as low-grade fever and anorexia. She noted significant pain during her ambulance ride to the hospital secondary to potholes. Laboratory studies were significant for WBC 12,700/μL. CT of the abdomen and pelvis was performed (Fig. 22).

The patient was admitted and treated with antibiotics. CT was repeated 3 days later to assess interval change (Fig. 23).

The patient underwent CT-guided percutaneous drainage of the periappendiceal abscess (Fig. 24).

After a few days of observation, the patient was discharged from the hospital with the drainage catheter in place. At 14 days after catheter placement, there was no further drainage, and the patient was feeling well. The catheter was removed. Six weeks later, the patient underwent an elective laparoscopic appendectomy.

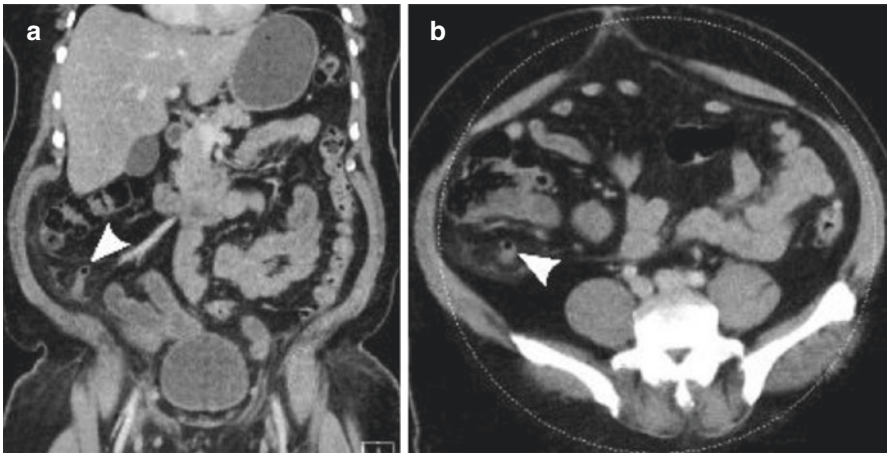

Fig. 21 *Technique:* CT of abdomen and pelvis with intravenous contrast. Coronal (**a**) and axial (**b**) images. *Findings:* The appendix (arrowheads) is dilated with surrounding fat stranding. No fecaliths. No fluid collections. *Diagnosis:* Acute appendicitis, uncomplicated

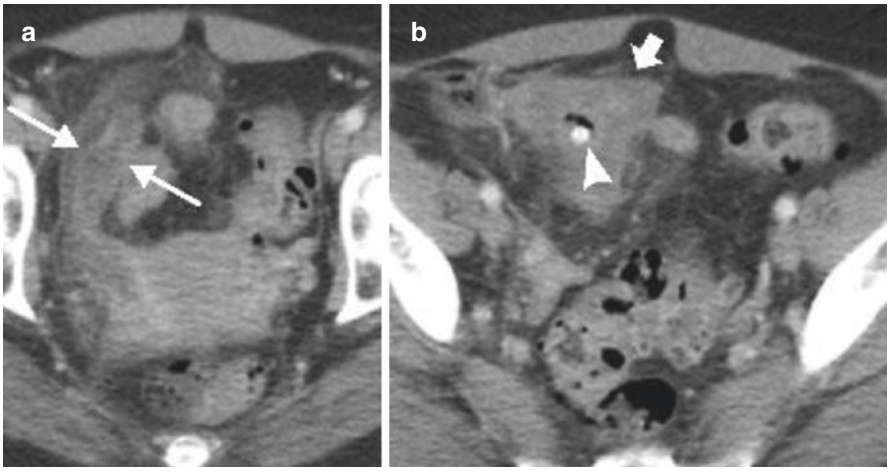

Fig. 22 *Technique:* CT of abdomen and pelvis with intravenous contrast. Axial images. *Findings:* (**a**) The appendix is markedly dilated and demonstrates thickened, hyperenhancing walls (arrows). (**b**) The tip of the appendix is not seen within the surrounding ill-defined fluid (block arrow). At the base of the appendix, an appendicolith is present (arrowhead). *Diagnosis:* Appendicitis with contained perforation and phlegmon

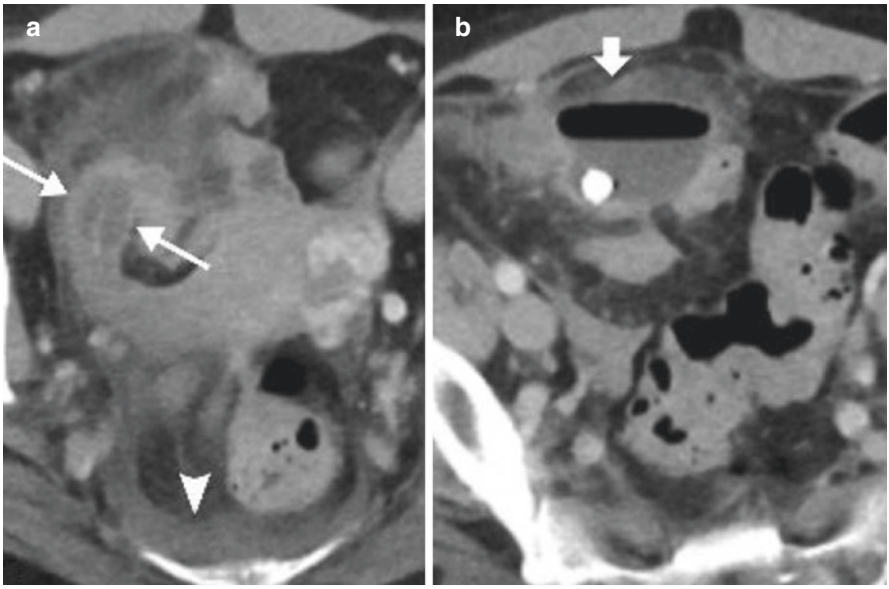

Fig. 23 *Technique:* CT of abdomen and pelvis with intravenous contrast. *Findings:* (**a**) The appendix remains markedly dilated (arrows). Trace free fluid is present in the pelvis (arrowhead). (**b**) There is a focal appendiceal perforation into a rim-enhancing fluid collection (block arrow) that contains the appendicolith and air. *Diagnosis:* Perforated appendicitis with abscess

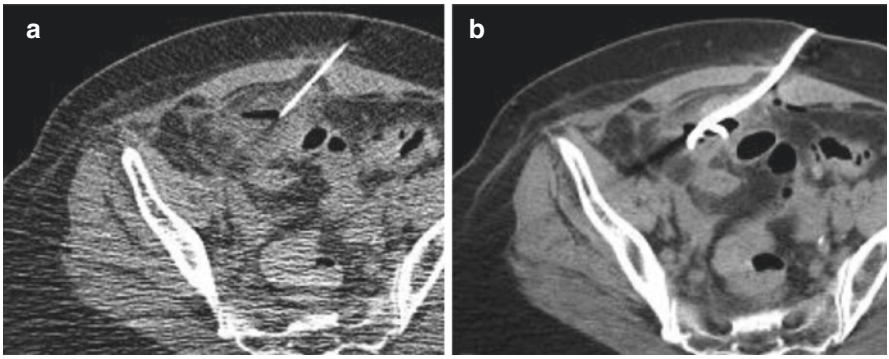

Fig. 24 *Technique:* Intra-procedure CT guidance. Axial images. *Procedure summary:* (**a**) A small centesis catheter with integrated needle was advanced into the abscess (direct or "trocar" technique) under intermittent CT guidance. The inner needle was removed. 1 mL of thick purulent fluid was aspirated through the centesis catheter and sent for microbiology studies. (**b**) The centesis catheter was exchanged over a wire for a larger drainage catheter, which was looped into the abscess. The catheter was then connected to a drainage bag for gravity drainage. (as an alternative to "trocar" technique, one may also use "Seldinger" technique: [wire through a needle and then catheter over wire] with sequential dilation over a wire, for drain placement)

10.1 Learning Points: Appendicitis

- Anorexia and epigastric pain later migrating to the right lower quadrant with an acute abdomen may warrant an ultrasound or an abdominopelvic CT in an adult to look for an inflamed or ruptured appendix.
- Common CT and ultrasound findings of acute appendicitis include
  - Dilated appendix (> 6 mm in diameter)
  - Appendiceal wall thickening (>3 mm)
  - Stranding of surrounding fat, best appreciated on CT, as evidence of periappendiceal inflammation
  - Appendicolith is occasionally present
  - Evidence of perforation: extraluminal air, surrounding fluid, phlegmon, and/or abscess
- Acute appendicitis may be *uncomplicated* (without perforation) or *complicated* (with perforation).
- For purposes of the boards, appendectomy is the treatment of choice for acute uncomplicated appendicitis. However, know that management of acute appendicitis is evolving, with a greater role for antibiotics as the initial therapy.
- Periappendiceal phlegmon is unorganized inflammatory material without drainable fluid. Phlegmon may resolve with antibiotic therapy or progress to abscess, an organized collection of inflammatory material/pus.
- Perforated appendicitis with periappendiceal abscess may be treated with IR percutaneous abscess drainage followed by interval appendectomy once appendiceal inflammation has decreased.

11 Abdominal Case 8

A 56-year-old man with several months of intermittent self-resolving epigastric pains presented with several hours of unremitting epigastric pain and nausea. Physical exam revealed only mild epigastric tenderness to palpation with a negative Murphy's sign. Laboratory studies demonstrated mild leukocytosis (WBC 10,900/μL) with left shift and normal liver function tests. Ultrasound of the right upper quadrant of the abdomen was performed (Fig. 25).

The patient underwent a laparoscopic cholecystectomy. Histology confirmed chronic necrotizing cholecystitis with cholelithiasis.

11.1 Learning Points: Acute Calculous Cholecystitis

- Right upper quadrant ultrasound is the initial imaging modality of choice to diagnose acute cholecystitis due to its high sensitivity, accessibility, lack of ionizing radiation, and ability to elicit the sonographic Murphy's sign [5]. Sonographic findings include
  - Gallstone
  - Sonographic Murphy's sign

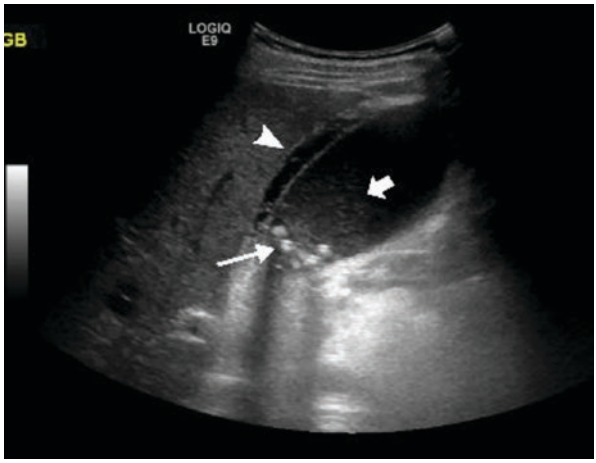

Fig. 25 *Technique:* Right upper quadrant ultrasound. B-mode image. *Findings:* Several echogenic foci with posterior acoustic shadowing are present (arrow), representing gallstones. Faint echogenicity is present in the gallbladder, representing biliary sludge (block arrow). Pericholecystic fluid is present (arrowhead). The sonographic Murphy's sign was positive. *Diagnosis:* Acute calculous cholecystitis

  - Gallbladder wall thickening (>3 mm)
  - Pericholecystic fluid
  - Gallbladder distension (less specific)
- CT is useful for evaluation of other potential causes of abdominal pain.
- HIDA cholescintigraphy is helpful in evaluating cases that are equivocal on ultrasound and CT.
- Cholecystitis without stones ("acalculous cholecystitis") may be seen in patients with vasculopathy, ICU, trauma, or burns.

12 Obstetric Case

A 37-year-old woman, G6P3023, at 7 weeks of gestation by last menstrual period and positive pregnancy test, presented with vaginal bleeding. Ultrasound of the pelvis was performed (Fig. 26).

The patient was taken to the operating room, where the right tubal ectopic pregnancy was confirmed. She underwent laparoscopic right salpingectomy.

12.1 Learning Points: Ectopic Pregnancy

- On ultrasound, the most important findings of ectopic pregnancy include the following:
  - No intrauterine pregnancy (the extremely rare heterotopic pregnancy, simultaneous intrauterine and extrauterine pregnancies, is an exception)
  - Adnexal or extra-adnexal mass with a solid ring of echogenic tissue that is highly vascular on color Doppler (*ring of fire* sign (Fig. 27), which is also seen in corpus luteum)
  - Live extrauterine pregnancy (as in this case)
- Free pelvic fluid suggests but is not specific for ruptured ectopic pregnancy.

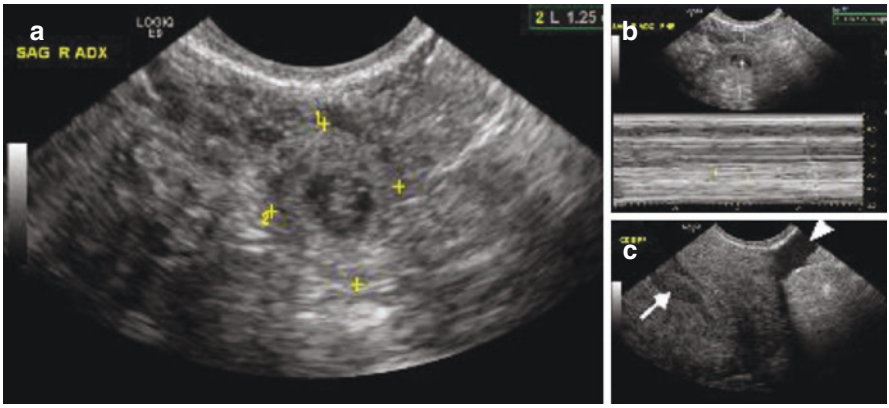

Fig. 26 *Technique:* Transvaginal ultrasound. B-mode images (**a**, **c**). M-mode image (**b**). *Findings:* No intrauterine pregnancy is seen (**c**, arrow). There is a gestational sac and fetal pole in the right adnexa surrounded by a solid ring of echogenic tissue (**a**). The fetal heart rate was normal at 138 beats per minute (**b**). Free fluid is present in the cul de sac (**c**, arrowhead). *Diagnosis:* Live right adnexal ectopic pregnancy with hemoperitoneum

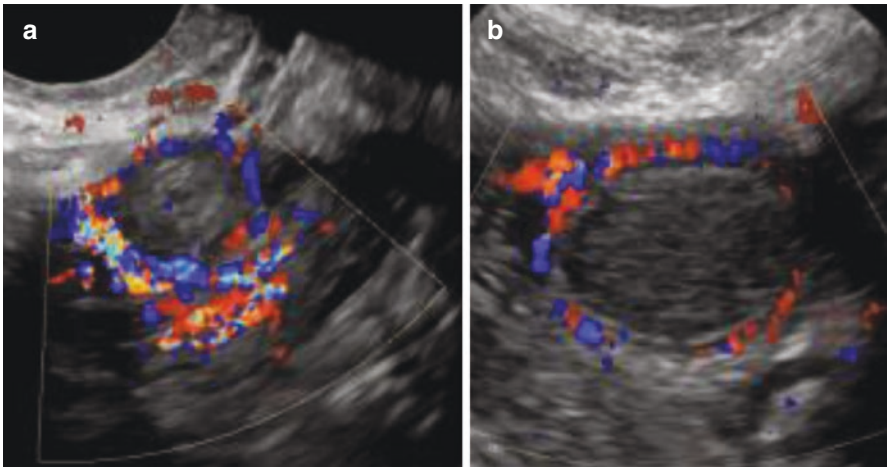

Fig. 27 Transvaginal color Doppler ultrasound images of the "ring of fire" sign seen in both (**a**) tubal ectopic pregnancy and (**b**) corpus luteum cyst. The two entities can be virtually indistinguishable on static images. However, note that a tubal ectopic pregnancy is outside the ovary, whereas a corpus luteum cyst is within the ovary. During an ultrasound examination, the sonographer would apply pressure with the transvaginal probe to see if the mass in question separates from the ovary (indicative of a tubal ectopic pregnancy) or is inseparable from the ovary (almost certainly indicative of a corpus luteum cyst). An ovarian ectopic pregnancy, although theoretically possible, is exceedingly rare

- The fallopian tube is the most common location for an ectopic pregnancy (tubal ectopic), which shows an adnexal mass separate from the ovary with a tubal ring sign.
- Mimic: corpus luteum, which has the following features:

- Within the confines of the ovary, rather than outside the ovary such as in a fallopian tube
- *Ring of fire* sign also seen (Fig. 27)

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Musculoskeletal and Bone Imaging

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Contents

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Acronyms

ABC	Aneurysmal bone cysts
AC	Appropriateness criteria
ACR	American College of Radiology
AP	Antero-posterior
CT	Computed tomography
DDH	Developmental dysplasia of hip
HIV	Human immunodeficiency virus
LL	Latero-lateral
MOF	Multiorgan failure
MPR	Multiplanar reformation
MRI	Magnetic resonance imaging
MSK	Musculoskeletal
NF	Necrotizing fasciitis
NOF	Non-ossifying fibroma
PA	Psoriatic arthritis
PD	Proton density
PM	Pyomyositis
RA	Rheumatoid arthritis
ROM	Range of motion
SLE	Systemic lupus erythematous
STIR	Short-tau inversion recovery
T	Tesla
TC-99m MDP	Technetium-99m methylene diphosphonate
US	Ultrasound
VR	Virtual reality
X-ray	Radiography
ZOT	Zone of transition
2D	Two-dimensional
3D	Three-dimensional

1 Introduction

Imaging allows us to analyze anatomy, detect pathology and monitor disease healing or progression. In the musculoskeletal system, imaging is invaluable to detect injuries and other pathologies.

Imaging interpretation requires experience, consistency, search patterns and checklists specific both to the anatomic area imaged and to the potential diagnostic/clinical concern. It is fundamental to "*describe first, diagnose second*." Precise verbal capture of the visual findings is necessary.

2 MSK Imaging Modalities

2.1 Radiography (x-ray)

X-ray is the first line in imaging technology, because it is highly specific and widely available. Radiography is a projection imaging using x-rays (ionizing radiation) and has a basic role in the diagnosis of many disorders. In radiography, a beam of x-rays, produced by an x-ray generator, is transmitted through an object, e.g., the part of the body to be scanned (Fig. 1). The x-rays are absorbed by the material they pass through in differing amounts depending on the density and composition of the material. X-rays that are not absorbed pass through the object and are recorded as a shadow on x-ray-sensitive detectors (or film).

X-rays are inexpensive and quick, although this depends on the number of parts of the body being examined and the general mobility of the patient.

In MSK, x-rays provide us optimal contrast between bone and "non-bone" structures. Plain x-ray (radiography) brings excellent depiction of bone details and anatomic relationships (e.g., joints) but poor depiction of soft tissues, because these appear as shades of grey that are difficult to interpret.

Different body areas need to be viewed from different directions to obtain enough information to make the diagnosis. This might require different scanning positions for each exam to better display the three-dimensional (3D) effect of the skeletal anatomy (Fig. 2). For joints like the pelvis, ankle, foot, elbow or wrist, we always take three standard views: antero-posterior (AP), latero-lateral (LL), and oblique. In children, obtaining a radiograph of a normal and affected limb for comparison is also wise. Furthermore, it is very important to provide an age-appropriate differential diagnosis for MSK disorders. Suspect abuse in the event of multiple fractures of varying ages (a sad but very common board question).

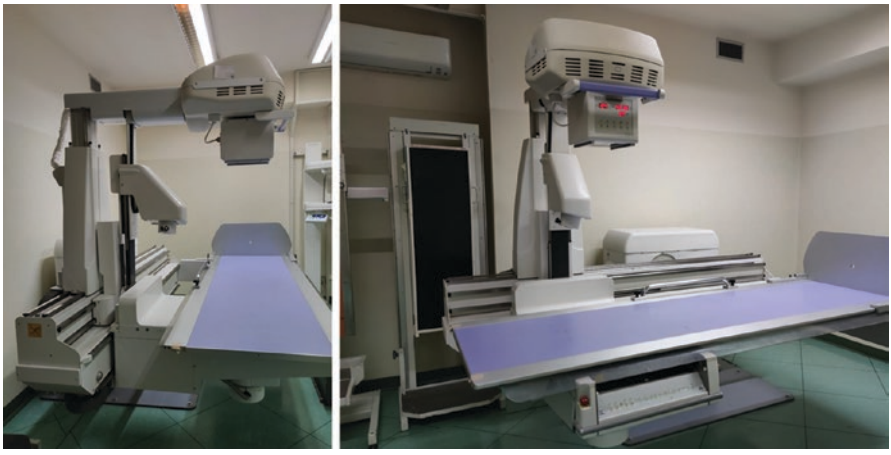


Fig. 1 X-ray generator and a detector used for acquisition of projectional radiography

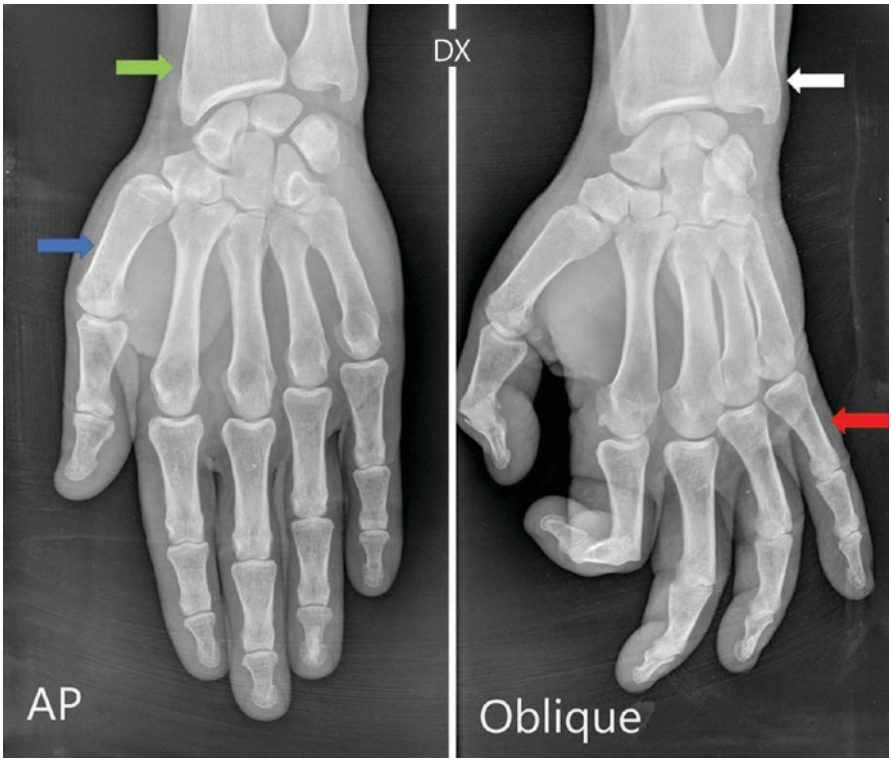


Fig. 2 AP and oblique view of right hand showing natural anatomical position of metacarpals (blue arrow), phalanges (red arrow), radius (green arrow), and ulna (white arrow)

In terms of radiation protection, it is important to cover as much anatomy as possible with the fewest number of films possible. X-rays are two-dimensional and can miss pathology; therefore, the correct diagnosis of MSK disorders may require other imaging studies.

Examples when plain radiography may be the initial study of choice:

- Fractures
- Some tendon and cartilage injury
- Degenerative joint disease – osteoarthritis
- Initial evaluation of bone lesions

2.2 Computed Tomography (CT)

Like x-rays, CT scan uses ionizing radiation. Main components of CT are x-ray source, detectors, and workstation or computer image processing system (Fig. 3). As the x-ray source and detector spin around the patient, a fast series of x-ray

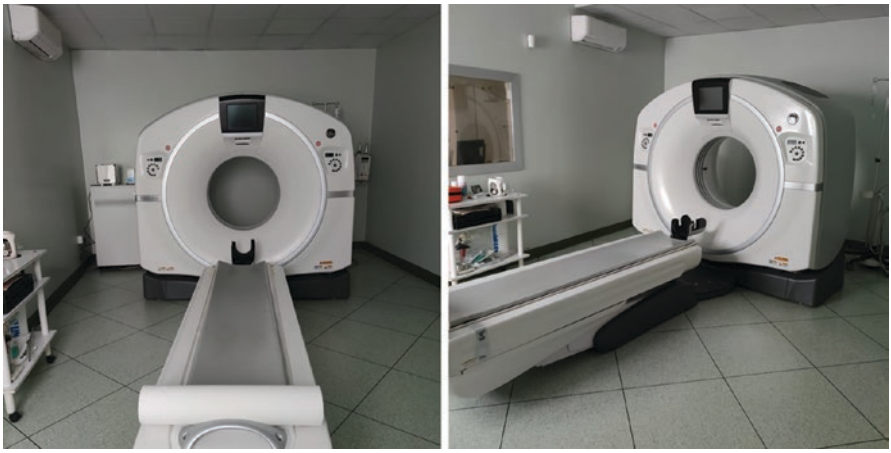


Fig. 3 Modern CT scanner

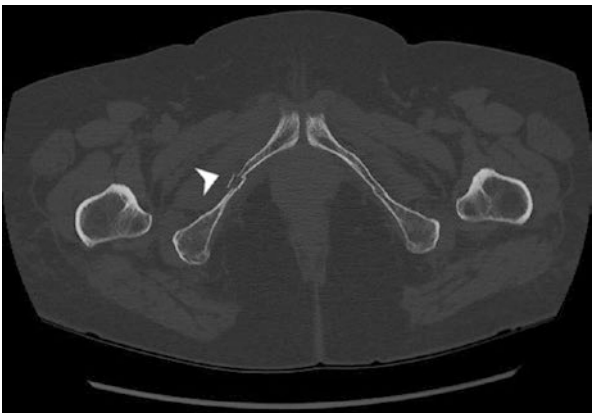


Fig. 4 CT scan image of 70-year-old female after high-energy trauma of the pelvis. Arrowhead showing superior right pubic ramus fracture. In order to accurately assess the complexity of the fracture, CT was the modality of choice

pictures are combined to create cross-sectional images of soft tissues and bones in the area that is scanned.

CT provides a more detailed radiographic picture of the anatomic area and detects almost all pathology related to cortical bone injury (e.g., characterizing fractures and detecting complex fractures) and is great for showing displacement or joint involvement (Fig. 4).

The possibility to convert 3D data from an imaging modality acquired or displayed in a certain plane into another plain is called multiplanar reconstruction or reformation ("MPR"), and has become an integral part of CT examination (Fig. 5). This technology provides us not only two-dimensional (2D) MPR imaging but also the possibility to create three-dimensional (3D) reconstruction images, or virtual reality (VR) (Fig. 6), which have the potential to facilitate the assessment of a variety of disorders, especially for trauma surgeons.

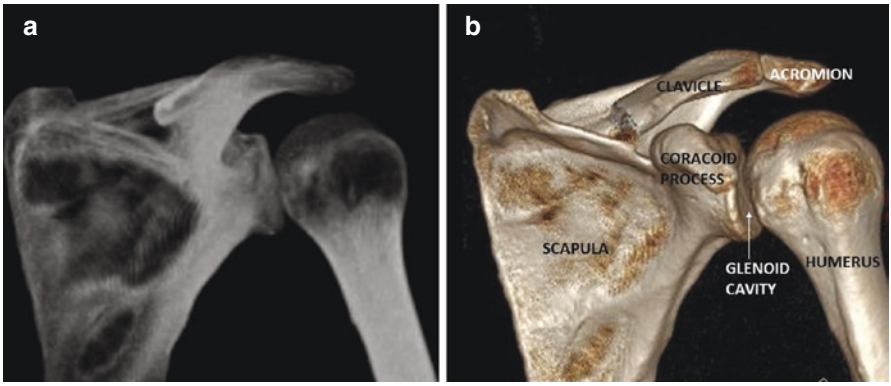


Fig. 5 CT scan (**a**) Maximum Intensity Projection (MIP) and (**b**) Virtual Reality (VR) images depicting anterior view of the left shoulder with normal bone anatomy

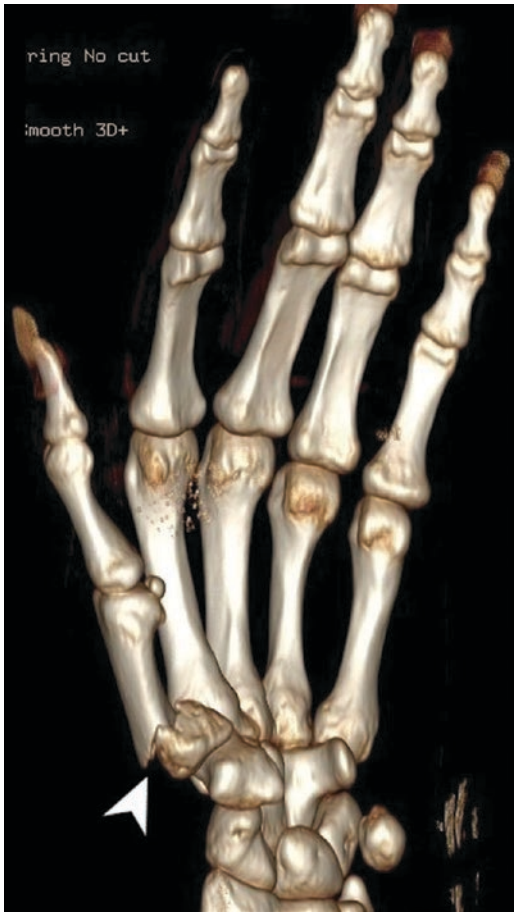


Fig. 6 CT scan in 20-year-old patient with acute hand trauma. VR image shows epibasal fracture of the left thumb (pseudo-Bennett fracture) with two-piece fractures of the proximal first metacarpal bone (arrowhead). This type of fracture is usually stable, depending on the degree of displacement. CT scan was performed to exclude unstable intra-articular type of fracture (Rolando fracture or Bennet fracture) which usually requires surgery

CT scans are costly, require larger radiation doses than traditional x-rays, and might often be unnecessary. It is not the primary imaging modality in the MSK imaging chain and is often used as a follow-up to an abnormal x-ray or is requested when radiographs fail to completely answer the clinical question, e.g., high clinical index of suspicion for a fracture, but no clear x-ray alterations are found on radiography. Overall, due to higher radiation exposure, CT scans require a more careful risk vs. benefit evaluation than conventional radiographs.

A few examples of when *CT* scan is the study of choice:

- Calcaneal fractures
- Fracture severity for surgical planning (e.g., depressed tibial plateau)
- Cervical spine fracture suspicion (time sensitive)
- If a patient can't have magnetic resonance imaging (MRI) (pacemaker, metal artifact)
- Sternoclavicular joint
- CT myelogram in spine (vs MRI)
- CT with 3D reconstruction for complex anatomy

2.3 Magnetic Resonance Imaging (MRI)

MRI is particularly well suited for the evaluation of the MSK system. It is the "gold standard" of soft tissue imaging and has the added advantage of requiring no ionizing radiation.

MRI scanners apply strong magnetic fields, magnetic field gradients, and radio waves to generate excellent contrast for detailed pictures of joints, soft tissues, and bones. MRI of the MSK system is done mostly at intermediate field strengths of 1.5T or lower, although lately clinical practice has moved towards 3.0T imaging for mostly evolutionary, not revolutionary reasons (Fig. 7a, b). Low-field office-based mini-MRI systems have recently proliferated (mainly for knees and elbows).

Contrary to CT scans that can obtain only axial images for most of the body areas (MPR images in other planes are elaborated in post-processing), MRI scanners can provide direct 3D multiplanar acquisition, e.g., direct arbitrary coronal or sagittal scans are acquired, being able to visualize anatomy in all three planes directly during the acquisition. MRI is a modality of choice when it comes to soft tissue evaluation, e.g., muscles, ligaments, tendons, and cartilage, as well as bone marrow evaluation.

MRI scanning protocols for MSK includes different types of pulse sequences, mainly T1-weighted sequences, and fluid-sensitive sequences (e.g., proton density (PD) fat-saturated or T2-weighted fat-saturated). The broad difference between these two types of sequences is that in fluid-sensitive sequences (T**2** = H**2**O), a lot of pathologies can be detected (e.g., effusions, soft tissue edema, hematoma), whereas in T1-weighted sequences, we can see better the anatomy, in particular, compartmentalization of structures.

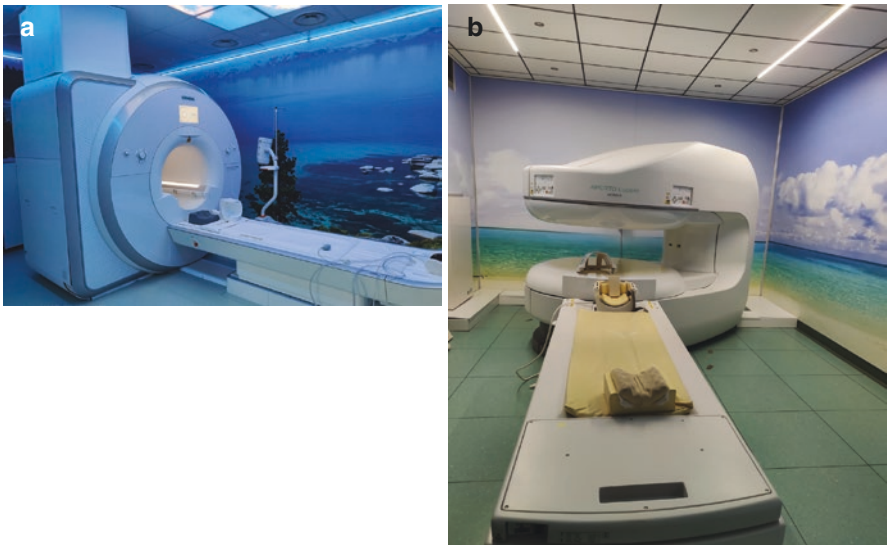


Fig. 7 (**a**) High-field superconducting MRI scanner (1.5 Tesla) with the closed configuration. The system includes a large magnet and radio wave that sends signals and receives signals to and from the patients' body. A computer that's attached to the scanner converts the returning signals into images. (**b**) Low-field open MRI scanner (less than 0.5 Tesla); these use a magnetic bottom and top and all four sides are open, reducing the risk of panic attacks and claustrophobia exponentially

The disadvantages of MRI are the cost, incomplete availability, claustrophobia for some patients, very high sensitivity to motion artifacts and low sensitivity for cortical bone evaluation.

MRI safety considerations [1]:

- Presence of metal fragments (bullets, shrapnel, shavings, etc.) ⟹ screen with x-ray first.
- Contraindication if metal objects are near vessels, nerves, eyes or bowels; no contraindication in bone but creates artifacts.
- Implantable cardioversion defibrillator, pacemaker, aneurysm clips, and pain pumps ⟹ consult with manufacturer, or look up tables. In about 2011, pacemakers began to be made that can be adjusted for MRI, with monitoring by the cardiologist.

Magnetic resonance arthrography of the joint is obtained with intra-articular injection of diluted gadolinium chelates and is the preferred imaging technique for the evaluation of the labroligamentous complex [2–4], articular cartilage [4], the intra-articular portion of tendons [2, 5], and postoperative joints. Introduction of the contrast media into the joint can be palpation-directed, ultrasound-guided (US-guided) or CT-guided [6]. MR arthrography is mainly used to detect cartilage lesions in the ankle joint [7], shoulder, knee, and elbow, although virtually any joint can be injected with contrast.

A few examples when *MRI* scan is the study of choice:

- Soft tissue tears and strains, sprains
- Missed fracture on x-ray including stress, insufficiency or acute fracture
- Tumor (benign or malignant)
- Infection

2.4 Ultrasound (US)

Ultrasound (US) uses sound waves to provide real-time imaging of anatomic structures. Ultrasound machines produce sound waves with frequencies which are higher than those audible to humans (>20,000 Hz). The ultrasound machine transmits high-frequency sound pulses into your body using a probe. The sound waves travel into your body and hit a boundary between tissues (e.g., between fluid and soft tissue, soft tissue and bone). Some of the sound waves get reflected back to the probe, while some travel on further until they reach another boundary and get reflected. The reflected waves are picked up by the probe and relayed to the machine. The machine then displays the distances and intensities of the echoes on the screen, forming a two-dimensional image.

It is commonly available, and free-hand imaging is low cost and fast and offers the advantages of real-time imaging without any ionizing radiation risk, and the US machine can be also portable, wheeled or even pocket-sized and wireless (Fig. 8) or connected to a smartphone or tablet.

Fig. 8 Portable ultrasound machines with the most common types of transducer probes, linear (white arrow), and convex (blue arrow)

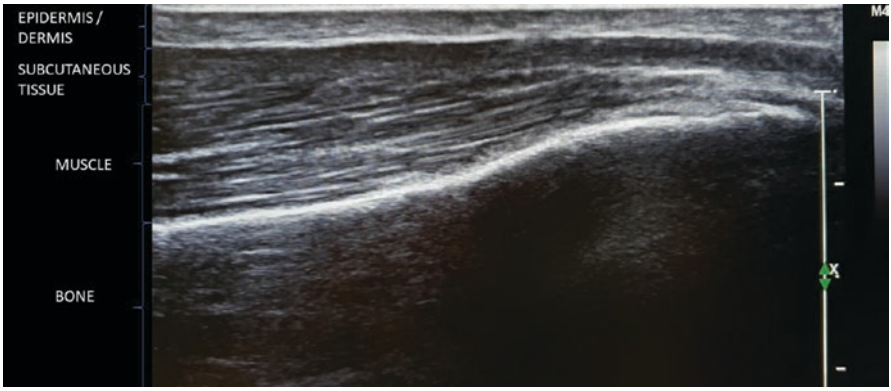


Fig. 9 Normal ultrasound appearance of soleus muscle. The ultrasound scan longitudinal to the muscle shows normal, hypoechoic, parallel fibrillar pattern

US is a highly effective primary diagnostic tool in traumatic, inflammatory, and degenerative soft tissue conditions, in particular for joints, ligaments, cartilage, and muscles (Fig. 9). In the skeletal system, US has limited use in the evaluation of other skeletal structures, since US waves cannot penetrate the bone. Proper US examination and interpretation requires accurate knowledge of relevant anatomy, artifacts, and techniques.

MSK ultrasound requires high-quality equipment with dedicated transducers and full software options to improve image quality, i.e. excellent soft tissue resolution, contrast, and the best possible artifact removal [8–10].

The minimum features of an ultrasound machine for musculoskeletal diagnosis include:

– A linear broadband transducer with a frequency of 5 to a minimum of 13 MHz with ultrasonic focusing at a depth of no more than 5 mm.
– Convex transducer with a frequency of about 2–6 MHz (for imaging obese patients or examining deep structures)
– Harmonic imaging
– Spatial compound imaging
– Doppler options: color, power, and tissue (microcirculation) imaging [10]

US is also very useful for image-guided interventional procedures (e.g., soft tissue lesion biopsies, ganglion or cyst aspiration, joint injections).

The disadvantage of US is that it is operator-dependent and results vary consistently with expertise of technologist and radiologist, as compared to CT and MRI.

Few examples when *US* is the study of choice:

- Pediatric patients' work-up (e.g., DDH - Developmental Dysplasia of Hip, joint effusions, trauma transient synovitis, osteomyelitis, foreign bodies, abscess, vascular malformation/lymphangioma)

- Initial evaluation of soft tissue masses
- Vascular disease
- Ligament/tendon/muscle injuries
- Articular disorders (cartilage, effusion, foreign bodies)
- Image-guided interventions

2.5 Nuclear Medicine (Bone Scan)

Bone scintigraphy uses ionizing radiation and is a highly sensitive method for demonstrating bone disease. Bone scan is used to detect bones undergoing rapid metabolic activity. After the venous injection of radioactive bone scan tracer that emits gamma rays (e.g., TC-99m MDP), the gamma detector (itself does not emit radiation) reveals the emitted radiation from sites with increased radioactive tracer uptake (so-called hot spots). It can detect occult fractures, infections, and bony metastases, often providing earlier diagnosis or demonstrating more lesions than are found by conventional radiographs, because it is highly sensitive. The disadvantages are low specificity, poor anatomic detail, limited availability, and moderate radiation risk (more than plain films but less than CT), depending on the type of radioactive tracer used.

Nuclear medicine procedures of any sort should be avoided in pregnant women. A relative contraindication to bone scan is breastfeeding; thus, women are asked not to nurse for at least 24 hours following the test (traces of TC-99m MDP in breastmilk).

Few examples when *bone scintigraphy* may be the study of choice (MRI may be indicated for some of these as well or instead):

- Stress fracture
- Differentiation of osteomyelitis from cellulitis
- Avascular necrosis or bone infarction
- Reflex sympathetic dystrophy
- Peripheral vascular disease
- Subtle lumbar lesions such as pars defects

The American College of Radiology (ACR) Appropriateness Criteria® (AC) are evidence-based guidelines widely used to assist referring physicians in making the most appropriate imaging and/or treatment decision for a specific clinical condition. Applying these guidelines helps providers increase quality of care and contribute to the most efficacious use of radiology (Table 1).

Guidelines for types of studies to be ordered for different indications https://www.acr.org/QualitySafety/Appropriateness-Criteria.

Table 1 Principal characteristics of MSK imaging modalities

	X-ray	CT	US	MRI	Bone scan
Energy	X-ray generator	X-ray generator	Ultrasound	Magnetic fields Radio waves	X-ray generator Radioactive tracer
Bone sensitivity	High	High	Low	High – bone marrow Low – cortical bone	Low
Soft tissue sensitivity	Low	Intermediate	High	Excellent	Low
Acquisition time	Short	Short	Real-time	Long	Long
Multi-planarity	No	Yes 3D with post-processing reformation	No	Yes Direct multiplanar acquisition	No
Availability	Wide	Wide	Wide	Low	Low
Cost	Low	High	Low	High	High
Contraindication	Pregnancy	Contrast media Pregnancy	None	Metal or electronic devices (e.g., pacemaker) Pregnancy Contrast media	Pregnancy Breastfeeding

3 MSK Anatomy Basics

3.1 Bones and Bone Marrow

Within the human body, there are long bones (appendicular skeleton), short bones, flat bones (sternum and ribs), sesamoid bones (within a tendon, e.g., patella), and irregular bones (e.g., vertebra).

Parts of the long bone are diaphysis, metaphysis, and epiphysis (Fig. 10). The long bone in a child is divided into four regions, the three aforementioned plus one called growth plate, an area which is almost completely replaced by bone at skeletal maturity (Fig. 11). Each bone has its cortex and medullary cavity. Bone marrow is a soft tissue that fills the medullary cavities. There are two types of bone marrow:

- *Red bone marrow*, or myeloid tissue, hematopoietically active due to hematopoietic cellular elements (red and white cells and platelets).
- *Yellow bone marrow*, or fatty tissue, hematopoietically inactive marrow, contains fat cells and increases with age.

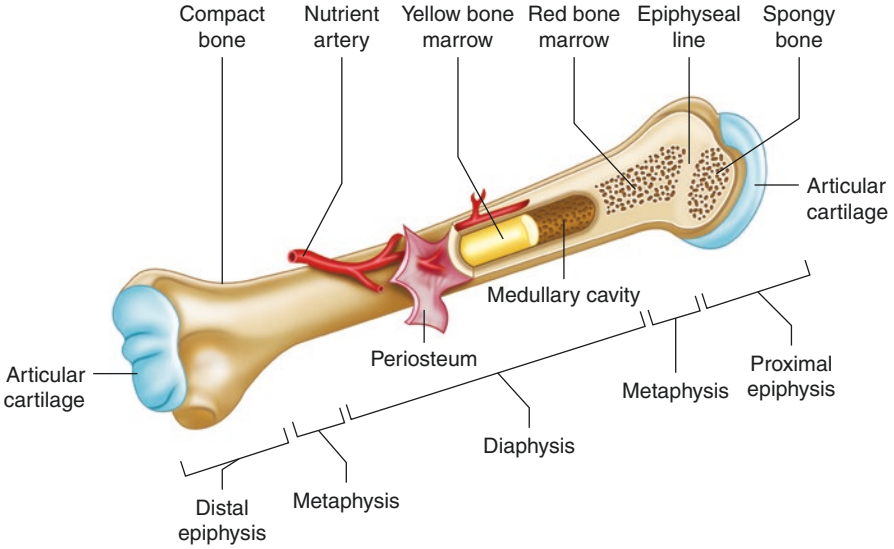


Fig. 10 Gross anatomical characteristics of a long bone. All of the bones in the arms and legs, except the patella, and bones of the wrist, and ankle, are long bones

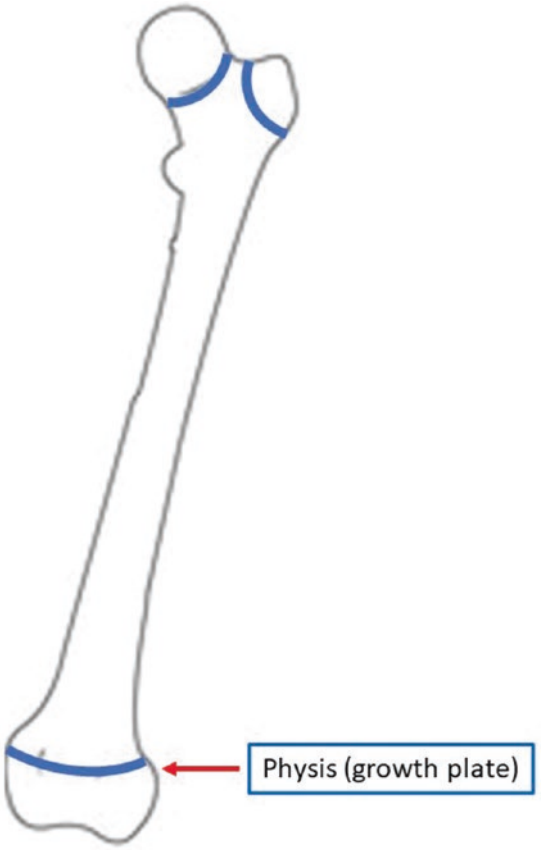


Fig. 11 Image of femur. In kids and adolescents, there are areas of growing tissue near the ends of the long bones called growth plates (blue lines) also known as physis or epiphyseal plates. These are the weakest part of the pediatric growing skeleton

Trabeculae serve as the architectural support for the marrow and as a mineral depot. Their number decreases with age [11].

3.2 Muscles and Tendons

There are approximately 700 muscles in the human body, divided into three main types: visceral or "smooth muscle" (for its smooth and uniform microscopic appearance, e.g., stomach and intestine wall and blood vessels), cardiac muscle (found only in heart), and skeletal muscle. Skeletal muscles are attached through tendons to the bones of the skeletal system and are the only voluntary muscle tissue in the human body (controlled consciously). It is very important to know the proper anatomy (location, origin, number of origins, insertion, shape, size, and direction) as well as function in meticulous search patterns when interpreting imaging of MSK disorders.

Tendons are stiff bands of solid connective tissue whose strong collagen fibers are rigidly attached and incorporated into the coverings of both muscles and bones.

3.3 Ligaments

A ligament is the fibrous connective tissue that connects bones to other bones. Their main function is to restrict joint motion and stabilize joints. Furthermore, ligaments have mechanoreceptors and free nerve endings that help with joint proprioception. Compared to tendons, ligaments carry only lower loads and recruit fibers gradually, because they do not plastically deform.

3.4 Cartilage

Cells called chondrocytes are capable of producing collagenous extracellular matrix and ground substance to form cartilage.

Articular cartilage is a smooth, white tissue covering the extremities of bones where they come together to form joints, allowing joints to move and glide over each other with very little friction. Cartilage has limited reparative capacities due to very low blood supply. Furthermore, chondrocytes are bound in lacunae and cannot migrate to damaged areas, which are often replaced by fibrocartilage scar tissue with different biomechanical properties (e.g., in osteoarthrosis).

4 MSK Disorders

The choice of imaging technique is dictated by the type of suspected abnormality. There are several traumatic and non-traumatic MSK disorders.

Obtaining a careful history and physical examination is required to establish the pretest probability of each competing diagnosis for the musculoskeletal disorder. Accurately choosing the first-line radiological study depends on identifying the affected joint(s) and/or surrounding tissue.

4.1 Trauma

Musculoskeletal emergencies and trauma are a major public health problem globally, causing a large burden of disability and suffering. MSK injuries needing urgent care are among the most frequent conditions at first referral in health facilities.

Pediatric MSK injuries of the upper and lower extremities represent one of the most important challenges for physicians, particularly for the radiologist, since a missed diagnosis can lead to delayed complications such as arrested development and osteoarthritis. Correct interpretation of MSK imaging studies of children requires extensive knowledge of normal skeletal growth and the physiologic changes that take place in growing bones.

The spectrum of disorders in musculotendinous trauma includes acute traumatic and subacute/chronic lesions due to repeated microtrauma.

Traumas affect patients of both sexes, at any age, and represent some of the most common causes of visits to the emergency department. Upper and lower extremity injuries often present pitfalls and challenges for the radiologist in the process of diagnosis and guiding the referring clinician. Awareness of mechanics of the injuries as well as extensive knowledge of a broad array of conditions extending from fractures to ligament, tendon, and neurovascular injuries are required.

Accurate imaging evaluation is essential to classify the lesions and address the patient to the most appropriate therapeutic management.

4.1.1 Fractures

X-rays are indicated when fracture or joint dislocation is suspected (pain, tenderness, deformity, and loss of function). The x-ray should be centered on the area to be examined; especially in traumatic lesions, it is necessary to x-ray the whole bone to avoid missing bordering fractures/injuries. At least two views of the bone involved at 90° angles to each other should be obtained (Fig. 12).

Four types of fracture mechanisms can be differentiated (*DIPS*):

- *D*irect fracture (adequate mechanical outer force)
- *I*ndirect fracture (strain and pressure on the bone, commonly resulting from abnormal rotation or leverage force)
- *P*athologic or spontaneous (on pre-existing bony abnormality, e.g., lesion or tumor, in the absence of adequate trauma)
- *S*tress fracture (abnormal repetitive forces on a normal bone or normal repetitive stress on abnormal bone, e.g., osteopenia).

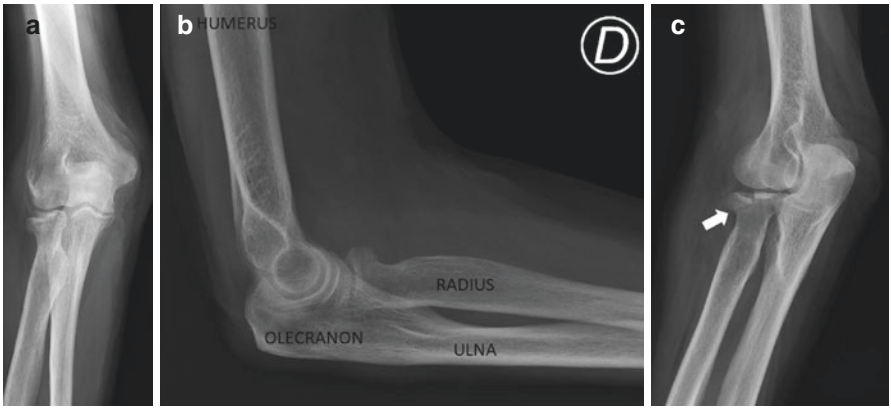


Fig. 12 X-ray AP (**a**), lateral (**b**), and oblique view (**c**) of the right elbow after traumatic injury. Comparing to other views in oblique view (**c**), the radial head fracture (white arrow) is best seen

Main fracture descriptors are the following:

- Open (the skin may be pierced by the bone)/closed (maintaining soft tissue and skin integrity)
- Type of fracture or break (stable, transverse, oblique, spiral, comminuted with three or more bone fragments, compression)
- Location within the bone (e.g., epiphyseal, intraarticular, metaphyseal)
- Displacement/axial malalignment, angulation
- Bone fragment orientation with various fragment dislocation

Eponymous fractures are fractures-dislocations often encountered in the emergency setting and are commonly denominated after the physician who first described them, or they may also be named after an activity with which they are correlated. Correct eponym usage allows rapid and concise communication of complex injuries to clinicians. We review most common extremity and spine fracture eponyms. We put focus on imaging features (radiographic findings and computed tomography images) to identify and differentiate these injuries.

1. Bankart's fracture is an injury of the anterior inferior glenoid labrum of the shoulder due to anterior shoulder dislocation. Mechanism of injury (MOI) - external rotation and abduction of shoulder (Fig. 13).
2. Barton's fracture is an intra-articular fracture of distal radius with dislocation (dorsal or ventral) of radio-carpal joint. MOI – fall on the outstretched hand (Fig. 14).
3. Bennett's fracture is an intra-articular fracture of the base of the first metacarpal bone with carpometacarpal joint involvement. MOI - axial load along metacarpal in partially flexed thumb (Fig. 15).

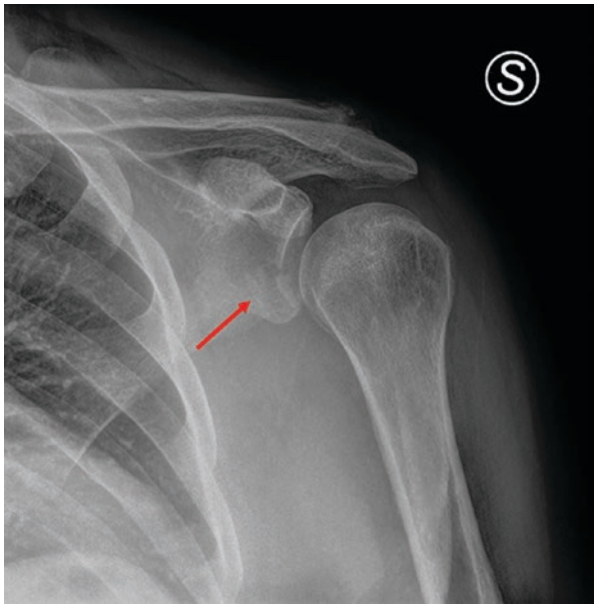


Fig. 13 X-ray anteroposterior (AP) projection showing a bony Bankart lesion (red arrow) after shoulder dislocation

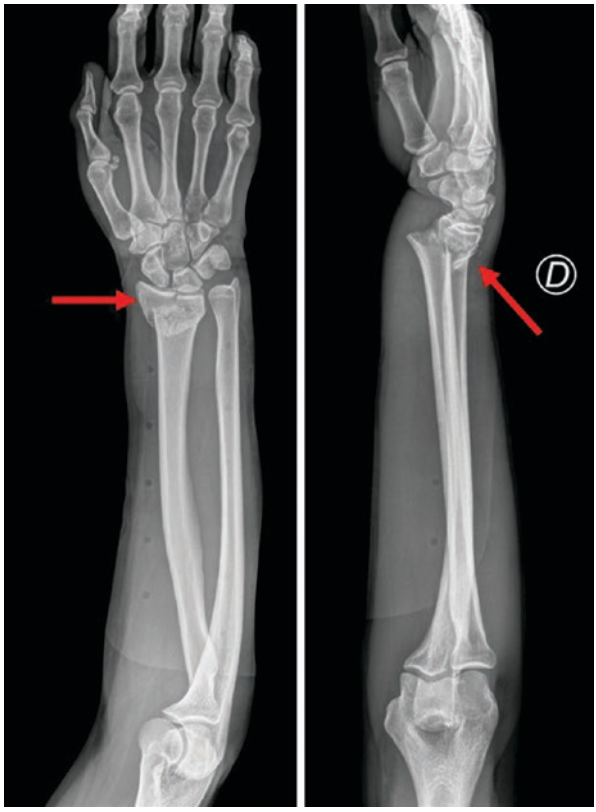


Fig. 14 X-ray (AP view and LL view) showing comminuted fracture of the distal radius with extension to the intra-articular surface along the dorsal aspect (red arrows)

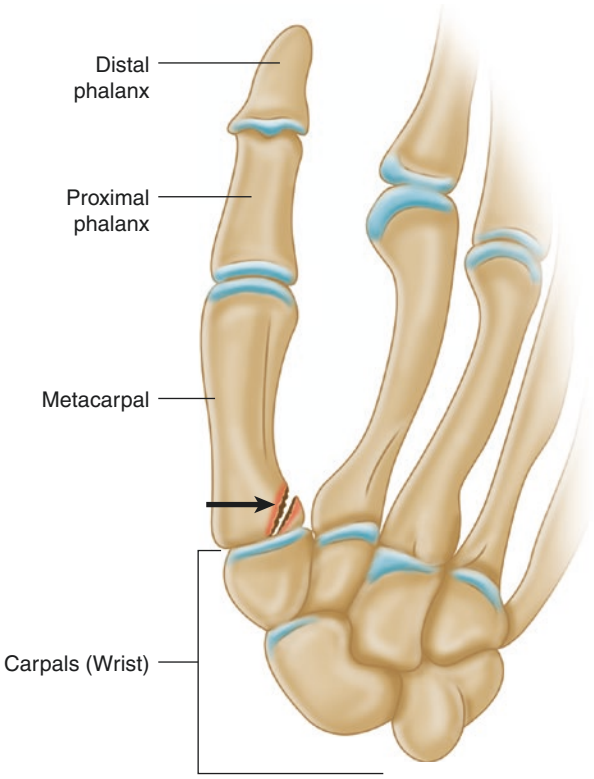


Fig. 15 Drawing of Bennett's injury illustrating an intra-articular fracture of the base of the first metacarpal (white arrow)

4. Bosworth fracture is a fracture of distal fibula with posterior dislocation of the proximal fibula behind the posterior tibial tubercle. MOI – severe external rotation of the foot (Fig. 16).
5. Boxer's fracture is a transverse fracture of the distal portion of the fifth metacarpal bone. These fractures are the most common metacarpal joint fractures. MOI – punching solid objects (Fig. 17)
6. *Bumper fracture* is a compression fracture of lateral tibial plateau. MOI – forced valgus of the knee when struck from the side by car bumper or might result from a fall or a direct blow, usually to the outside of the knee (Fig. 18).
7. Chance fracture is a horizontal compression fracture of the vertebral body. MOI – violent forward flexion seen in car accidents when lap belts were used (Fig. 19).
8. Chauffeur's fracture is an intra-articular fracture of the styloid process of the distal radius. MOI – forced ulnar deviation of the wrist causing avulsion of the radial styloid (Fig. 20).
9. Chopart's fracture-dislocation is a dislocation of mid-tarsal (talonavicular and calcaneocuboid) joints of the foot often with associated fractures of calcaneus, cuboid, and navicular. MOI – falls from height, traffic collisions, twisting injuries in sports (basketball players) (Fig. 21).

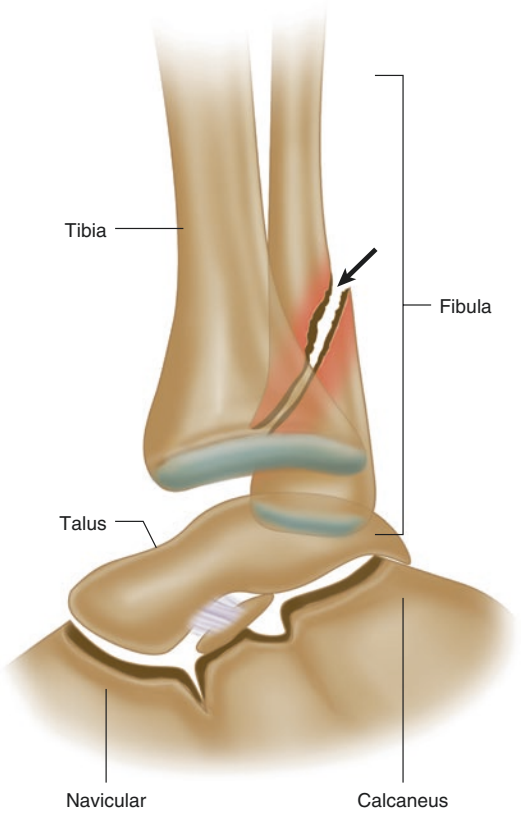


Fig. 16 Representative drawing in lateral view of Bosworth fracture with fracture of distal fibula (arrow) and proximal fibular fragment trapped behind tibia

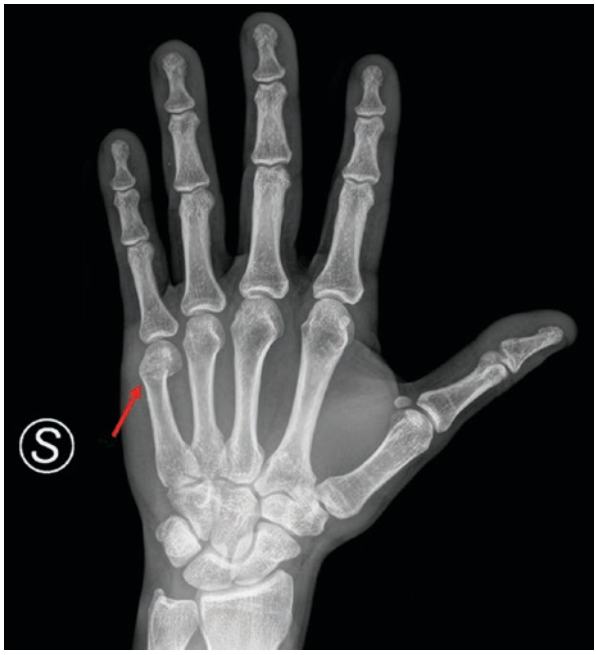


Fig. 17 X-ray (AP view) of the right hand shows an extra-articular fracture of the fifth metacarpal head of the left hand (red arrow) from punching a wall

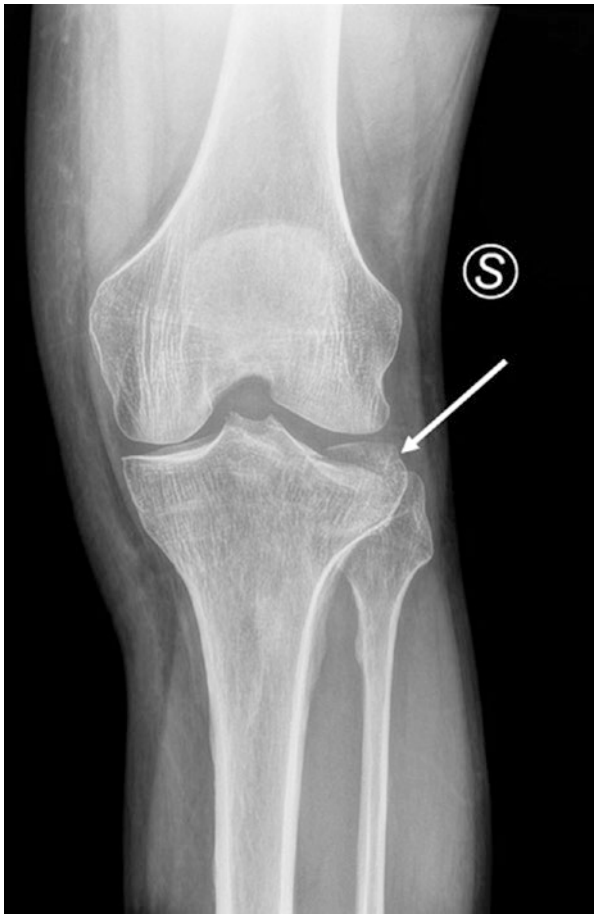


Fig. 18 X-ray (AP view) of the left knee showing a fracture of the lateral tibial plateau (arrow)

10. Clay-shoveler's fracture is a stable fracture of the spinous process of a vertebra most commonly occurring at any of the lower cervical or upper thoracic vertebrae (usually C6, C7 or T1). MOI – forced hyperflexion of neck (Fig. 22).
11. Colles' fracture is a fracture of the distal radius with dorsal angulation, impaction, and radial drift. MOI – fall on outstretched hand (Fig. 23).
12. Dupuytren/Pott's fracture is a bi/trimalleolar ankle fracture. MOI – eversion of ankle (Fig. 24).
13. Duverney fracture is an isolated pelvic fracture involving only the iliac wing. It is considered a stable pelvic fracture since it does not disrupt the weight bearing pelvic ring. MOI – direct trauma (Fig. 25).
14. Essex-Lopresti fracture is a fracture of the radial head with interosseous membrane disruption and concomitant dislocation of distal radio-ulnar joint. MOI – fall from height (Fig. 26).
15. Galeazzi fracture is radial shaft fracture with dislocation of distal radio-ulnar joint. MOI – blow to forearm (Fig. 27).

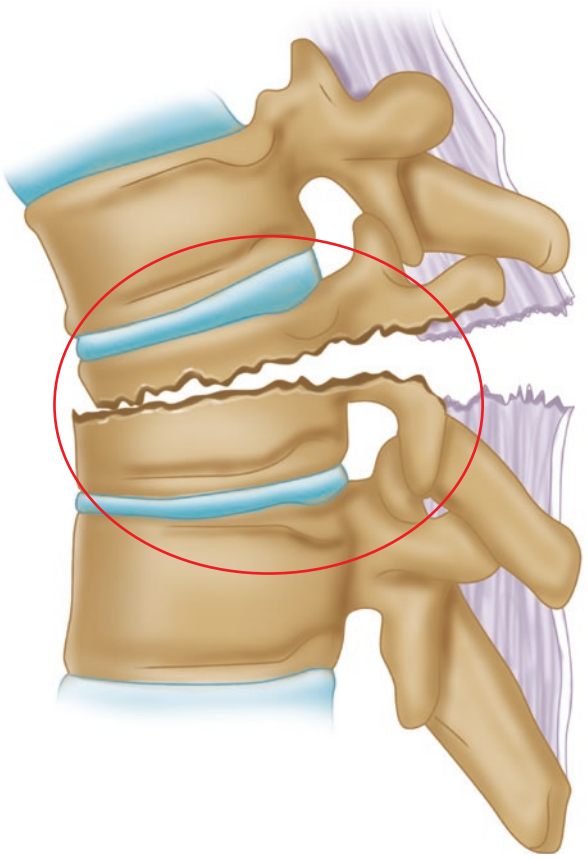


Fig. 19 Drawing of the dorsal vertebral bodies (lateral view) depicts a fracture line extending through the spinous process, pedicles, and vertebral body

16. Gosselin fracture is a V-shaped distal tibia fracture extending into the tibial plafond dividing it in anterior and posterior segments (Fig. 28).
17. Greenstick fracture – common pediatric fractures are "greenstick" and "torus or buckle" fractures (children's type of fracture due to major bone elasticity also known as bowing fracture deformity). These fractures occur when the force applied to a bone results in bending of the bone such that the structural integrity of the convex surface is overcome, and it breaks. MOI – bending forces after fall (Fig. 29).
18. Hangman's fracture is a fracture of both pedicles of the axis vertebra (C2). MOI – distraction and extension of neck (Fig. 30).
19. Hill-Sachs fracture is a cortical depression in the posterolateral head of the humerus occurring during anterior shoulder dislocation. It is a result of forceful impaction of the humeral head against the anterior-inferior glenoid rim (Fig. 31).
20. *Holstein-Lewis fracture* is a spiral fracture of the distal third of the shaft of humerus resulting in entrapment of the radial nerve (Fig. 32).

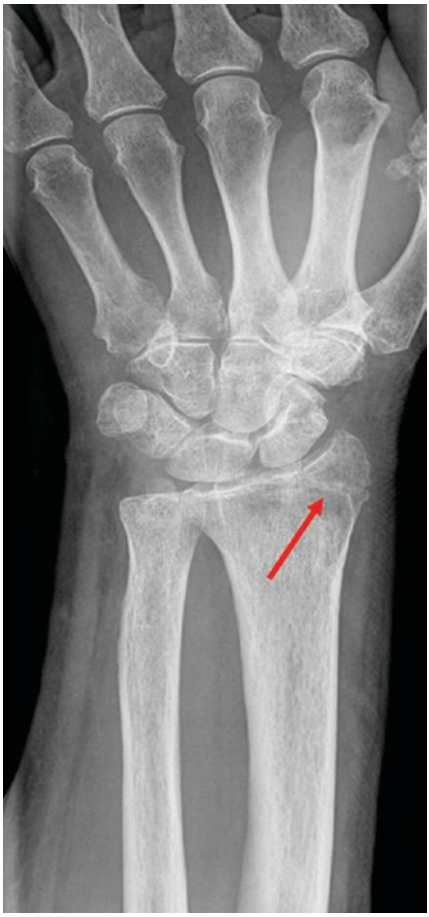


Fig. 20 X-ray (AP view) of the right wrist showing isolated fracture of the radial styloid process (red arrow)

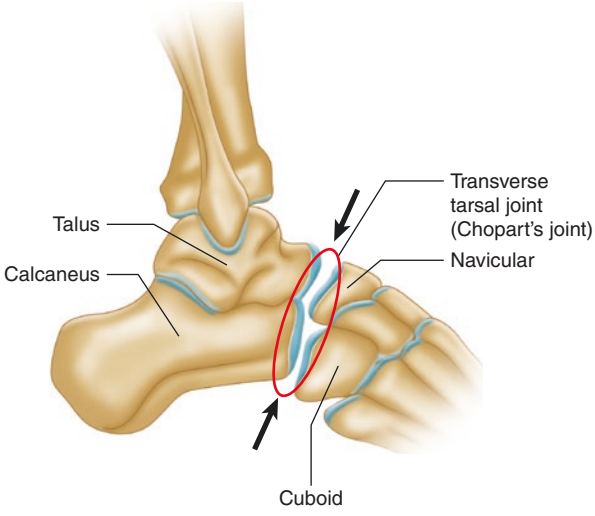


Fig. 21 An illustration of Chopart's fracture-dislocation (arrows)

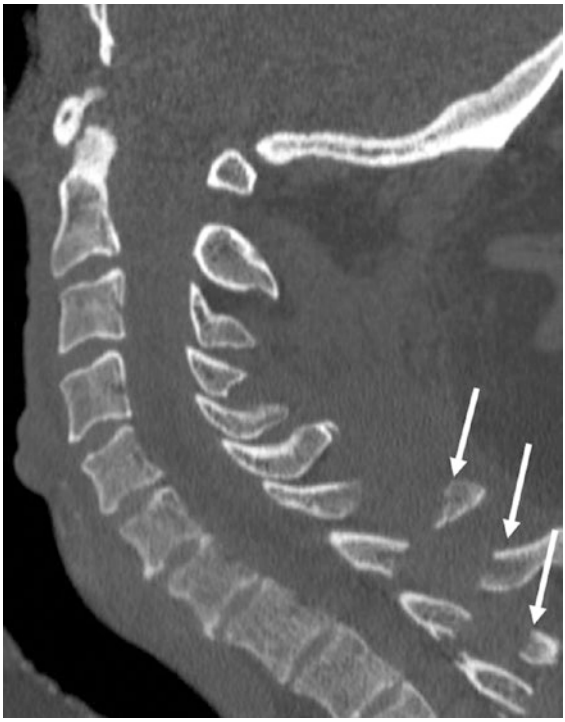


Fig. 22 Sagittal reformatted computed tomography image shows a distracted fracture of the spinous process of T1, T2, and T3 (arrows)

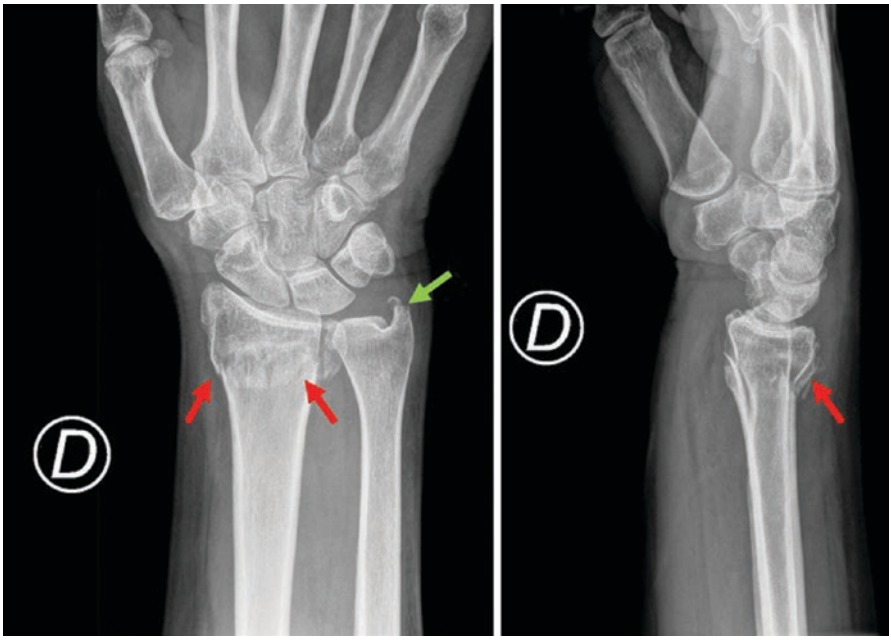


Fig. 23 X-ray (AP view and LL view) showing fracture of the distal metaphysis of the radius with dorsal angulation and displacement (red arrows). Also notice the fracture of the ulnar styloid (green arrow)

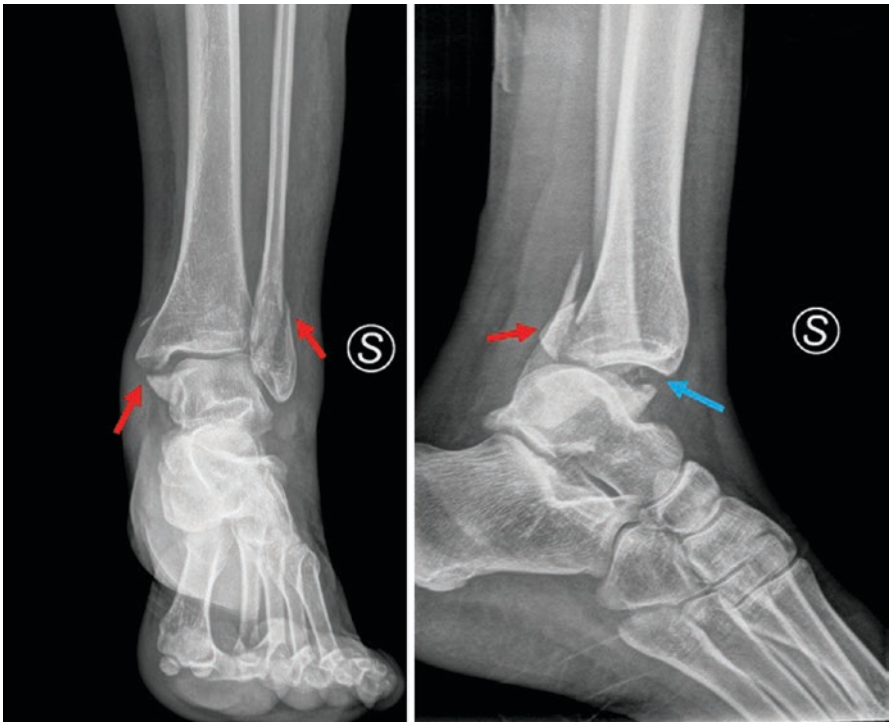


Fig. 24 X-ray (AP view and LL view) of the left ankle showing trimalleolar fracture (red arrows) accompanied by dislocation of the ankle joint (blue arrow)

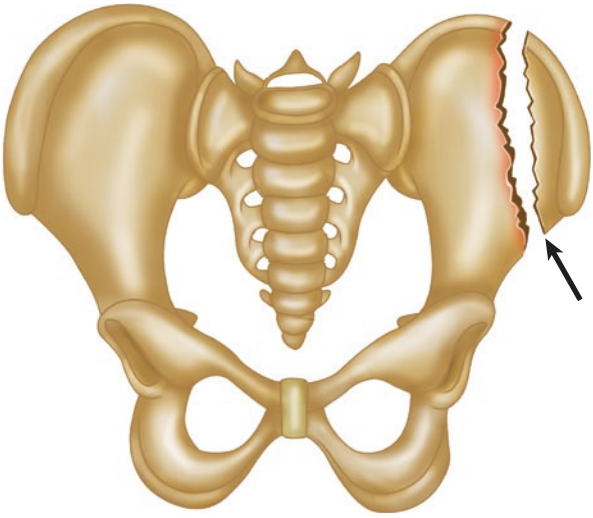


Fig. 25 An illustration of stable pelvic fracture involving iliac wing (arrow)

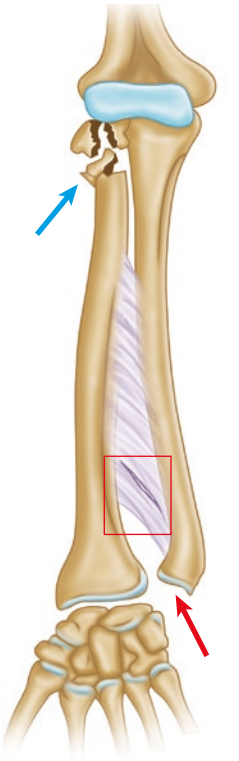


Fig. 26 Representative drawing of Essex-Lopresti fracture with comminuted displaced fracture of radial head (blue arrow), disruption of the interosseous membrane (red rectangle), and subluxation or dislocation of distal radio-ulnar joint (red arrow)

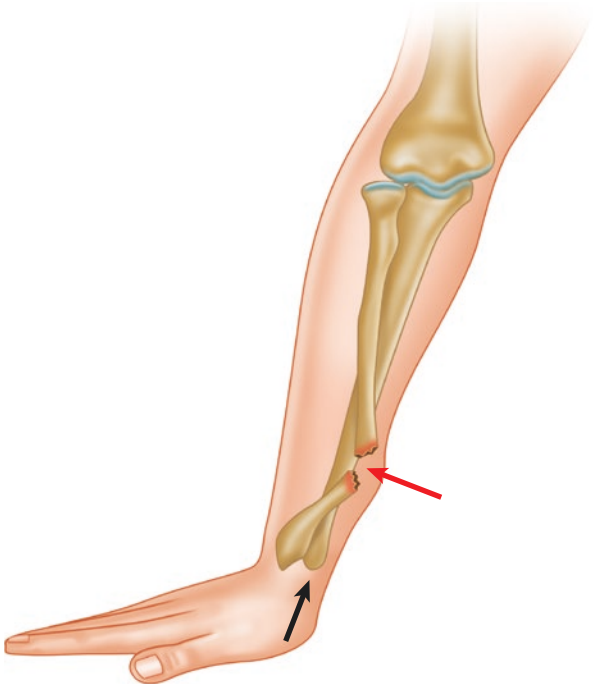


Fig. 27 Diagram of Galeazzi injury illustrates volar fracture of a radial shaft (red arrow) and dislocation of distal radio-ulnar joint (black arrow)

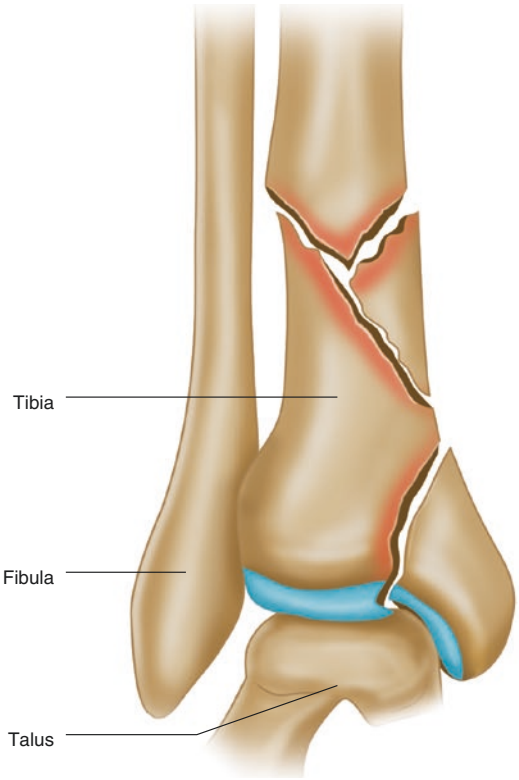


Fig. 28 Representative illustration of a Gosselin fracture (AP view) showing the V-shaped fracture of the distal tibia that extends into the tibial plafond, dividing the plafond into anterior and posterior fragments

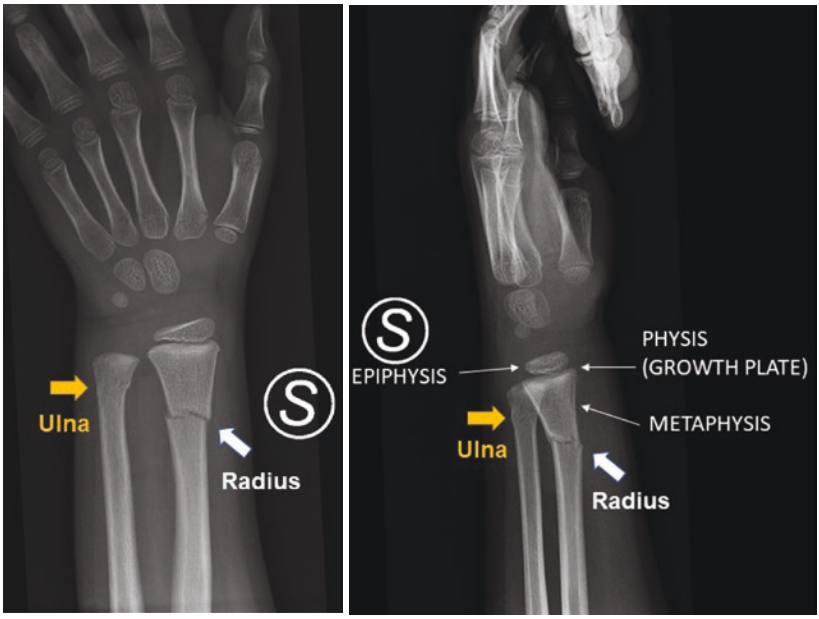


Fig. 29 AP (**a**) and LL (**b**) x-ray views showing a radial greenstick fracture (white arrow) and ulnar torus fracture (yellow arrow)

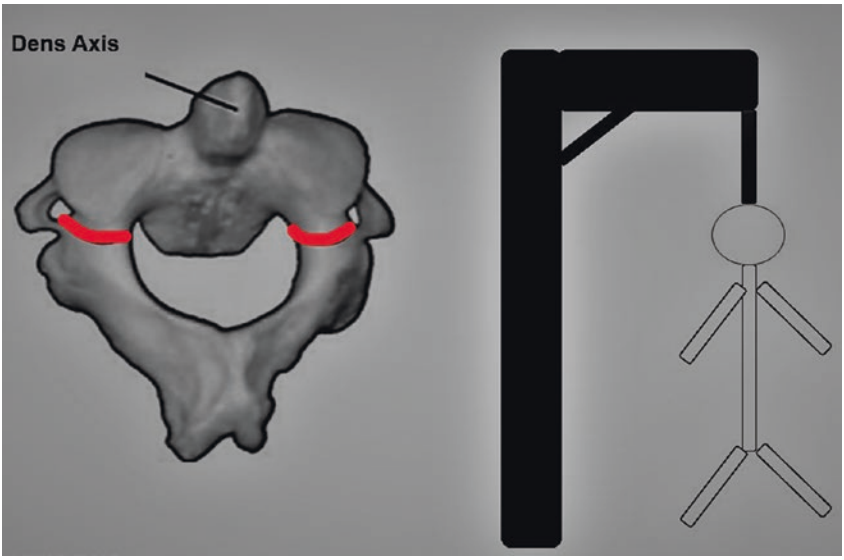
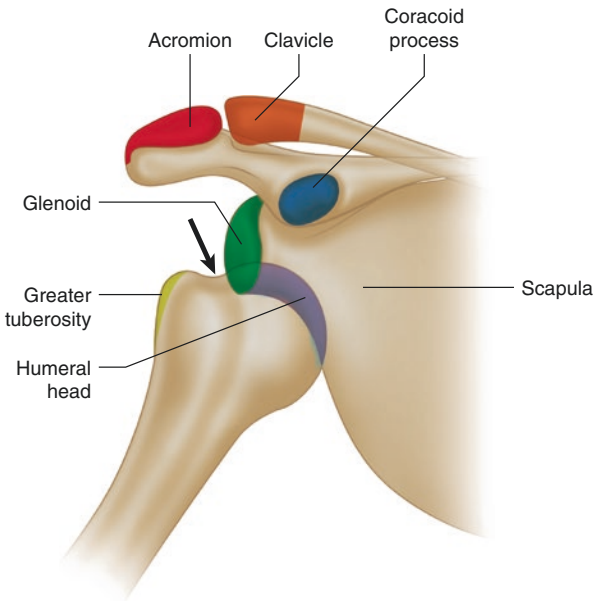


Fig. 30 An illustration of a bilateral fracture of the pars interarticularis (red lines) resulting from a forcible hyperextension of the head. This would happen during judicial hanging

Fig. 31 Drawing of Hill-Sachs injury depicts anterior shoulder dislocation causing large cortical depression in the posterolateral head of the humerus (white arrow)



21. Holdsworth fracture is an unstable spinal fracture-dislocation at the thoracolumbar junction. The injury comprises a fracture through a vertebral body, rupture of posterior spinal ligaments, and concomitant fracture of facet joints (Fig. 33).
22. Hume fracture is an injury of the elbow comprising a fracture of olecranon with concomitant anterior dislocation of the radial head. This injury occurs in children. MOI – hyperextension of elbow with pronation of forearm (Fig. 34).

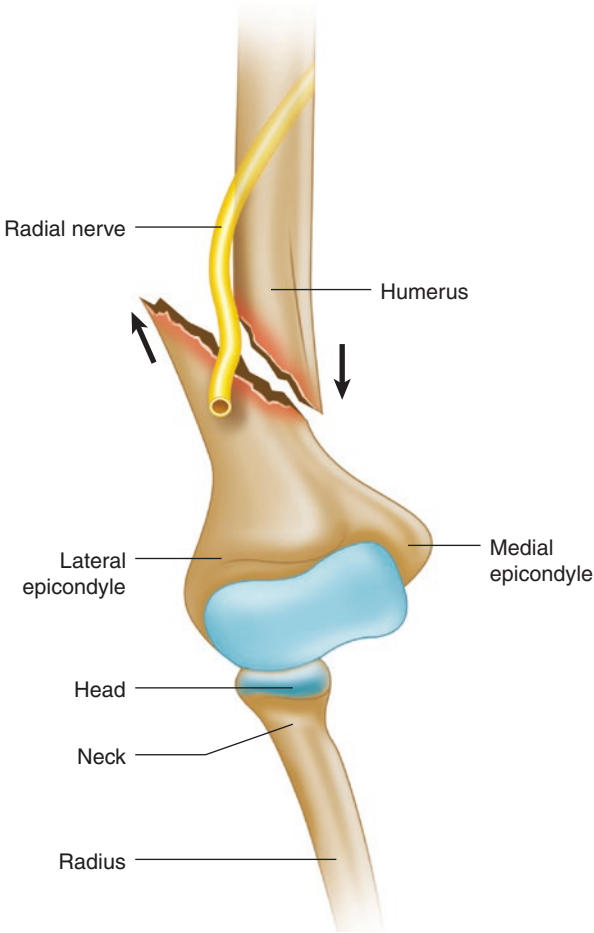


Fig. 32 Illustration of spiral fracture through the distal humeral diaphysis with proximal foreshortening and lateral displacement commonly associated with radial nerve injury

23. Jefferson fracture is a fracture of the anterior and posterior arches of first cervical vertebra C1. MOI – compression of neck (Fig. 35).
24. Jones fracture is a fracture of the base of the fifth metatarsal of the foot. MOI – inversion of ankle (Fig. 36).
25. Le Fort fractures of the skull are a series of facial fractures involving the maxillary bone and the surrounding structures in either horizontal, pyramidal or transverse direction. MOI – direct trauma to face (Fig. 37).
26. Le Fort's fracture of the ankle is a vertical fracture of the anteromedial part of the distal fibula with avulsion of anterior tibiofibular ligament. MOI – avulsion of ankle (Fig. 38).
27. Lisfranc injuries, also called Lisfranc fracture-dislocations, are the most common injuries of the foot in which one or more of the metatarsal bones are displaced from the tarsus. MOI – direct crush injury or an indirect load onto a plantarflexed foot (Fig. 39).

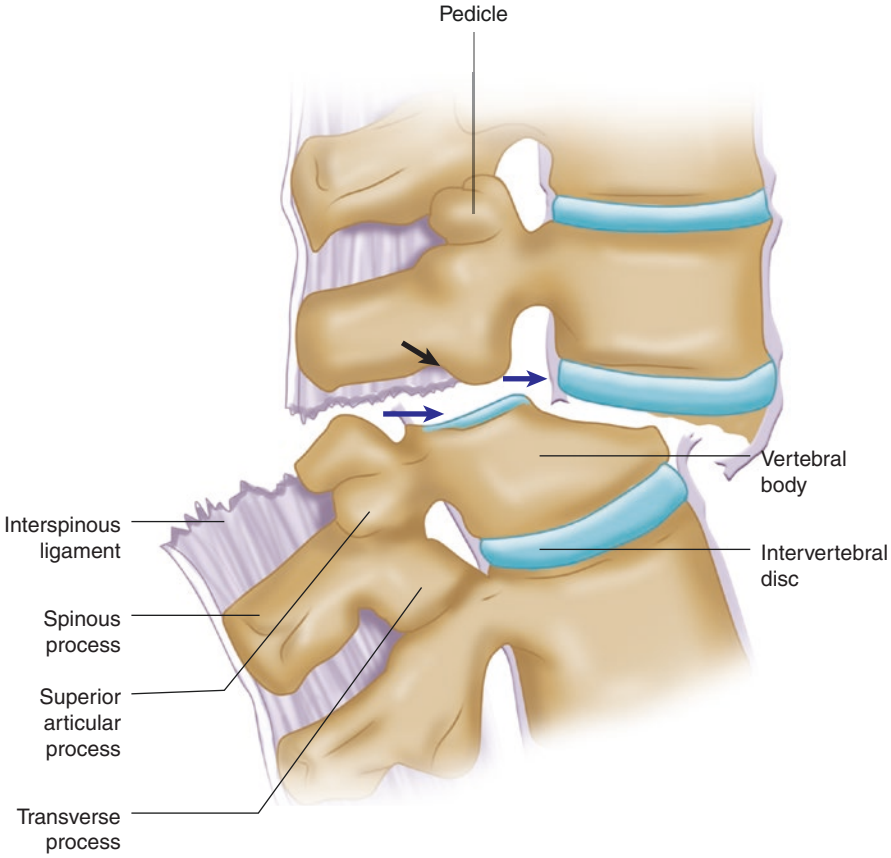


Fig. 33 Drawing of a Holdsworth injury depicts a fracture through the vertebral body, which is one component of this unstable thoracolumbar junction fracture-dislocation. This injury is associated with rupture of the posterior ligaments (blue arrows) and fracture of the articular processes (black arrow)

28. Maisonneuve fracture is a spiral fracture of the proximal fibula. MOI – external rotation of ankle (Fig. 40).
29. Malgaigne's fracture is an unstable type of pelvic fracture characterized by vertical pelvic fracture with bilateral sacroiliac dislocation and fracture of the pubic rami. MOI – high-energy axial loading, such as from falls or other high-energy trauma (Fig. 41).
30. March fracture is a fracture of the distal third of the metatarsals occurring due to recurrent stress. These fractures most commonly occur in the second and third metatarsal bones of the foot. It is a common cause of foot pain, especially when people suddenly increase their activities. MOI – repetitive micro-traumas or heavy exercise (Fig. 42).
31. *Monteggia fracture* is a fracture of the proximal third of ulna with dislocation of the head of the radius. MOI – blow to forearm (Fig. 43).

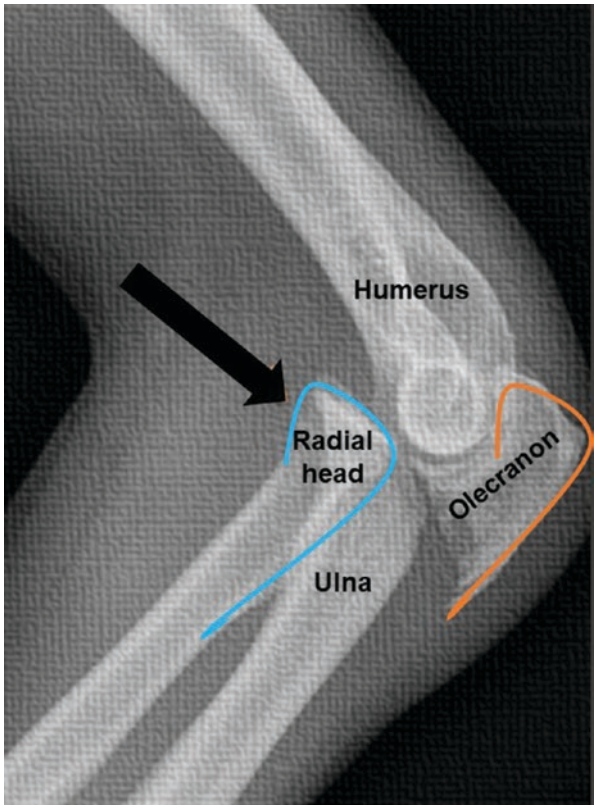


Fig. 34 LL view of elbow illustrating fracture of olecranon associated with anterior dislocation of radial head (black arrow)

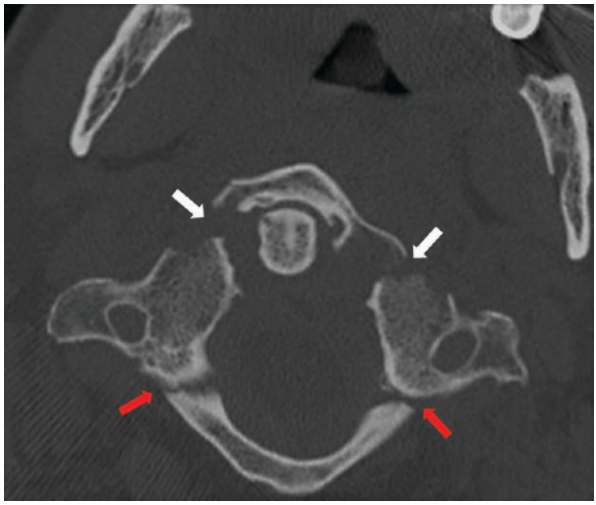


Fig. 35 Axial CT image shows double fractures of the anterior arch (white arrows) with mild displacement and a double fracture of the posterior arch of atlas (C1) (red arrows)

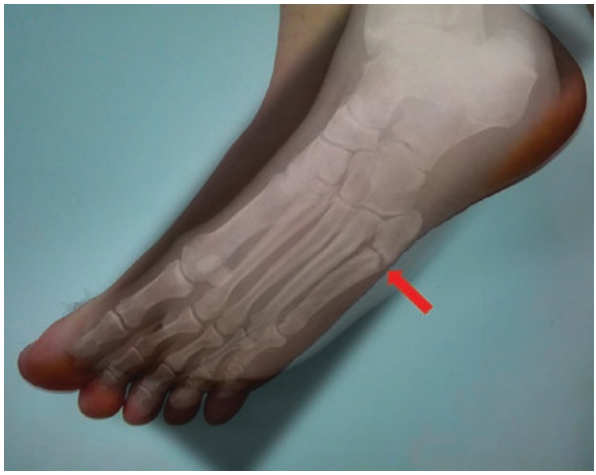


Fig. 36 Extra-articular fracture at the base of the fifth metatarsal bone (red arrow)

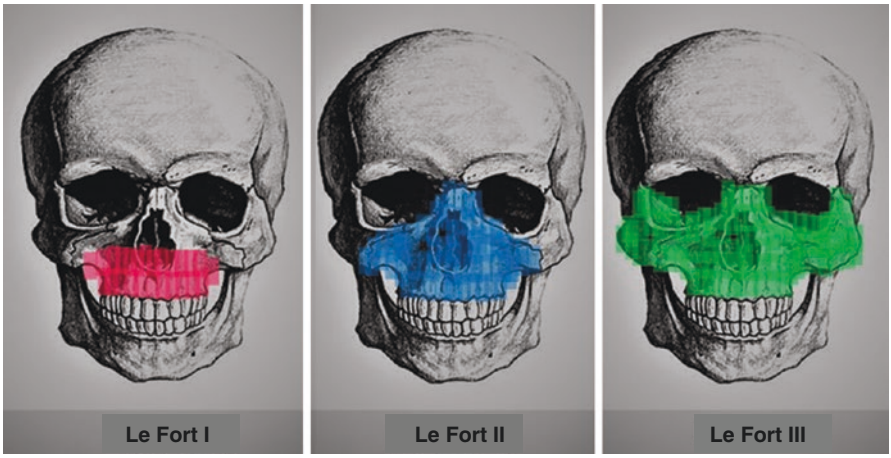


Fig. 37 Drawing of Le Fort injuries illustrating complex fractures of the midface. These fractures are classified into three groups based on the direction of the fracture: horizontal (type I), pyramidal (type II), or transverse (type III)

32. Moore's fracture is a distal radius fracture with ulnar dislocation and entrapment of styloid process under annular ligament (Fig. 44).
33. Pipkin fracture-dislocation is a posterior dislocation of the hip with avulsion fracture of fragment of femoral head by the ligamentum teres. MOI – impact to the knee with the hip flexed (dashboard injury) (Fig. 45).
34. Rolando fracture is a comminuted intra-articular fracture of base of first metacarpal. MOI – axial load along the metacarpal causing splitting of the proximal articular surface (Fig. 46).
35. Runner's fracture is a stress fracture of the distal fibula approximately 3–8 cm above the lateral malleolus. MOI – repeated axial stress on fibula (Fig. 47).

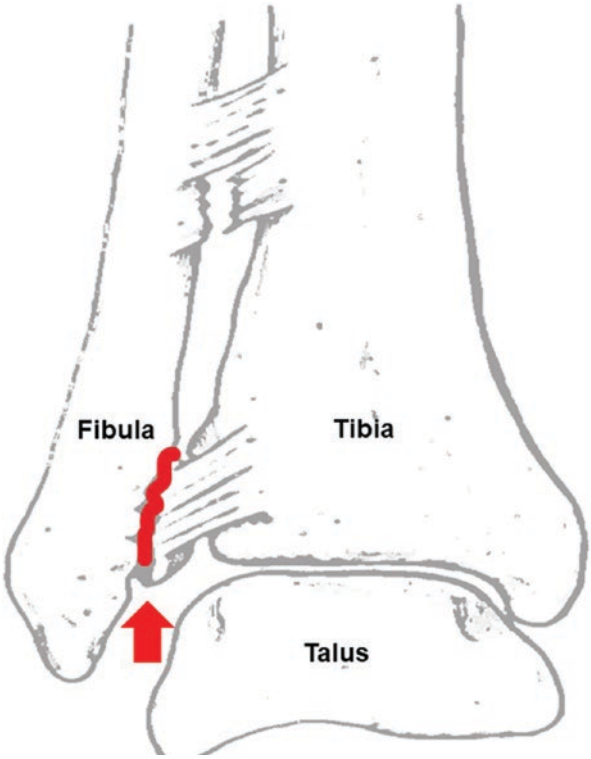


Fig. 38 Diagram of ankle (anterior view) depicts vertical avulsion fracture of the anteromedial portion of the distal fibula (red arrow)

36. Salter-Harris fractures are commonly found in children. These fractures involve a physeal plate or growth plate. The Salter-Harris classification describes five types of physeal fractures (Fig. 48). MOI – various (Fig. 49).
37. Segond fracture is a type of avulsion fracture from the lateral tibial condyle of the knee, immediately beyond the articular surface of the tibia. These fractures are often associated with anterior cruciate ligament tear. MOI – internal rotation of the knee (Fig. 50).
38. Shepherd's fracture is a fracture of the lateral tubercle of the posterior process of the talus. It is sometimes mistaken for symptomatic Os trigonum. MOI – inversion ankle injury or extreme ankle plantar flexion, equinus (Fig. 51).
39. Smith's fracture (reverse Colle's fracture) is a fracture of the distal radius with volar displacement. MOI – fall onto flexed wrist.
40. Stieda fracture is an avulsion fracture of the medial femoral condyle at the site of the proximal attachment of the medial collateral ligament. MOI – valgus injury to the knee (Fig. 52).
41. Tillaux fracture is an injury characterized by Salter-Harris III fracture anterolateral distal tibial epiphysis. These fractures occur almost exclusively in adolescents. MOI – forced lateral rotation of the foot.
42. Toddler's fracture is an nondisplaced spiral fracture of distal third of the tibia. Found in infants and young children. MOI – low energy trauma, often rotational.

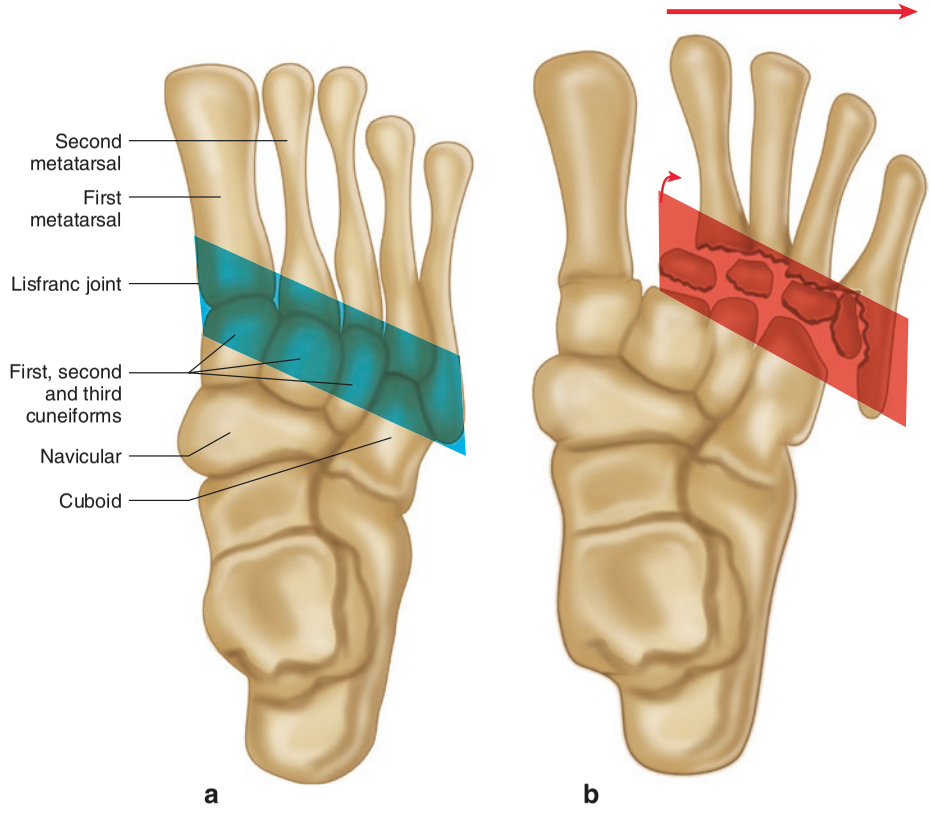


Fig. 39 An illustration of normal Lisfranc joint (**a**) and Lisfranc injury (**b**) showing fracture and lateral dislocation of four metatarsals (II-V). According to Quénu and Küss classification modified by Myerson, this is a type B2 (complete) Lisfranc injury

Fig. 40 Schematic illustration of Maisonneuve injury showing spiral fracture of proximal third of fibula (black arrow) with disruption of distal tibiofibular syndesmosis and fracture of medial malleolus (red arrow)

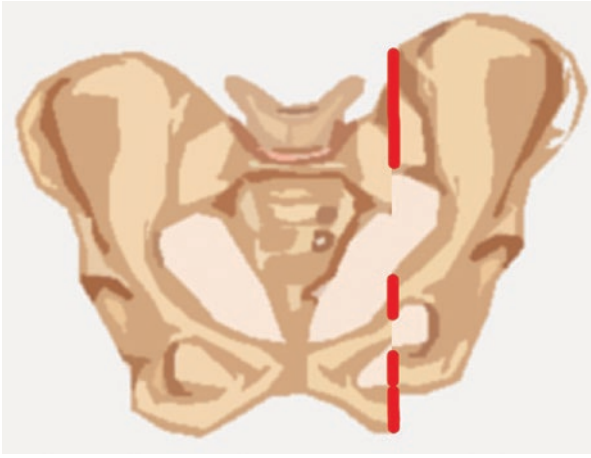


Fig. 41 An illustration of Malgaigne fracture, an unstable type of pelvic fracture, which involves one hemipelvis, and results from vertical shear energy vectors

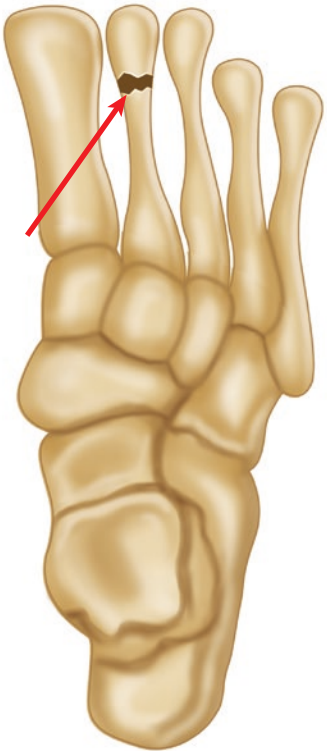


Fig. 42 An Illustration showing stress fracture in second metatarsal (red arrow)

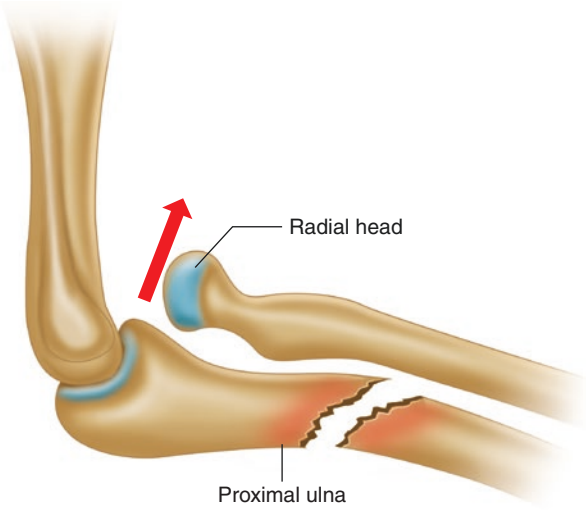


Fig. 43 Drawing of Monteggia injury showing proximal ulna fracture and anterior dislocation of radial head (red arrow)

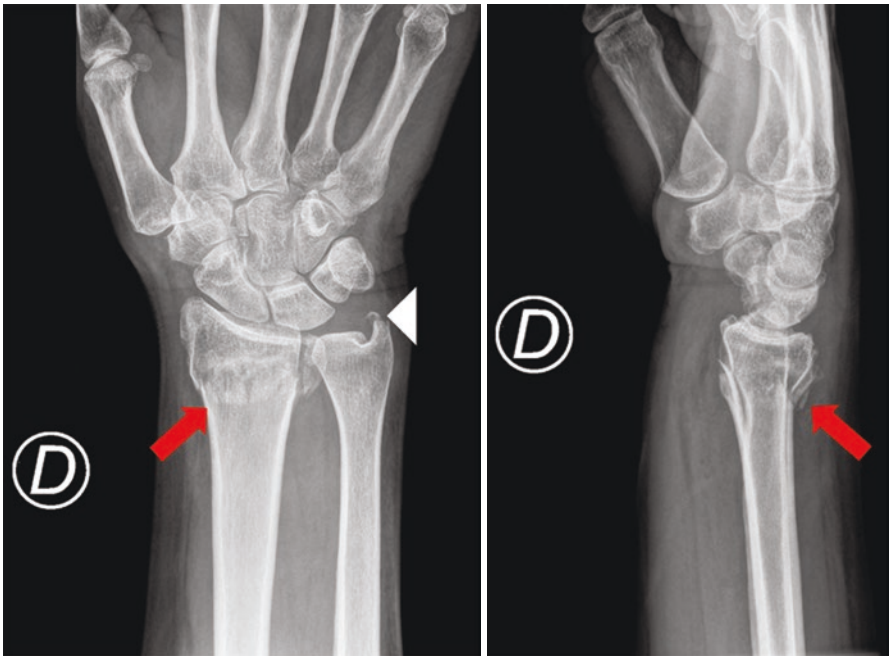


Fig. 44 X-ray images (AP view and LL view) of the right wrist showing fracture of distal radius (red arrows) and of ulnar styloid process (white arrowhead) with mild dislocation of distal ulna

When interpreting complex fractures or intra-articular fractures, CT scan may provide additional details of the severity and extent of a trauma with the possibility of 3D reconstruction and is helpful in planning a surgical repair if needed (e.g., small bony fragments, complex hip fractures). However, plain films should always precede other types of imaging. Initially, many fractures can be hidden as extremely subtle or microscopic; thus, prophylactic immobilization and follow-up repeat imaging at 10–14 days may be useful (typical question is in an elderly patient after a fall on hip).

4.1.2 Soft Tissue Injuries

Soft tissue injuries include a broad range of disorders which are commonly seen in primary care [12]. Assigning a specific diagnosis can be challenging given complex anatomy and potentially overlapping symptoms from a variety of syndromes. It is most helpful to approach these disorders using broad diagnostic and treatment principles, while being alert for cases that do not follow an expected course so that they may be referred for specialist opinion expeditiously [13].

Diagnostic considerations – definitions:

Strain Stretch/tear of muscle or musculotendinous structure through macrotrauma, tissue invasion, or repetitive microtrauma. There is generally muscle

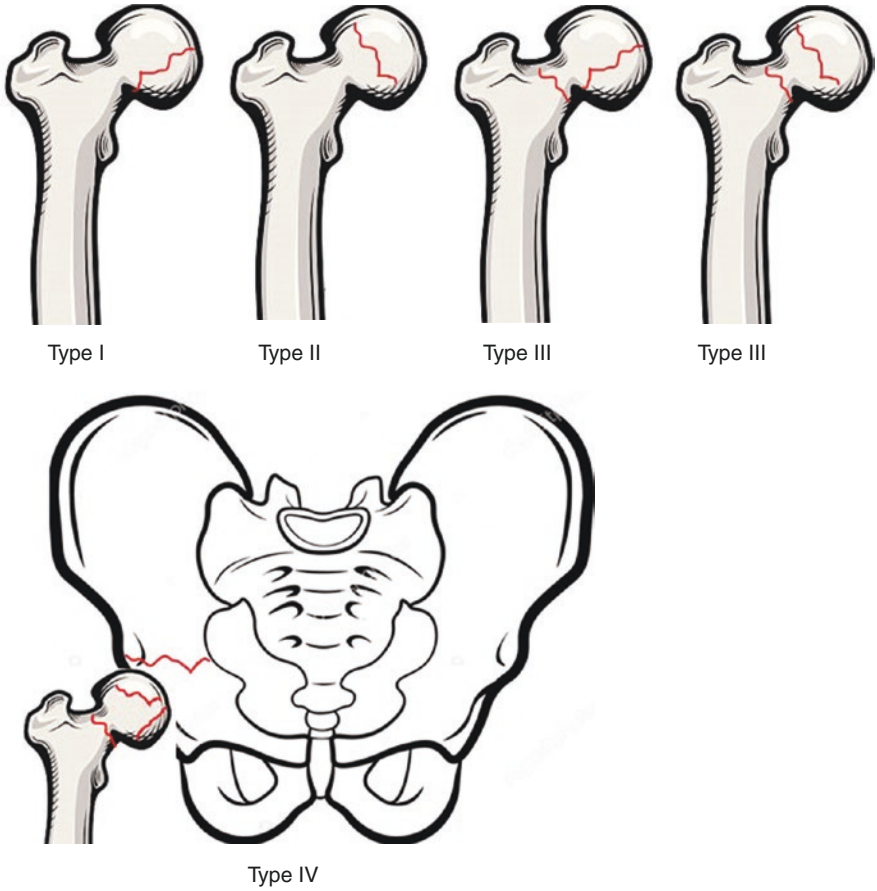


Fig. 45 Pipkin classification of femoral head fractures with posterior hip dislocations. Type I is below the fovea with the fracture outside of the weight-bearing joint parts. Type II fractures involve the more cranial, weight-bearing parts. Type III is any fracture of the head associated with a femoral neck fracture. Additional fractures of the acetabulum are classified as type IV

contusion after direct trauma and muscle tear after indirect mechanism of accident. A direct trauma can induce a bleeding deep within the muscle, resulting in a hematoma. In sporadic cases, direct muscle contusions can cause compartment syndrome. Indirect trauma is induced by eccentric contraction [14]. Typical features of indirect muscle injuries are hematoma, fiber disruption, and muscle edema. The muscles with a high risk for tears are types with a high proportion of type II fibers, muscles with multiple heads, and muscles extending over two joints (e.g., biceps femoris muscle) (Fig. 53) [15].

Localization of injury is also age dependent. In young patients, the muscle typically tears at the non-fused apophysis. In elderly people, the tear is most commonly located at the tendon, often being already affected by degenerative changes.

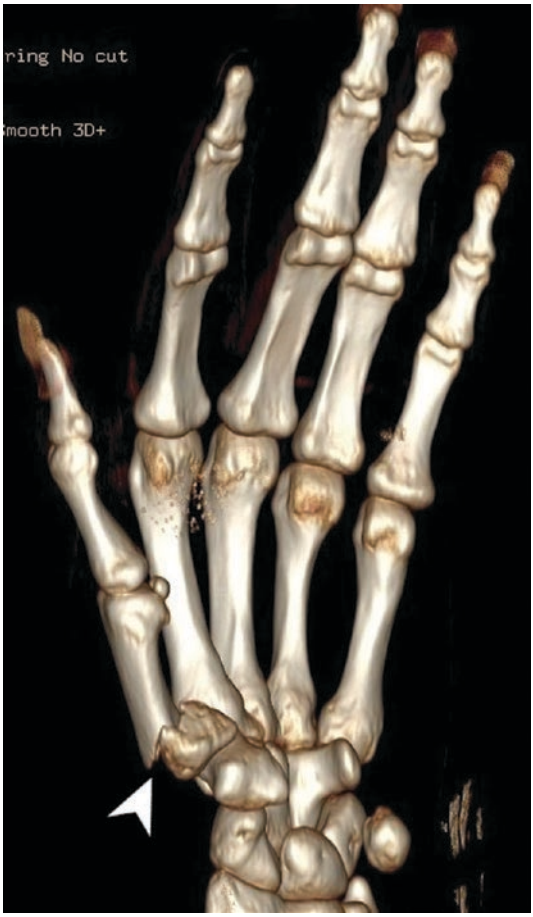


Fig. 46 3D-computed tomographic image of a Rolando fracture defined as comminuted intra-articular fracture-dislocation of the base of the thumb metacarpal

In adults of intermediate age, the location of the tear is usually the myotendinous junction, which is the weakest link in the muscle-tendon-bone chain [14, 16].

Tendinopathy Acute (tendinitis/tenosynovitis) or chronic (tendinosis) damage that occurs to tendons, the connective tissues that transfer power from muscle to joint. Tendon rupture with fluoroquinolone use is a common board question.

Myofascial Pain Soft tissue pain originating from a tense band of skeletal muscle or its fascia. Associated trigger points elicit pain in a myotomal distribution and may cause decreased range of motion (ROM)/strength. This must be distinguished from fibromyalgia.

Sprain Stretch/tear of a ligament or joint capsule through large forces at extremes of ROM beyond tissue tolerance, e.g., hyper-extension.

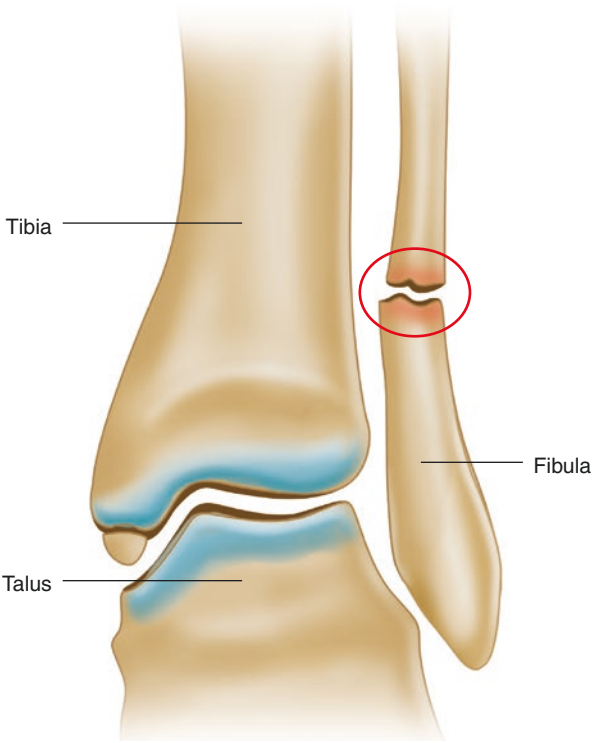


Fig. 47 An illustration of distal fibular stress fracture which occurs after repetitive loading of the foot and ankle in athletes

Bursitis Inflammation of a bursa, the fluid filled sac that reduces friction at key anatomic locations such as joints. Bursitis may occur through stress or infection.

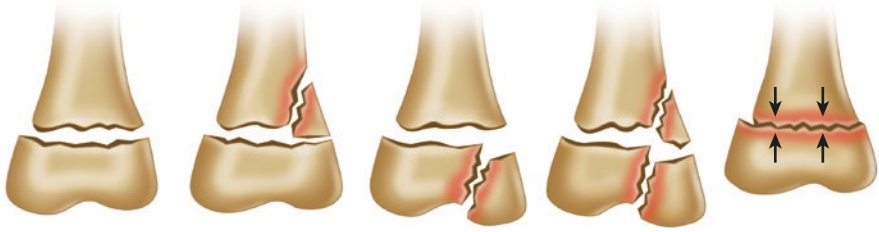
Both ultrasound and MRI are generally suitable to evaluate soft tissue injuries [17, 18]. However, MRI is considered the "gold standard" in diagnostic imaging of muscle injuries.

US is often used as the first modality, as it is widely available and is fast, easy to use, and affordable. However, US is known to be highly operator dependent. Furthermore, the determination of the length of a muscle tear might be challenging, and small hematomas can be missed, especially within the first 24 hours after injury.

MRI is a more elaborate imaging technique that allows for an excellent depiction of the most relevant findings in muscle injuries [19] and has a high sensitivity in identifying acute and chronic soft tissue alteration [20].

To assess soft tissue injuries on MRI, fluid-sensitive T2-weighted short-tau inversion recovery sequences (STIR) or PD fat-saturated sequences on the one hand and T1-weighted sequences on the other hand should be acquired as minimal. As aforementioned, fluid-sensitive sequences allow visualization of edema, muscle tear, hematoma, and bone bruise. PD fat-saturated sequences provide more anatomical information. T1-weighted sequences allow for an assessment of (stress) fractures including bony avulsions and hematoma. Contrast-enhanced sequences are not mandatory after acute trauma [21].

Salter-Harris (SH) physeal injury classification	
Type	**Characteristics**
I	Separation through the physis, usually through areas of hypertrophic and degenerating cartilage cell columns.
II	Fracture through a portion of the physis that extends through the metaphyses.
III	Fracture through a portion of the physis that extends through the epiphysis and into the joint.
IV	Fracture across the metaphysis, physis and epiphysis.
V	Crush injury to the physis.



SH classification from I-V

Fig. 48 Salter-Harris (SH) physeal injury classification

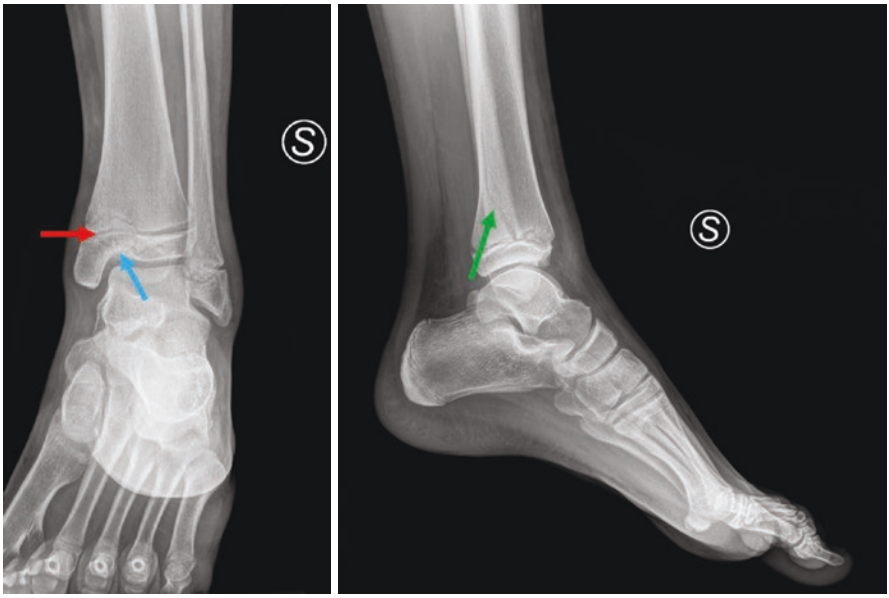


Fig. 49 X-ray (AP view and LL view) of the left ankle showing a type IV fracture that involves all three elements of the distal tibia, the growth plate (red arrow), metaphysis (green arrow), and epiphysis (blue arrow)

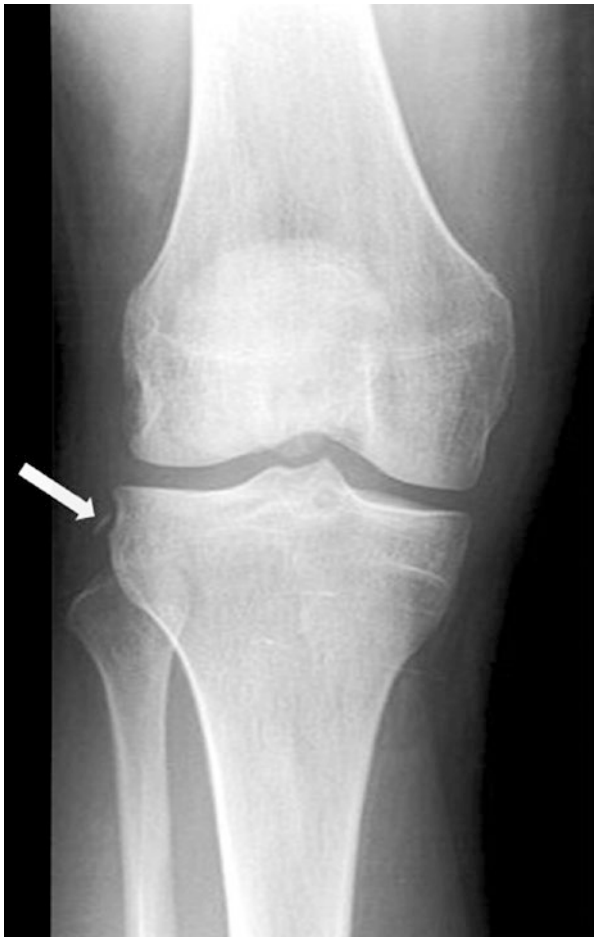


Fig. 50 X-ray (AP view) image of the right knee showing linear fracture fragment (white arrow) in proximity of the lateral tibial condyle, cranial to fibular head. Segond avulsion fracture is frequently associated with detachment of the capsular portion of the lateral collateral ligament

4.2 Infection

4.2.1 Bone Infection

Osteomyelitis is inflammation of the bone marrow secondary to infection, which can progress to osteonecrosis, bone destruction, and septic arthritis and is an important cause of permanent disability in both children and adults worldwide [22, 23]. Imaging plays a crucial role in establishing a timely diagnosis and guiding early management, with the aim of reducing long-term complications. Osteomyelitis arises from infection with a variety of microorganisms via different mechanisms [23]. Most common pathogens include *Staphylococcus aureus*, *Staphylococcus epidermidis*, and *Enterobacter* species. Fungal osteomyelitis most commonly occurs in immunocompromised patients [24].

Osteomyelitis has three main routes of spread [25]:

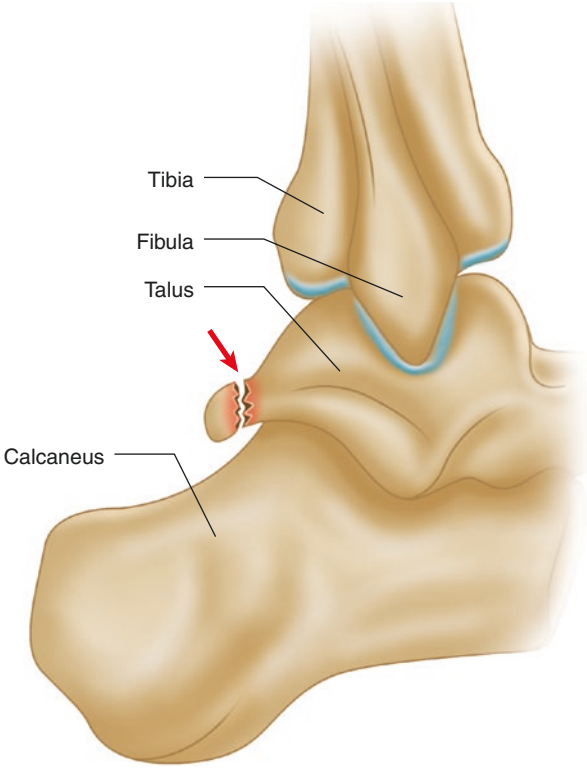


Fig. 51 Schematic drawing showing fracture involving the lateral tubercle of the posterior process of the talus (red arrow)

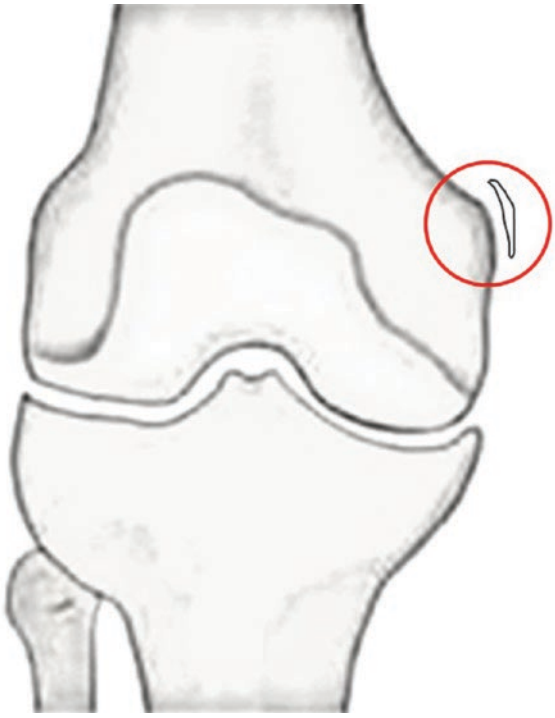


Fig. 52 Diagram of Stieda fracture showing bony avulsion injury of the medial collateral ligament at the medial femoral condyle

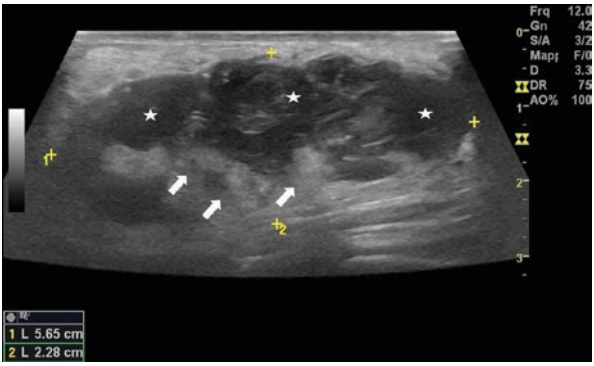


Fig. 53 Ultrasound image showing complete rupture of femoral biceps muscle. In this longitudinal image, there is a heterogeneous mass (arrows) surrounded by fluid (fresh blood; stars). Fibers are completely interrupted in the biceps femoris muscle, and there is an anechogenic gap due to hematoma and retraction of the muscle

1. *Hematogenous* – blood-borne bacteria deposit in the medullary cavity and form a nidus of infection. Most commonly in the metaphysis of long bones because of the large amount of slowly flowing blood, which creates a great environment for bacterial growth and proliferation.
2. *Contiguous* – soft tissues and joints as sources of infections that spread contiguously to the bone. Predisposing factors are repeated trauma commonly of lower extremities in patients with vascular insufficiency (e.g., patients with diabetes mellitus or peripheral vascular disease). Poor perfusion of the infected region reduces the immune response.
3. *Direct inoculation* – seeding of bacteria directly into the bone, e.g., open fractures, insertion of metallic implants or joint prostheses, human or animal bites, and puncture wounds [26].

There are two types of osteomyelitis, acute and chronic. Acute osteomyelitis is an acute suppurative response to hematogenous spread of infection or direct inoculation of pathogens which can lead to intramedullary formation of well-circumscribed intraosseous abscess, also known as a Brodie's abscess [27]. The presence of pus raises the intramedullary pressure and, in some cases, can lead to bone cortex rupture and further spread of pus to subperiosteal space and to surrounding soft tissues.

Chronic osteomyelitis is a consequence of inadequately treated acute osteomyelitis. Chronic osteomyelitis leads to osteonecrosis, caused by disruption of the intraosseous and periosteal blood supply during the acute stage of disease. A fragment of dead and infected bone separates from viable bone and is called sequestrum. The bacteria within the devascularized sequestrum are protected from antibiotics and the endogenous immune response, consequently forming a focus for chronic infection which may persist for several years [23]. Discitis, its appearance on MRI, a classic board question, can cause adjacent bony vertebral body osteomyelitis and destruction (Fig. 54).

4.2.2 Muscle Infection

Pyomyositis (PM) is a rare primary infection of skeletal muscle, and it is usually caused by *Staphylococcus aureus*. It is characterized by suppuration within the skeletal muscles, manifesting eventually as single or multiple abscesses. Pyomyositis

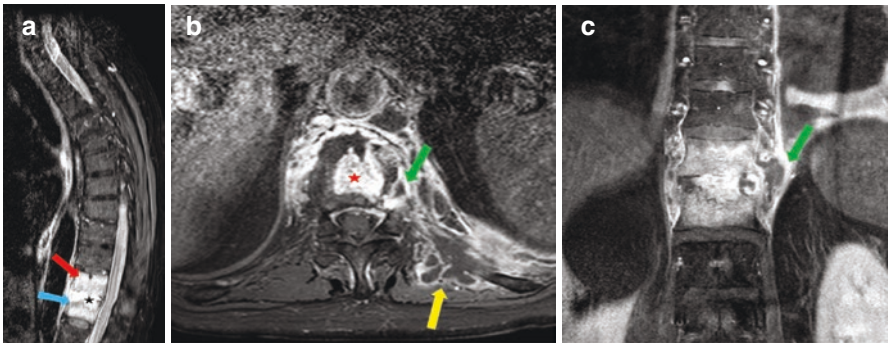


Fig. 54 Case of T12-L1 vertebral osteomyelitis. (**a**) T2 short-tau inversion recovery (STIR – fat suppression technique) image in sagittal plane shows high signal in disc space caused by fluid collection (black star), a high signal in adjacent vertebral bodies due to bone marrow edema (red arrow) and loss of low signal cortex at endplates (blue arrow). (**b**, **c**) T1 contrast-enhanced (Gadolinium contrast medium) images in axial and coronal planes demonstrate peripheral enhancement around fluid collections (green arrows), enhancement of vertebral endplates (red star), and enhancement of paravertebral soft tissues (yellow arrow)

can be acute, subacute, or chronic. In the past, PM was most commonly encountered in children and in patients from the tropics. However, more recently, patients infected by human immunodeficiency virus (HIV) have been recognized to be at high risk for deep muscular infection [28–30]. Other reasons of immunodeficiency have been reported as risk factors for PM such as steroid use, intravenous drug abuse, diabetes mellitus, leukemia, lymphoma, or sickle cell anemia. The most commonly affected areas are the buttock and thigh. Local symptoms consist of swelling, fluctuance, myalgia, and inflammatory skin. In rare cases, the infection can be associated with extensive gas formation due to myonecrosis, also known as "gas gangrene," caused by multiple organisms – polymicrobial PM (classically associated with *Clostridium perfringens*) [28]. The patient's condition is extremely severe, and the mortality rate is high. Several other pathogens have also been described (bacteria, virus, and parasites).

Treatment relies on antibiotic therapy and urgent surgical drainage.

US is a diagnostic tool that shows muscular heterogeneity and purulent collection. Diagnosis can be challenging in the early stages. However, US can be performed promptly at the bedside and is less expensive than other imaging modalities (CT and MRI). It allows the clinician to examine the deeper tissue planes of muscle, in which purulent fluid collections will develop as pyomyositis advances. It can be used as a guidance for interventional procedures (puncture, biopsy, or catheter drainage).

CT can confirm the diagnosis before abscess formation. Hypodensity of enlarged muscle can be seen, and when abscess is present, fluid collection with rim enhancement can be found.

MR is the most sensitive imaging method and it determines both localization and extension of the disease. A hyperintense rim on unenhanced T1-weighted images, muscle edema, and fluid collection on T2-weighted images and peripheral enhancement after gadolinium-based contrast medium intravenous injection are all useful for identifying the number, size, and location of abscesses.

4.2.3 Joint Infection

Septic arthritis is generally secondary to hematogenous seeding and less commonly due to trauma or to recent instrumentation of the joint. Large joints with rich blood supply to the metaphysis are most susceptible to bacterial infection, with the shoulder, hip, and knee being the most commonly affected. Prompt diagnosis and treatment are crucial, in order to avoid irreversible joint damage due to the proteolytic enzymes of the white blood cells that inundate the joint space.

Main risk factors for this infection include advanced age, an immunocompromised condition, rheumatoid arthritis (RA), history of intra-articular injections, prosthetic joints, and bacteremia. Septic arthritis is most frequently encountered in intravenous drug abusers and HIV-infected patients, with *Staphylococcus aureus being* the most common pathogen. The patient usually presents with a painful joint, fever, and purulent synovial fluid collections.

Radiographs may be normal in early stages, but joint space narrowing and juxta-articular lucencies or sclerosis can be seen if untreated. Ultrasound can be helpful in superficial joints and in pediatric patients. US can show joint effusion with echogenic debris (Fig. 55), perisynovial hyperemia on color-Doppler, and is very useful as guidance for joint aspiration. CT demonstrates joint effusion and bone erosions

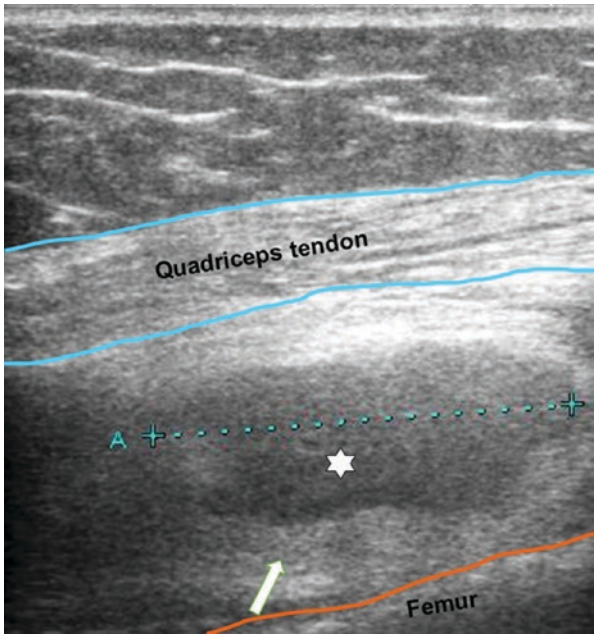


Fig. 55 Ultrasound image (longitudinal plane) of the right knee just above patella shows an effusion (white star) and synovial thickening (white arrow) in an adult patient with septic arthritis

around the joint. A fat-fluid level can be a specific sign in the absence of trauma [31]. MRI shows hypointense subchondral bone on T1-weighted images, perisynovial edema on T2-weighted images, and synovial enhancement on postcontrast sequences (Fig. 56a, b). Destructive arthritis erosions in any single joint may be difficult to differentiate from infection. Distribution can help differentiate the two entities. A solitary joint effusion in a young patient should raise the possibility of infection (gonococcus).

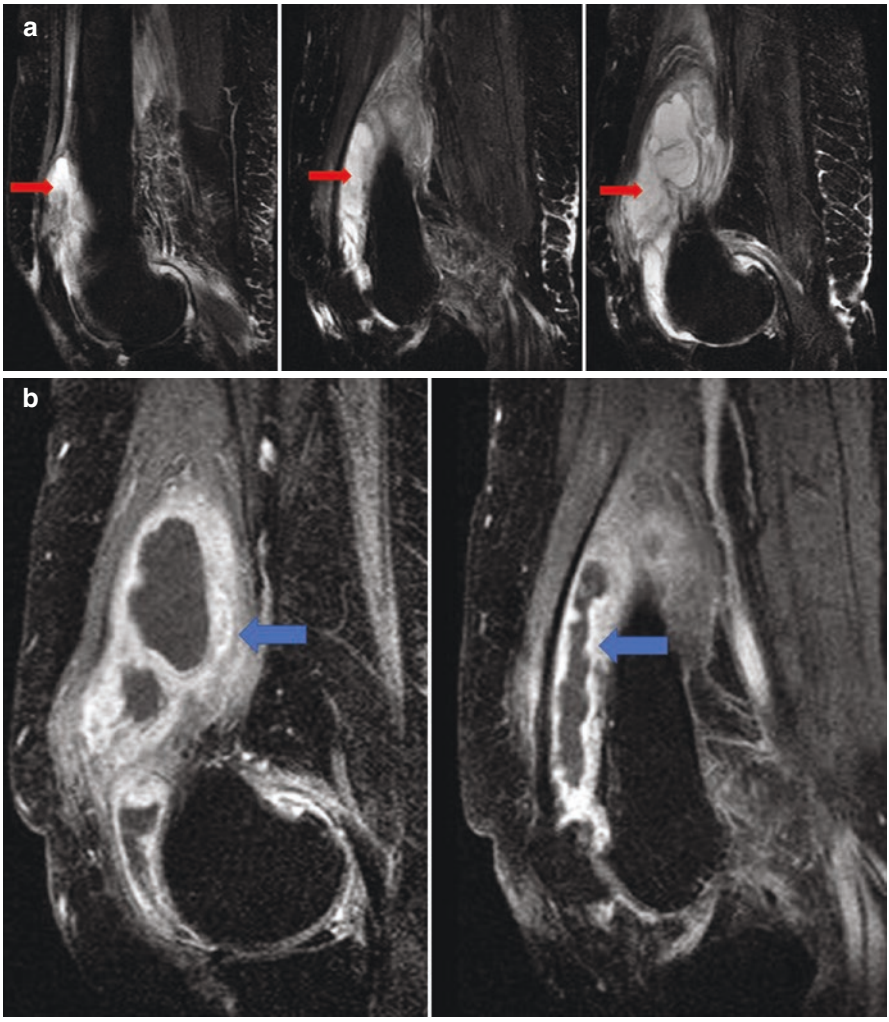


Fig. 56 (**a**, **b**) Same patient underwent a knee MRI. T2-weighted sagittal images (**a**) showed large amount of joint effusion (red arrows) and intense synovial enhancement (blue arrows) on T1-weighted contrast-enhanced images in sagittal plane (**b**)

4.2.4 Soft Tissue Infection

Soft tissue infection of the musculoskeletal system may be challenging and can be associated with high mortality and morbidity, if not rapidly and accurately diagnosed and treated. The diagnosis of soft tissue infection is frequently clinical and often delayed. Clinical and laboratory findings sometimes lack sensitivity and specificity, and a definite diagnosis may not be possible. In some situations, imaging is performed to confirm the diagnosis, evaluate the extent of the disease, and support the treatment planning.

Radiography generally represents the primary examination in patients with soft-tissue infections. The main findings pointing to an inflammatory process include soft tissue swelling, effacement of fat planes, and skin interruption in the setting of deep ulcers. These findings are nonspecific and can be found in other settings (e.g., trauma, systemic causes of subcutaneous edema, venous insufficiency, deep venous, thrombosis). Plain radiography can identify the presence of gas in the soft tissue or the foreign bodies, which usually supports underlying infection.

After initial radiography, US can play an important role in the first-line management of musculoskeletal infections. US can exclude non-inflammatory causes of soft tissue swelling, such as deep venous thrombosis or soft tissue tumors. As aforementioned, US also provides a real-time guidance for interventional procedures, such as tissue sampling or immediate needle aspiration of liquid collections.

Cross-sectional imaging, including CT and MRI, provide detailed anatomic information in the evaluation of soft tissues because of their intrinsic high spatial and contrast resolution. Intravenous contrast media can be used to better define extent of disease, delineate abscess collections or sinus tracts, and identify non-enhancing devitalized soft tissue.

Imaging findings of soft-tissue infections can be nonspecific and can overlap with noninfectious processes. In fact, it is crucial to combine imaging with the clinical history and laboratory findings.

Cellulitis is an acute inflammation of the skin and superficial subcutaneous tissues. *Staphylococcus aureus* and *Streptococcus pyogenes* are the most common offending agents gaining access through the disrupted areas of skin (cuts, cracks, or surgical wounds). Radiographs show nonspecific diffuse soft tissue swelling. US gray-scale imaging findings are also nonspecific, showing subcutaneous tissue edema with hypoechoic stranding insinuated between the echogenic fat lobules. Increased vascular flow at color or power Doppler US may also be seen. MRI demonstrates diffuse linear or ill-defined soft-tissue thickening with hyperintensity at T2-weighted imaging and STIR imaging, hypointensity at T1-weighted imaging and postcontrast enhancement. CT may show infiltration of the subcutaneous fat and identify underlying abscess. Local lymphadenopathy can be associated.

Infectious tenosynovitis refers to infection of a tendon and its protective sheath. The most common sites of this affection are the finger, hand, and wrist. Main causes of tenosynovitis are infection, systemic inflammatory arthropathy, crystal deposition (e.g., gout, pseudogout), or overuse. Patients usually present with tenderness, swelling, erythema, and painful ROM of the affected tendon. The presence of gas or complex tenosynovial fluid orientate for infectious cause, while multiple joint

involvement (e.g., wrist) with associated tenosynovitis favors inflammatory or crystal-induced arthropathy. MR imaging is the modality of choice in the evaluation of tenosynovitis. It demonstrates fluid distending the tendon sheath, thickening of the synovial sheath, and intense enhancement on postcontrast images. Tendons lose their low signal intensity and may look thickened and show intermediate signal intensity. However, it is not sensitive in differentiating the causes of tenosynovitis. US may show fluid distention of the tendon sheath and hypervascularity of a thickened synovium.

Septic bursitis is caused by direct bacterial inoculation of the synovial bursa, *Staphylococcus aureus* being the most common pathogen found. The most commonly affected superficial bursae are prepatellar and olecranon bursae, often due to direct transcutaneous inoculation due to penetrating injury. Deep bursal infection is less common, generally caused hematogenously. Clinical manifestations include tenderness over the inflamed bursa, soft tissue swelling and erythema, fever, and local lymphadenopathy. US, CT, and MR imaging show thickened bursal wall, bursal distention with infected fluid (complex fluid with debris), inflammatory edema within the adjacent soft tissues, and sometimes gas bubbles (reliable sign to differentiate septic from non-septic bursitis). However, a horizontal fluid level in a bursa may indicate fracture ("sail sign" in elbow x-ray from blood in bursa).

Necrotizing fasciitis (NF) is a very serious condition caused by rapidly progressive infection (mono-bacterial vs polymicrobial) of the deep soft tissue with a high mortality rate. The main risk factors are injectable drug abuse, chronic diseases (e.g., diabetes mellitus, immunosuppression, obesity), and peripheral vascular comorbidities. The most commonly affected sites are the extremities, followed by the perineum, trunk, and head-and-neck area. Patients present with signs and symptoms of sepsis, including fever, hypotension, and multiorgan failure (MOF). MR imaging is the modality of choice in demonstrating deep fascia involvement. The characteristics of NF include (*i*) extensive involvement of the deep intermuscular fascia, (*ii*) thickening of fascia measuring 3 mm or more at STIR or T2-weighted fat-suppressed imaging, and (*iii*) involvement of three or more compartments. Soft tissue gas might be present and has a high specificity for NF.

4.3 Arthritis

Derives from *"arthros,"* joint, and *"itis,"* inflammation, inflammation of joint. Clinically joints appear stiff and warm, with swelling, redness, and pain.

Types of arthritis:

- *Degenerative* arthritis*/ osteoarthritis* – (*i*) primary (idiopathic/spontaneous), associated mainly with aging, a degenerative condition affecting mostly joints that bear a lot of weight like the hip, knee, and interphalangeal joints as well as the spine (Fig. 57); (*ii*) secondary caused by previous injury to affected bone and can begin at young age. There is no abnormality in laboratory studies.

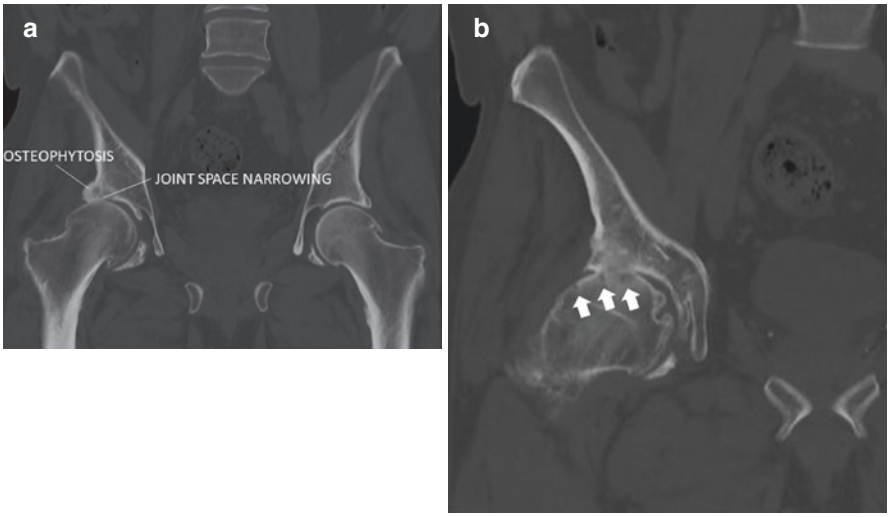


Fig. 57 60-year-old patient CT scan of the pelvis with MPR in coronal plane and bone window: osteoarthritis of the hip, more advanced on the right side. We can find joint space narrowing, sclerosis, osteophytosis (**a**), and subchondral cyst also called geode (**b**; arrows)

- *Inflammatory arthritis* – RA, psoriatic arthritis (PA), ankylosing spondylitis, Reiter syndrome, erosive osteoarthritis.
- *Metabolic arthritis* – (*i*) Gout caused by deposition of monosodium urate monohydrate crystal; (*ii*) pseudogout caused by deposition of calcium pyrophosphate crystal and hydroxyapatite deposition disease or the rare tumoral calcinosis (benign).
- *Infectious arthritis* – As aforementioned, septic arthritis is a life- and limb-threatening bacterial infection.
- *Connective tissue arthritis* – Systemic lupus erythematous (SLE).

Common radiological features of arthritis are soft tissue swelling, subchondral sclerosis, joint space narrowing, joint effusion, formation of osteophytes (degenerative bony outgrowth continuous with underlying cortex), subchondral cystic lesions, and periarticular osteoporosis.

X-ray is the most widely used investigation, very useful in detecting disease distribution and monitoring treatment response. However, x-rays are not very sensitive in the early stage of the disease.

US is used mainly in the detection of joint effusion, synovial thickening, and hypervascularity as well as in the monitoring of disease progression.

CT has only a limited role, while MRI is the gold standard for synovial imaging, for early detection of erosions and for bone marrow evaluation.

Main approach for arthritis assessment: "**ABCDEs**":

- **A**lignment
- **B**one density
- **C**artilage/bone space

- **D**istribution
- **E**rosions
- **S**oft tissue changes

4.4 Tumors and Tumor-Like Lesions

Bone tumors are a relatively rare finding in musculoskeletal radiology [32]. When evaluating osseous lesions, the radiologist's main goal is to assess whether the lesion is benign or aggressive in appearance and whether further workup is necessary. The list of potential osseous lesions is substantial; this review of bone tumors does not include metabolic or degenerative lesions. To provide a meaningful differential diagnosis to the referring clinician, several features of every osseous lesion should be generally assessed.

Usually, seven radiographic features of a bone lesion should be examined.

– Zone of transition (ZOT)
– Presence or absence of periosteal reaction
– Location in the bone (transversal and longitudinal)
– Pattern of cortical destruction
– Age of the patient and associated symptoms (pain)
– Matrix
– Number of lesions (mono-ostotic or polyostotic)

The differential diagnosis mainly depends on the review of the conventional radiographs (being the most useful examination) and the age of the patient.

The most significant determinants in the analysis of a potential bone tumor are the following:

– MORPHOLOGY of the bone lesion on a plain radiograph (well-defined osteolytic; ill-defined osteolytic; sclerotic)
– AGE of the patient (<30 years old vs > 30 years old)

However, further imaging and clinical clues need to be considered (ZOT, periosteal reaction, localization within the skeleton, cortical destruction, matrix, etc.).

The ZOT represents the outer margin of the lesion that corresponds to the change from pathologic to normal bone. A wide ZOT manifests when the lesion cannot be clearly outlined or circumscribed; this feature is usually associated with an aggressive lesion (malignancies such as Ewing's sarcoma and osteosarcoma). However, infections and other benign processes can also present wide margins (eosinophilic granulomas). The ZOT only applies to osteolytic lesions since sclerotic lesions usually have a narrow transition zone. Remember that a wide ZOT is not directly associated with malignancy, but it is very rare for a narrow ZOT to be associated with any other condition than a benign lesion.

- Patients less than 30 years of age with a narrow ZOT ⟹ Benign
- Patients with >40 years of age with well-defined lesions ⟹ Metastases and myeloma must be considered in the differential diagnosis

Periosteal reaction is often subtle and can mislead the radiologist attempting to classify a lesion as aggressive. It is a nonspecific reaction and can occur whenever the periosteum is irritated by a malignant tumor, benign tumor, infection, or trauma (Fig. 58).

There are two patterns of periosteal reaction: benign (solid) and aggressive type [33]. Detecting a benign periosteal reaction may be very helpful, since malignant lesions never cause a benign periosteal reaction.

A benign type of periosteal reaction is a thick, wavy, and uniform callus formation resulting from chronic irritation.

Typical aggressive-appearing periostitis is described as having an "*onion-skin*," "sunburst," or "hair-on-end" appearance.

The *location* of the lesion (transversely and longitudinally) might be helpful in narrowing the differential diagnosis. For example:

– Eccentrical location and cortex involvement (osteoid osteoma, parosteal osteosarcoma, and non-ossifying fibroma (NOF))
– Epiphysis involvement (chondroblastoma)
– Central location (solitary bone cysts, enchondromas, and Ewing's sarcoma)

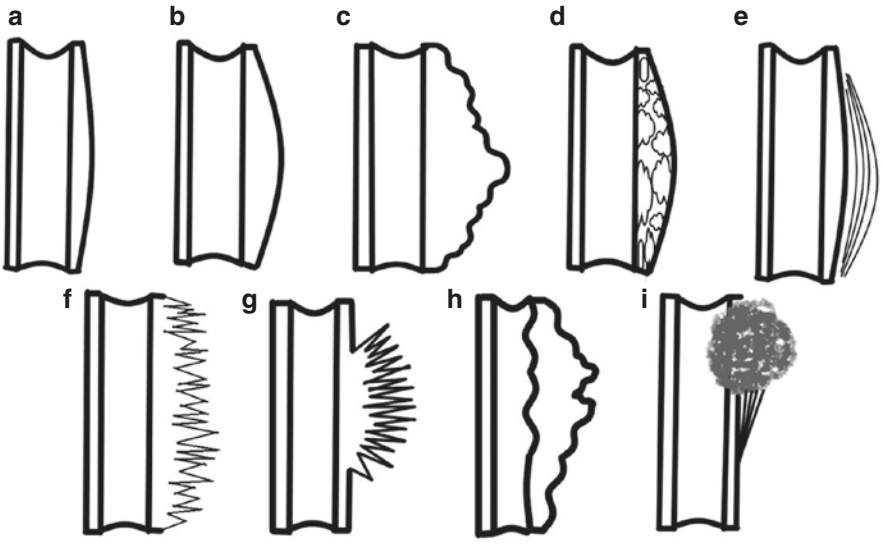


Fig. 58 Periosteal new bone formation: (**a**) solid thin; (**b**), solid thick; (**c**), ondulated; (**d**), soap bubbles; (**e**), multilayered (onion skin); (**f**), spiculated; (**g**), sunburst; (**h**), irregular; (**i**), Codman's

Pattern of cortical destruction provides a clue for determining the growth rate of an osteolytic lesion and its aggressiveness. It is not the most helpful finding in distinguishing between malignant and benign lesions.

Several other characteristics, such as the presence of sclerotic margins, soft tissue involvement, endosteal scalloping, and the pattern of matrix (osteogenic/cartilaginous) can also support the differential diagnosis.

Suspected soft-tissue extension of an osseous lesion should be further characterized with contrast-enhanced MRI in order to determine not only tumoral extent but also the risk of complications (e.g., neurovascular compromise).

MRI has the potential to narrow the list of differential considerations by showing cystic or necrotic components, encapsulation, contrast enhancement, and the presence of fluid levels or perilesional edema on MRI.

Benign bone lesions*:*

1. Solitary bone cysts
2. Aneurysmal bone cysts (ABC)
3. Fibrous dysplasia
4. Non-ossifying fibroma (NOF)
5. Giant cell tumor
6. Eosinophilic granuloma
7. Enchondroma
8. Osteochondroma

Malignant bone lesions*:*

1. Osteosarcoma
2. Ewing's sarcoma
3. Chondrosarcoma
4. Undifferentiated pleomorphic sarcoma (malignant fibrous histiocytoma) and fibrosarcoma
5. Metastatic disease and myeloma

Lytic Bone Lesion Mnemonic – a good start for remembering a differential for a lytic bone lesion.

FOG MACHINE

- **F**ibrous dysplasia
- **O**steoblastoma
- **G**iant cell tumor
- **M**etastasis myeloma
- **A**BC
- **C**hondroblastoma
- **H**yperparathyroidism
- **I**nfection
- **N**OF
- **E**osinophilic granuloma enchondroma

Sclerotic Bone Lesion Mnemonic
I VINDICATE
- **I**atrogenic
- **V**ascular
- **I**nfection
- **N**eoplasm
- **D**rugs/degenerative
- **I**nflammatory/idiopathic
- **C**ongenital
- **A**utoimmune
- **T**rauma
- **E**ndocrine

Sclerotic bone neoplasms include both lesions with osteoid matrix (e.g., osteosarcoma, osteoid osteoma, osteoblastic metastases) and with chondroid matrix (e.g., enchondroma, chondrosarcoma, osteochondroma).

Educational Websites
- http://www.learningradiology.com
- http://uwmsk.org/RadAnatomy.html
- https://radiopaedia.org/encyclopaedia/all/musculoskeletal
- https://skeletalrad.org/web-resources
- http://www.radiologyassistant.nl/en/
- http://www.wikiradiography.net
- http://xrayhead.com
- https://www.radiologymasterclass.co.uk/
- https://www.acr.org/QualitySafety/Appropriateness-Criteria
- https://www.essr.org/subcommittees/ultrasound/
- https://www.appliedradiology.com/articles/bone-tumors-and-tumor-like-conditions-of-bone
- http://www.mrisafety.com/

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Part III

Other Imaging & Q Bank

Vascular, Interventional Radiology, and Interventional Oncology

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Contents

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1 Introduction

Whereas the other chapters mainly cover diagnostic radiology, here the matter is really different!

Interventional radiology is a branch of medical radiology that includes all invasive or minimally invasive diagnostic and therapeutic procedures performed through imaging guidance and control, such as fluoroscopy, CT, CBCT, MR, fusion, or US. The goal of interventional radiology is to achieve equal or better results, costs, recovery time, or efficacy, morbidity, and mortality than the analogous surgical procedure, with the advantage of a minimally invasive approach.

The basis of interventional radiology is the knowledge of the specific tools and devices and how to apply those tools toward specific clinical needs with a reliance upon expert knowledge of anatomy, imaging, and common anatomic variations and anomalies. This really is as important as knowing the clinical aspect of the job (Figs. 1, 2, and 3). IR and IO are clinical specialties, with a special independent training pathway (recent creation of independent residency in the US). IR and IO rely upon clinical knowledge, hands-on clinical care and patient clinics, and a foundation of diagnostic radiology, radiology physics, and an expert skill in image

Fig. 1 Protection from X-rays involves the use of lead aprons and thyroid shields

Fig. 2 Standard angiographic table

interpretation, often used in real time to perform surgical-like procedures, with video game-like guidance and navigation, manipulating tools at the skin, via needle or catheter-sized holes, while watching on a monitor which sometimes displays multiple modalities fused on one image with the device for exquisite localization and manipulation.

Needles The outer diameter of the needles is measured in gauges (G) and the most used range from 22 G (0.91 mm) to 16 G (1.63 mm) (Fig. 4). The diameter of the inner lumen is instead measured in inches.

A common feature is the sharp stylet and the coaxial structure with two or three elements. Coaxial means "needle within a needle." Exchanging needles and wires and catheters of different sizes, one after the other, is a core IR technique.

Thinner needles (<20G) are considered almost atraumatic and are normally used for diagnostic purposes that involve highly vascularized organs such as the liver and kidney (percutaneous transhepatic cholangiography, percutaneous pyelography, nephrostomy or biliary drainage, cholecystostomy, or biopsy, for cytology analysis).

Larger needles (19-14G) are used to obtain specimens for histological examinations, called "core biopsy" because it samples a larger core of tissue (typically an 18G core biopsy gun, via a 17 G outer needle).

Fig. 3 Standard modern angiographic suite with a C-arm angiography capable of CBCT = cone beam CT, with a spinning fluoro C-arm, and creates CT-like images (with smaller field of view)

Guides The second step after having obtained access to the structure or organ of interest is to position in the lumen of the needle/cannula or guidewire.

The caliber of guidewires are measured in inches and range from 0.014 to 0.045 (Fig. 5), but the most common size in interventional radiology is 0.035/0.038 and 0.018. Guidewires are measured by outer diameter, whereas catheters may report inner lumen and outer diameter (such as a 4 French outer cobra catheter with an 0.035 inch lumen) [3 French = 1 mm]. Sheaths are often measured by their inner lumen (5F sheath receiving a 4F catheter). This 5 French sheath is the outer element of the system, but the 5 French refers to the inner lumen. Size control and compatibility can be critical.

Many different guidewires are available:

Standard: These are metal guides usually covered with a Teflon coating that increases smoothness.

Hydrophilic Guidewires: The main advantages of this type of guide are elasticity and slippery smoothness. These guides often make it possible to overcome severe stenosis of blood vessels, bile ducts, or urinary tracts. The tip can be straight or "J type." They can be challenging on which to hold or secure.

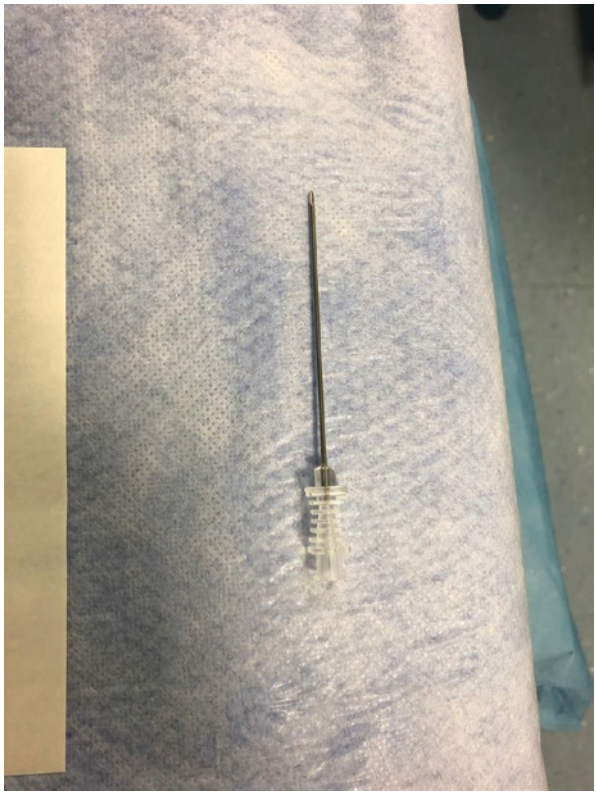

Fig. 4 Standard 18 G single-wall puncture needle for arterial and venous access

Fig. 5 (**a**). common hydrophilic 0.35″ j-tip glide-wire guidewire; (**b**). 2.7F coaxial microcatheter

"Amplatz Type" Metal Guidewires: They are very rigid in the proximal portion and gradually softening in the last 6–10 cm. They are widely used in the most complex interventional radiology procedures because, thanks to their high stiffness and greater stability, they provide safe and stable access to catheters, biliary and urinary prostheses of big caliber (10–16 F), without buckling.

Microguidewires: Their caliber is lower than 0.025." They are particularly useful when it is necessary to navigate in the peripheral vascular systems (liver, intracranial, iliac vessels, gastrointestinal) and usually require coaxial catheter systems (e.g., microcatheters), of 3F caliber or less. A 2.8, 2.7, or 2.4 or 2.0 French microcatheter receives an 0.018 or 0.014 inch microwire. Both fit through an 0.035 inch lumen catheter (typically 4 or 5 Fr) via a Y-adapter with a flushable valve, called a Tuohy Borst adapter.

Introducer Sheaths These are fundamental tools that allow you to maintain safe access to the vascular, biliary, or renal systems. They consist of two parts: an inner straight stiff catheter (usually 15 cm long), with a very tapered tip, called dilator, and an outer often Teflon sheath (normally 10 cm long) (Fig. 6).

After having placed them into the artery over the wire via Seldinger technique (usually with a 21G needle micropuncture system under ultrasound guidance), only the sheath is left in place. The introducer sheaths are valved to avoid blood reflux during catheter manipulation and usually have a side tube with a three-way stop cock that allows flushing and continuous perfusion of the sheath. Their caliber is measured in French (3–14F).

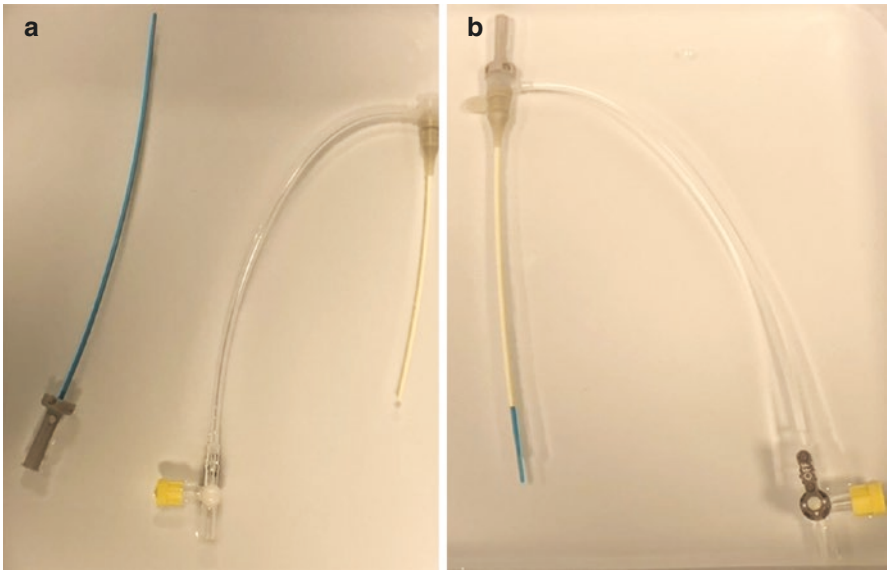

Fig. 6 Standard 5F angiographic sheath

Catheters Their main features are the following:

- The external diameter (measured in French, which varies from 2 to 5).
- The length, which varies from 60 to 120 cm, or longer for "exchange length" which allows exchanging of catheters without moving the wire internally.
- The shape of the tip. The most common forms are shown in Fig. 7 and include cobra, hockey stick, Simmons, and pigtail.

Special Catheters They have a specific therapeutic use

- Occlusion balloon catheters: they consist in a roundish soft compliant balloon placed at the tip of the catheter, which can be inflated through a double-way stop cock; they are used for vascular occlusions or to remove biliary or urinary stones. The balloon diameter varies from 8 to 40 mm and gently and softly conforms to the size and shape of the vessel.
- *Angioplasty balloon catheters*: the balloons, when dilated, have a variable diameter from 2 to 40 mm and can be inflated up to pressures of 18 atm (Figs. 8 and 9). They are non-compliant, which means that with an increase of pressure, the balloon will experience only a small change in diameter. It is stiff when fully inflated to a certain size and is used to intentionally crack open a stenosis (a therapeutic dissection) or expand a balloon-expandable stent, covered stent, or stent graft.
- *Drainage catheters*: they are normally made in polyethylene, polyurethane, or even silicone; the most common shape of the tip is "J" or "pig tail." Near the distal tip and in the distal straight portion, they usually have holes that allow an

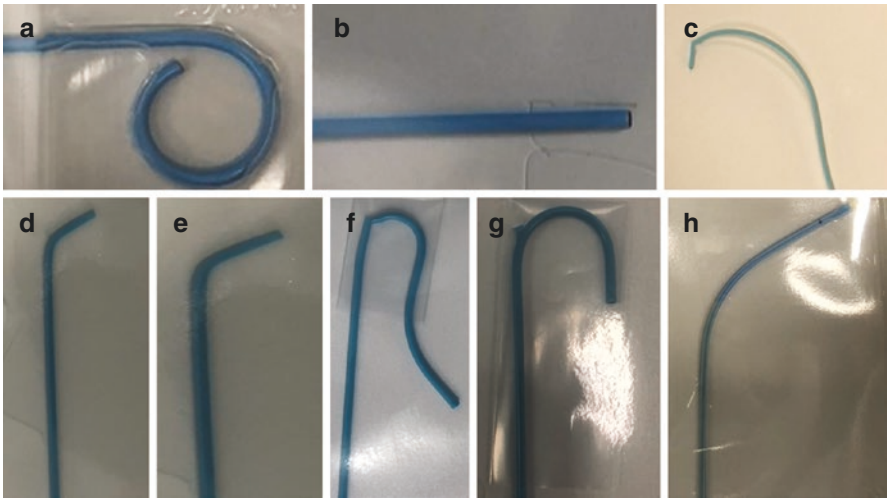

Fig. 7 Common vascular catheter shapes. (**a**), "pig-tail"; (**b**), straight; (**c**), Cobra; (**d**), Ber; (**e**), Vertebral; (**f**), Simmons; (**g**), "J tip" or RC1; (**h**), Multipurpose (MPA)

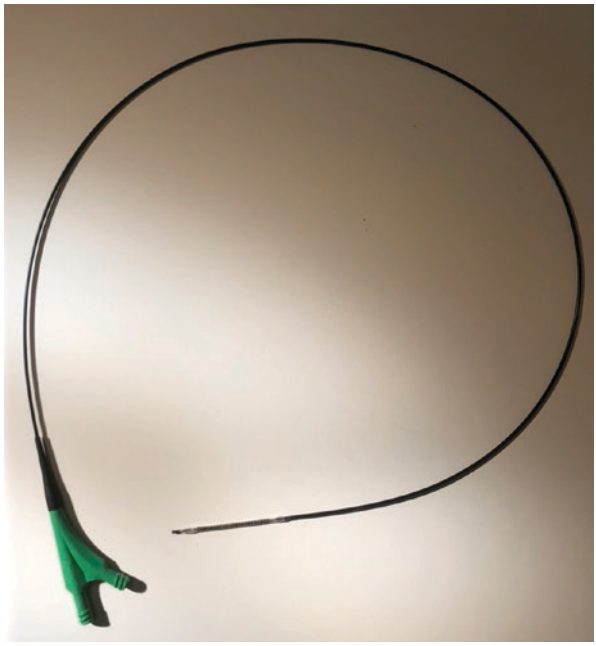

Fig. 8 Angioplasty balloon catheter

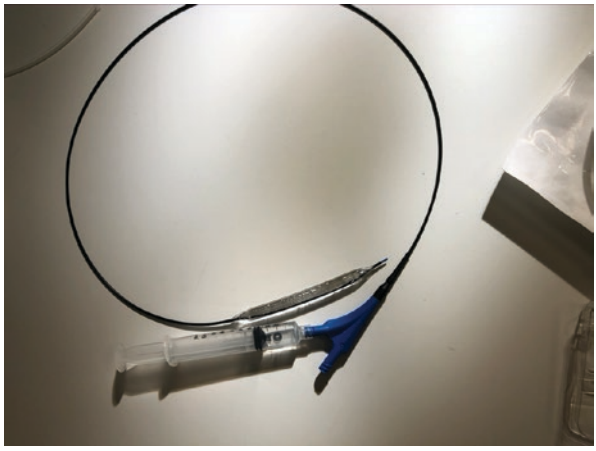

Fig. 9 Inflated angioplasty catheter

easy drainage or discharge of the material to be drained (pus, urine, bile, seroma, lymphocele, stomach, bladder, etc.).

Metallic Stents They are particular types of prosthesis that can be positioned in the blood vessels and biliary tract in case of stenosis in order to restore patency. In the arterial vascular environment, they are used to complete angioplasty (PTA) in selected cases (Fig. 10).

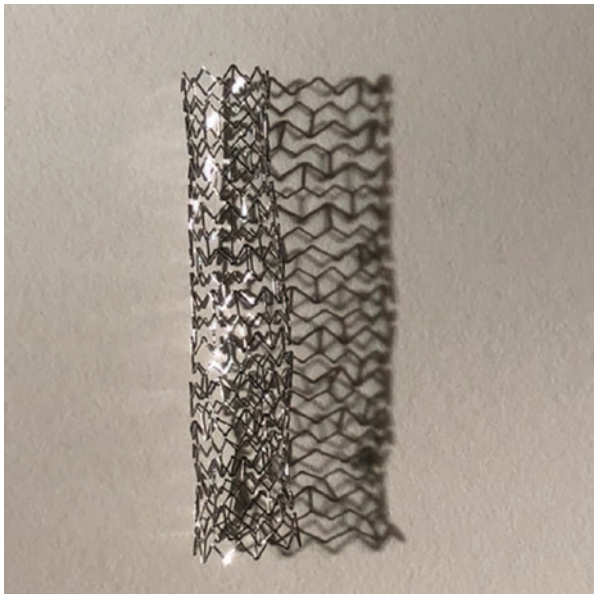

Fig. 10 A bare metal stent

In the venous system, their use is more limited (such as for SVC syndrome or stenosis of an arteriovenous dialysis graft whose flow rates reduced from an efferent stenosis on the venous side).

Covered stents (stent grafts): they are extensively used to treat conditions such as aneurysm or pseudoaneurysm of the thoracic and abdominal aorta, arteriovenous fistulas, traumatic perforations, or complications or tumors in the biliary tract.

Inferior vena cava filters Inferior vena cava (IVC) filters are percutaneous devices designed to prevent the onset of pulmonary embolisms (Fig. 11). Absolute indications for IVC filter placement is the presence of DVT or PE with any of the following conditions [1]:

- Contraindication for anticoagulation.
- Recurrent PE in spite of anticoagulation.
- Anticoagulation-related complication.

IVC filter use may be controversial in some settings, and temporary placement is amendable to later removal, to avoid chronic IVC syndromes.

Embolizing agents This includes a large group of tools and agents used in interventional radiology to occlude venous or arterial vessels both as an elective or in an emergency setting.

The embolic agents are divided into temporary or definitive. Among the temporary ones, the most used material is the gelatin sponge which, in a mixture with iodinated contrast media, may achieve hemostasis. Gelatin sponges are usually

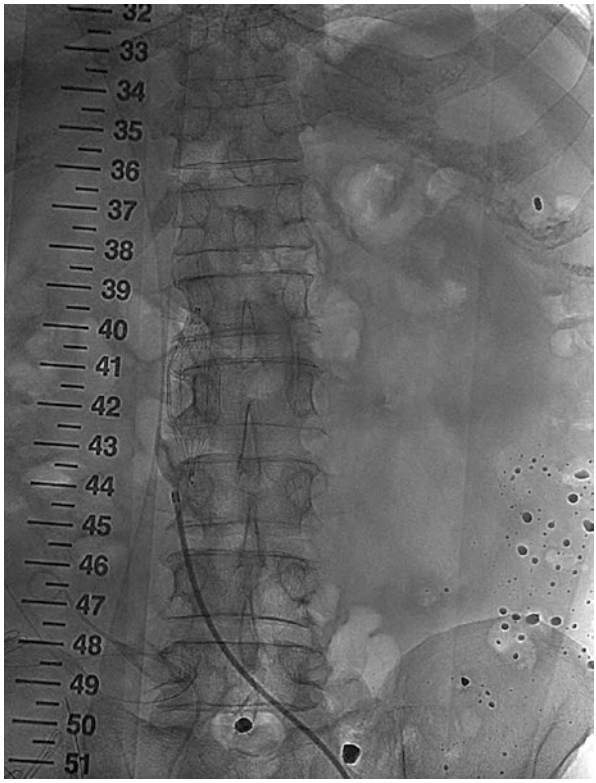

Fig. 11 Inferior vena cava filter placement via a left femoral vein approach

absorbed completely within four to six weeks; however, any temporary occlusion may become permanent.

Among the definitive ones, polyvinyl alcohol microparticles (which come in different sizes: from 40 to 900 μm) may be radio-opaque or drug eluting. Drug eluting beads (usually delivering doxorubicin or irinotecan at present) may also deliver therapeutics, most commonly in TACE (chemoembolizations) for liver cancer. Y-90 glass micro-spheres or lipiodol oil may also be delivered via catheters, typically for liver cancer as well.

Other embolics include acrylic glue, a derivative of isobutyl cyanoacrylate, which is usually mixed with an oily contrast agent to make it visible and more controllable, AVPs (Amplatzer Vascular Plug) which consist of mesh disks of braided nitinol, and a wide variety of platinum or steel metal coils in various shapes, with or without added fibers. Some are deployable and immediately removable, if not perfectly placed. Coils are the most commonly used for stopping bleeding.

Coils, in particular, have been for decades the pillar of vascular interventional radiology. They are made in platinum or steel (covered or not with Dacron to strengthen the embolizing effect) and are available in different shapes and sizes, to be compatible with specific catheter systems.

2 Vascular Accesses

In general, interventional radiologists are responsible for the positioning and management of long-term venous access devices (LTVADs) (Tesio, Hickman, Groshong-type CVC, vascular access in hemodialysis, or apheresis) and totally implantable venous access devices (TIVADs) such as Port-a-Cath (Fig. 12).

There are no clear indications in the literature regarding when to place a long-term CVC or which device to use. However, there are important factors to consider:

- Frequency and duration of therapy.
- Nature of therapy.
- Need for supportive care.
- Need to perform bone marrow transplant or to collect stem cells.
- Patient preference.

Before the procedure, it is necessary to accurately evaluate the vascular anatomy or to review prior imaging to be absolutely certain of the patency of the access vein and to exclude venous stenoses.

These procedures are considered "clean" and require maximum attention to sterile technique [2].

Before the procedure (<30 days), the operator should check the patient's allergies and relevant laboratory values, particularly coagulation parameters, such as prothrombin time and platelet count. Current recommendations are to avoid venipuncture in patients with platelet counts <50,000 or INR > 1.5 and to hold antiplatelet medications, such as Plavix. It may not be necessary instead to hold aspirin [3]. Safe vascular access may be performed with lower values, however, with careful ultrasound technique, but risks go up.

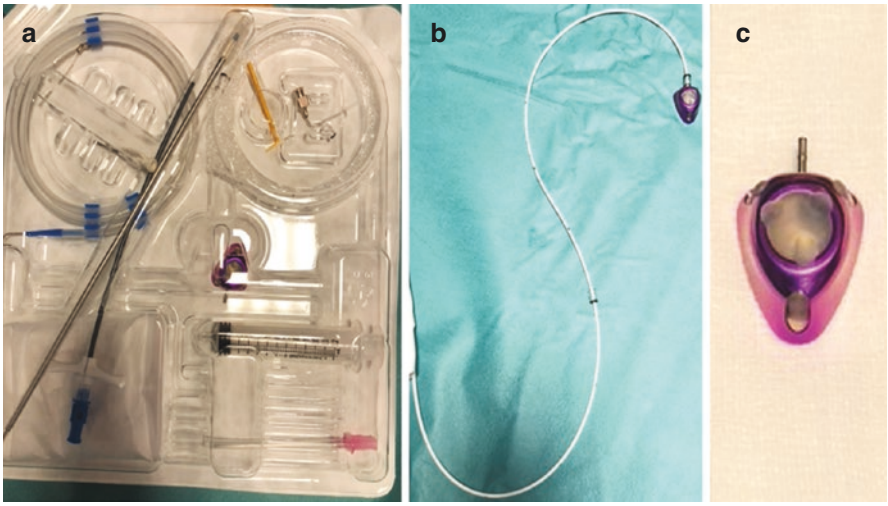

Fig. 12 A Port-a-Cath, the most common totally implantable venous access device (TIVAD) (**a**), a complete Port-a-Cath set; (**b**), Port-a-Cath reservoir attached to its catheter; (**c**), a close-up of a Port-a-Cath reservoir

3 Vascular Interventional Radiology

In vascular interventional radiology (IR), we are dealing with two very different worlds: the arterial and the venous system. Both types of procedures must be preceded by a preliminary vascular diagnostic examination: the angiography (or phlebography). It consists in the injection of a iodinate contrast medium through a diagnostic catheter into the vessel that needs to be studied.

The access to the arterial vessel is performed using the technique introduced in 1953 by the Swedish radiologist Dr. Sven Ivar Seldinger.

4 Arterial System

4.1 Visceral Arteries

There are several causes and manifestations of an inadequate blood supply to the splanchnic district. The first distinction must be made between acute and chronic forms, based on the onset and the clinical characteristics.

The most common cause of chronic mesenteric arterial steno-occlusive disease is atherosclerosis, which accounts for 35–75% of cases. In most cases, the etiology of acute forms is a thromboembolic disease. The main clinical picture has been describes as "abdominal angina." Because of the extensive mesenteric arterial collateral network, chronic mesenteric ischemia (CMI) (Fig. 13) usually occurs only

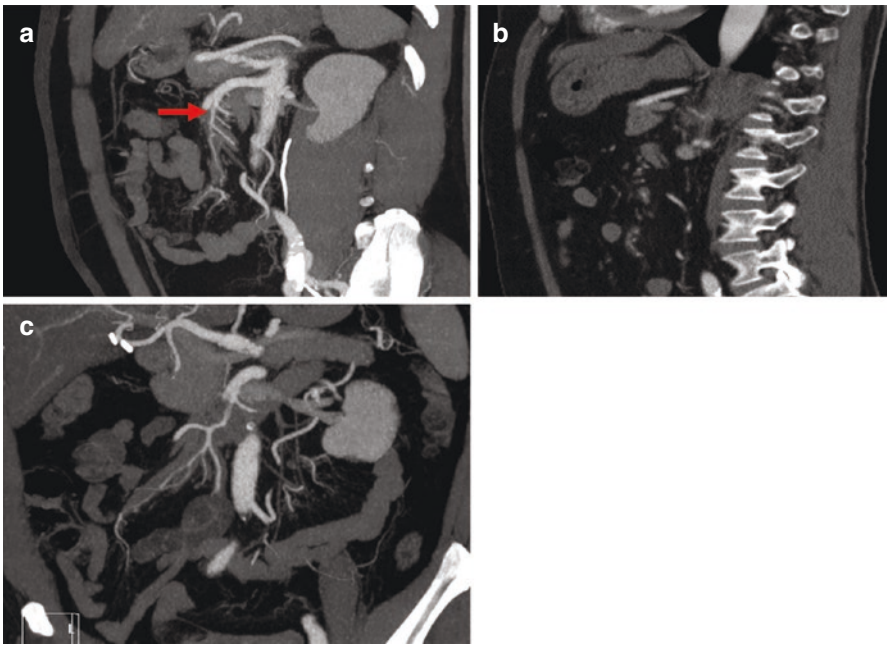

Fig. 13 CT angiography of the upper abdomen. A, *arrow* a dissection flap in the proximal part of the superior mesenteric artery that causes chronic abdominal pain

when there is a severe stenosis or an occlusion of at least two of the three visceral arteries. Therefore, the indications for mesenteric angioplasty and/or stent placement are a symptomatic patient (unintentional weight loss, postprandial abdominal pain, and/or food aversion) with at least two-vessel disease on imaging or aortic/mesenteric artery dissection that impairs mesenteric perfusion.

4.2 Arteries of the Lower Limb

The obstructive peripheral artery disease represents the most frequent localization of arteriosclerotic disease. It mainly affects males over 65 years of age, with risk factors like smoking, hypertriglyceridemia, hypercholesterolemia, hypertension, and diabetes. The anatomical localization most prone to disease are the aortic bifurcation, the common iliac artery (with consequent compromised sexual function and *claudicatio glutea*), the superficial femoral artery in its middle-distal tract, the popliteal artery, and the so called "below the knee" vessels (generally involved in diabetic patients and/or with Buerger disease).

In addition to the physical examination, a mainstay in the diagnosis of obstructive peripheral artery disease lies in the CT angiography (Fig. 14).

Angioplasty can be defined as a procedure with a balloon-tipped catheter with the aim to enlarge a narrowing in a stenotic artery. The balloon is placed in the middle of the obstruction and inflated until the pre-established size. In case of insufficient dilation or damage to the vessel wall, a metallic or covered stent can be deployed [4].

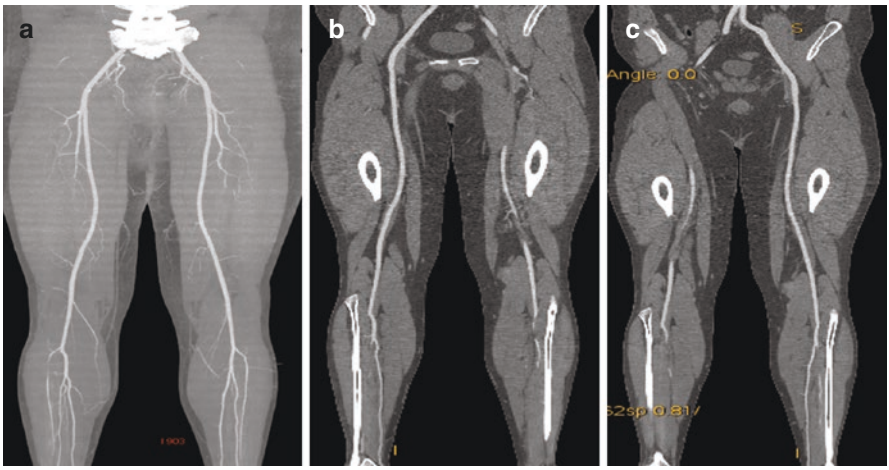

Fig. 14 CT angiography of the lower limbs, almost normal findings. Maximum intensity projection (MIP) coronal reconstruction with stenosis of the right anterior tibial artery refers to figure (**a**). almost normal findings refers to figure (**b**) and (**c**)

4.3 EVAR/TEVAR

An aneurysm is defined as an increase in the diameter of the vessel above 50% of its normal proximal tract size. Aortic aneurysms have a multifactorial etiology that includes risk factors like cigarette smoking and hypertension associated with atherosclerosis and inflammatory processes. For most aortic aneurysms, treatment is indicated at a diameter of ≥5 cm. The endovascular procedure consists in positioning a stent-graft or endoprosthesis to exclude the aneurysmal segment (sealing) from the blood stream (Fig. 15). The sealing is associated with the exclusion of the aneurysmal sac from the arterial flow, and it is mediated by the adhesion of the proximal and distal tract of the endoprosthesis to the non-dilated vessel's wall. This causes the consequent formation of a stratified thrombus into the flow excluded part.

The stent-graft or endoprosthesis consists of three main elements: a metallic skeleton (stent) generally in nitinol, an internal or external coating (graft) in polytetrafluoroethylene (PTFE), and a delivery system.

With these devices, it is possible to treat the aneurysms of the thoracic aorta (TEVAR) through the positioning of a single device that can be extended up to the first visceral branches and aneurysms of the abdominal subrenal aorta (EVAR) through the positioning of an inverted Y endoprosthesis with the two distal branches anchored to the iliac arteries.

4.4 Hemorrhagic Pathology

In an emergency setting, transarterial embolization are common procedures, and IRs must be ready to deal with these problems! Bleeding can be caused by various

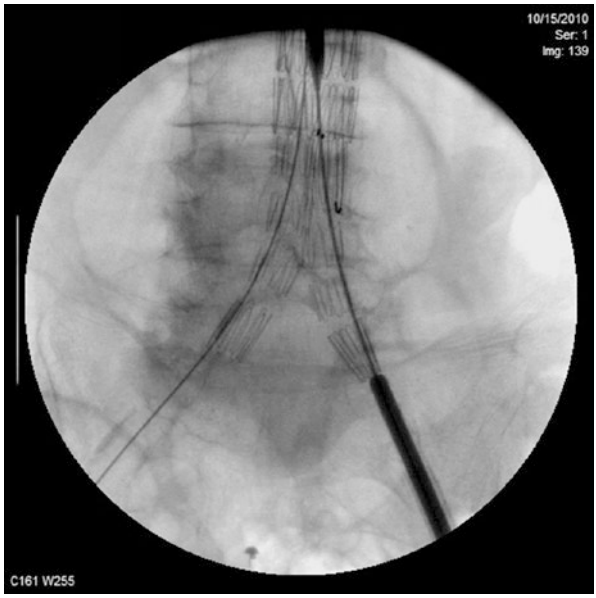

Fig. 15 Fluoroscopic image showing the positioning of a left iliac branch during an EVAR procedure

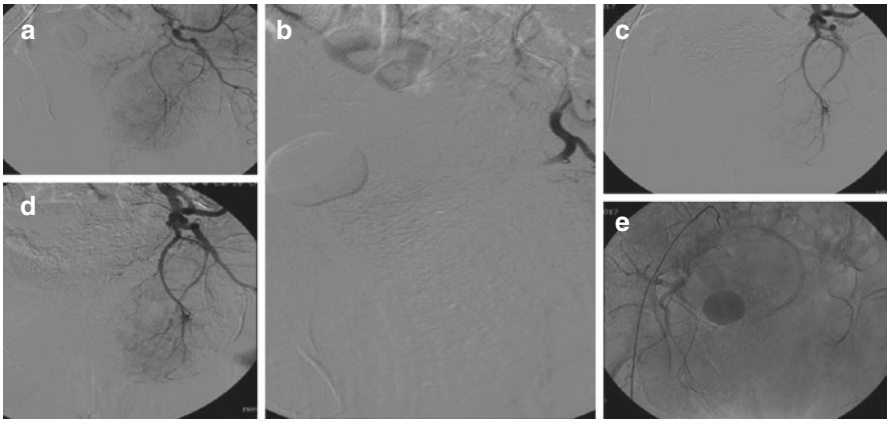

Fig. 16 Various phases of an lilac artery catheterization prior to PAE

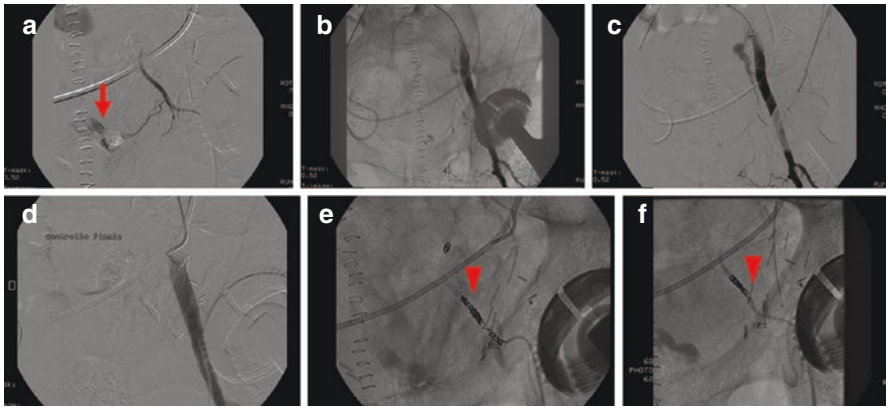

Fig. 17 Superficial circumflex iliac artery embolization after hip replacement surgery. (**a**), *arrow* active extravasation of contrast media represents active bleeding; (**e**, **f**), *arrowhead* coils deployment with the aim to stop the bleeding

conditions: polytrauma, stabbing or gunshot wounds, overdose of anticoagulant drugs, cancer, surgery, or iatrogenic. The first step is the identification of the bleeding vessel with a CTA exam. Subsequently, a selective arteriography of the vessel involved should be performed to confirm the bleeding (Figs. 16 and 17).

The bleeding site can subsequently be embolized with one or a combination of the agents mentioned above.

4.5 Tips

The treatment of acute or chronic bleeding from esophageal varices in cirrhotic patients is performed trough a very complex intervention, the transjugular intrahepatic portosystemic shunt (TIPS) (Fig. 18). The aim of this procedure is to reduce the pressure gradient between the portal vein and hepatic vein with the aim to bring

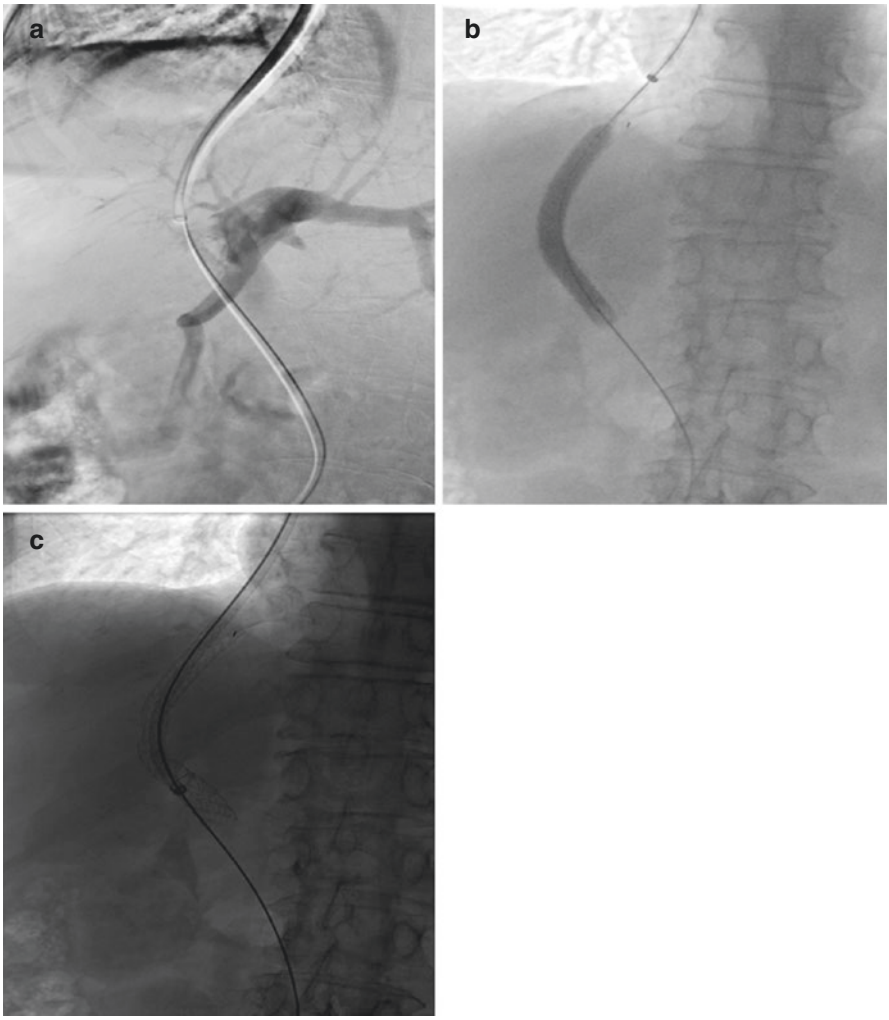

Fig. 18 Three key moments of a TIPS procedure. (**a**), metallic guidewire into the superior mesenteric vein, after having successfully managed to create a shunt between the systemic circle and the mesenteric circle. (**b**). an inflated angioplasty catheter with the aim do dilate the intrahepatic tract. (**c**), correct placement of the covered stent

it below 12 mmHg. It is performed through an internal jugular venous access, preferably right, and consists in positioning a stent-graft that connects the hepatic vein and the portal vein through a small tract of liver parenchyma. The main goal is often to decongest the esophageal portal varicose veins; furthermore, it allows more accessible further interventions in the portal system, and the varicose veins may also be embolized at a different time with a better bleeding control.

4.6 Uterine Artery Embolization – UAE

Uterine fibroids are non-cancerous growths of the uterus that are very common in the female population over 30 years of age (20–25% general population). Fibroids can be subserosal, intramural, or submucosal. Fibroids can develop as a solitary tumor or in multiple nodules with sizes ranging from 1 cm to 10 cm and even more. This condition can be treated with arterial embolization. The purpose is to interrupt the tumor arterial blood supply and to reduce the volume of the fibroid and therefore the patients' symptoms. This intervention is a valid alternative to surgery due to the minimal invasiveness approach. The treatment is carried out with a femoral arterial access and the subsequent injection via a microcatheter (2.7F) of microspheres with an average diameter of 500–900 μm inside the uterine arteries branches afferent to the fibroid [5]. Considerable attention must be paid to avoid non-target embolization (i.e., ovarian arteries to avoid early menopause).

4.7 Prostatic Artery Embolization – PAE

Benign prostatic hyperplasia (BPH) is present in about 80% of men between 70 and 79 years; symptoms may vary but in general they are known as lower urinary tract symptoms (LUTS). They consist of urinary irritative and obstructive disorders that impair the quality of life. The goal of prostatic artery embolization is to reduce the prostate volume with consequent resolution of symptoms. This minimally invasive approach implicates a short hospitalization time compared to the surgical gold standard (transurethral resection of the prostate) and prevents annoying side effects such as retrograde ejaculation. The technique consists in a femoral (or radial) arterial access and subsequent cannulation of the prostatic arteries with the injection of 300–500 μm microspheres until complete stasis of blood flow. The procedure is effective in improving BPH symptoms with improvement or resolution in 80% of patients [6].

5 Venous System

5.1 Deep Vein Thrombosis

Deep vein thrombosis (DVT) is the partial or complete obstruction of one or more veins in the deep circulation of the lower limbs, usually below the knee. The most feared complication of DVT is pulmonary embolism (PE) caused by migration of an

embolus from the deep vein thrombus. The severity of this condition is directly proportional to the size of the migrated thrombus. In order to prevent PE, in selected cases, it is possible to position a specific filter into the inferior cava vein below the renal vein through a jugular or femoral approach. Vena cava vein filters reduce mortality and morbidity of these acute events. Filters can be either permanent or temporary. Long-term sequelae of IVC filters have seen interest in recent years.

5.2 Varicocele Sclero-Embolization

Varicocele consists in a dilation of the pampiniform venous plexus of the left testicle (97% of cases). This condition affects approximately 9—15% of the male population and represents the most frequent cause of male infertility. The cause of the pathology is linked to the vertical outlet of the left spermatic vein into the renal vein with a failure of the vein valves and a subsequent congestion of the pampiniform plexus. This may cause an increase of the basal temperature of the testicles that implicate a compromised production of sperm. Therefore, the purpose of the procedure is to close this vein and its branches. The method that involves fewer recurrence rates is the sclero-embolization. It consists of the injection of a drug (polidocanol or sodium-tetradecyl-sulfate) mixed with air with a 2:1 or 3:1 ratio into the spermatic vein catheterized with a femoral or jugular access (Fig. 19). The advantages of this technique are a 3.6% recurrence rates that is comparable to inguinal microsurgery and a very low invasiveness.

6 Interventional Oncology

Interventional oncology is a branch of interventional radiology that deals with the diagnosis and treatment of cancer and cancer-related problems using targeted minimally invasive procedures performed under image guidance and has among its objectives the treatment, control of the disease, or palliation.

Available techniques can be divided into two main categories:

- Endovascular interventions.
- Percutaneous interventions.

7 Endovascular Interventions

The main application of endovascular treatment in interventional oncology is in liver lesions (both primary and secondary), through the injection of beads, oil, or other embolics, with a chemotherapeutic agent (or radiotherapeutic Y-90 glass spheres into the hepatic artery).

In both types of procedures, through guides and catheters, the vessel tributaries to the liver lesion have to be super selectively catheterized; in most cases, the hepatic artery arises from the celiac trunk, although anatomical variants are not uncommon (which is why a preoperative planning with a level II imaging is essential). Hepatic

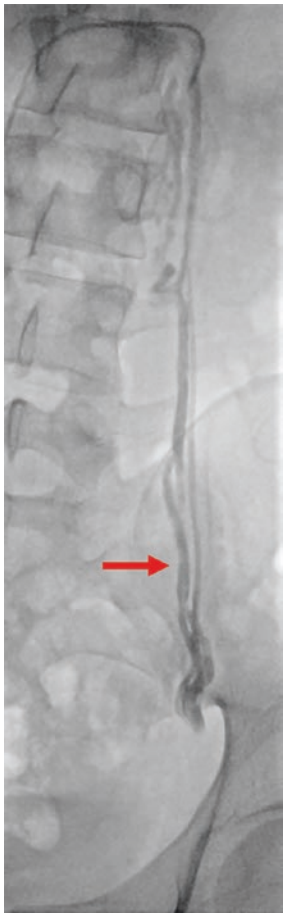

Fig. 19 *Arrow* phlebography of a dilated spermatic vein prior to varicocele embolization

arteries may be anomalous in 12–18% of patients and be "replaced" or "accessory." Replaced right hepatic artery comes off the SMA, and a replaced left hepatic artery comes off the left gastric artery, instead of the proper hepatic artery. The anatomy is key so you can avoid delivery or embolic or drug to the wrong location (gut instead of liver, which could cause ischemic ulcer). With the help of microcatheters (2.0–2.7F), the arterial branches afferent to the lesion to be treated can be super-selectively catheterized, and, at that level, the injection of the therapeutic agent can be eventually performed (Figs. 19 and 20).

When a chemotherapeutic agent is injected, the procedure is called TACE (transarterial chemoembolization), and it is subdivided mainly according to the agent that carries the drug:

- Conventional TACE (cTACE): the chemotherapeutic agent is mixed with an oily contrast agent, Lipiodol (ethiodized oil).
- Drug eluting beads TACE (DEB-TACE): the embolizing particles are previously loaded with the drug.

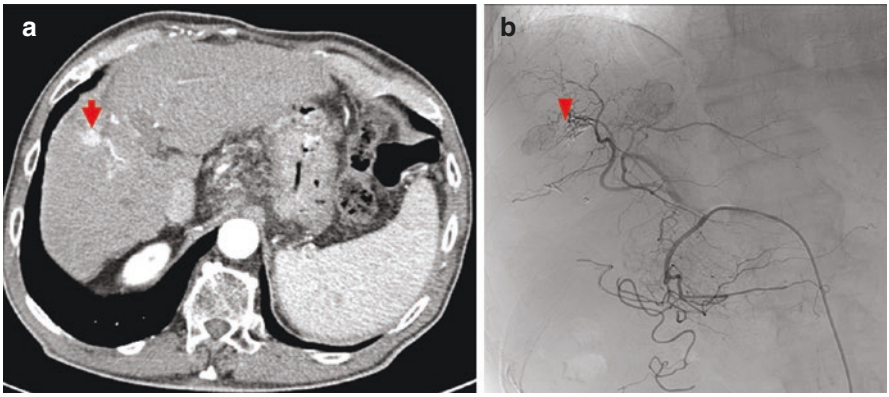

Fig. 20 (**a**), contrast CT scan, arterial phase, *arrow* shows a enhancing round mass with a capsule and rapid washout (shown on later phases) compatible with HCC (a LI-RADS score 4); (**b**), *arrowhead* shows the tip of a microcatheter ready to perform a chemoembolization

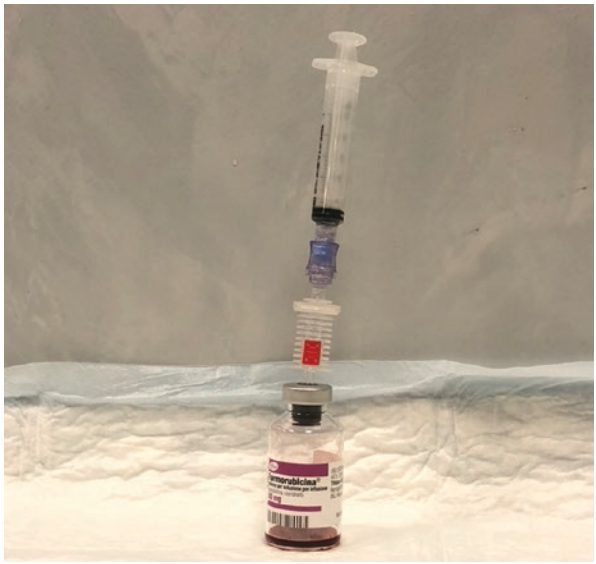

Fig. 21 Mixing of drug eluting beads and a chemotherapeutic agent prior to deb-TACE

Both techniques fulfill two main goals: first, ischemia, directly related to the embolization and with the deprivation of the tumor's oxygen supply, and second, the cytotoxic action of the anticancer drug. There is also another technique, TAE (also called bland embolization), in which the lesion is targeted only with an embolizing agent (more common for neuroendocrine liver metastases).

The most commonly used drugs are anthracyclines, in particular doxorubicin, although there is no certain superiority of one drug over another [7] (Fig. 21).

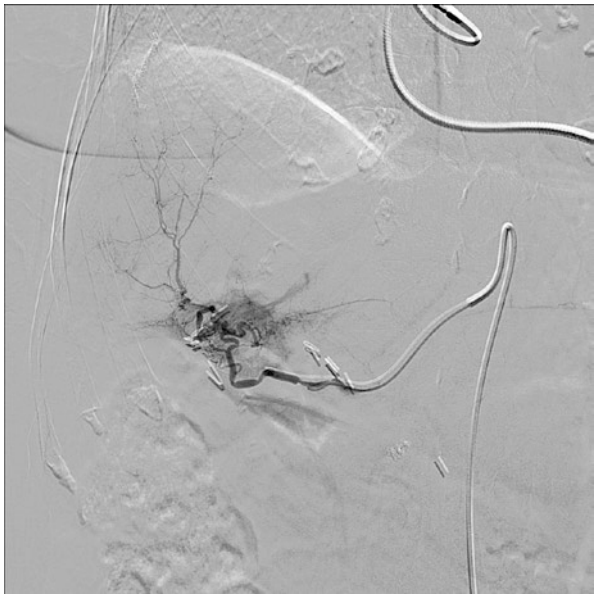

Fig. 22 Right hepatic artery catheterization prior to TARE

When a radioactive agent is instilled, we speak of TARE (transarterial radio embolization) or SIRT (selective internal radiation therapy): today Yttrium-90 or Holmium-166 are more commonly used (Fig. 22).

Prior to the actual treatment, it is essential to perform a preliminary examination, which consists of an injection of radioactive particles (albumin marked with Technetium-99) from a microcatheter positioned at the exact level from where the treatment will be next carried out.

This allows to verify the disposition of the particles, to define with certainty the treatment area, and to evaluate the possible presence of aberrant vascular communications or shunts to the lung that could cause non-target radionecrosis.

8 Percutaneous Interventions

Percutaneous treatments are procedures that use an energy source (either heat, cold, or chemical) to destroy cancerous tissue in a target organ. They can be performed either with a percutaneous or laparotomy approach.

The main targets of this type of treatment are solid lesions of the liver (Fig. 23), kidneys, and lungs or bones; nevertheless, it is possible to treat any type of solid lesion that can be clearly identified with imaging methods.

In the liver, ultrasound and CT are the preferred imaging modalities used to guide the procedures, while for lung tumors, CT or CBCT scan alone is generally sufficient. In case of renal lesions, both modalities are usable, according to the

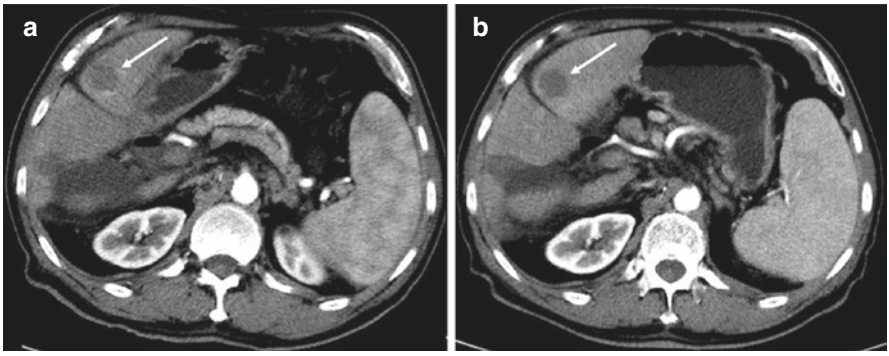

Fig. 23 Contrast CT of the upper abdomen, arterial phase. *Arrow* shows an hypodense round area with no sign of residual enhancing tissue after MW ablation of a liver lesion

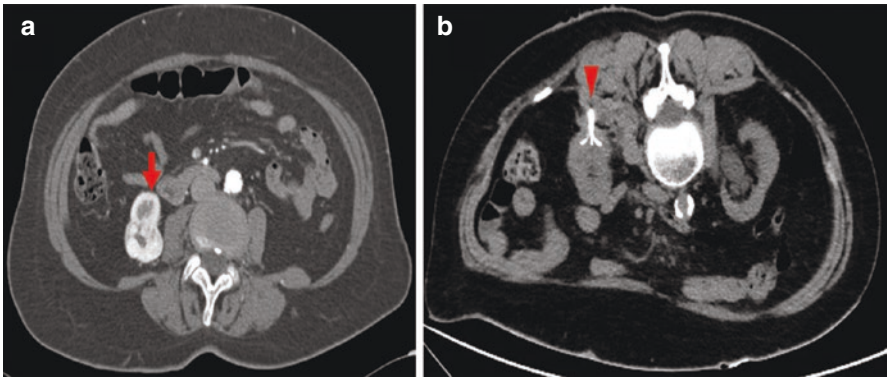

Fig. 24 (**a**). contrast CT scan, arterial phase; *arrow* shows an enhancing, solid exophytic mass in the right kidney, compatible with RCC. Necrosis or unenhancing high interstitial pressure simulates a renal medulla here. (**b**), *arrowhead* shows the distal shaft of a RF needle ready to ablate the renal mass

operator's preference. Obviously, in case of a laparotomic approach, US guidance becomes absolutely necessary.

Treatments that utilize a heat source are the following:

- Radiofrequency: the passage of high-frequency AC through the needle determines the formation of radio frequencies that generate heat (Fig. 24).
- Microwaves: a microwave generator determines the oscillation of water molecules (H_2O) inside the tissue with consequent generation of heat (2–5 billion molecular tilts per second) [8].

Treatments that utilize a cold source are the following:

- Cryoablation: after positioning the needle, a gas (in general argon) is pumped through the device, freezing the surrounding tissues, thus inducing cell death by osmosis and necrosis inside a so-called "ice ball." This modality, unlike the previous ones, has the advantage in making the treatment area much more predictable: when the "ice ball" is formed, we are almost sure that the tissue that lies inside it gets fully covered.

To date, there is no scientific evidence that demonstrates that one ablative modality is superior to the other; still, some suggestions can be made based on the lesion's characteristics. For example, in case of lesions near vascular branches >3 mm, the use of MW is recommended to reduce what is called "heat sink effect." Cryoablation may injure less renal collecting system for renal medullary lesions.

More recently, new techniques that exploit non-thermal mechanisms have been developed, and irreversible electroporation (IRE) is probably the most known. The physics behind it consists in a flow of electric pulses that create nanometric defects to the cell membrane: the result is cell death via apoptosis due to the inability to maintain the physiological homeostasis.

In any case, before the procedures, it is necessary to accurately evaluate size, localization, and type of the target lesion in order to choose how to treat your patient in the best possible way. Also, pay attention: before any percutaneous treatment, it is mandatory to evaluate the coagulation parameters and platelet count. SIR guidelines recommend to correct INR if>1.5 and transfuse patients if platelets <50,000 [3].

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Women's Imaging/Mammography

Lucy Chow and Hyung Won Choi

Contents

1 Gynecologic Imaging

1.1 Pelvic Ultrasound

The initial imaging modality of choice to evaluate obstetric and gynecologic complaints is a pelvic ultrasound. Common indications for pelvic ultrasound include pelvic pain, abnormal vaginal bleeding, pregnancy, and infertility. The advantage of pelvic ultrasound is real-time imaging of the pelvic structures without ionizing

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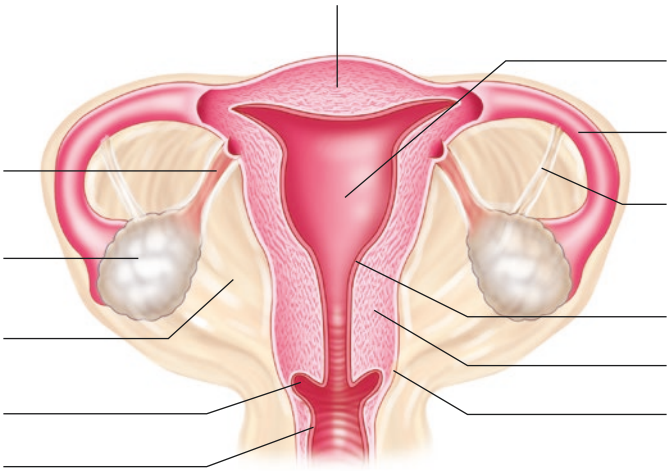


Fig. 1 Pelvic anatomy

radiation. Transabdominal ultrasound provides an overview of the pelvic structures with a large field of view of the pelvis. Detailed images of the endometrial cavity and ovaries are better obtained with transvaginal ultrasound because the transducer probe is placed closer to the pelvic organs of interest (Figs. 1 and 2).

1.2 Other Pelvic Imaging Studies

Sonohysterogram (SHG) is a detailed sonographic examination of the endometrial cavity and endometrium [1]. Saline is instilled into the endocervical canal to distend the endometrial cavity which provides improved visualization of the endometrial cavity and lining (Fig. 3). SHG is often performed after an initial pelvic ultrasound demonstrates findings that require more detailed imaging of the endometrium, such as irregular endometrial thickening. Common indications for SHG are abnormal vaginal bleeding and infertility. Pathologies such as endometrial polyps, submucosal myomas, endometrial hyperplasia, uterine cavity anomalies, and intrauterine adhesions can be visualized. SHG is contraindicated if there is a concern for pelvic infection, pregnancy, or excessive vaginal bleeding at the time of the procedure.

Hysterosalpingogram (HSG) is a fluoroscopic study where contrast is injected into the endocervical canal to evaluate the uterus and fallopian tubes. The most common indication for HSG is infertility. Other indications include recurrent spontaneous abortion, preoperative and postoperative evaluation for patients undergoing tubal ligation, or myomectomy. Abnormalities such as congenital uterine anomalies, synechiae, submucosal fibroids, polyps, salpingitis, tubal occlusion, hydrosalpinx, and peri-tubal adhesions may be visualized (Figs. 4, 5, and 6). Similar to SHG,

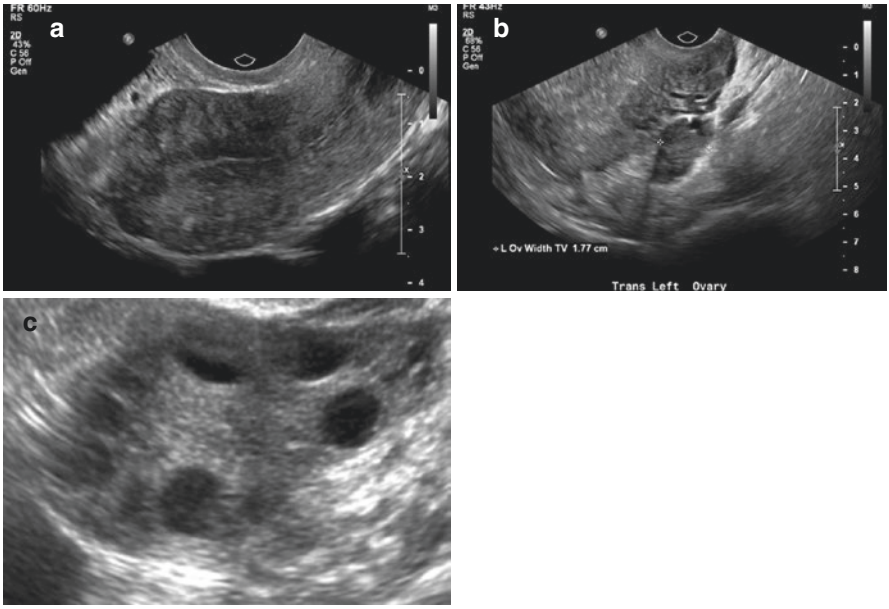


Fig. 2 Normal pelvic anatomy on ultrasound. (**a**) Uterus. (**b**) Ovary. (**c**) Normal physiologic follicles within the ovary

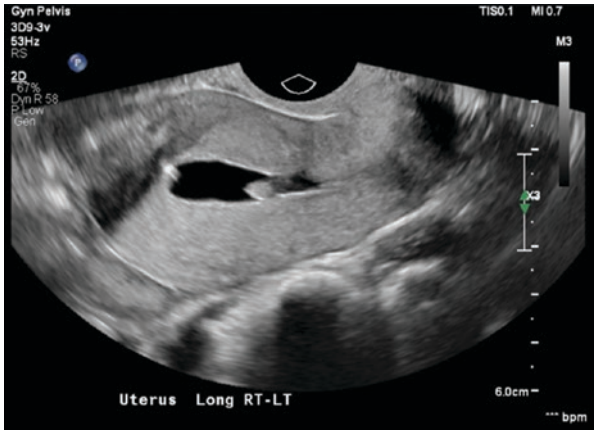


Fig. 3 SHG showing an endometrial polyp

HSG is contraindicated if there is a concern for pelvic infection, pregnancy, or heavy vaginal bleeding.

CT examination is infrequently performed as the initial imaging evaluation of the female pelvis, due to high radiation dose exposure and its limited utility in characterizing pelvic soft tissues. CT is helpful in detecting other pathologies that may cause acute pelvic pain such as appendicitis, colitis, diverticulitis, and nephrolithiasis.

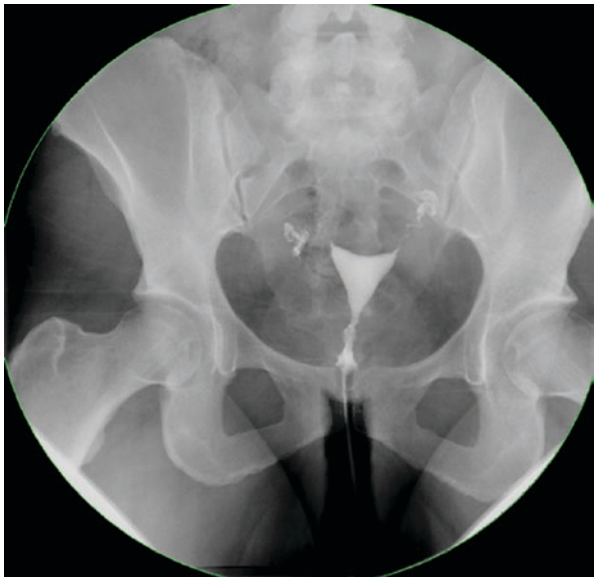


Fig. 4 Normal HSG demonstrating a normal appearance of the uterus and patent fallopian tubes

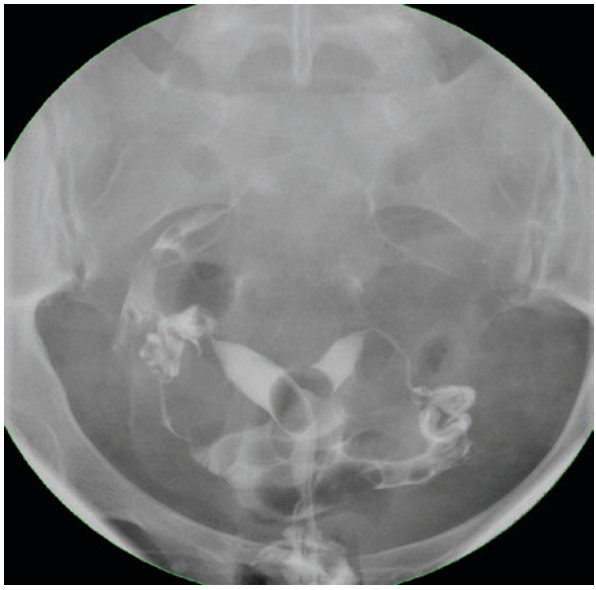


Fig. 5 HSG showing a uterine anomaly – a bicornuate uterus

MRI of the pelvis is usually reserved for outpatient, non-emergent indications, such as further evaluation of uterine anatomy, fibroids, pelvic masses, or ovaries. MRI is rarely performed for emergent situations due to its long acquisition time.

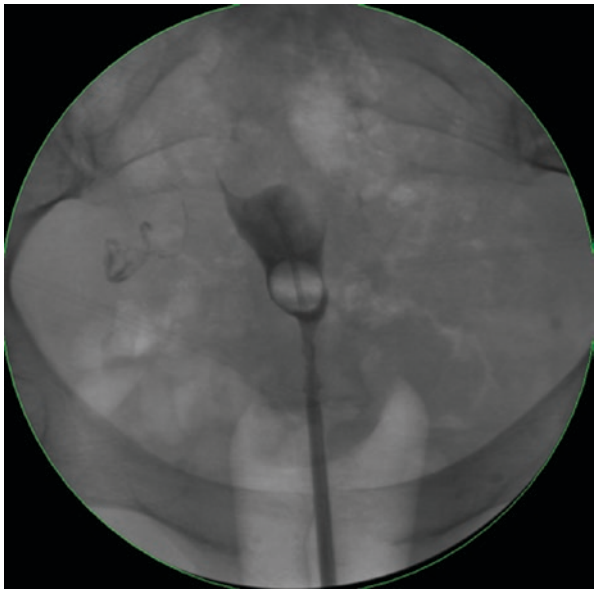


Fig. 6 HSG showing a blocked left fallopian tube, probably due to scarring

1.3 Pelvic Pain

One of the most common chief complaints seen in the emergency department is pelvic pain. When a female patient of childbearing age presents with pelvic pain or abnormal bleeding, a urine pregnancy test or serum b-hCG should be obtained prior to, or in conjunction with, imaging. Knowledge of the patient's pregnancy status narrows down the differential diagnosis and helps focus the imaging workup (Fig. 7).

1.3.1 Differential Diagnoses in the Non-pregnant Patient

Ovarian torsion occurs when the ovary or fallopian tube twists on its ligamentous structures, resulting in venous and lymphatic congestion [2] (Fig. 8). Infarction of the ovary can ensue if the torsion is not treated promptly. Ovarian torsion typically arises in the setting of a large ovarian mass greater than 5 cm, during fertility treatments, or prepubertal ovaries with long infundibulopelvic ligaments. Ultrasound typically demonstrates an asymmetrically enlarged ovary with peripherally displaced follicles. Absence of color and spectral doppler flow confirms torsion; however, normal ovarian flow does not exclude an ovarian torsion. Torsion-detorsion of ovaries can show normal or diminished flow on imaging depending on when the exam is performed.

Pelvic inflammatory disease (PID) (Fig. 9) is caused by ascending infection through the pelvic structures (Fig. 10). As the infection progresses, the fallopian tube fills with purulent material, and the infection and inflammation can extend into the adjacent ovary, resulting in a tubo-ovarian abscess. Ultrasound demonstrates a dilated fallopian tube filled with echogenic debris and increased vascularity in the fallopian tube wall. A tubo-ovarian abscess is seen as a multi-loculated adnexal

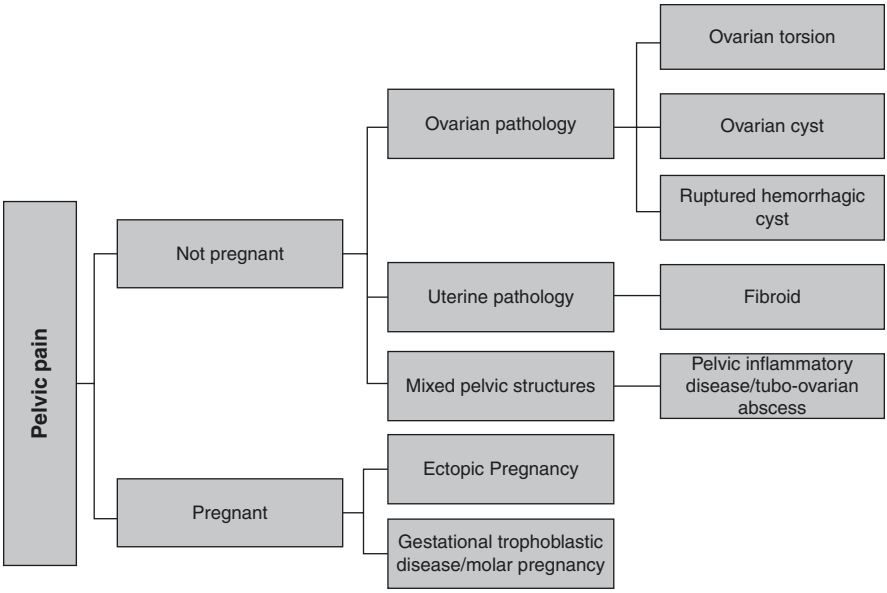


Fig. 7 Differential diagnoses for pelvic pain

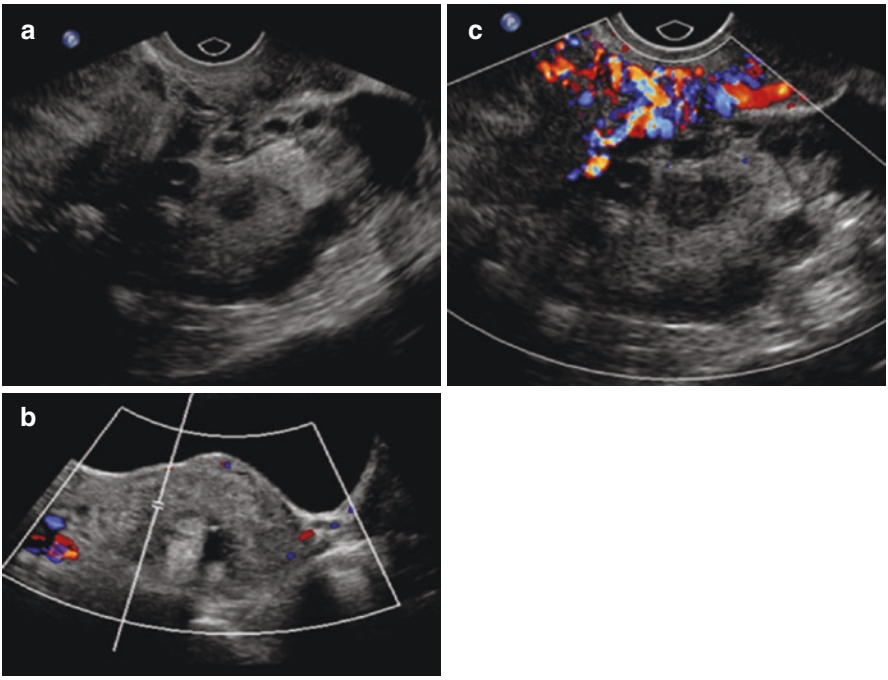


Fig. 8 Ovarian torsion. (**a**) Enlarged ovary with follicles in the periphery. (**b**, **c**) No blood flow detected in the ovary

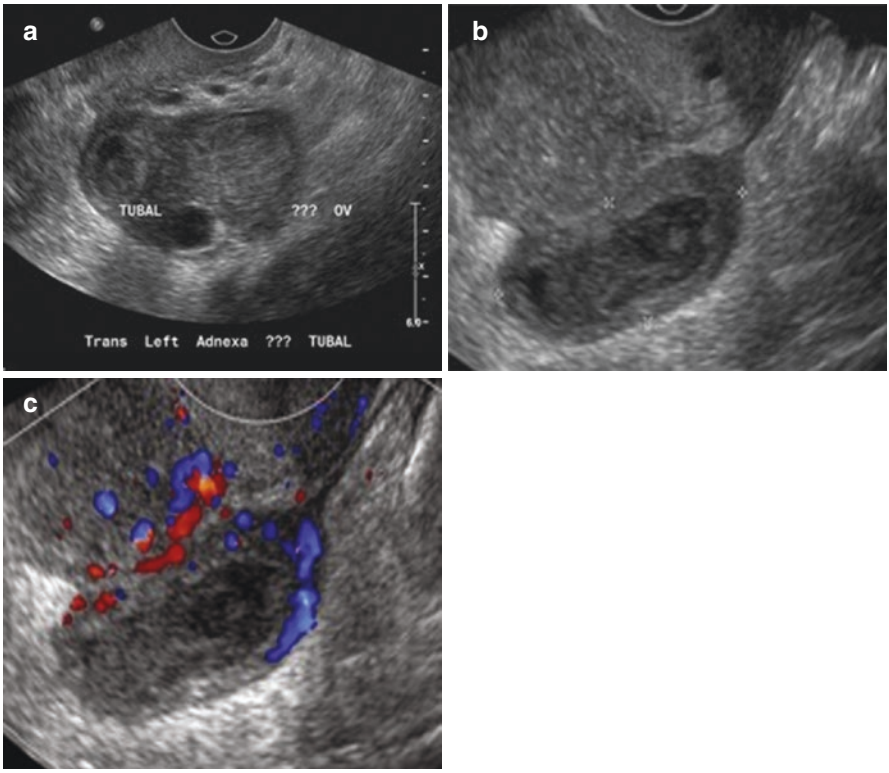
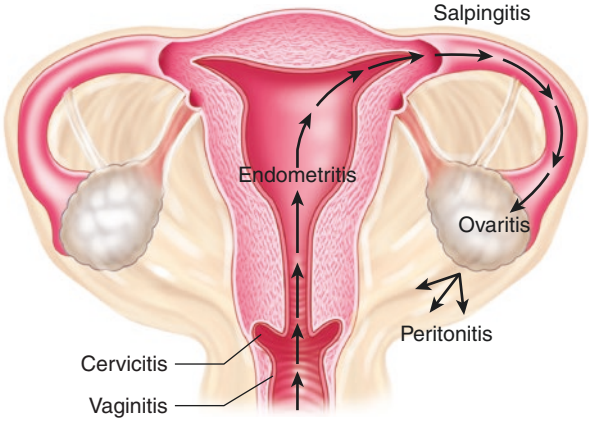


Fig. 9 Pelvic inflammatory disease. (**a**) Dilated fallopian tube seen adjacent to the ovary. (**b**) Dilated fallopian tube filled with complex debris and (**c**) increased vascular flow in its wall [5]

Fig. 10 PID route of spread



mass with incomplete septations and irregularly thickened walls. Pelvic free fluid is present, sometimes with complex debris. Clinical exam, fever, elevated WBC, and history is key.

Hemorrhagic cyst rupture occurs when an ovarian cyst bleeds internally and bursts open into the peritoneal cavity. A rupture frequently involves the right ovary and is associated with sexual intercourse, exercise, trauma, and pregnancy. Potential complications are massive hemoperitoneum and hemodynamic instability. A hemorrhagic cyst with fibrin strands appears as a mixed echogenicity adnexal structure with "lacelike" internal appearance (Fig. 11a). Mixed echogenicity material within the cyst represents internal blood clot. Complex pelvic free fluid represents hemoperitoneum (Fig. 11b). In contrast, an uncomplicated ovarian cyst is filled with simple fluid and appears anechoic with posterior acoustic enhancement and an imperceptible wall (Fig. 12). The treatment for hemorrhagic cyst rupture is conservative management, but surgery is indicated if the patient is hemodynamically unstable. Ultrasound may differentiate simple cysts from complex ones. Cystic cavities are dark on ultrasound and bright on T2-weighted MRI.

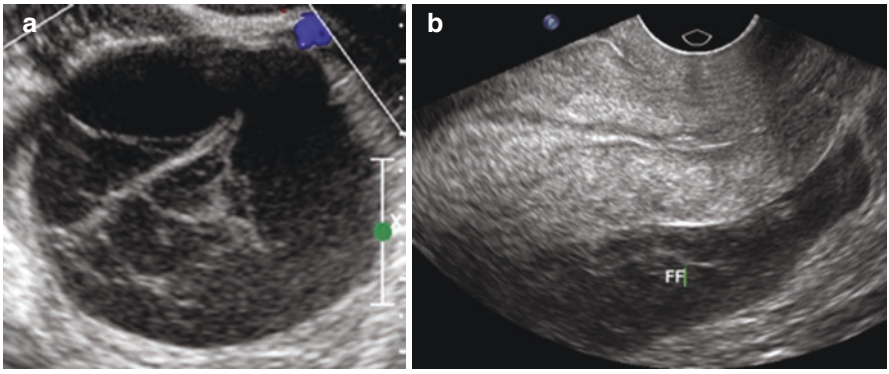


Fig. 11 Hemorrhagic cyst. (**a**) "Lacelike" internal appearance. (**b**) Complex pelvic free fluid (ff) in the cul-de-sac, consistent with hemoperitoneum

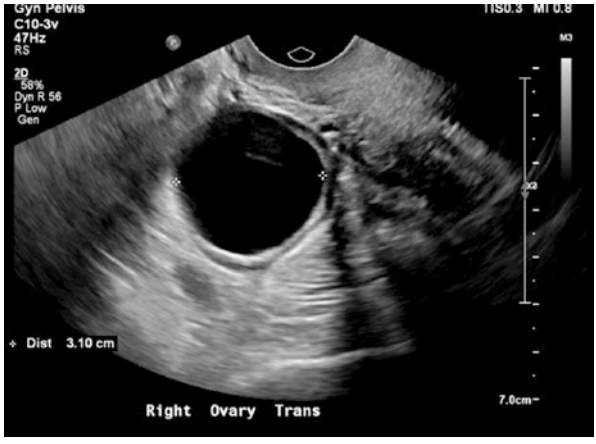


Fig. 12 Simple ovarian cyst

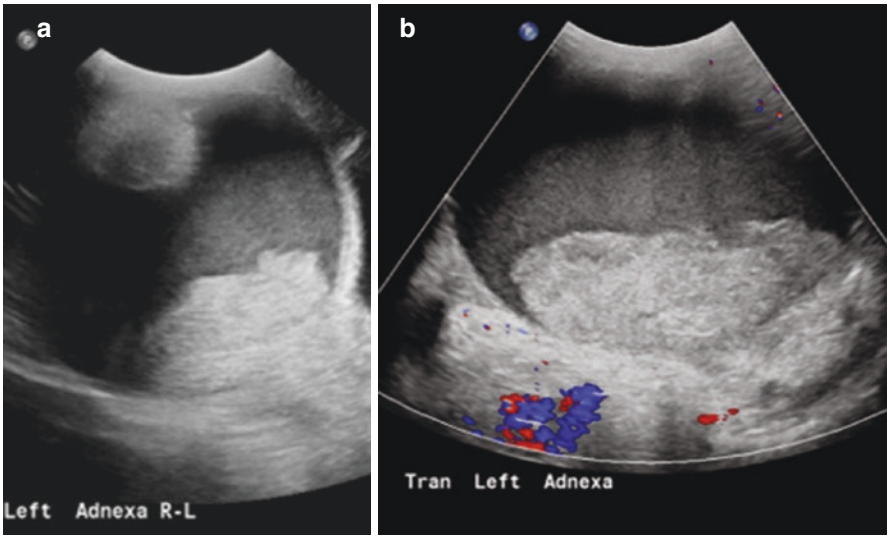


Fig. 13 Dermoid in the left adnexa. A complex cystic mass with layering hyperechoic material and hyperechoic mural soft tissue nodules

Ovarian dermoid cyst, also known as a mature cystic ovarian teratoma, is the most common ovarian neoplasm and is usually benign. It contains structures from multiple germ cell layers, including fluid, fat, hair, and teeth. Sonographic features of these elements within the dermoid include an echogenic mass with posterior acoustic attenuation, hyperechoic mural nodules, echogenic shadowing calcific components, the presence of fluid-fluid levels, and multiple echogenic thin bands (Fig. 13). Notably, no internal vascularity is identified on color doppler. Further workup is needed to exclude a malignant lesion if a complex ovarian mass demonstrates internal vascularity. Complications of a dermoid cyst include ovarian torsion, cyst rupture, and malignant transformation.

1.3.2 Differential Diagnoses in the Pregnant Patient

An ectopic pregnancy occurs when a fertilized egg implants outside the uterine cavity. A majority of ectopic pregnancies implant within the fallopian tube, and less commonly within the cervix, ovary, peritoneal cavity, or uterine scars. Risk factors include pelvic inflammatory disease, presence of an IUD, infertility, and prior tubal ligation. Fallopian tube or uterine rupture and hemorrhage are potential complications. Ultrasound usually demonstrates an adnexal mass as a sac-like structure or an embryo in the adnexa, as well as fallopian tubal dilation and thickening, in the absence of an identifiable intrauterine pregnancy (Fig. 14). Hematosalpinx and hemoperitoneum can be visualized if the ectopic gestation ruptures. A definite ectopic pregnancy is defined as an extrauterine gestational sac with a yolk sac, with or without an embryo demonstrating a heartbeat. A probable ectopic pregnancy consists of an irregular adnexal mass or extrauterine sac-like structure. Look for rupture. This is a favorite boards question.

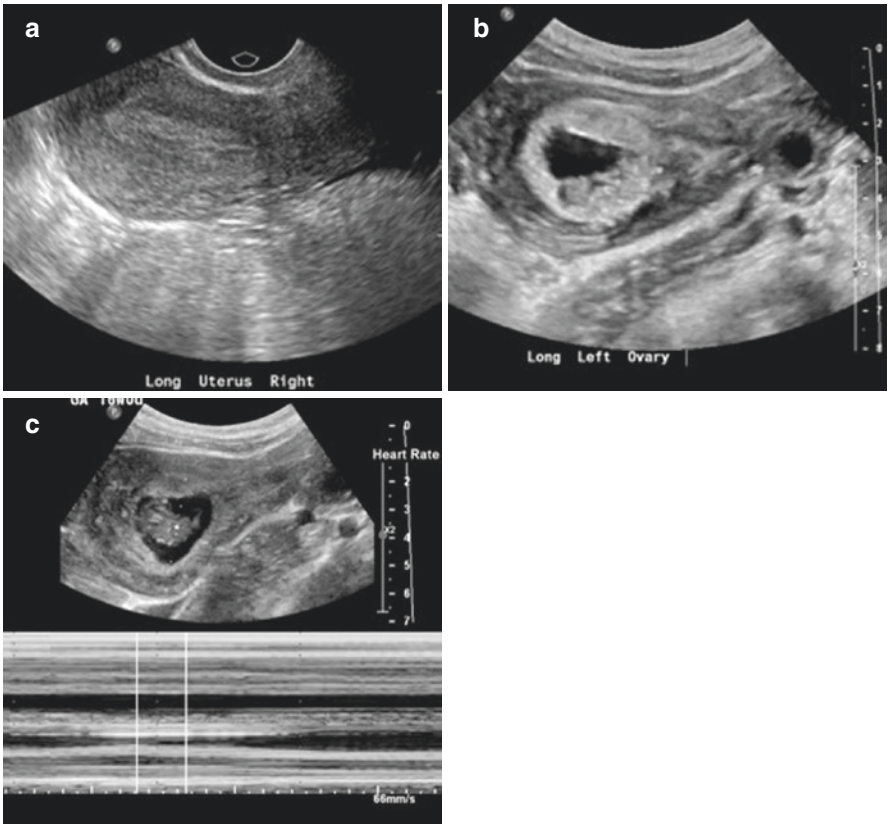


Fig. 14 Ectopic pregnancy. (**a**) No intrauterine pregnancy seen. (**b**) Gestational sac in the left adnexa, separate from the ovary. (**c**) Embryo with a heartbeat within the gestational sac [5]

Gestational trophoblastic disease is the abnormal proliferation of trophoblastic tissue and composes of different diseases including hydatidiform mole, invasive mole, and choriocarcinoma [3] (Fig. 15a). A complete mole is an intrauterine mass without any fetal components and is characterized by multiple cystic spaces that have a "snowstorm" or "clusters of grapes" appearance. Bilateral theca-lutein cysts may be seen within the ovary, resulting from an exaggerated physiological stimulation of the ovary from elevated b-hCG level (Fig. 15b).

1.4 Abnormal Vaginal Bleeding

Common causes of abnormal vaginal bleeding can be classified by structural or nonstructural causes (Fig. 16). Structural causes such as polyp, adenomyosis, leiomyoma of the submucosal type, malignancy, and hyperplasia can often be visually identified on imaging techniques. Nonstructural causes include coagulopathy,

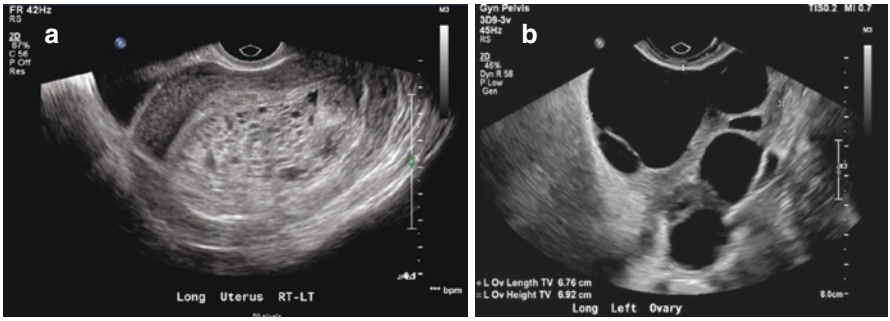


Fig. 15 Molar pregnancy. (**a**) "Snowstorm" or "cluster of grapes" within the uterus. (**b**) Theca lutein cysts in the ovary due to hyperstimulation of the ovary from elevated b-hCG level

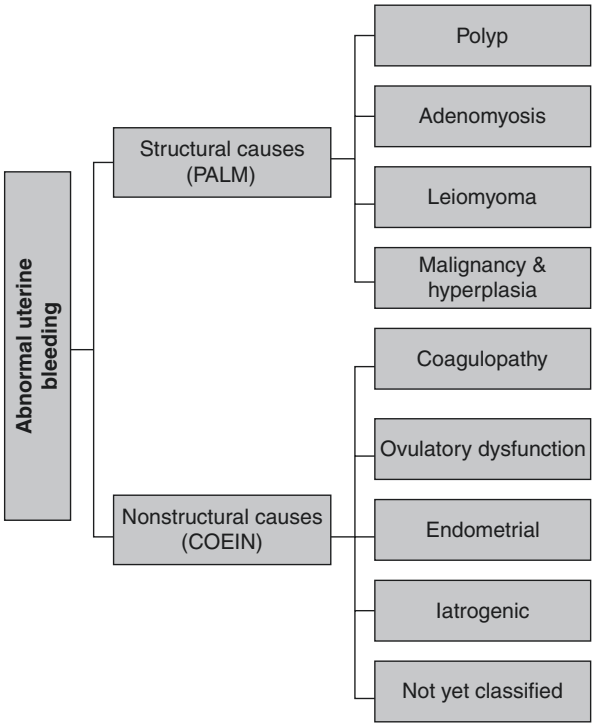


Fig. 16 Abnormal uterine bleeding (AUB) differentials

ovulatory dysfunction, endometrial, iatrogenic, and not yet classified. PALM-COEIN is a mnemonic for the structural causes (PALM) and the nonstructural (COEIN) causes, respectively [4].

Endometrial polyps are benign soft tissue nodules arising from the endometrial surface. Polyps may appear as thickened endometrium or a discrete polypoid mass with a stalk or feeding vessel extending to the polyp on color doppler imaging. Endometrial polyps are best visualized with a sonohysterogram (Fig. 3).

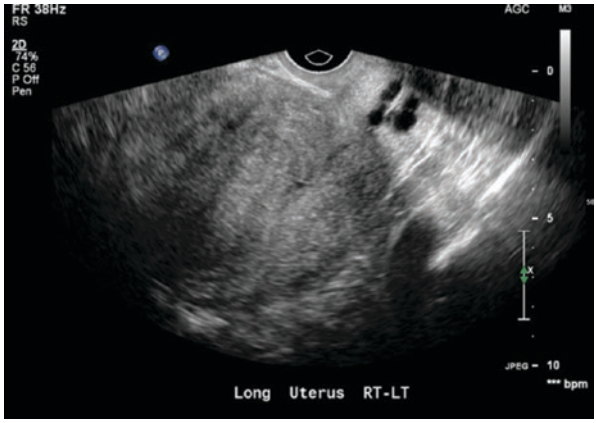


Fig. 17 Adenomyosis. "Venetian blind" appearance

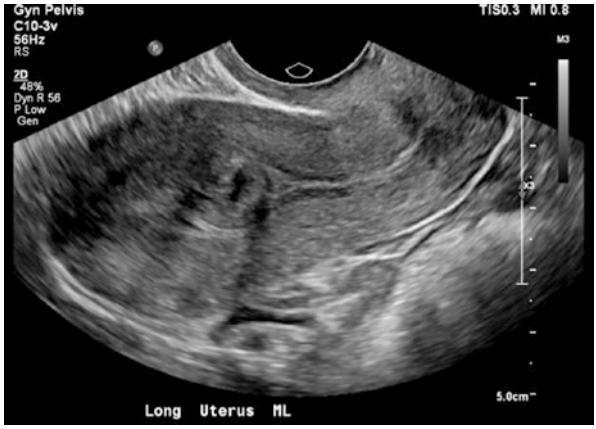


Fig. 18 Submucosal leiomyoma. Submucosal leiomyoma protruding into the endometrial cavity and distorting the endometrial lining

Adenomyosis refers to an entity where ectopic endometrial tissue is found within the myometrium. Sonographic features of adenomyosis include subendometrial echogenic linear striations also known as "Venetian blind" appearance, multiple tiny subendometrial cysts, myometrial thickening, and increased myometrial vascularity (Fig. 17).

Submucosal leiomyoma is a subtype of fibroid that protrudes into the endometrial cavity (Fig. 18). On ultrasound, a submucosal fibroid is seen as a broad-based hypoechoic shadowing mass, often with multiple feeding vessels.

Endometrial hyperplasia is abnormal proliferation of the endometrium and can potentially undergo malignant transformation to endometrial carcinoma (Fig. 19). The thickness of the endometrium depends on the patient's menstrual status and stage of the menstrual cycle. In a premenopausal patient, a thickness of up to 15 mm may be within normal limits in the secretory phase. In a postmenopausal patient, the normal endometrial thickness is generally less than 5 mm. Endometrial biopsy is recommended in the setting of abnormal endometrial thickening to exclude endometrial carcinoma. Post-menopausal uterine bleeding or recurrence of menstruation is a classic history for a boards question.

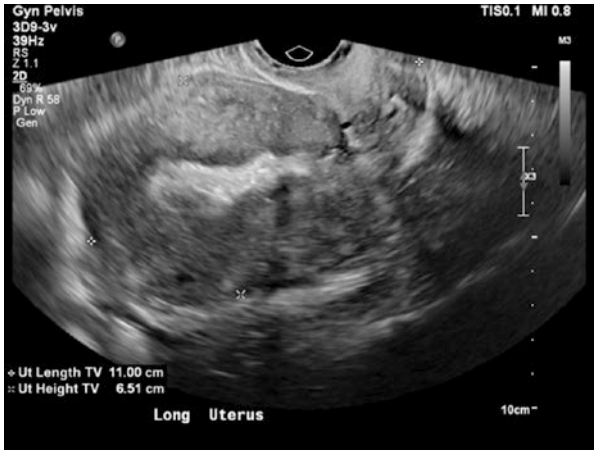


Fig. 19 Endometrial hyperplasia. Irregular thickening of the endometrium

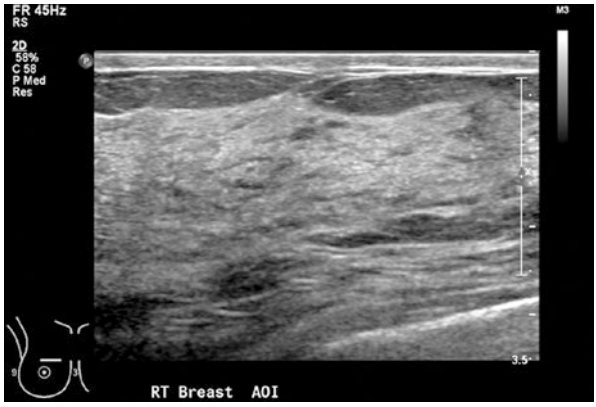


Fig. 20 Normal breast anatomy on US

2 Breast Imaging

2.1 Imaging Modalities

A mammogram is an X-ray image of the breast. A screening mammogram is used to detect breast cancer on patients who have no breast symptoms or complaints. A diagnostic mammogram is performed on patients with a specific breast symptom or complaint and may include additional views targeted to the area of concern. A mammogram is the best modality for evaluation of microcalcifications which are often occult on ultrasound imaging.

Breast ultrasound (Fig. 20) is used to further characterize abnormal findings seen on a mammogram and is often included in the diagnostic workup of a palpable breast lump or pain. In patients who are less than 30 years of age, diagnostic evaluation of breast complaints should begin with an ultrasound examination to reduce radiation exposure. Cysts or cyclical changes of breast tissue may be commonly seen on ultrasound imaging, but look closely for family history of breast cancer (i.e., BRCA mutation).

Breast MRI is used for screening women with elevated estimated risk of breast cancer (greater than 20%), *BRCA* gene mutation, strong family history of breast cancer, and history of chest radiation during childhood. Diagnostic breast MRI is used to evaluate for silicone implant integrity, nipple discharge, metastatic axillary lymphadenopathy with an unknown primary malignancy, extent of disease in breast cancer patients, and response to chemotherapy. Sometimes MRI is used for screening in dense breasts, where the sensitivity of mammography may be reduced. MRI examination should be performed between menstrual cycle days 7–12 to decrease background parenchymal enhancement.

2.2 Description of Findings

The location of the area of interest is described by its clock face and its distance from the nipple (Fig. 21).

Findings are given "BI-RADS" categories based on the level of suspicion of the imaging features and its probability of being malignant (Fig. 22).

2.3 Palpable Abnormality – Common Benign Entities (Fig. 23)

A simple cyst (Fig. 24) is identified sonographically as a round or oval anechoic structure, with circumscribed margins, smooth thin wall, and posterior acoustic enhancement. Simple cysts are benign (BI-RADS 2), and no further workup is required. Aspiration may be performed on large cysts for symptomatic relief.

A complicated cyst (Fig. 25) is a cystic breast lesion with internal debris. It appears as a circumscribed thin-walled hypoechoic ("gray") structure with fluid-fluid levels or internal echoes that alter with patient's movement. Complicated cysts are often considered probably benign (BI-RADS 3), and a short interval follow-up is recommended to evaluate for stability, usually in 6 months. A biopsy is performed if there is interval growth or development of worrisome features.

Fibroadenoma (Fig. 26) is a common benign breast mass that generally develops in women less than 40 years of age. The typical clinical presentation is a mobile,

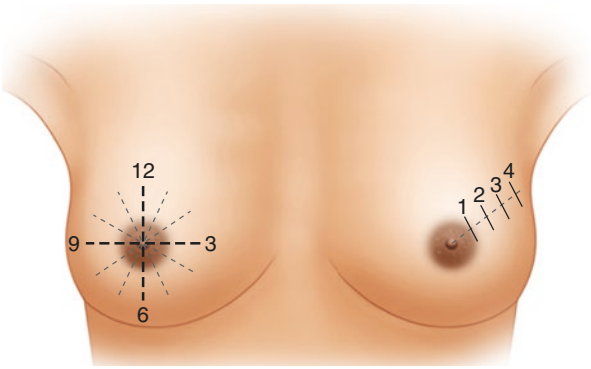


Fig. 21 Describing the location of breast findings

palpable lump. On a mammogram, fibroadenomas may have an appearance of a circumscribed oval mass and develop coarse calcifications over time as the mass involutes. On ultrasound, fibroadenomas typically present as a round or oval mass with circumscribed margins and are homogeneously hypoechoic. Indications for biopsy generally include atypical sonographic features, enlargement, and large size greater than 2.5 cm.

Sebaceous cyst and epidermal inclusion cyst (Fig. 27), are dermal based lesions located in the skin or subcutaneous tissues. They are identified sonographically as a

Category		Management
0	Incomplete	Need additional imaging evaluation
1	Negative	Routine annual screening
2	Benign	Routine annual screening
3	Probably benign (≤2% likelihood)	Short-interval follow up
4	Suspicious of malignancy (2-95% likelihood)	Biopsy
5	Highly suspicious of malignancy (≥95% likelihood)	Biopsy
6	Known biopsy-proven malignancy	Appropriate action to be taken

Fig. 22 **B**reast **I**maging **R**eporting **a**nd **D**ata **S**ystem (BI-RADS) Assessment Categories

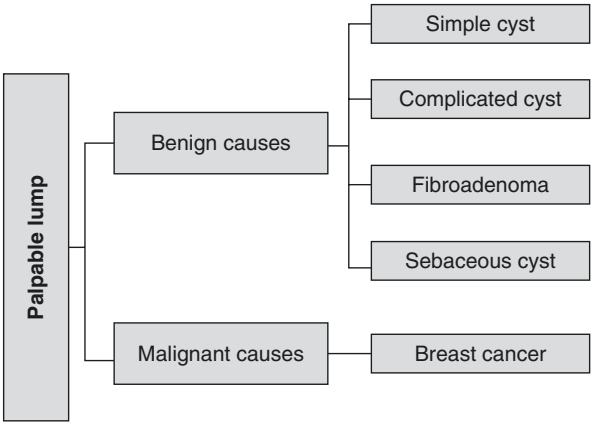


Fig. 23 Palpable breast lump differentials

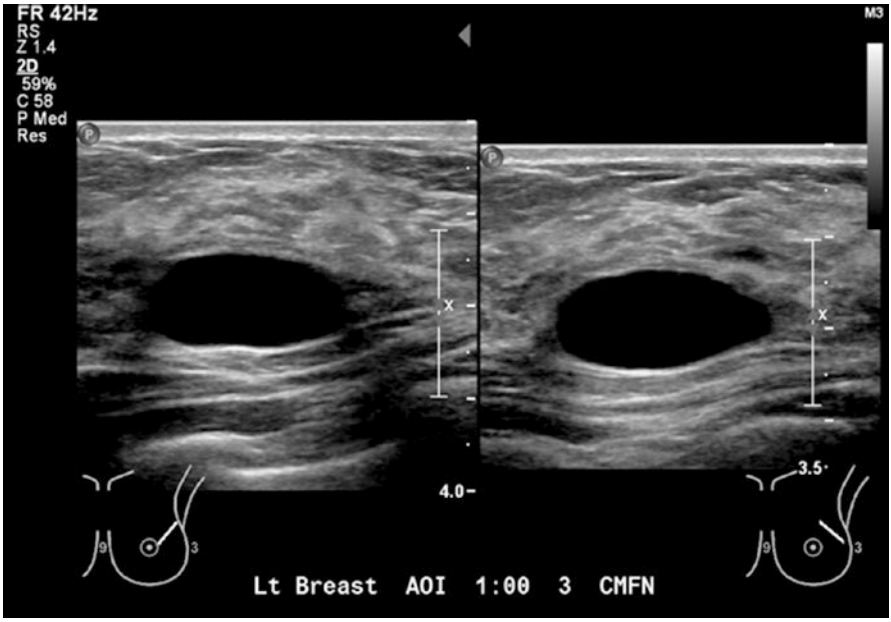


Fig. 24 Simple cyst

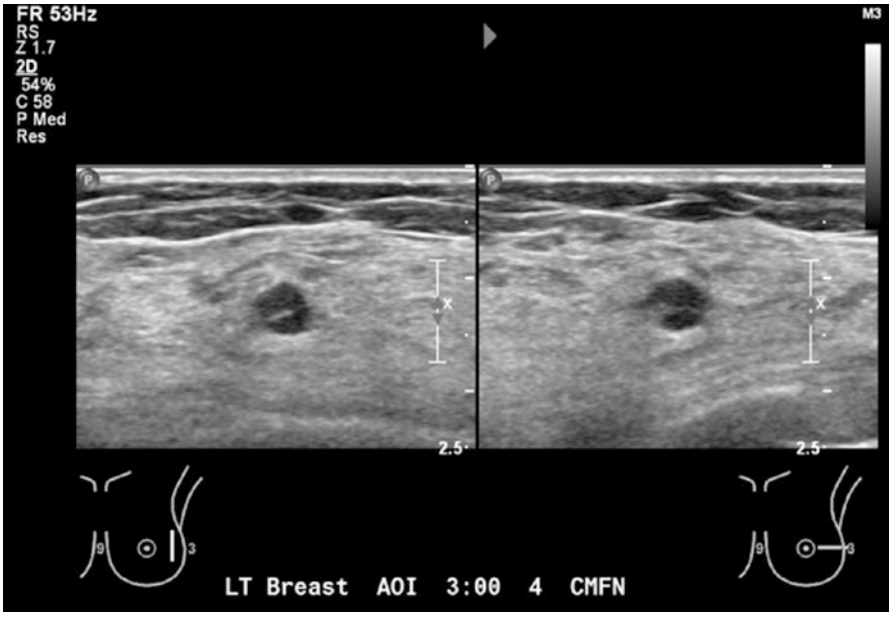


Fig. 25 Complicated cyst

superficial circumscribed, round or oval mass that maintains continuity with the epidermis and may have a tract extending to the skin surface.

2.4 Breast Cancer

Breast cancer is detected on screening mammograms as a *mass, architectural distortion or group of calcifications with suspicious features* (Fig. 28). Features of a suspicious mass include irregular shape and spiculated margins. The most suspicious calcifications have a fine linear branching or pleomorphic morphology. Architectural distortions are identified as irregular pulling of tissue toward a central point. If the finding is sonographically visible, then the radiologist may proceed with an ultrasound-guided biopsy. If the abnormal finding is only detectable on a mammogram, the radiologist may perform a stereotactic/mammogram-guided biopsy.

2.5 Male Breast

The initial imaging study of choice to evaluate a male breast complaint is a mammogram, regardless of age.

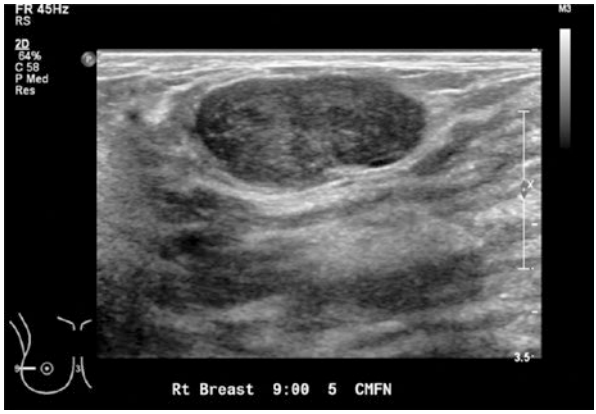


Fig. 26 Fibroadenoma

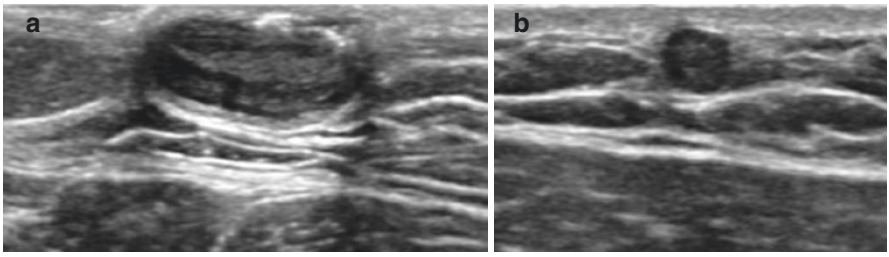


Fig. 27 Sebaceous gland cyst/epidermal inclusion cyst

Gynecomastia (Fig. 29) is a benign condition of excessive male breast tissue. It can present as a palpable lump, tenderness, or pain. Gynecomastia is best evaluated on a mammogram since ultrasound is limited at evaluating the area deep to the nipple due to artifact. It appears as a subareolar flame-shaped lesion and can be asymmetric.

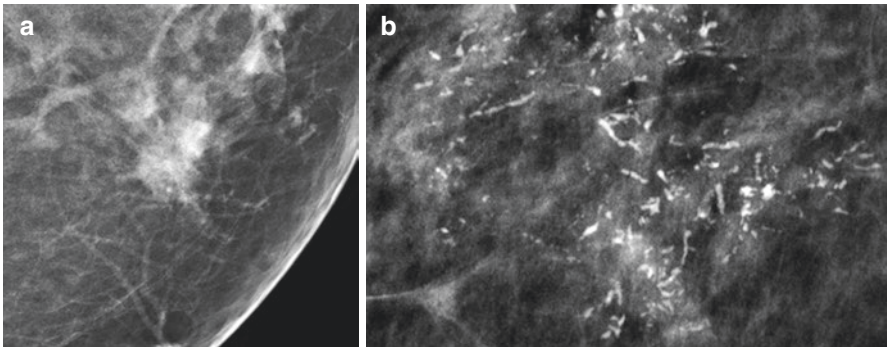


Fig. 28 Suspicious breast mass and breast calcifications. (**a**) Breast mass. (**b**) Breast calcification

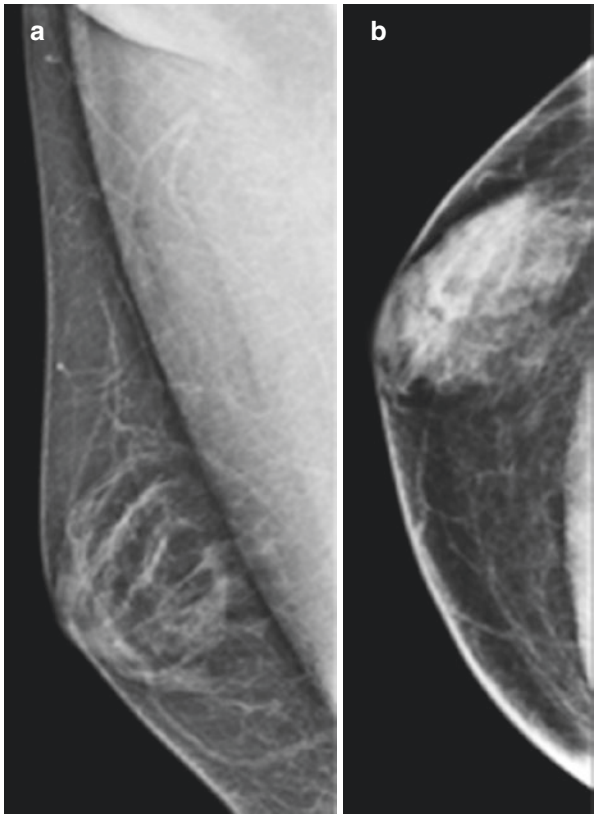


Fig. 29 Gynecomastia on mammogram. (**a**) Mediolateral oblique view of right breast gynecomastia. (**b**) Craniocaudal view of right breast gynecomastia

Males can also develop breast cancer, including invasive ductal carcinoma. Since male breast does not have lobules, pathologies of lobule origin usually do not occur (Fig. 30). For example, lobular carcinoma, fibroadenoma, and phyllodes are of lobular origin and thus very rarely seen in males.

2.6 Breast Emergencies

Hematoma and abscess (Fig. 31) are best evaluated with ultrasound, seen as a complicated fluid collection with internal debris. A hematoma has a typical clinical history of recent trauma, surgery, or anticoagulation use. An abscess can develop from mastitis or prior infection and often presents with skin thickening and erythema. If clinically indicated, fluid collection in the breast can be drained with ultrasound guidance.

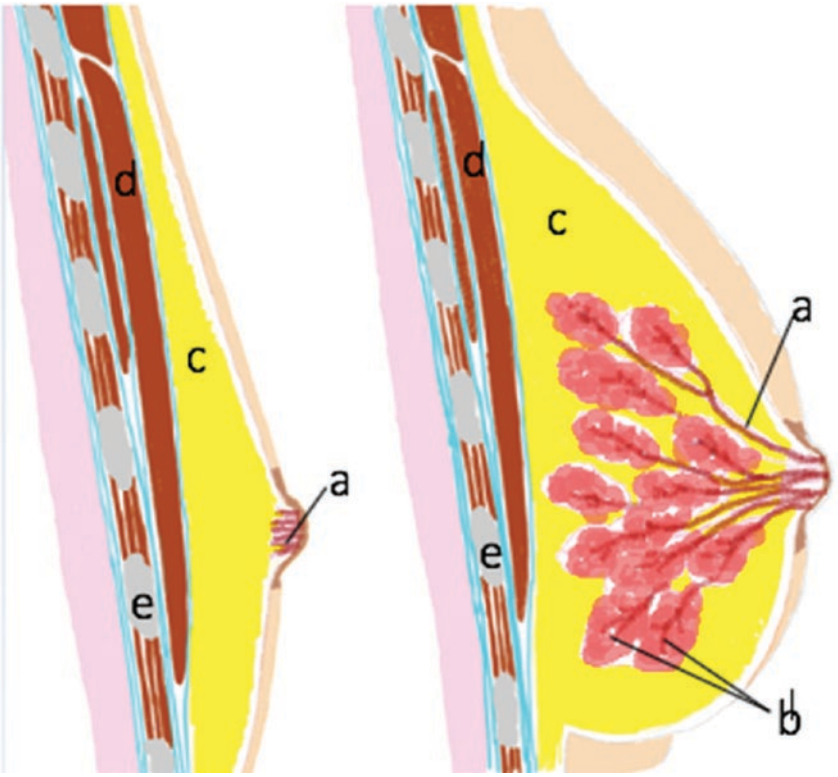


Fig. 30 Anatomy of the male (left) and female (right) breast. (**a**) Duct. (**b**) Lobule. (**c**) Breast fat. (**d**) Pectoralis muscle. (**e**) Rib

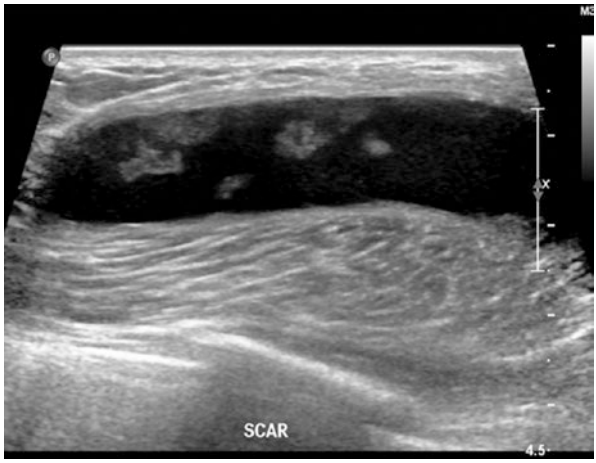


Fig. 31 Complicated fluid collection with internal debris

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Ophthalmology Imaging

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Contents

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1 Neuro-Ophthalmology

1.1 Visual Field Defects

Visual field defects can be caused by a variety of lesions or vascular events which produce visual changes depending on their location along the visual axis. Posterior lesions, such as occipital lobe abscesses or infarcts, can cause a contralateral homonymous hemianopsia (Fig. 1). The visual deficit usually does not affect the macula, due to redundant macular coverage by the opposite occipital cortex. Lesions affecting the optic chiasm, including pituitary adenomas or craniopharyngiomas, can

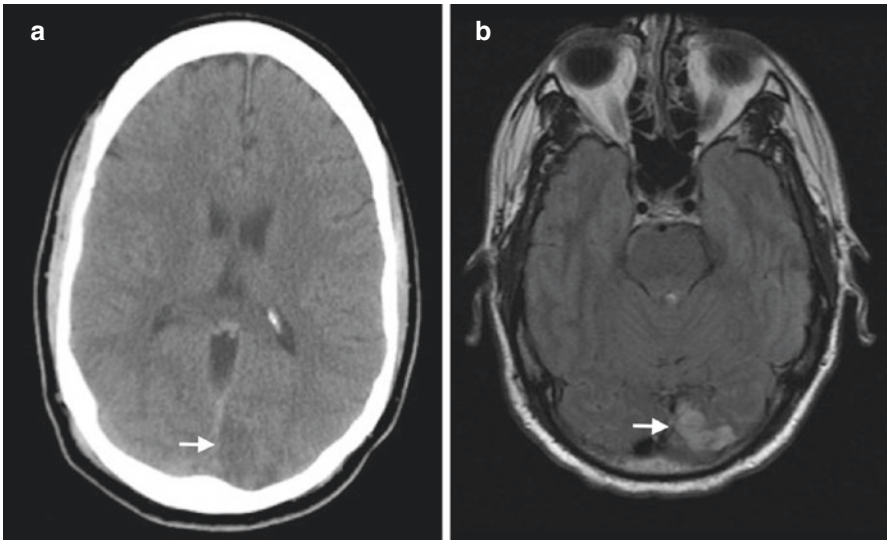

Fig. 1 (**a**) Axial CT scan showing area of hypointensity in the left occipital lobe (white arrow) and (**b**) axial T2-weighted flair MRI showing hyperintensity in this same area (white arrow), consistent with occipital lobe stroke. (Picture courtesy of Dr. Mark Moster, Neuro-Ophthalmology Service, Wills Eye Hospital)

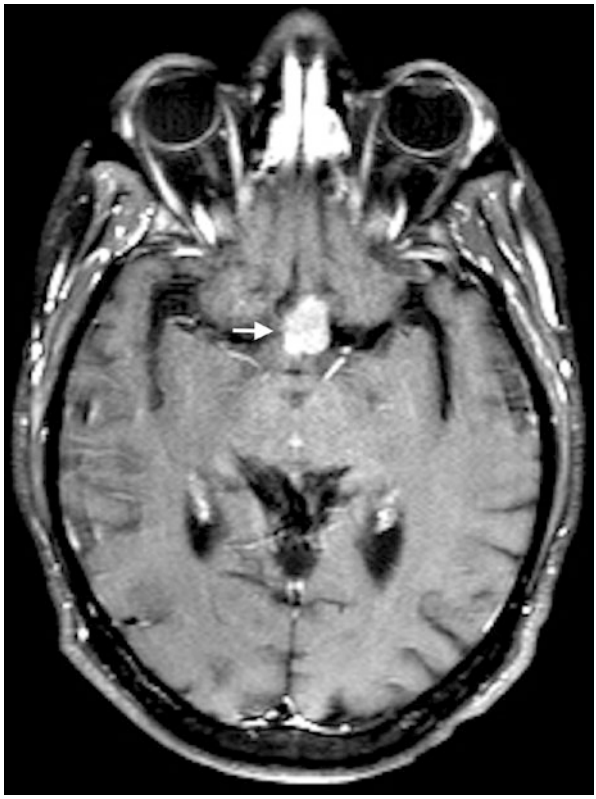

Fig. 2 Axial contrast-enhanced T1-weighted MRI showing enhancement of a suprasellar mass (white arrow), consistent with pituitary adenoma. (Picture courtesy of Dr. Kalla A. Gervasio, Wills Eye Emergency Room, Wills Eye Hospital)

cause a bitemporal hemianopsia (Fig. 2). Lesions affecting the optic nerve, such as optic nerve glioma, cause unilateral vision loss [1, 2].

1.2 Horner's Syndrome

Horner's syndrome consists of a constellation of clinical findings including unilateral pupillary constriction (miosis), drooping eyelid (ptosis), and absence of facial sweat (anhidrosis) (Fig. 3). It can be caused by a variety of pathologic events that affect the sympathetic pathway, including dissections of the internal carotid artery (Fig. 4), or Pancoast lung tumors [1, 2].

1.3 Pseudotumor Cerebri (Idiopathic Intracranial Hypertension)

Pseudotumor cerebri (idiopathic intracranial hypertension) is a condition characterized by increased intracranial pressure without any identifiable cause. Patients

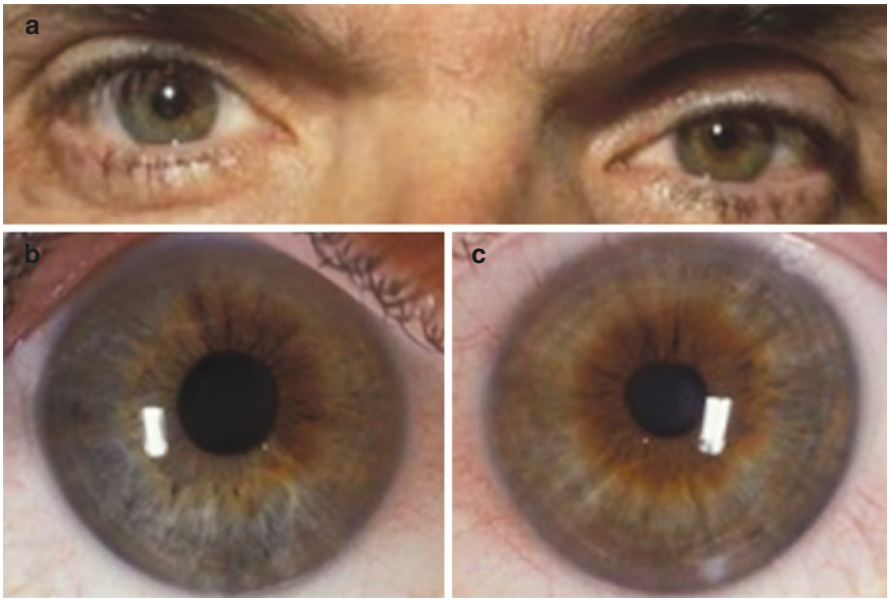

Fig. 3 (**a**) Drooping of the left lid (ptosis), (**b**) normal right eye, and (**c**) constriction (miosis) of the left eye in Horner's syndrome. (Picture courtesy of Dr. Mark Moster, Neuro-Ophthalmology Service, Wills Eye Hospital)

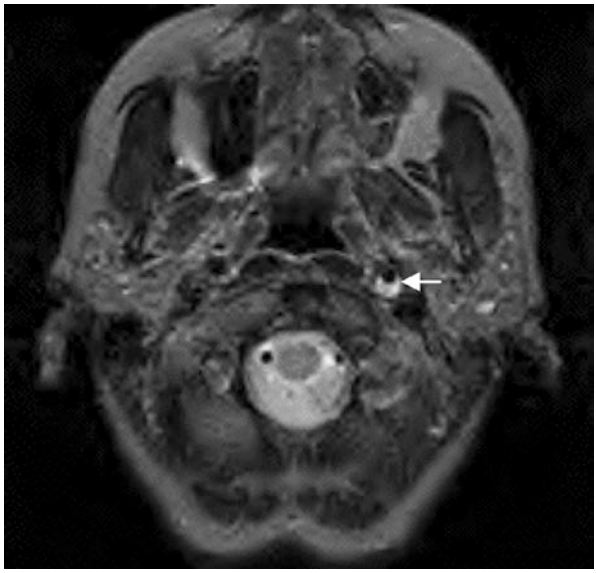

Fig. 4 Internal carotid artery dissection (white arrow). (Picture courtesy of Dr. Mark Moster, Neuro-Ophthalmology Service, Wills Eye Hospital)

present with symptoms such as headache, nausea/vomiting, pulsatile tinnitus, diplopia, and transient visual obscurations (episodes of black out vision bilaterally lasting less than 30 seconds at a time). Risk factors for the disease include female gender, obesity, oral contraceptive pills, and vitamin A supplements. Examination of the retina will demonstrate optic nerve edema bilaterally (papilledema) (Fig. 5). Medical treatment is directed toward decreasing cerebrospinal fluid production with carbonic anhydrase inhibitors. Surgical intervention with a ventriculoperitoneal (VP) shunt or optic nerve sheath fenestration is indicated for those who fail medical management [1, 2].

1.4 Multiple Sclerosis

Multiple sclerosis (MS) is a demyelinating disease of the central nervous system, characterized most often by periventricular white matter lesions on MRI. Optic nerve involvement of the disease can cause optic neuritis (Fig. 6), with symptoms including vision loss, pain with eye movement, and altered color perception. Brain stem involvement, particularly of the medial longitudinal fasciculus, can cause the inability to adduct the ipsilateral eye on lateral gaze with a contralateral abducting nystagmus (internuclear ophthalmoplegia) (Fig. 7) [1, 2].

1.5 Neurofibromatosis

Neurofibromatosis type 1 (NF-1) is an autosomal dominant disorder caused by a mutation of the NF1 gene on chromosome 17. The disease has variable expression and can present with cutaneous neurofibromas, café au lait spots, axillary or

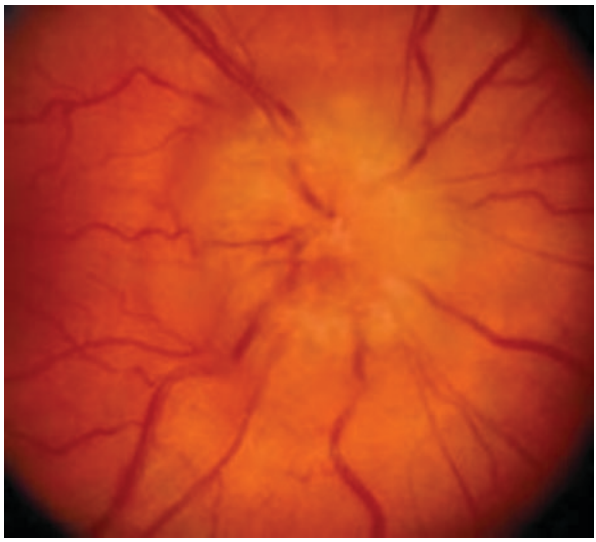

Fig. 5 Swelling of the optic disc (optic nerve edema). In the setting of increased intracranial pressure, bilateral optic nerve edema can be considered papilledema. (Picture courtesy of Dr. Mark Moster, Neuro-Ophthalmology Service, Wills Eye Hospital)

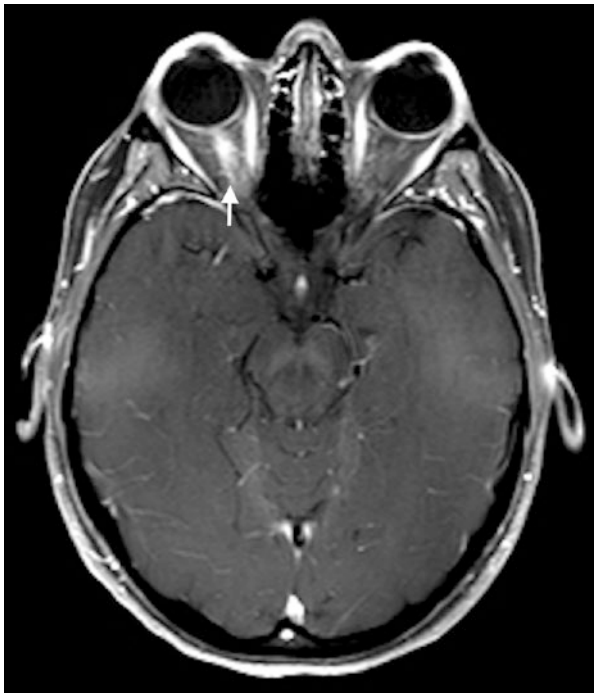

Fig. 6 Axial contrast-enhanced T1-weighted MRI showing enhancement of the right optic nerve, suggestive of optic neuritis. (Picture courtesy of Dr. Ranjodh S. Boparai, Wills Eye Emergency Room, Wills Eye Hospital)

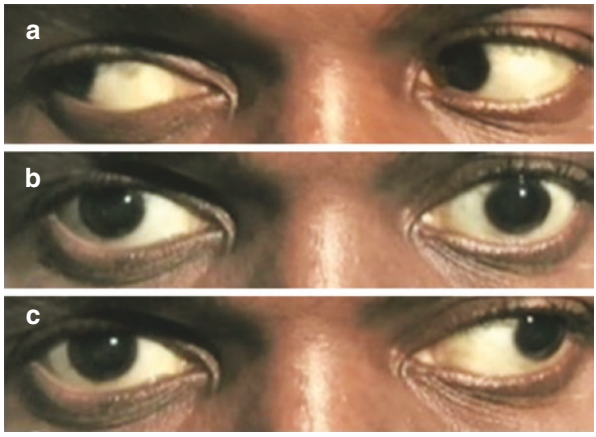

Fig. 7 (**a**) Normal right gaze, (**b**) slight right exotropia in primary gaze, and (**c**) restriction of right eye adduction in left gaze. Contralateral abducting nystagmus is not shown, but this pattern is consistent with internuclear ophthalmoplegia. (Picture courtesy of Dr. Mark Moster, Neuro-Ophthalmology Service, Wills Eye Hospital)

inguinal freckling, or pheochromocytoma. Ophthalmologic manifestations of NF-1 include Lisch nodules on the iris and optic nerve gliomas (Fig. 8) [1–3].

1.6 Cranial Nerve Palsies

The eye is controlled by six extraocular muscles, including the superior, inferior, medial, and lateral recti and the superior and inferior oblique muscles. These

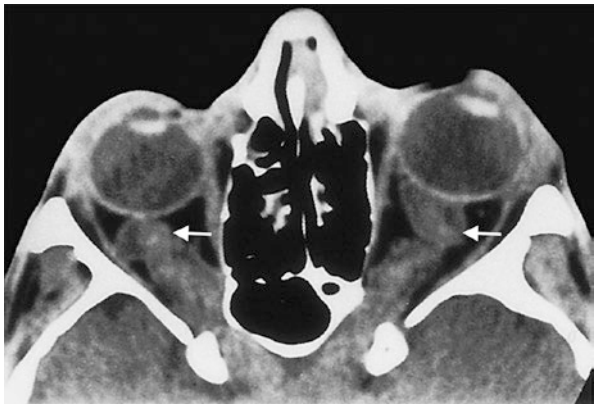

Fig. 8 Axial T1-weighted MRI showing bilateral optic nerve enlargement (white arrows) in a patient with neurofibromatosis type I, consistent with bilateral optic nerve glioma. © 2019 American Academy of Ophthalmology

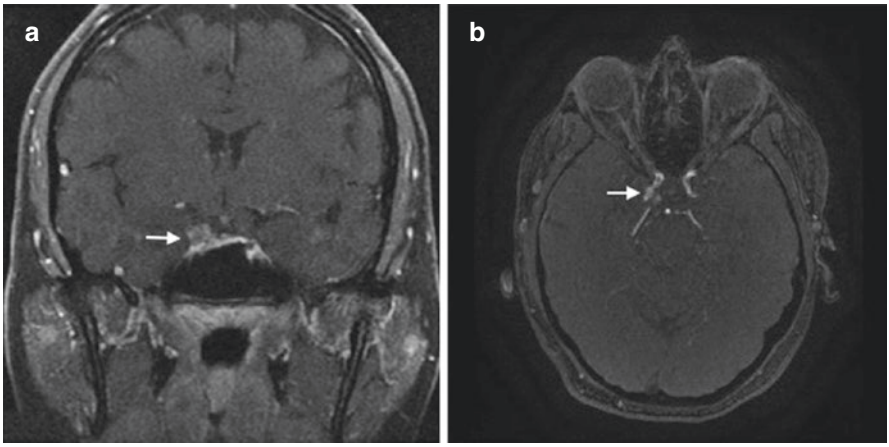

Fig. 9 (**a**) Coronal T1-weighted MRI demonstrating rounded area of hyperintensity posterior to the communicating segment of the right internal carotid artery (white arrow), (**b**) confirmed on axial MRA as a right posterior communicating artery aneurysm (white arrow). (Picture courtesy of Dr. Mark Moster, Neuro-Ophthalmology Service, Wills Eye Hospital)

muscles are controlled by three cranial nerves. The oculomotor nerve (CN III) innervates the superior rectus, inferior rectus, medial rectus, and inferior oblique muscles. The trochlear nerve (CN IV) innervates the superior oblique muscle, and the abducens nerve (CN VI) innervates the lateral rectus muscle. Compression, demyelination, or ischemia of any of these nerves can result in cranial nerve palsies with restriction of ocular motility. "SO4LR6" sounds like a fast car name but is an easy mnemonic, which helps one recall :"Superior oblique is CN4, and lateral rectus is CN6."

Posterior Communicating Artery (PCOM) **Aneurysms** can cause a third nerve palsy, affecting all muscles except the superior oblique and lateral rectus (Fig. 9). As a result, the eye will be "down and out." There may also be partial to complete ptosis, as CN III also controls the levator palpebrae muscle responsible for lifting the

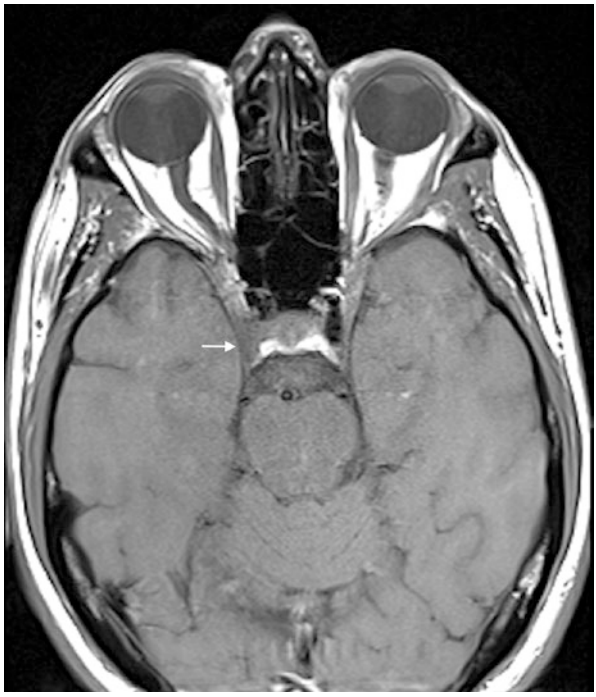

Fig. 10 Axial contrast-enhanced T1-weighted MRI showing enlargement of the right cavernous sinus and compression of the right internal carotid artery, suggestive of cavernous sinus thrombosis. (Picture courtesy of Dr. Mark Moster, Neuro-Ophthalmology Service, Wills Eye Hospital)

eyelid. Moreover, autonomic fibers that control pupillary reactions also run on the outside of the nerve. Thus, compressive damage to CN III usually presents with pupillary dysfunction, while ischemic changes that affect the interior of the nerve will often spare the pupil [1, 2]. This is a common board question.

Cavernous Sinus Thrombosis is usually caused by contiguously spreading infection from the sinuses, nose, or mouth and can affect all of the extraocular muscles, since all three related cranial nerves traverse this anatomic space (Fig. 10). The first nerve affected is classically CN VI, which runs through the middle of the cavernous sinus and is, therefore, most susceptible to injury [1, 2].

2 Oculoplastics

2.1 Dacryocystitis

The lacrimal drainage system is responsible for draining tears from the eye into the nasal cavity. Dacryocystitis is inflammation of the lacrimal sac, one of the components of this drainage system. The inflammation is usually caused by blockage of the nasolacrimal duct, which becomes secondarily infected. It presents clinically as an erythematous swelling near the medial canthus of the eye; the sac can also rupture and cause a fistula into the skin (Fig. 11). Treatment of acute dacryocystitis

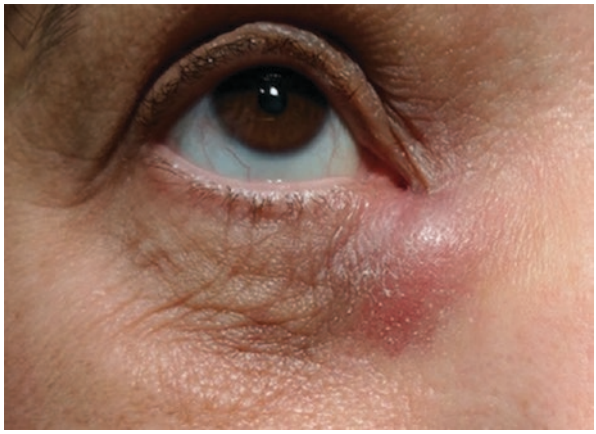

Fig. 11 Erythema and edema near the medial canthus of the right eye, suggestive of dacryocystitis. (Picture courtesy of Dr. Robert Penne, Oculoplastics Service, Wills Eye Hospital)

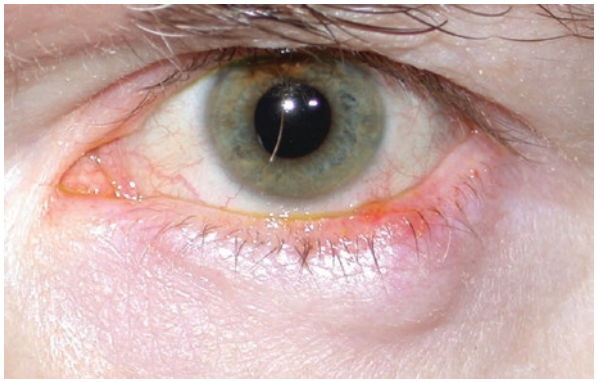

Fig. 12 Swelling of the left lower eyelid margin, consistent with chalazion. (Picture courtesy of Dr. Robert Penne, Oculoplastics Service, Wills Eye Hospital)

involves incision and drainage of the abscess, along with systemic antibiotics to treat the infection and possible future surgical intervention with a dacryocystorhinostomy (DCR) to prevent recurrence [1, 4].

2.2 Hordeolum/Chalazion

Hordeolum (stye) is an acute infection of the sebaceous (Meibomian) glands that line the eyelid. Persistent hordeolums can evolve into chalazia (chronic inflammation of that gland). Both present as tender swellings on or just below the eyelid margin (Fig. 12), but hordeola can be more erythematous and inflamed. Initial treatment for hordeola is conservative, involving warm compresses and antibiotic ointment, while chalazia can be treated similarly or with excision and/or corticosteroid injections [1, 4].

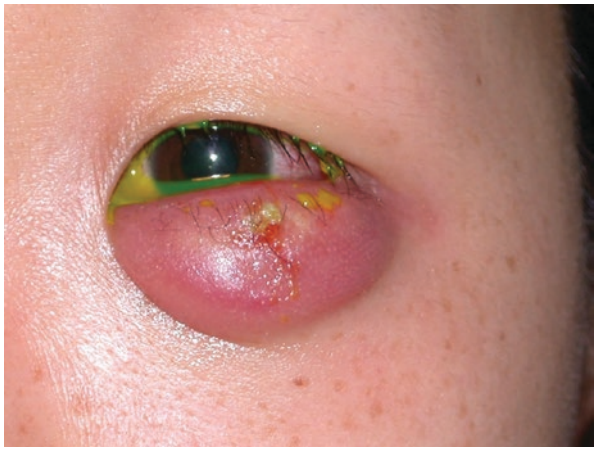

Fig. 13 Erythematous swelling confined to the left lower eyelid in preseptal cellulitis. (Picture courtesy of Dr. Robert Penne, Oculoplastics Service, Wills Eye Hospital)

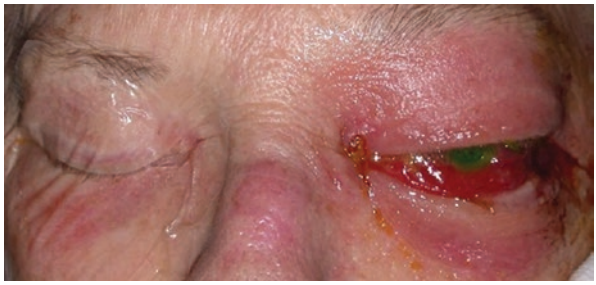

Fig. 14 Swelling of the left upper and lower eyelids, proptosis of the left eye, and chemosis (conjunctival swelling) of the left eye in orbital cellulitis. (Picture courtesy of Dr. Robert Penne, Oculoplastics Service, Wills Eye Hospital)

2.3 Preseptal Vs. Orbital Cellulitis

Cellulitis in the ocular region can be either preseptal or orbital in nature. Preseptal cellulitis is an infection of the eyelids, while orbital cellulitis is a deeper infection posterior to the orbital septum. Preseptal cellulitis is usually secondary to a superficial skin infection and presents with erythematous swelling of the eyelids (Fig. 13). Orbital cellulitis is most commonly due to contiguous spread of a sinus infection and can present with proptosis, pain, and restriction of extraocular motility (Fig. 14). CT or MRI may also demonstrate the presence of a subperiosteal abscess. While preseptal cellulitis can be managed with oral antibiotics, orbital cellulitis is more severe and should be managed with immediate IV antibiotics with gram-negative coverage and may require surgical drainage of an abscess and/or sinus [1, 4].

2.4 Thyroid Eye Disease

Thyroid eye disease (TED) is one of the many consequences of systemic thyroid disease, most commonly from hyperthyroidism. In this condition, antibodies

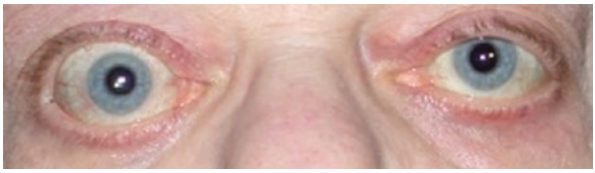

Fig. 15 Bilateral proptosis and upper lid retraction in a patient with Grave's disease. (Picture courtesy of Dr. Robert Penne, Oculoplastics Service, Wills Eye Hospital)

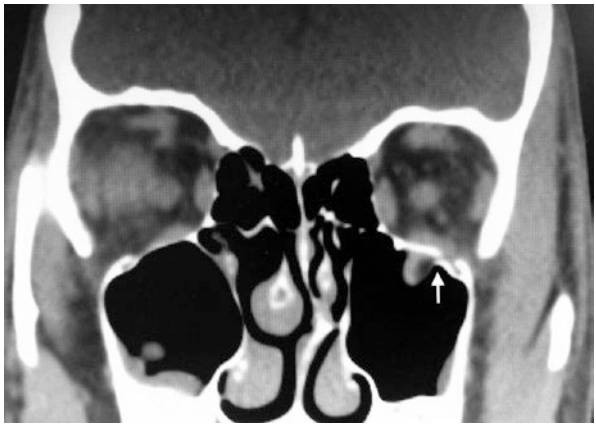

Fig. 16 Coronal CT scan shows left orbital floor fracture and herniation of orbital contents (white arrow) into the left maxillary sinus. (Picture courtesy of Dr. Robert Penne, Oculoplastics Service, Wills Eye Hospital)

directed at the thyroid also react to receptors in extraocular muscles and soft tissues of the orbit causing muscle and tissue enlargement. TED presents clinically with proptosis, chemosis (swelling of the conjunctiva), eyelid retraction, and restriction of extraocular motility (Fig. 15). CT or MRI may demonstrate enlargement of extraocular muscles that spares the muscle tendons [1, 4].

2.5 Orbital Fracture

Periocular trauma can result in orbital fractures. The most commonly affected regions are the orbital floor, composed of the zygomatic and maxillary bones, and the thin medial wall (lamina papyracea). Clinical presentation of orbital floor fractures ("blow-out fractures") can involve pain, swelling, ecchymosis, diplopia, an inferiorly dislocated globe (hypoglobus) or an inward displaced/sunken globe (enophthalmos). CT or MRI will show fracture of the orbital floor and may demonstrate herniation of orbital contents into the maxillary sinuses (Fig. 16). If the inferior rectus muscle is found to be entrapped within the fracture, patients may demonstrate restriction of ocular motility and an oculocardiac reflex with nausea/vomiting and bradycardia on exam (Fig. 17). Extraocular muscle entrapment is most commonly found in fractures of children given their pliable bones. This is considered a surgical emergency in order to avoid permanent muscle ischemia and chronic diplopia [1, 4].

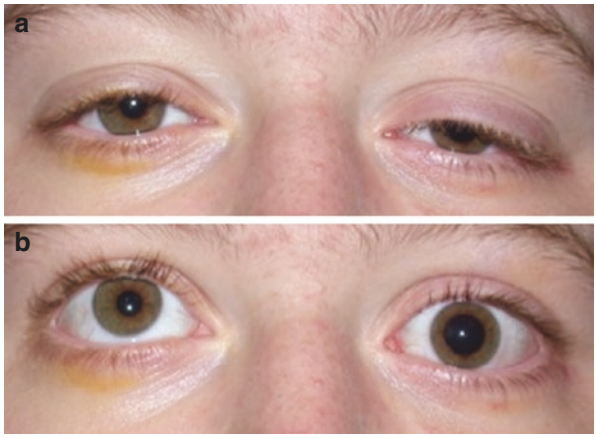

Fig. 17 (**a**) Left eye ptosis, mydriasis, and enophthalmos in primary gaze, and (**b**) restriction of motility in upgaze. (Picture courtesy of Dr. Robert Penne, Oculoplastics Service, Wills Eye Hospital)

3 Cornea and External Disease

3.1 Herpes Simplex Keratoconjunctivitis

Herpes simplex keratoconjunctivitis is a viral infection of the cornea and conjunctiva, usually caused by HSV-1. The herpes virus can lay dormant in the trigeminal nerve ganglion and reactivate to cause damage to ocular tissue. Symptoms include ocular pain, blurry vision, photophobia, and mucous discharge. Examination can reveal swelling and inflammation of the conjunctiva (chemosis), vesicles on the lid margin, and/or corneal dendritic ulcers highlighted by fluorescein (Fig. 18). Treatment involves oral antivirals plus topical antibiotic drops and/or ointment [1].

3.2 Corneal Ulcer

Corneal ulcers are open wounds on the cornea, usually caused by infection. Although bacteria are the most common cause of such ulcers, protozoa, fungi, and viruses may also cause ulcers. Risk factors for corneal ulcers include contact lens use, dry eye, inadequate eyelid closure (history of Bell's palsy or stroke), or trauma. Clinical presentation involves pain, photophobia, visual changes, and possible discharge. Examination will reveal an area of corneal defect, highlighted by fluorescein (Fig. 19) [1].

3.3 Endophthalmitis

Endophthalmitis is severe inflammation of the eye, usually of bacterial or fungal origin. The infection can be due to exogenous causes such as trauma or after surgery. It can also be due to endogenous causes such as septicemia due to endocarditis

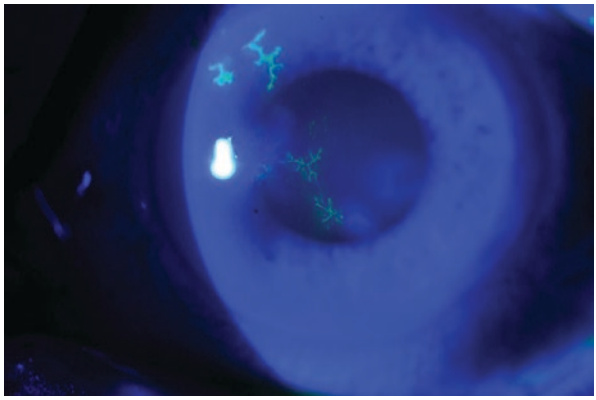

Fig. 18 Dendritic ulcers highlighted by fluorescein in HSV keratitis. (Picture courtesy of Dr. Christopher Rapuano, Cornea Service, Wills Eye Hospital)

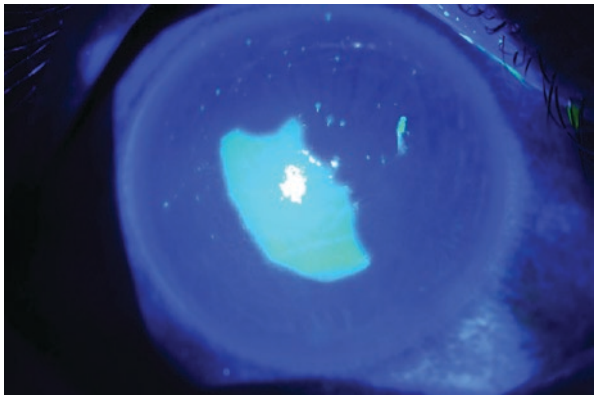

Fig. 19 Corneal ulcer highlighted by fluorescein. (Picture courtesy of Dr. Christopher Rapuano, Cornea Service, Wills Eye Hospital)

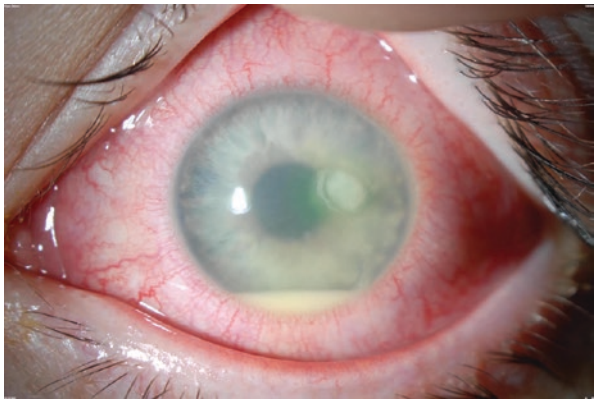

Fig. 20 Inflammatory cells in the anterior chamber of the eye (hypopyon) in endophthalmitis. (Picture courtesy of Dr. Christopher Rapuano, Cornea Service, Wills Eye Hospital)

or fungemia. Clinical symptoms include eye pain and vision loss. Examination can show swelling of the eyelids, swelling and redness of the conjunctiva, and hypopyon (Fig. 20). Treatment is directed at the causal infection and can involve systemic and/or intravitreal antibiotics or antifungals [1].

4 Glaucoma

4.1 Glaucoma

Glaucoma involves damage to the optic nerve, causing peripheral visual field loss. Increased intraocular pressure is the most important risk factor for glaucoma, as it can compress retinal blood flow and lead to optic nerve damage.

4.2 Open-Angle Glaucoma

Open-angle glaucoma is an insidious disease process that involves slow loss of the peripheral visual field. There are no external findings in this case, but examination of the optic nerve can reveal an increased "cup-to-disc" ratio greater than 0.3, indicating optic nerve damage (Fig. 21) [1].

4.3 Acute Angle Closure Glaucoma

Acute angle closure glaucoma is characterized by abrupt, painful visual loss. External examination will reveal a fixed, mid-dilated pupil, corneal clouding, and redness of the conjunctiva (hyperemia). Patients usually present with extreme pain, nausea and/or vomiting, light sensitivity, and blurry vision. Treatment is directed toward reducing intraocular pressure and can include oral or intravenous acetazolamide and/or mannitol, as well as topical beta-blockers or parasympathomimetics [1].

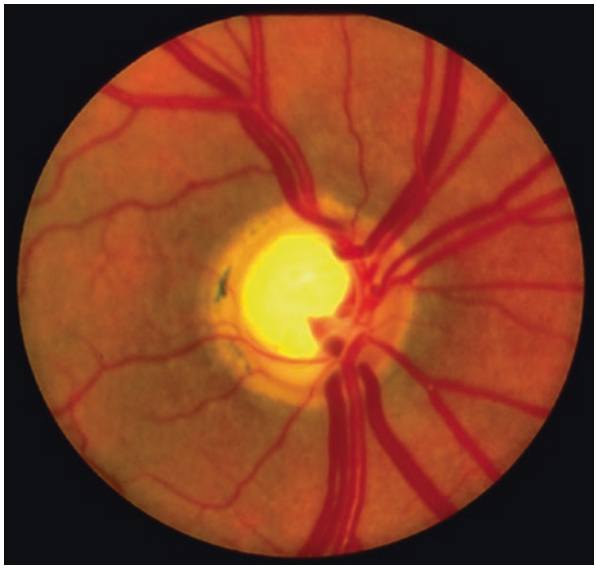

Fig. 21 Enlargement of the optic cup in a patient with open-angle glaucoma. © 2019 American Academy of Ophthalmology

5 Retina

5.1 Diabetic Retinopathy

Chronic hyperglycemia in diabetes can cause microvascular disease in many parts of the body, including the eye. Diabetic retinopathy begins as a non-proliferative microvascular disease, characterized by retinal hemorrhages, hard exudates, and microaneurysms (Fig. 22). It can progress into proliferative retinopathy, characterized by neovascularization and worsening hemorrhages. Treatment for diabetic retinopathy involves strict control of underlying diabetes, along with intravitreal anti-vascular endothelial growth factor (VEGF) and laser photocoagulation if there are neovascular changes [1, 5].

5.2 Hypertensive Retinopathy

Hypertensive retinopathy is a retinal vascular disease caused by long standing high blood pressure. Characteristic retinal changes include "copper-wiring" of retinal vessels, retinal hemorrhages, cotton wool spots (ischemic areas of the retina), and possible optic nerve edema (Fig. 23) [1, 5].

5.3 Age-Related Macular Degeneration (AMRD)S

Age-related macular degeneration (AMRD) is a degenerative retinal disease that produces central vision loss. Dry macular degeneration is caused by deposition of yellow extracellular material (drusen) under the retina and presents with gradual vision loss and metamorphopsia (Fig. 24). Wet macular degeneration is caused by

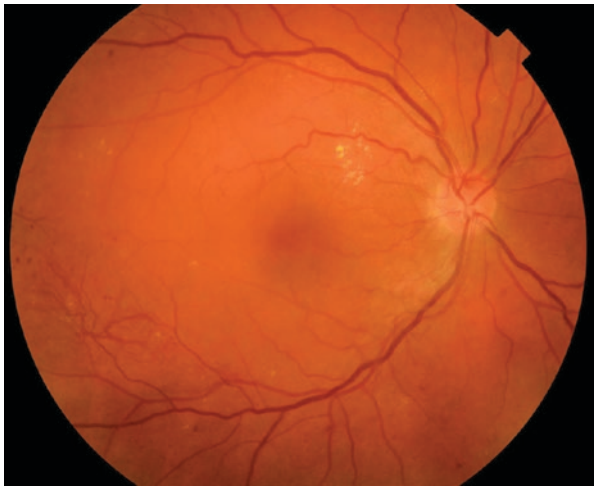

Fig. 22 Dark dot-blot hemorrhages and lighter hard exudates in non-proliferative diabetic retinopathy. (Picture courtesy of Retina Service, Wills Eye Hospital)

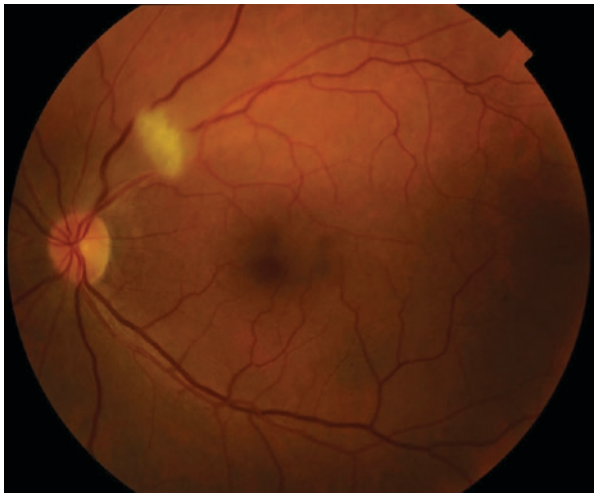

Fig. 23 "Copper-wiring" of retinal vessels and fluffy cotton-wool spot in hypertensive retinopathy. (Picture courtesy of Retina Service, Wills Eye Hospital)

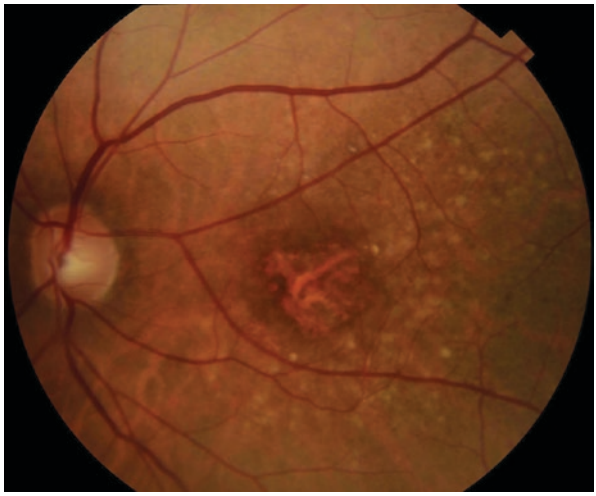

Fig. 24 Extracellular material (drusen) deposits in the macula of a patient with age-related macular degeneration. (Picture courtesy of Retina Service, Wills Eye Hospital)

deeper layer neovascularization and presents with retinal hemorrhage, fluid accumulation, and further vision loss. Dry AMRD is treated with a combination of zinc, copper, lutein, zeaxanthin, and antioxidant vitamins C and E (the Age-Related Eye Disease Study [AREDS] formula recommended by the National Eye Institute). Wet AMRD is treated most commonly with intravitreal anti-VEGF injections [1, 5].

5.4 Cytomegalovirus (CMV) Retinopathy

Opportunistic infections such as cytomegalovirus (CMV) can affect the retina, causing retinitis. In patients with AIDS with a CD4 count less than 50/mm^3, CMV

can spread to the retina and cause visual changes along with large cotton wool spots and hemorrhages on examination. Treatment involves intravitreal antiviral injections with ganciclovir or foscarnet, along with systemic antiviral therapy, HAART therapy, and hospital admission [1, 5].

5.5 Central Retinal Artery Occlusion (CRAO), Branch Retinal Artery Occlusion (BRAO)

Occlusion of the central retinal artery (CRAO) can produce sudden painless unilateral vision loss, most commonly caused by an atherosclerotic embolus. Occlusion can be total or can involve a branch of the central retinal artery (branch retinal artery occlusion, BRAO). Risk factors for CRAO and BRAO include carotid atherosclerosis, atrial fibrillation, diabetes mellitus, hypertension, and hypercholesterolemia. Examination reveals ischemic retinal whitening and a characteristic macular cherry red spot, indicating the unaffected choroidal vasculature supplying the macula (Fig. 25). There are no effective treatments for CRAO, but hyperbaric oxygen and ocular massage are considered [1, 5].

5.6 Central Retinal Vein Occlusion (CRVO), Branch Retinal Vein Occlusion (BRVO)

Occlusion of the central retinal vein (CRVO), or a branch of the retinal vein (BRVO), can also cause sudden, painless visual loss. Risk factors for this disease include age, hypertension, diabetes mellitus, smoking history, and hypercoagulability. Examination of the retina can reveal dilation of retinal veins, retinal hemorrhages,

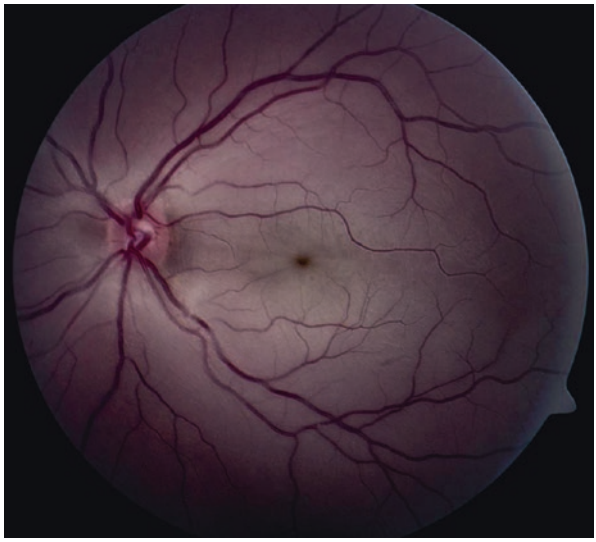

Fig. 25 Pale ischemic retina with cherry red spot in the macula in central retinal artery occlusion. © 2019 American Academy of Ophthalmology

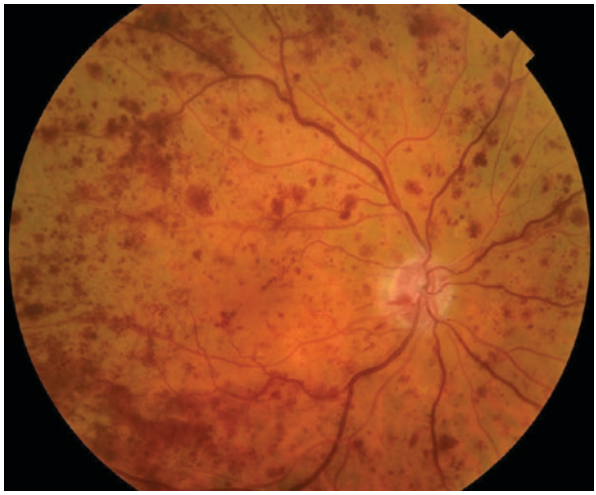

Fig. 26 Dilated retinal veins and numerous retinal hemorrhages in central retinal vein occlusion. (Picture courtesy of Retina Service, Wills Eye Hospital)

and retinal edema (Fig. 26). Treatment can include laser treatment and/or intravitreal anti-VEGF injections, to prevent ischemia-induced neovascularization [1, 5].

6 Oncology

6.1 Basal Cell Carcinoma (BCC)

Basal cell carcinoma (BCC) is the most common malignant eyelid lesion. It most commonly presents on the lower eyelid as a nodule or ulcerated lesion with rolled, pearly borders and telangiectasias (Fig. 27). There can be associated eyelash loss (madarosis) in the region of the lesion. BCC rarely metastasizes but can be locally invasive into the lacrimal drainage system, orbit, or rarely the cranial cavity. Treatment involves surgical excision with frozen sections to ensure negative margins [1, 6].

6.2 Squamous Cell Carcinoma (SCC)

Squamous cell carcinoma (SCC) is the second most common malignant eyelid lesion. It most commonly presents on the upper eyelid or medial canthus as a well-defined nodule with frequent hyperkeratosis or ulceration with a red base. It can also present as a papillomatous or cutaneous horn lesion (Fig. 28). Eyelid SCC can be locally invasive, through contiguous soft tissue extension or neural invasion. Treatment involves surgical excision using frozen sections or Mohs microsurgery to ensure negative margins [1, 6].

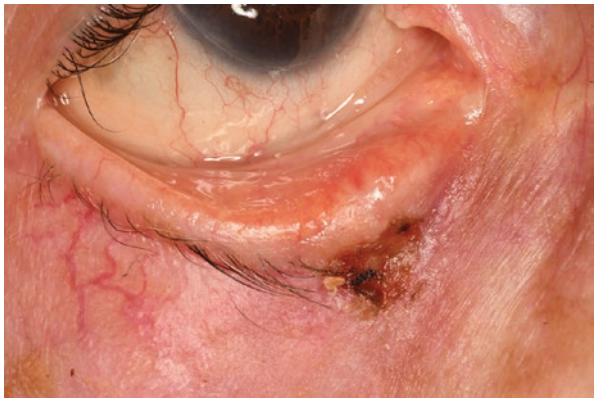

Fig. 27 Ulcerated lesion with pearly margins on the lower eyelid margin, consistent with basal cell carcinoma. (Picture courtesy of Dr. Carol Shields, Ocular Oncology Service, Wills Eye Hospital)

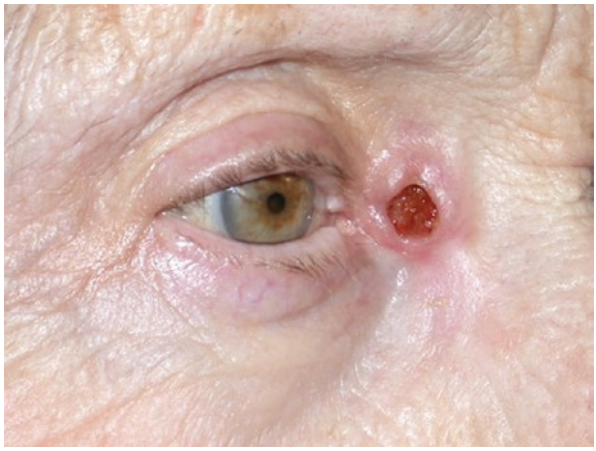

Fig. 28 Extensive ulcerated lesion with red base near the medial canthus, consistent with squamous cell carcinoma. (Picture courtesy of Dr. Robert Penne, Oculoplastics Service, Wills Eye Hospital)

6.3 Choroidal Melanoma

Choroidal melanoma is the most common primary intraocular tumor in adults in the United States. Choroidal melanoma presents clinically as a dome or mushroom-shaped mass under the retina and can be pigmented or nonpigmented (Fig. 29). It can cause visual loss and can grow to extend into the anterior chamber of the eye or the orbit. The most common site of metastasis for melanoma is the liver [1, 7].

6.4 Retinoblastoma

Retinoblastoma is the most common intraocular malignancy in children. It presents clinically with a white pupillary light reflex (leukocoria, Fig. 32) and can cause visual loss and strabismus. Careful examination will reveal a white lesion in the retina, with feeding vessels (Fig. 30). Retinoblastoma can be associated with other tumors, including pinealoblastoma and osteosarcoma [1, 7].

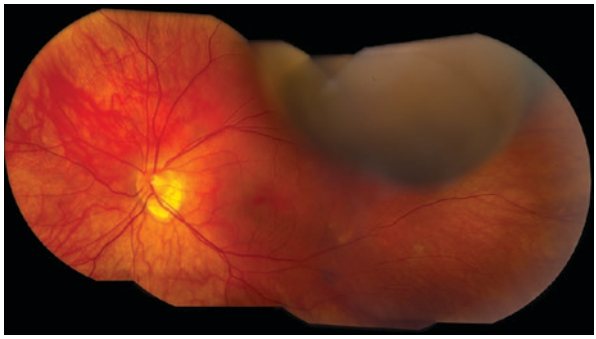

Fig. 29 Pigmented, mushroom-shaped melanoma visible on fundus exam. (Picture courtesy of Dr. Carol Shields, Ocular Oncology Service, Wills Eye Hospital)

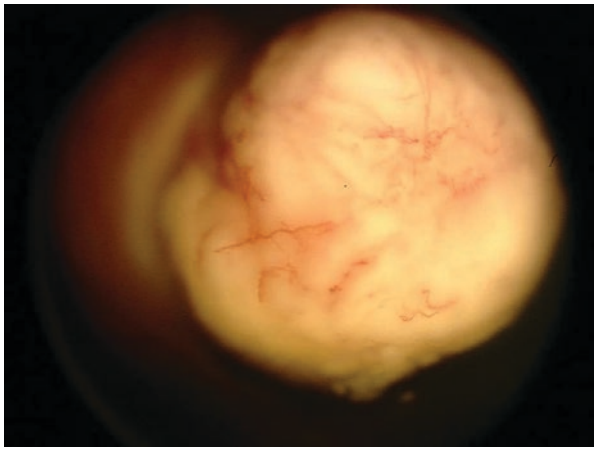

Fig. 30 White retinal lesion in the eye of a child, consistent with retinoblastoma. (Picture courtesy of Dr. Carol Shields, Ocular Oncology Service, Wills Eye Hospital)

6.5 Rhabdomyosarcoma

Rhabdomyosarcoma is a malignant skeletal muscle tumor, most commonly presenting as an orbital mass in children. Patients can present with bulging of the eye (proptosis), drooping of the eyelid (ptosis), a palpable mass, or pain. The mass can enlarge and can invade the adjacent bones and sinuses. Computed tomography (CT) will show a well-circumscribed orbital mass that spares the extraocular muscles. Treatment depends on tumor staging and may involve a combination of chemotherapy, radiation, and/or surgery [1, 6].

7 Pediatrics

7.1 Sturge-Weber Syndrome

Sturge-Weber syndrome is a congenital neurocutaneous disorder caused by GNAQ mutations. It is characterized by capillary-venous malformations of the skin, eye,

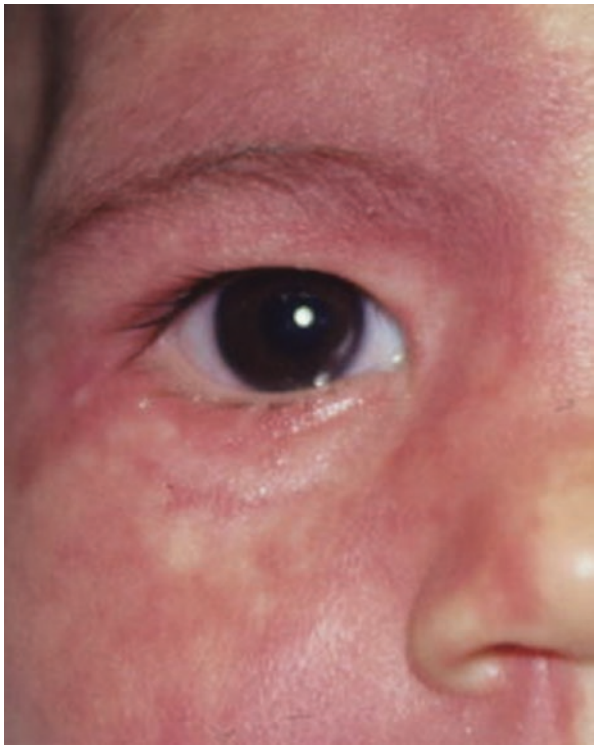

Fig. 31 Unilateral pigmentation (nevus flammeus, port wine mark) in the V1-V2 distribution of a patient with Sturge-Weber syndrome. (Picture courtesy of Dr. Alex Levin, Pediatrics, Wills Eye Hospital)

Fig. 32 White pupillary light reflex (leukocoria) in a patient with retinoblastoma. (Picture courtesy of Dr. Carol Shields, Ocular Oncology Service, Wills Eye Hospital)

and brain. A common presentation includes capillary malformation of the face in the V1-V2 distribution of the trigeminal nerve (nevus flammeus, port wine mark), which can be associated with glaucoma (Fig. 31). Associated capillary-venous malformations in the brain can cause seizures [1–3].

7.2 Leukocoria

A normal pupillary red light reflex is caused by the reddish reflection of light from the retina. Leukocoria, or white pupillary light reflex, can be caused by a number of conditions (Fig. 32). As mentioned previously, retinoblastoma can be the cause of

Table 1 Neonatal conjunctivitis differential [1,3]

Etiology	Clinical features	Time of presentation	Treatment	Prophylaxis	Additional information
Neisseria gonorrhea	Swelling, conjunctival injection, excessive mucopurulent discharge	2–3 days after birth	Intramuscular ceftriaxone	Topical erythromycin	
Chlamydia trachomatis	Swelling, conjunctival injection, mucoid discharge, pseudomembranes	5–14 days after birth	Oral erythromycin	Treatment of maternal chlamydia	
Herpes simplex	Swelling, conjunctival injection, vesicles on eyelid skin, corneal ulcers	1–2 weeks after birth	Intravenous and topical antivirals	Avoid contact with active HSV infection	
Chemical (silver nitrate)	Conjunctival injection, tearing	<24 hours after birth	Supportive (artificial tears)		Commonly caused by silver nitrate

such a reflex. Other causes of leukocoria include retinal detachment or congenital cataract, commonly caused by congenital infections such as rubella. Traumatic cataract can also present with a white pupillary reflex [1–3].

7.3 Neonatal Conjunctivitis

Neonatal conjunctivitis can be caused by several etiologies, as listed below (Table 1).

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Dermatology Imaging

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Abbreviations

ACD	Allergic contact dermatitis
AD	Autosomal dominant
BSA	Body surface area
CS	Corticosteroid/s
DRESS	Drug reaction with eosinophilia and systemic symptoms
HIV	Human immunodeficiency virus
HSV	Herpes simplex virus
IVDA	IV drug abuse
MC	Most common
NSAIDS	Non-steroidal anti-inflammatory drugs
PCT	Porphyria cutanea tarda
PG	Pyoderma gangrenosum
SIADH	Syndrome of inappropriate antidiuretic hormone secretion
SJS	Stevens-Johnson syndrome
SLE	Systemic lupus erythematosus
TEN	Toxic epidermal necrolysis
UV	Ultraviolet

1 Pyoderma Gangrenosum (PG)

PG is a diagnosis of exclusion. The usual history is of a pustule progressing into painful purulent ulcers with dusky, raised/bullous, undermined borders (Fig. 1). It may be concerning for a serious infection or even necrotizing fasciitis; however, the PG patient is typically nontoxic appearing. PG appears in the setting of systemic

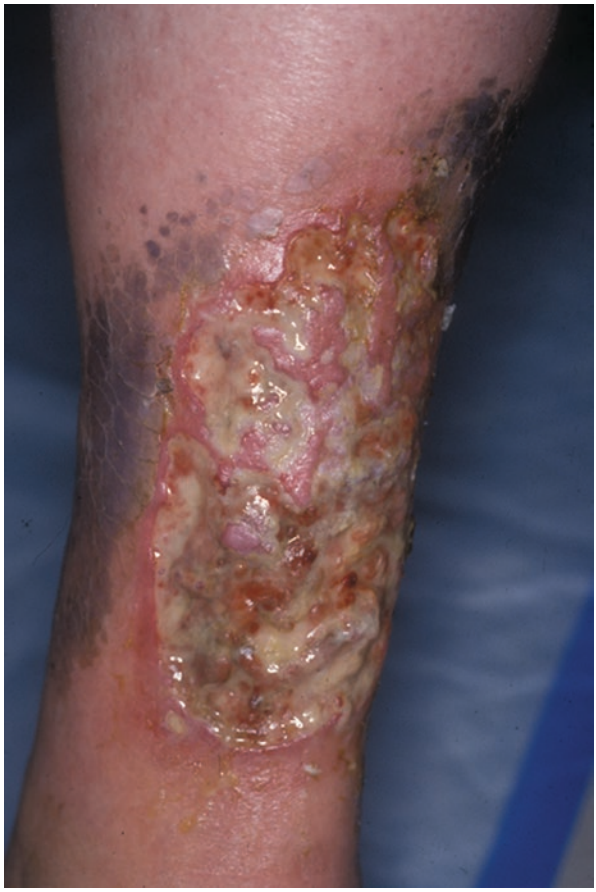


Fig. 1 Purulent ulcers on the extremity with dusky, raised borders. (Courtesy of Charles E. Crutchfield III, MD's Dermatology Image Library Archive)

disease 50% of the time (e.g., inflammatory bowel disease, myeloma, or monoclonal gammopathy). Treatments are anti-inflammatory topical, intralesional, or systemic steroids, cyclosporine, TNF-alpha inhibitors, and/or topical tacrolimus [1, 2].

2 Necrotizing Fasciitis

Patients are extremely toxic appearing (fever, shock, tachycardia); the infection is malodorous and may exhibit crepitus; and pain is out of proportion to physical findings. NSAIDS can mask symptoms and should be avoided. The infection of the skin and deeper subcutaneous tissues tracks along fascia beyond clinical visibility. The course is rapidly progressive and 50% fatal; patients require immediate critical care support, fluid resuscitation, broad spectrum IV antibiotics, and an emergent surgery consult. Surgery will collect tissue cultures and can perform a test incision (showing pathologic undermining), debridement, and/or fasciotomy. Treatment should be initiated *without delay for imaging;* gas shows inconsistently on plain X-rays. CT or MRI can show fascial plane edema. One specific finding is *lack of fascial enhancement on enhanced CT* [1, 2].

For diagnosis, the white blood cell count, hemoglobin, sodium, glucose, serum creatinine, and C-reactive protein values comprise the Laboratory Risk Indicator for Necrotizing Fasciitis score. Risk factors for developing necrotizing fasciitis include immunosuppression, diabetes, elderly patients, peripheral vascular disease, IVDA history, recent surgery, or trauma. Females, the elderly, longer time to debridement, and increased lactic acid or creatinine levels carry increased mortality risk. Group A strep is thought to be the most common cause [1, 2].

3 Burns

Burns may be due to heat, radiation, electrical exposure, chemical exposure, and frictional trauma (Fig. 2a–c). Patients should be referred to a burn center for inhalational, electrical, or chemical burns; burns of the face, hands, feet, and/or genitals; partial thickness burns of >10% BSA; or any size full thickness burns. Also refer

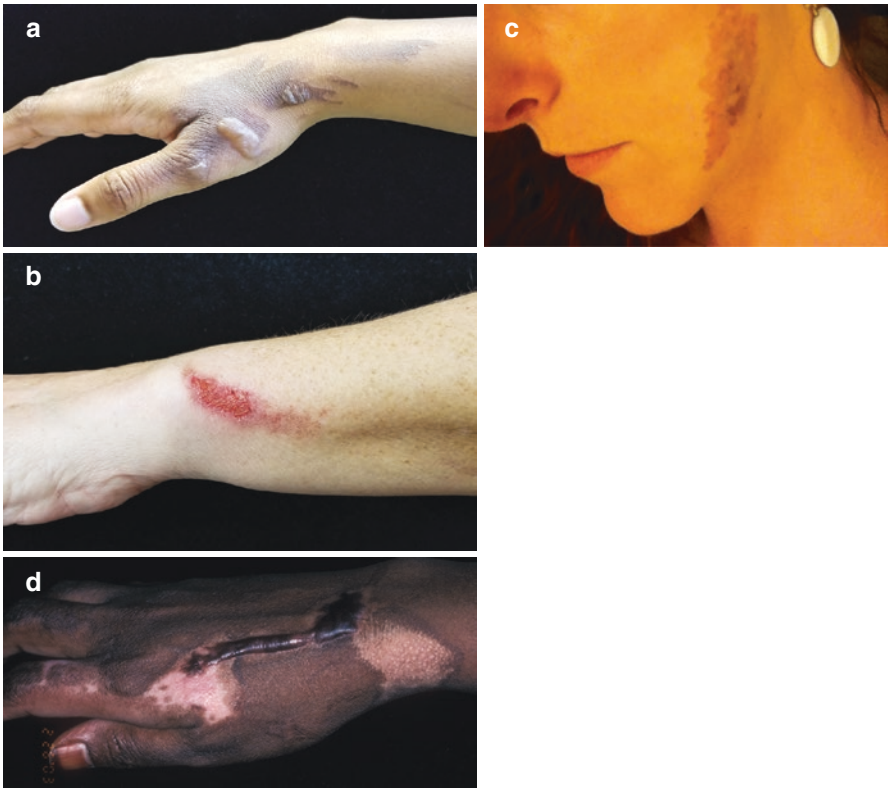


Fig. 2 (**a**) Epidermal/superficial dermal burn on the hand/forearm showing hyperpigmentation and blistering. (**b**) Epidermal/superficial dermal burn on the forearm showing erosion/crusting, erythema, and hyperpigmentation. (**c**) Hyperpigmentation after at least epidermal burn from curling iron on the face. (**d**) Hypertrophic/keloid and hyper/hypopigmented scarring after at least deep dermal burns on the hand/forearm. (Courtesy of Charles E. Crutchfield III, MD's Dermatology Image Library Archive)

Table 1 Summary of 2009 American Burn Association Revised Burn Classification System [2, 3]

Depth	Appearance	Exam	Classification	Treatment	Healing
Epidermis	Non-blistering, discoloration	Painful, blanches with pressure	Superficial	None needed; *gentle supportive care* includes cleansing with soap and water, Vaseline, cool compresses, sun protection	~1 week, no scarring
Superficial dermis	Serous or hemorrhagic bullae, deep redness, erosion, exudative	Severe pain, blanches with pressure	Superficial partial thickness	Change dressing daily with topical antibiotic and non-stick gauze in addition to *gentle supportive care*	~3 weeks, mild scarring
Most of dermis (including follicular structures)	Fragile bullae, erosions, deep red, pale, or speckled color	Severe pain, sensation only to pressure, does not blanch with pressure	Deep partial thickness	Surgical intervention (e.g., excision, skin substitution)	>3 weeks, hypertrophic scars, contractures (Fig. 2d)
Full-thickness of dermis	Waxy white, gray, or charred black, dry and stiff	Only sensation to deep pressure intact, does not blanch with pressure	Full-thickness	Surgical intervention	Significant scarring
Beyond the skin	Penetrates through underlying fat, fascia, muscle or bone	Only sensation to deep pressure intact	Fourth degree	Surgical intervention	Does not heal without surgical intervention, can be lethal

any patients with complicating comorbidities (whether concomitant traumatic injuries, or requiring special medical, psychological, or social interventions) [1–3].

Thermal burns may be deeper than expected on thinner skin or in pediatric or elderly patients. The extent of injury can progress over several days. Classification and treatment are based on the depth of injury using the 2009 American Burn Association revised burn classification system (Table 1). The most accurate BSA tool to calculate the extent of burn injury in children and adults is the Lund-Browder chart; however, it may underestimate the BSA if obese. The rule of nines method for adults is less accurate [1–3].

4 Porphyria Cutanea Tarda (PCT)

Porphyrias result from disorders of heme synthesis. PCT is the most common porphyria; dysfunctional uroporphyrinogen decarboxylase causes porphyrins to build up in the skin. It is triggered or worsened by liver injury (e.g., excess alcohol use, hepatitis, HIV, exogenous estrogens, iron overload states). It leads to photosensitivity, excess facial hair or pigmentation, and fragile skin. Erosions and blisters form after minor trauma, leading to scarring and milia in sun-exposed areas (Fig. 3). *Acquired* Type 1 PCT is the most common form. Autosomal dominant Type II PCT is less common [1, 2].

Diagnosis is confirmed by elevated urinary uroporphyrins/coproporphyrins, plasma uroporphyrins, fecal isocoporphyrins, skin biopsy, or urine that fluoresces pink under Wood's lamp (black light). Order liver function tests, and screen for iron overload, hepatitis B or C, HIV, the hemochromatosis gene if ferritin elevated, and hepatocellular carcinoma with alpha-fetoprotein. Imaging is rarely needed to evaluate the liver for size, iron content, and tumors. Treatment includes sun protection (physical zinc or titanium sunscreens, protective clothing, sun avoidance), phlebotomy for treatment of iron overload or hemochromatosis, and antimalarials [1, 2].

5 Urticaria (+/− Angioedema)

Urticaria, or hives, appear as wheals of edematous pink-red papules or plaques (Fig. 4a, b), with or without central pallor, pruritus, or associated angioedema (deeper subcutaneous/mucosal swelling, Fig. 5a, b). *Individual lesions last <24 h.* Urticaria/angioedema can affect the mucosa, respiratory, and gastrointestinal systems.

Urticaria results from triggering and degranulation of mast cells, releasing histamine and pro-inflammatory/vasoactive factors. Most cases of acute urticaria are idiopathic; others are triggered by infection/URI, drugs, or food. Chronic urticaria is either idiopathic or autoimmune much more commonly than related to chronic

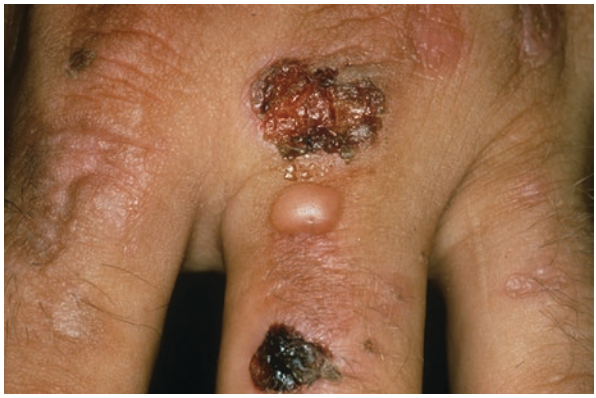


Fig. 3 Erosions, blisters, crusts, and scars on the sun-exposed dorsal hands in PCT. (Courtesy of Charles E. Crutchfield III, MD's Dermatology Image Library Archive)

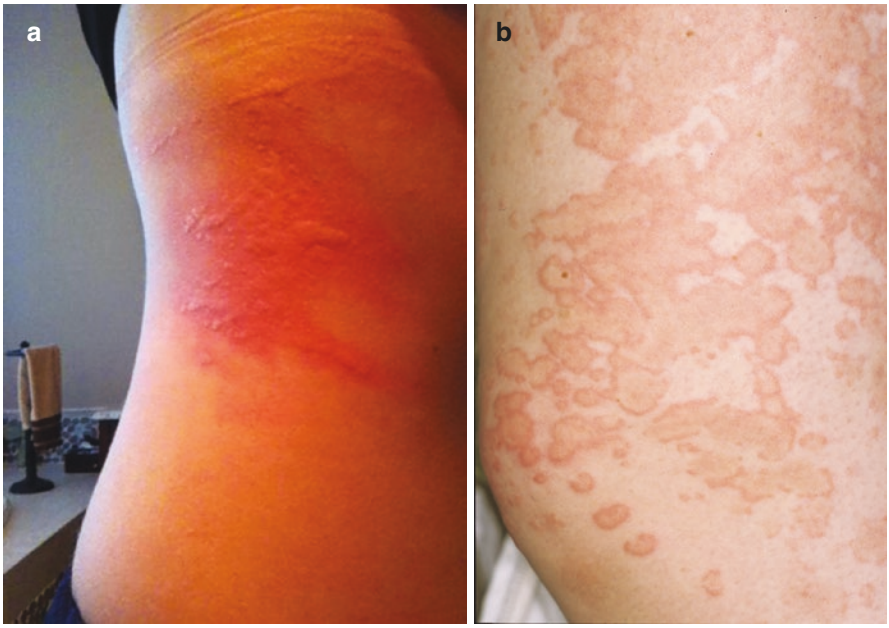


Fig. 4 (**a**) Edematous plaques with erythematous flare on the trunk. (**b**) Edematous erythematous annular wheals with central pallor. (Courtesy of Charles E. Crutchfield III, MD's Dermatology Image Library Archive)

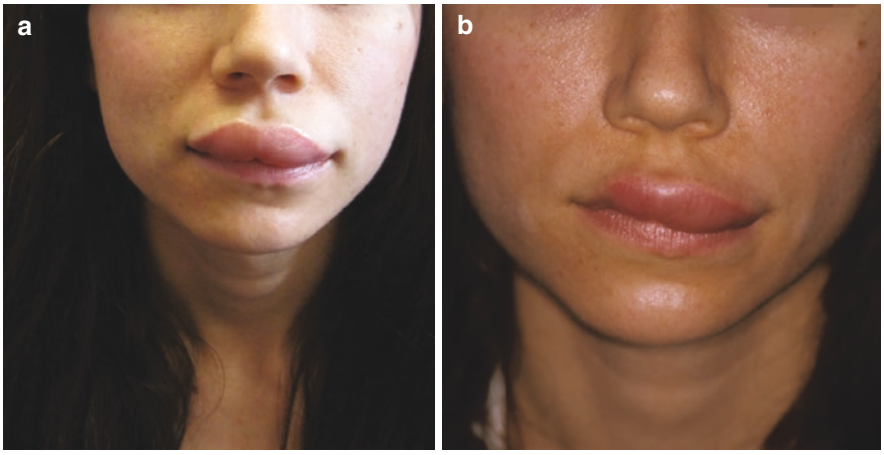


Fig. 5 (**a**, **b**) Subcutaneous/mucosal swelling of the lips in angioedema; angioedema can occur in isolation or in association with urticarial skin lesions. (Courtesy of Charles E. Crutchfield III, MD's Dermatology Image Library Archive)

infections. Long-acting antihistamines are the mainstay of treatment, up to 2–4xs the usual dose of H1 antihistamines daily, with sedating H1 antihistamines nightly and H2 antihistamines if needed. Oral CS should be avoided due to the risk of rebound in most cases. Avoid triggers, e.g., drugs (aspirin, NSAIDS, codeine, morphine) or physical (cold, pressure) [1, 2].

6 Angioedema Without Urticaria: Acquired v. AD Hereditary (Types I-III)

Angioedema is tissue edema *without* skin findings (Fig. 5a, b), due to increased bradykinin levels and vascular permeability. Angioedema lasts 2–3 days, commonly affecting the face, genitals, and GI/respiratory tract (can be mistaken for acute abdomen). There are either acquired or hereditary types of angioedema without urticaria. *C4 levels are low* in hereditary Types I, II, and acquired angioedema. In hereditary angioedema Type I, both C1 inhibitor protein levels and activity are low. In hereditary angioedema Type II, C1 inhibitor protein levels are normal, but activity is impaired. Acquired angioedema is associated with disease states such as lymphoma or MGUS; C1 inhibitor levels/activity and C1q levels are low. *C4 levels are normal* in hereditary angioedema Type III, idiopathic, or drug-induced cases of angioedema. Treatments for hereditary angioedema include C1 inhibitors, ecallantide or icatibant, and long-term PO danazol [1, 2].

7 Atopic Dermatitis (AD, Eczema)

Atopy results from a complex interplay between genes, the environment, and Type-I (IgE-mediated) hypersensitivity reactions. AD onset is mostly from 1 to 5 years, manifesting with itchy, inflamed, red, scaly skin that may ooze, crust, and become secondarily infected (Fig. 6a–d). Chronic itch-scratch cycles can lead to hyper- or hypo-pigmentation and skin thickening (Fig. 6b–d). Infants usually present with cheeks/extensors involved. Children and adults typically show flexural involvement (Fig. 6a–c). There is epidermal barrier dysfunction with impaired filaggrin, decreased ceramides, and increased risk of secondary skin infections (staph, herpes, molluscum). The atopic march refers to AD patients' increased risk of accompanying asthma and allergic rhinitis. Triggers for AD flares include skin dehydration, microbial colonization/superinfection, humidity/temperature extremes, and irritant detergents/fabrics (wool). Some environmental allergies (dust mites, pollens, contact allergens) and rarely certain foods (eggs, milk, peanuts, soybeans, fish, wheat) can be triggers [1, 2].

Treatment is directed at daily maintenance with topical emollients and avoidance of triggers. Mild cases are controlled by intermittent topical CS and/or topical calcineurin inhibitors. Flares are managed with stronger topical CS until clear, antihistamines, and antibiotics if infected. Severe or recalcitrant cases may benefit from narrow-band UVB phototherapy or short courses of systemic CS as a bridge to biologics or steroid sparing systemic immunosuppressants (e.g., cyclosporine, methotrexate) [1, 2].

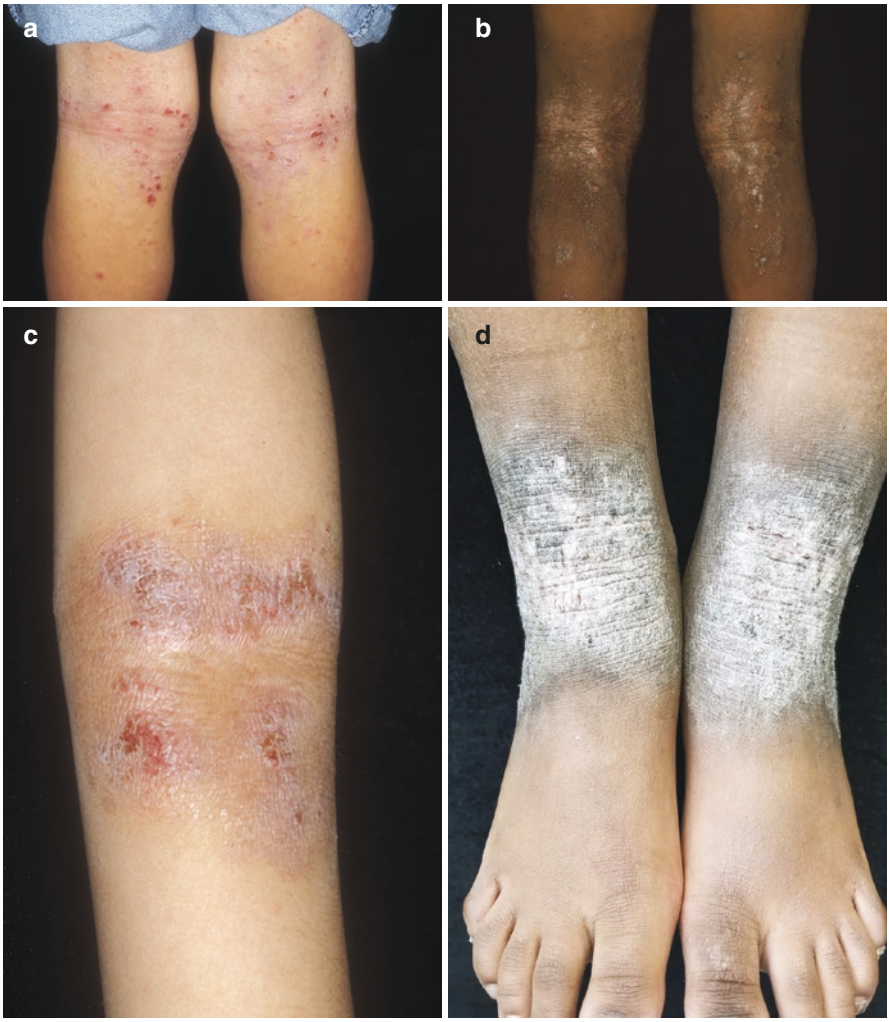


Fig. 6 (**a**, **b**) Erythematous, crusted, excoriated papules/plaques in flexures with pigment alteration. (**c**, **d**) Thickened, crusted, lichenified plaques in flexures with pigment alteration. (Courtesy of Charles E. Crutchfield III, MD's Dermatology Image Library Archive)

8 Herpes Simplex Virus (HSV, Human Herpes Virus 1 and 2)

HSV lesions appear as grouped vesicles, pustules (or ruptured eroded/ulcerative lesions) with scalloped borders on a red base (Fig. 7). The double-stranded DNA virus is transmitted through bodily fluids and remains latent in the dorsal root ganglion. Orolabial infection of HSV-1 is most common (MC). Most often this is asymptomatic in adults (90% seroprevalence). Children can present with gingivostomatitis, and young adults can present with pharyngitis and mono-like syndrome

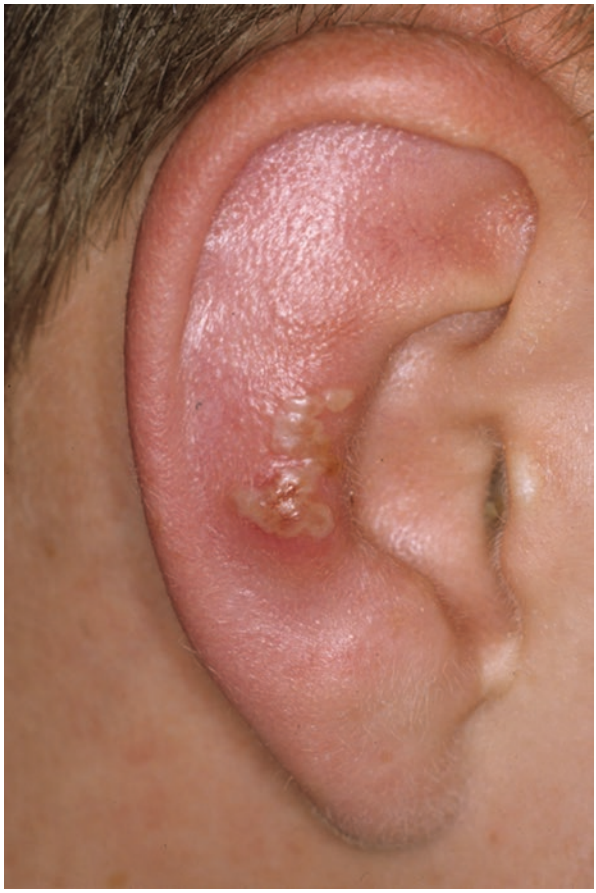


Fig. 7 Yellow-crusted, grouped vesicles on an erythematous base with scalloped borders. (Courtesy of Charles E. Crutchfield III, MD's Dermatology Image Library Archive)

with drooling and dysphagia. HSV-2 more commonly affects the genitals and may appear as perifollicular pustules or chronic, painful ulcers. Sequelae of HSV infection include erythema multiforme (HSV-1 is the MC cause of EM minor), aseptic meningitis, encephalitis (MC cause of fatal viral encephalitis, no skin findings), and eczema herpeticum (widespread infection in areas of atopic dermatitis +/− systemic symptoms, *can be life threatening in children*) [1, 2].

HSV reactivations are usually heralded by prodromes of burning, itching, pain, or tingling. They are less severe than the initial presentation, dermatomal, and triggered by UV radiation, stress, injury, fever, immunosuppression, or spontaneous. Outside of the common oral/genital locations, herpes gladiatorum is so named for wrestlers transmitting HSV to the neck/face. Herpetic whitlow refers to infections on the fingers among children or dental workers. Neonatal HSV has the highest risk of transmission if the mother is infected with a primary HSV outbreak during delivery (<3% risk if mother is having a recurrence). Newborn ocular HSV is MC with vaginal delivery [1, 2].

Diagnosis can be confirmed by Tzanck prep, culture, PCR (most sensitive/specific), skin biopsy, serology, or direct fluorescent antibody testing. Topical treatments are not as effective as oral antivirals (acyclovir, valacyclovir, famciclovir). Consider daily oral prophylaxis for >6 episodes per year or frequent severe episodes; viral shedding is also decreased. Episodic therapy is most helpful if a patient experiences a prodrome. Foscarnet and cidofovir are options for acyclovir resistance (often immunosuppressed). IV acyclovir is needed in severe cases (eczema herpeticum, neonatal HSV, or the immunosuppressed) [1, 2].

9 Scabies

Mites create linear burrows or vesicopustules favoring wrists (Fig. 8a–c), web spaces of digits (Fig. 8d, e), trunk, umbilicus, or papules and nodules of genitals. In the crusted (Norwegian) type, patients are often immunocompromised and may have secondary bacterial infections. Risk factors for infection include overcrowded

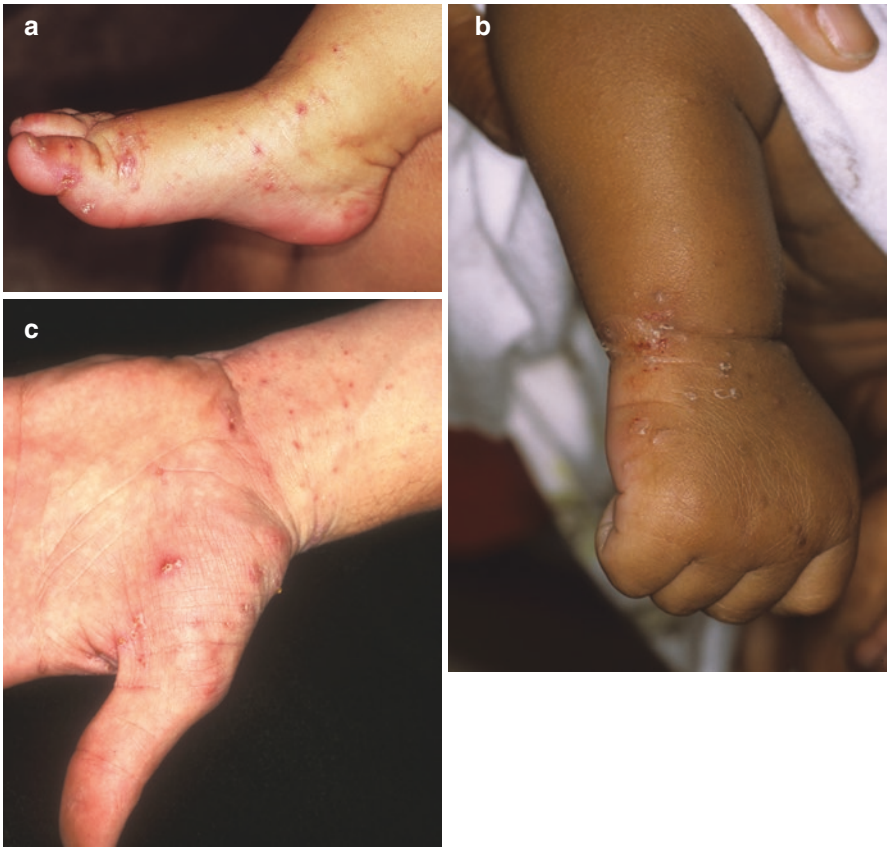


Fig. 8 (**a–c**) Minute, erythematous vesicopustules, and linear burrows on the wrists, ankles. (**d**, **e**) Papules/burrows of the web spaces. (**f**) Mineral oil prep showing scabies mite and eggs. (Courtesy of Charles E. Crutchfield III, MD's Dermatology Image Library Archive)

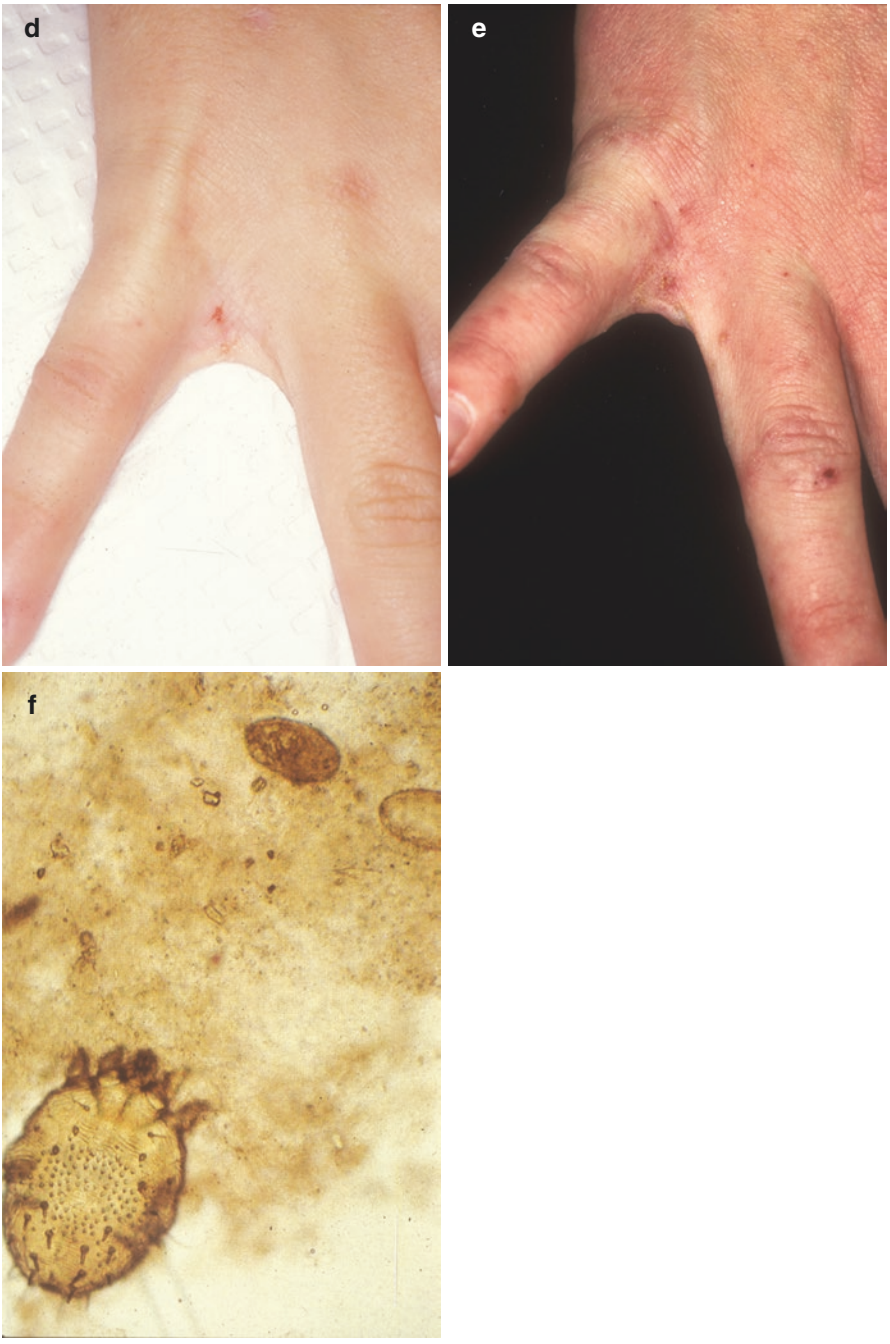


Fig. 8 (continued)

environments. Mites have a 30-day life cycle and can survive 1 week off a human host. Mineral oil scraping can aid in diagnosis (Fig. 8f). Treatments include permethrin 5% cream from the neck down, PO ivermectin or lindane [1, 2].

10 Infantile Hemangioma (IH)

IHs (Fig. 9a, b) appear in the first few weeks of life, in ~5% of infants, more common if female or premature, and are GLUT-1 positive. They exhibit a proliferative phase for the first 6–9 months, involute over the next several years, and resolve by

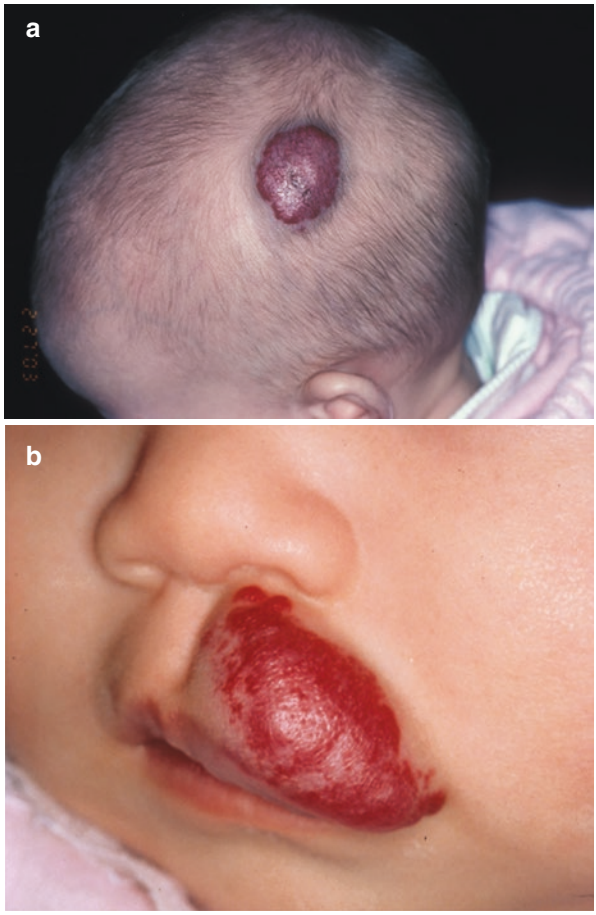


Fig. 9 (**a**, **b**) Bright red plaques of the scalp and upper lip. (Courtesy of Charles E. Crutchfield III, MD's Dermatology Image Library Archive)

5 years in 50% of cases, sometimes with residual atrophic or fibrofatty skin changes. Superficial IHs are bright red. Deep IHs are light blue-purple or deeper/subcutaneous masses. Mixed IHs share features of both. Distribution may be focal or segmental [1, 2].

Special considerations include ulcerative IHs (10%) which carry risk of infection and scarring. Periocular IHs require ophthalmologic evaluation due to risk of interference with the globe, visual axis, and musculature. Nasal tip, columella, lip, or vermillion IHs carry risk of disfigurement. IHs of the beard area/lower face require otolaryngologic evaluation for associated airway hemangiomas. Large facial IHs, especially segmental, carry the risk of *PHACES* syndrome (posterior fossa malformation, hemangioma, arterial abnormalities, cardiac/coarct, eye abnormalities, sternal defect, or supraumbilical raphe). Large lower body IHs, especially segmental, carry the risk of *LUMBAR* syndrome (lower body hemangioma and lipoma, urogenital anomalies, myelopathy, bony deformity, anorectal or arterial anomalies, renal anomalies). Midline lumbosacral IHs carry risk of spinal dysraphism. Visceral involvement should be ruled out by hepatic ultrasound if ≥5 IHs are present, especially hepatic involvement, which carries the risk of high output cardiac failure and hypothyroidism. Doppler ultrasound can distinguish from high flow lesions [1, 2].

Management is simply observation for uncomplicated lesions. Other therapies include topical timolol, topical high potency CS or steroid injection, or pulsed dye laser for thin lesions. When there is potential for vision or airway compromise; disfigurement (Fig. 9a); large, rapidly growing or severely ulcerating lesions; or high output cardiac failure, oral propranolol is the first-line systemic therapy [1, 2].

Note: In contrast to IH, *vascular malformations* (port-wine stains, venous malformations, lymphangiomas, arteriovenous malformations) are typically present at birth, M=F, not associated with prematurity, GLUT-1 negative, and life-long [1, 2].

11 Tinea (Pityriasis) Versicolor

Transformation of *Malassezia* furfur (normal flora) from yeast to hyphae leads to this superficial, non-inflammatory skin infection characterized by multiple, coalescing, pink/tan/light/brown round macules, patches, or thin plaques with subtle scale (Fig. 10a–c). The condition is often more noticeable in summer. Treatment options include antifungal shampoo BIW × 2–4 weeks, then taper for maintenance, ketoconazole cream BID × 2 weeks, fluconazole 200–400 mg PO QW × 2–3 weeks [1, 2].

12 Tinea, Dermatophytoses

T. Capitis (Head/Scalp), T. Faciei (Face), T. Corporis (Body), T. Cruris (Groin), (Foot), T. Manuum (Hand), T. unguium (Nail)

Tinea refers to a group of inflammatory, superficial, dermatophyte fungal infections of the skin (Fig. 11a–c), hair (Fig. 11d, e), and nails. *Trichophyton*

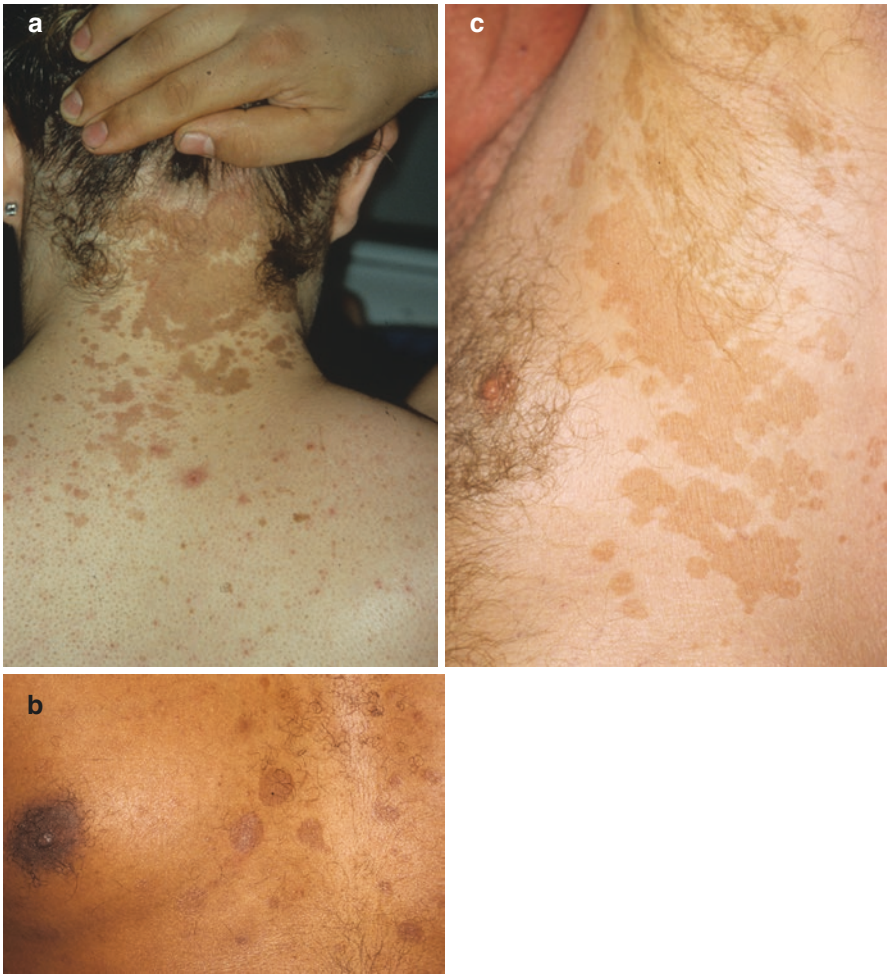


Fig. 10 (**a–c**) Multiple, coalescing, tan/brown round macules and patches, and plaques with subtle scale on the neck, chest, and axillae. (Courtesy of Charles E. Crutchfield III, MD's Dermatology Image Library Archive)

rubrum and mentagrophytes are the MC causes. The exception is in children with T. capitis; *T. tonsurans* is the MC cause (Fig. 11d, e). The skin shows "ringworm" annular, erythematous, scaly plaques with raised edges +/− pustules/vesicles (Fig. 11a–c). "Tinea incognito" presents atypically due to mistreatment with topical CS. Diagnosis can be confirmed by KOH scraping +/− fungal culture. Topical antifungals are the mainstay of treatment, applied twice daily for 2–4 weeks. Oral options include fluconazole, griseofulvin, terbinafine, or itraconazole (not approved for children). Typical oral courses last 2–4 weeks daily for the body, 4–8 weeks for hair bearing areas, and 6–12 weeks for nails (or longer with pulse-dosed regimens) [1, 2].

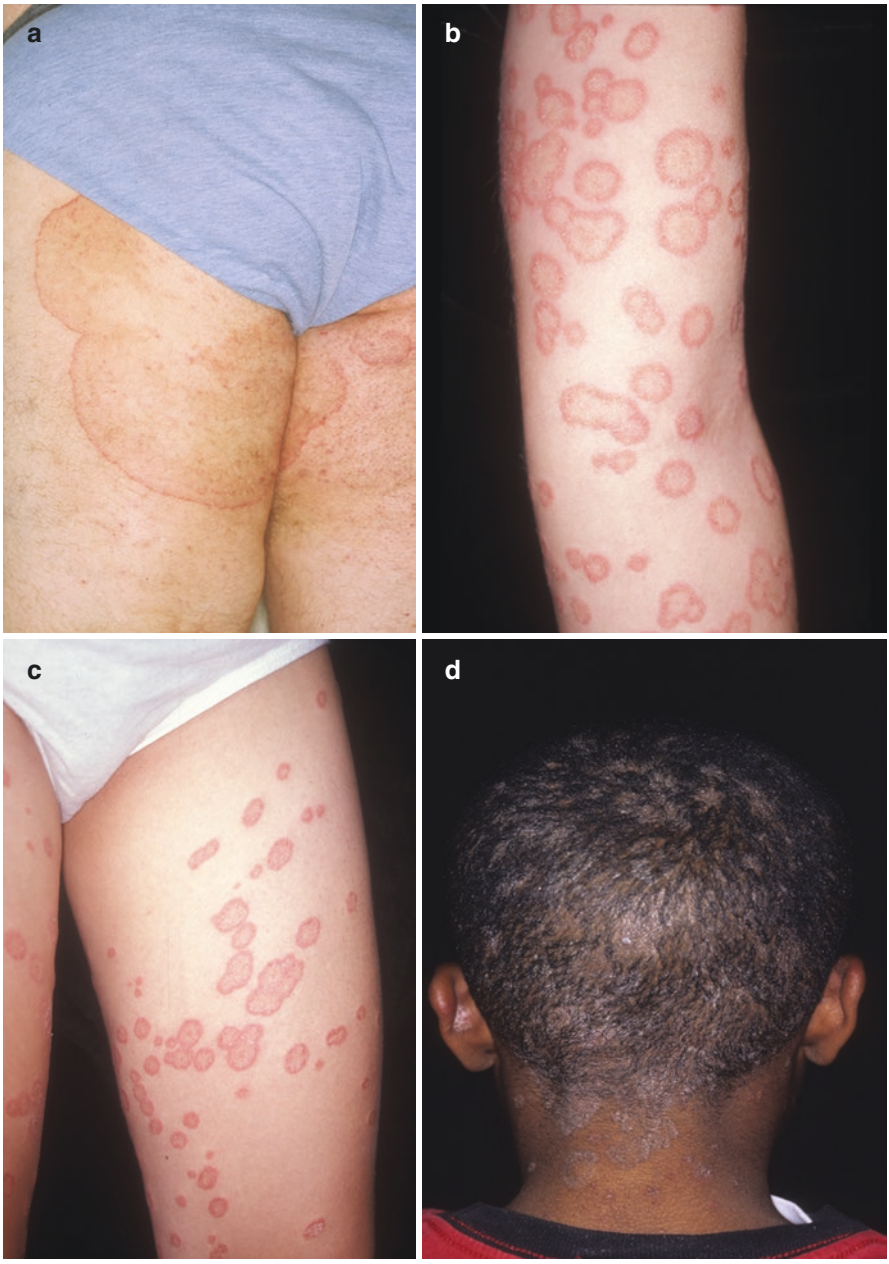
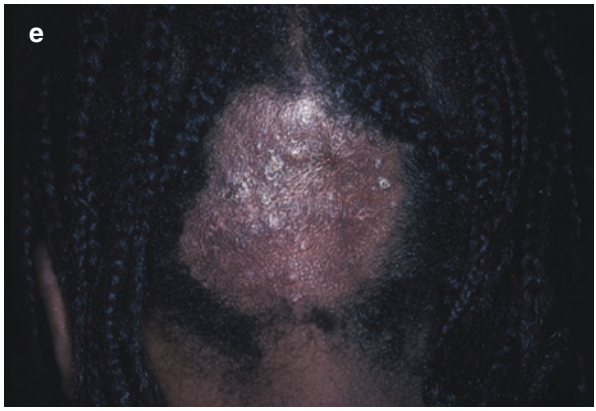


Fig. 11 (**a–c**) Annular erythematous scaly plaques on the trunk and extremities in tinea corporis. (**d**, **e**) Annular alopecic scaly patches on the scalp in tinea capitis. (Courtesy of Charles E. Crutchfield III, MD's Dermatology Image Library Archive)

Fig. 11 (continued)



13 Bullous Pemphigoid (BP)

BP is an autoimmune bullous disease causing *tense* bullae, and/or itchy urticaria-like edematous erythematous plaques that are fixed (Fig. 12) (as opposed to <24 h urticarial lesions). Oral lesions are less common. This is MC in the elderly or associated neurologic conditions. *Nikolsky's sign is negative* (deep blisters; not extended by firm rubbing of unaffected skin adjacent to/between lesions), and *Asboe–Hansen sign is negative* (blisters not expanded by direct downward pressure) [1, 2].

BP autoantibodies bind hemidesmosomal proteins BP antigen 1 (BP230) and BP antigen 2 (BP180) that anchor the epidermis to the dermis. It can be medication induced (loop diuretics, neuroleptics). Diagnostic studies include skin biopsy showing subepidermal (=*tense*) bullae, eosinophils, and positive direct immunofluorescence studies with IgG/C3 along the basement membrane, anti-BP180/BP230 enzyme-linked immunosorbent assay (ELISA), and peripheral eosinophilia in half of patients. Treatment is directed toward review and elimination of potential medication culprits and super potent topical steroids. If extensive, add oral CS +/− steroid-sparing immunosuppressive agents (e.g., azathioprine, mycophenolate mofetil, methotrexate) [1, 2].

14 Hypertrophic and Keloid Scars

Firm variably colored papules or plaques develop at the sites of surgery, piercing, or injury (even acne). Keloids (Fig. 13a, b) are more common in darker skin types and often extend beyond the wound margins (unlike hypertrophic scars). Treatments include silicone sheets, intralesional triamcinolone or 5-fluorouracil injections, excision (recurrence risk), lasers, radiation, and topical imiquimod [1, 2].

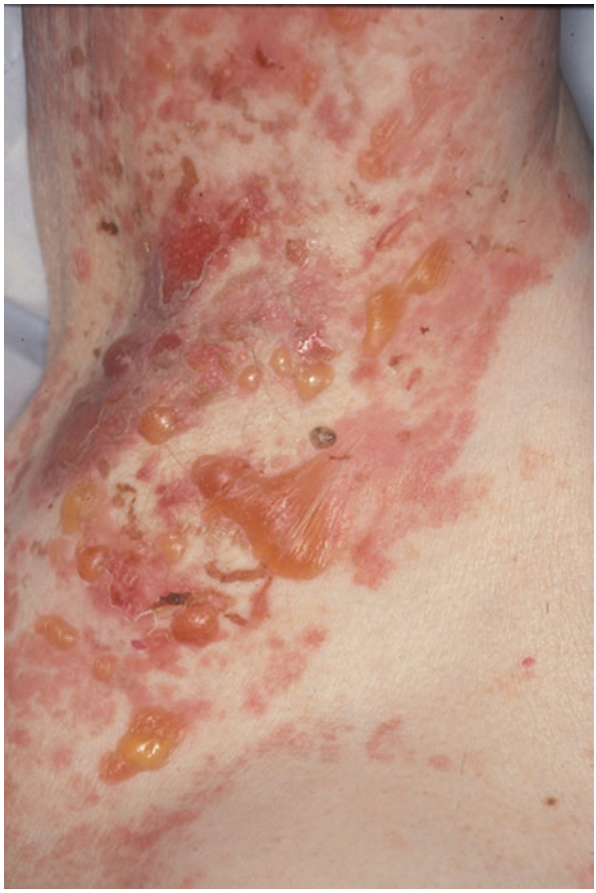


Fig. 12 Tense blisters, erosions, and urticaria-like plaques of the axilla in BP. Nikolsky sign will be negative. (Courtesy of Charles E. Crutchfield III, MD's Dermatology Image Library Archive)

15 Psoriasis

Psoriasis is an inflammatory disorder of the skin with hyperproliferative skin lesions and sometimes nail and joint involvement. There is a genetic component; the strongest HLA association is HLA-Cw6. There are increased Th1 cytokines, IL-23 from dendritic cells, IL-17, IL-22, and antimicrobial peptides (which leads to decreased risk of superinfections). The skin shows symmetric pink-red-hyperpigmented papules and plaques with silvery scale, MC on extensor surfaces (Fig. 14a–c). There may be nail pitting, oil spots, or thickening and overlap with scalp seborrheic dermatitis. Inverse psoriasis lesions of folds/genitals may lack scale. *Erythrodermic* psoriasis– generalized redness over the entire body—is a medical emergency. Psoriatic arthritis is present up to 1/3 of patients, usually rheumatoid factor negative, and associated with HLA-B27. *Triggers* include post-Strep pharyngitis or other infections, stress, alcohol, smoking, obesity, drugs (e.g., lithium, beta-blockers, systemic cortricosteroid rebound; TNF-alpha inhibitors can cause pustular

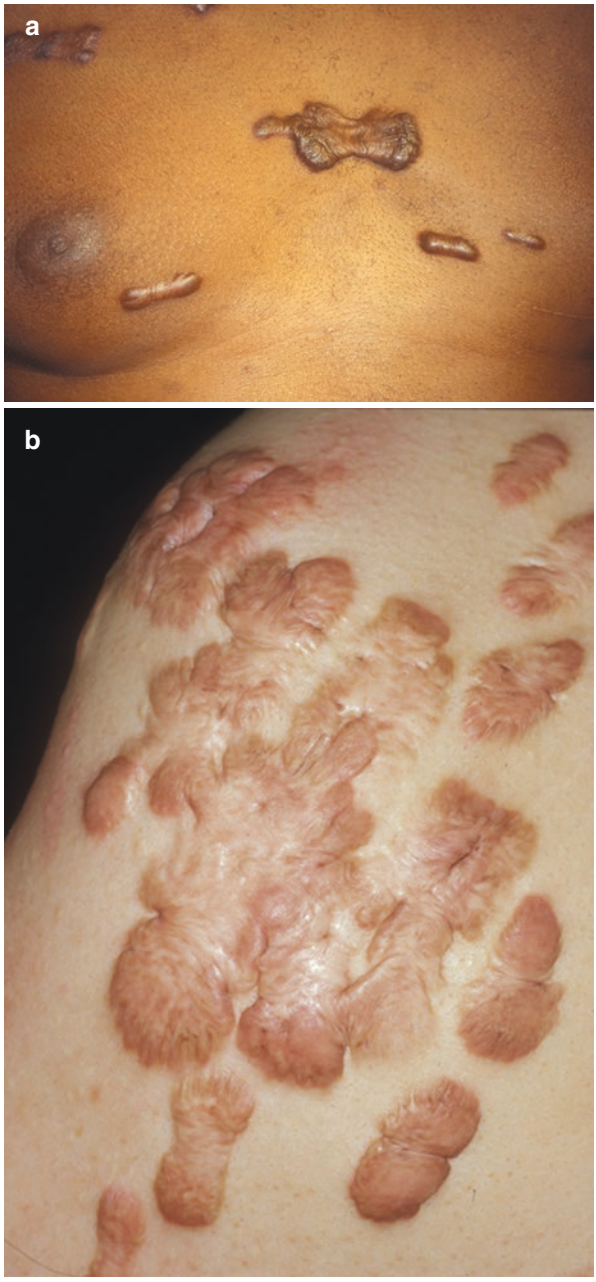


Fig. 13 (**a**, **b**) Keloids are hypertrophic plaques extending beyond the borders of original injury. (Courtesy of Charles E. Crutchfield III, MD's Dermatology Image Library Archive)

psoriasis). Diagnosis can often be made clinically. Punch biopsy of skin shows acanthosis, hyperkeratosis, parakeratosis, and decreased to absent granular layer. Therapeutic regimens often include topical CS +/− vitamin D analogues (calcipotriene), topical retinoids (tazarotene), and narrow band UVB +/− systemic

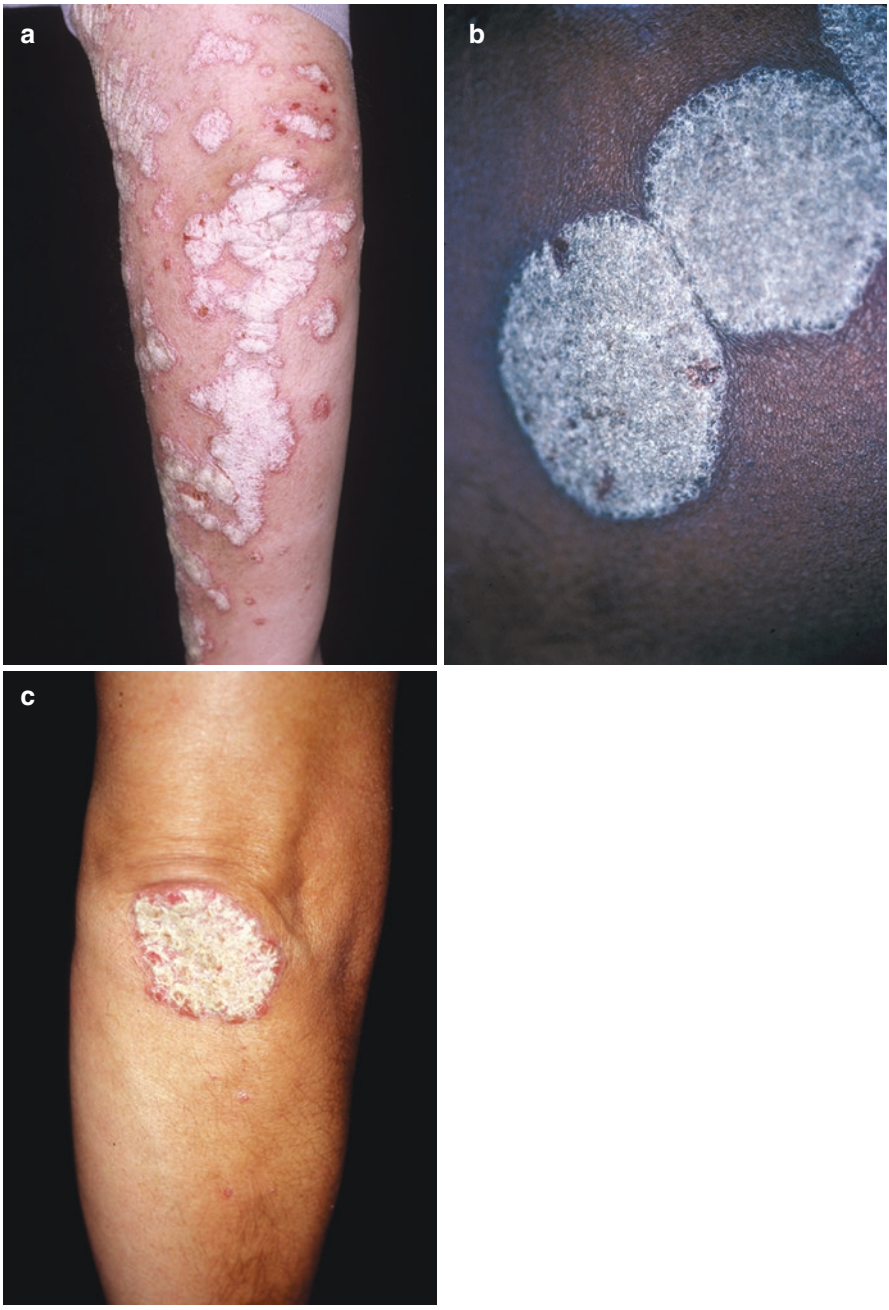


Fig. 14 (**a–c**) Psoriasis causes pink/hyperpigmented plaques with thick silvery scale most common on the extensor surfaces. (Courtesy of Charles E. Crutchfield III, MD's Dermatology Image Library Archive)

immunosuppressants. Patients should be screened and treated for comorbid cardiovascular disease. Psoriatic arthritis requires systemic treatment (methotrexate, TNF-alpha inhibitors (infliximab, adalimumab, certolizumab pegol, golimumab, etanercept), cyclosporine, acitretin, apremilast (PDE-4 inh), tofacitinib (Jak 1/3 inh), biologics (IL-12 and IL-23 inh ustekinumab, IL-23 inh guselkumab, IL-17 inh ixekizumab, secukinumab, brodalumab) [1, 2].

16 Hypersensitivity Reactions

Type I *allergic* hypersensitivity reactions are antibody mediated and immediate. Examples include asthma, allergic rhinitis, and anaphylactic reactions to bee stings, food, or drugs. Initial antigen exposure causes TH2 cells to stimulate B-cell production of IgE. Upon second exposure, the antigen cross-links IgE on mast cells, causing their release of vasoactive mediators/histamine and resulting vasodilation, inflammation, and bronchoconstriction. Anaphylaxis is a sudden, severe, systemic hypersensitivity reaction (urticaria, angioedema, hypotension, bradycardia) common after exposure to a known allergen (peanuts, fish, tree nuts), drug (*penicillins*>cephalosporins, NSAIDS, anesthetics/opioids/relaxants), insect venom, less commonly latex, IV contrast, or blood products; about 1/3 of cases are idiopathic. Treatment is SC/IM epinephrine for anaphylaxis or several pharyngeal angioedema [1, 2].

Type II hypersensitivity reactions are *cytotoxic* and antibody mediated. Antibodies to tissue-specific cell receptors activate opsonization, complement, and NK cells, resulting in the death of self-cells. Examples include pemphigus vulgaris: the antibody targets desmosomes, which provide epidermal cell-cell adherence. Patients present with widespread fragile blisters. In immune thrombocytopenic purpura, the antibody targets platelet membranes. In autoimmune hemolytic anemia, the antibody targets red blood cell membrane proteins. Others include Goodpasture syndrome, myasthenia gravis, and Grave's disease [1, 2].

Type III hypersensitivity reactions are *immune complex/*antibody mediated. IgM or IgG antibodies bind to antigen and create immune complexes that deposit in tissues, activating the complement cascade. Examples include immune complex formation in SLE or post-streptococcal glomerulonephritis [1, 2].

Type IV reactions are *cell-mediated*, thus delayed. Antigen sensitization occurs; subsequent exposure causes T-cell-mediated release of cytokines and chemotactic factors leading to inflammation in the target tissue. Examples include diabetes, the skin test for TB, and allergic contact dermatitis (poison ivy, neomycin, nickel, fragrance mix) [1, 2].

17 Contact Dermatitis

Contact dermatitis can be allergic or irritant. Irritant contact dermatitis is MC; it often affects hands>face and varies from blisters to dryness, scale, skin thickening, or acneiform in appearance. No prior sensitization is necessary; it is due to direct skin damage common in manufacturing/agriculture/automotive/wet work occupations. Allergic contact dermatitis (ACD) *acutely* appears as sharply demarcated edematous, erythematous, papules/vesicles/oozing in patterns depending on the exposure (Fig. 15a, b) (linear, rhus; florist's fingertips, tulips; eyelids, cosmetics; hands, gloves; earrings/belt buckle, nickel). *Chronically* more scaling, skin thickening, and post-inflammatory pigment appears. ACD is a delayed Type IV hypersensitivity reaction, requiring prior sensitization. The MC allergens are nickel and poison ivy [1, 2].

Treatment for both includes irritant/allergen avoidance and topical CS. Patch testing can be helpful to work up ACD. Severe poison-ivy dermatitis usually requires at least 2–3 weeks of oral CS with minimal tapering to avoid rebound [1, 2].

18 Molluscum Contagiosum

The *pox virus* of molluscum causes *umbilicated* pink pearly papules (Fig. 16a–c) spread by skin to skin or fomite contact (common in children, +/− sexually transmitted in adolescents or adults). In atopic dermatitis or immunosuppressed, lesions can become widespread or giant. Diagnosis is often clinical; skin biopsy shows molluscum bodies. Management begins with reassurance, since molluscum tends to be self-limited over weeks to years without treatment. Destructive options include curettage, cryotherapy, cantharidin, and sometimes topical retinoids, oral cimetidine, intralesional candida antigen immunotherapy, or topical imiquimod [1, 2].

19 Seborrheic Dermatitis

Dandruff is a mild form of seborrheic dermatitis. Classically, there are patches of greasy scale +/− a pink to erythematous base in seborrheic areas (Fig. 17a, b) (brows, eyelids, melolabial fold, scalp, postauricular, sternal, body folds), +/− pruritus. Other presentations include infants with cradle cap or more macerated erythematous plaques in creases. There is an increased sensitivity to and/or increased *Malassezia furfur and* increased or altered sebum composition. The course is chronic, may flare with stress, or be of increased severity in the setting of HIV or neurologic disorders (TBI, stroke, Parkinson's). Treatment includes ketoconazole shampoo/cream, +/− topical CS; other options include selenium sulfide, salicylic acid, tar or zinc shampoos, ciclopirox, or calcineurin inhibitors for the face and baby/mineral oil for infants [1, 2].

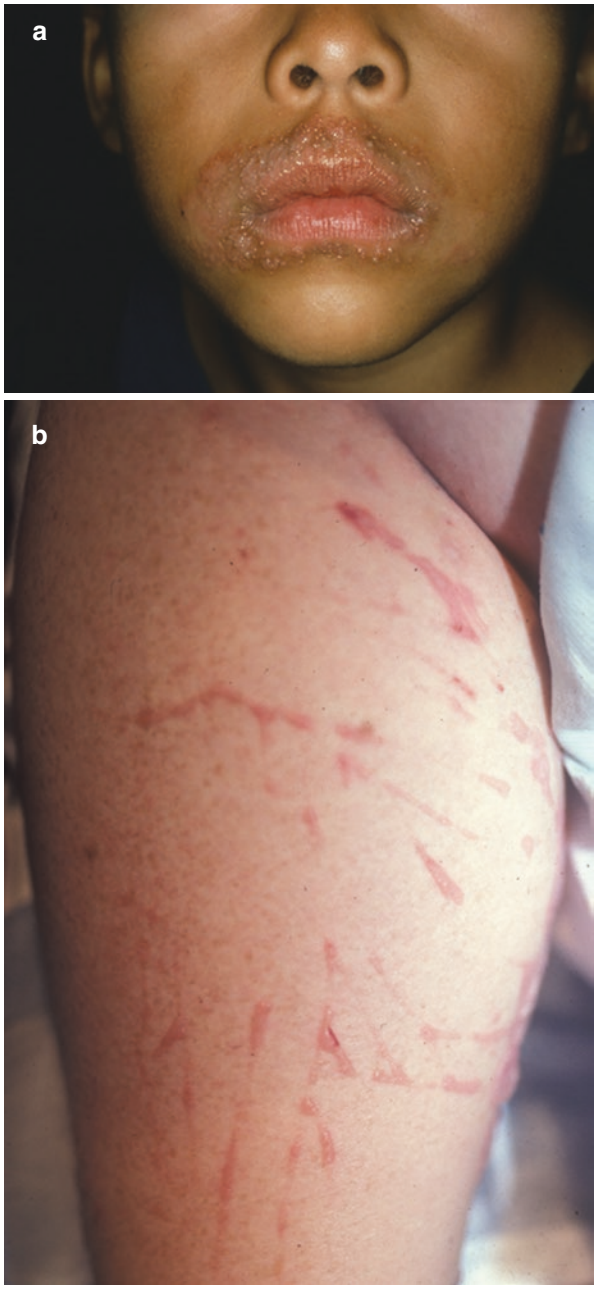


Fig. 15 (**a**) Allergic contact dermatitis causes sharply demarcated areas of edematous, erythematous papules/vesicles with oozing. (**b**) Linear patterns are seen in rhus dermatitis, in this case due to poison ivy. (Courtesy of Charles E. Crutchfield III, MD's Dermatology Image Library Archive)

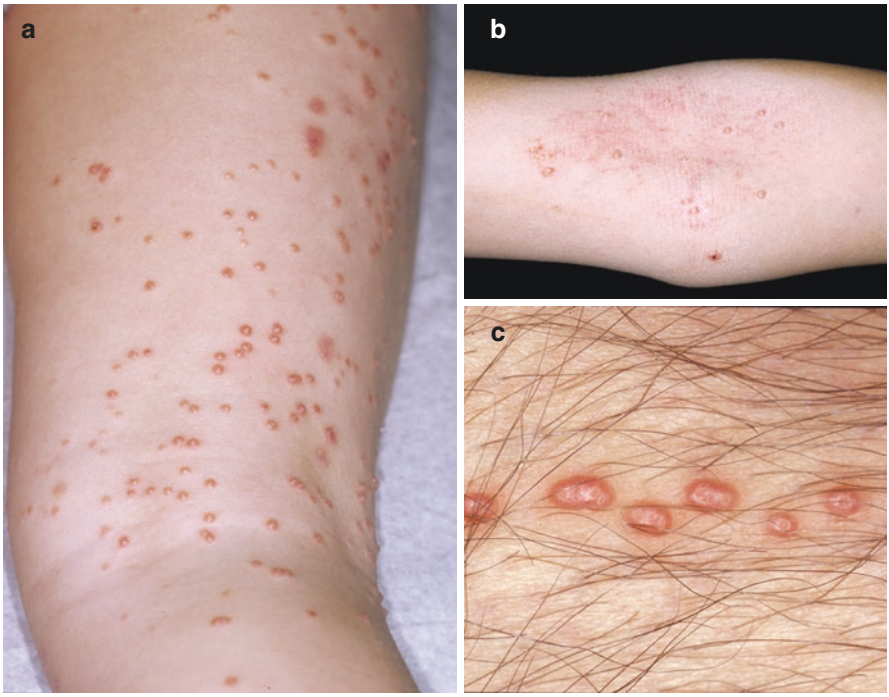


Fig. 16 (**a–c**) Molluscum presents with scattered umbilicated papules. (Courtesy of Charles E. Crutchfield III, MD's Dermatology Image Library Archive)

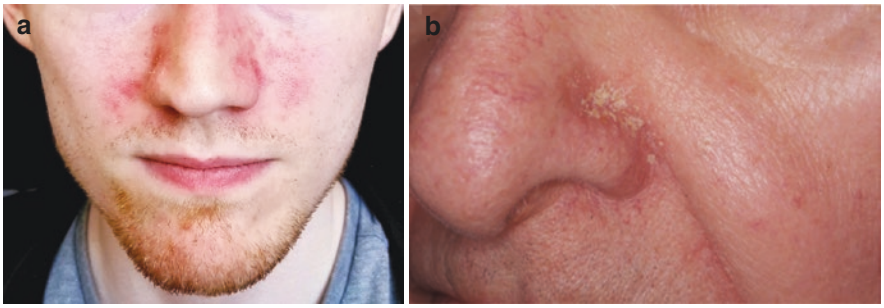


Fig. 17 (**a**, **b**) Greasy, scaly patches on a red base in a seborrheic distribution (nasofacial grooves, brow). (Courtesy of Charles E. Crutchfield III, MD's Dermatology Image Library Archive)

20 Vitiligo

Vitiligo results from multifactorial, genetic, and environmental etiologies, leading to acquired autoimmune destruction of melanocytes. Clinically, well-circumscribed

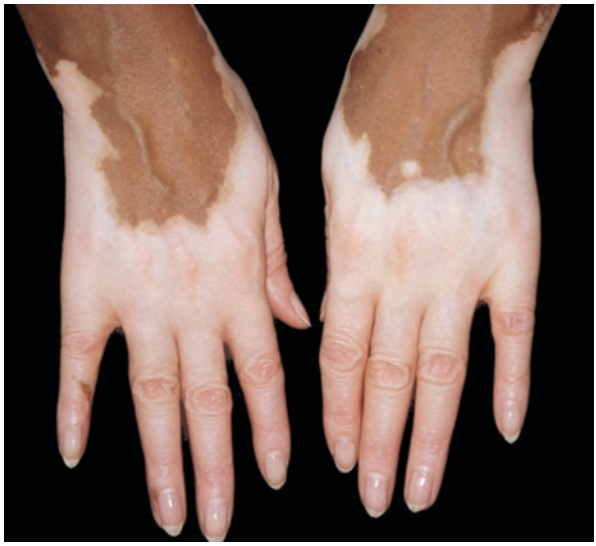


Fig. 18 Depigmented patches on the hands in vitiligo. (Courtesy of Charles E. Crutchfield III, MD's Dermatology Image Library Archive)

depigmented macules/patches of skin and mucous membranes around the eyes, mouth, fingers, wrists, axillae, groin, and genitals are noted (Fig. 18). Vitiligo is associated with other autoimmune diseases, MC thyroid > DM, Addison's pernicious anemia, alopecia areata, and uveitis. The diagnosis is often clinical; there is increased contrast to normal skin with Wood's lamp examination and an absence of melanocytes upon skin biopsy. Treatment includes sun protection, topical CS/calcineurin inhibitors, narrow-band UVB, excimer laser, calcipotriene, systemic immunosuppressants, surgical grafting, depigmentation, and off-label use of topical JAK kinase inhibitor tofacitinib [1, 2].

21 Impetigo

Impetigo is a superficial bacterial skin infection (stratum corneum) and the MC childhood bacterial infection. It appears as "honey-colored" crusts on injured/eczematous skin (Fig. 19). Non-bullous types are *S. aureus* > *Strep*, commonly on the face, and can self-resolve by 2 weeks. If *Streptococcus pyogenes*, 5% develop post-streptococcal glomerulonephritis (but no risk of rheumatic fever). In the bullous type, *S. aureus*, phage group II types 55 and 71 produce exfoliatoxins A and B, leading to more generalized flaccid bullae and erosions. There is increased risk in *S. aureus* carriers (>1/3 of population), and MC carriage in anterior nares > perineum, axilla, toe webs. Treat localized disease with topical mupirocin, retapamulin, and fusidic acid. For widespread disease, use oral antibiotics (first generation cephalosporin, B-lactamase resistant penicillin, clindamycin). If severe, use IV

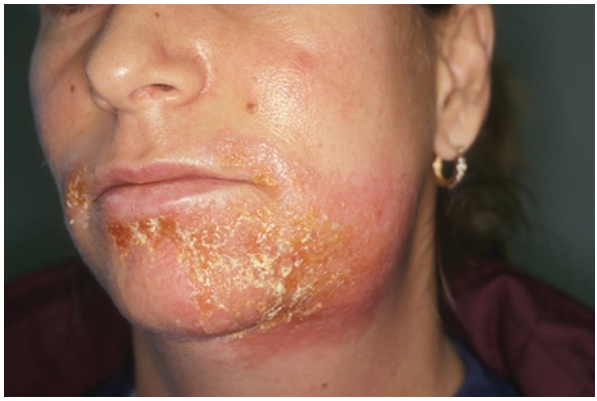


Fig. 19 Honey-colored crusting on eczematous skin in impetigo. (Courtesy of Charles E. Crutchfield III, MD's Dermatology Image Library Archive)

ceftriaxone. If *recurrent*, consider decolonization with mupirocin ointment to nares BID for 7–10 days +/− chlorhexidine body wash [1, 2].

Bullous impetigo can disseminate into staphylococcal scalded skin syndrome with fever, skin tenderness, and dramatic radial wrinkling and superficial sloughing of the skin, beginning periorificial on the face, then in intertriginous areas. It carries low mortality, <5%, and begins healing in about 1 week without scarring. Etiology is *S. aureus* in a distant site (most commonly nasopharynx or conjunctiva in children); the exfoliatoxins spread hematogenously. Thus, bullae cultures will be negative; instead culture nasopharynx, nostrils, conjunctiva, blood, or any other source of infection. The diagnosis is clinical or with frozen section biopsy. Treatment includes copious emollients and PO dicloxacillin/cephalexin/clindamycin for mild cases, and IV nafcillin or methicillin if severe [1, 2].

Ecthyma is a deeper variant of impetigo that eventually extends into the dermis leading to "punched out" ulcers, hemorrhagic crust, and heals with scarring, sometimes due to scratched bug bites. Treat with PO antibiotics [1, 2].

22 Acne Vulgaris

Acne presents with open and closed comedones, pustules, and cysts/nodules (Fig. 20a–c). Rare associated syndromes include (1) *SAPHO* (synovitis, acne, pustulosis, hyperostosis, osteitis) and (2) *PAPA* (sterile pyogenic arthritis, pyoderma gangrenosum, and acne). Acne fulminans is acute onset severe cystic acne, ulceration, and scarring +/− systemic findings of fever, increased WBC, ESR, and sterile osteolytic bone lesions on radiograph. This may occur after isotretinoin; treatment is oral steroids then oral isotretinoin [1, 2].

Acne is a multifactorial pilosebaceous disease common in adolescence due to overproduction of sebum, inflammation, abnormal keratinization, and *Cutibacterium acnes*. Acne can also be associated with androgen excess/syndromes, stress, and

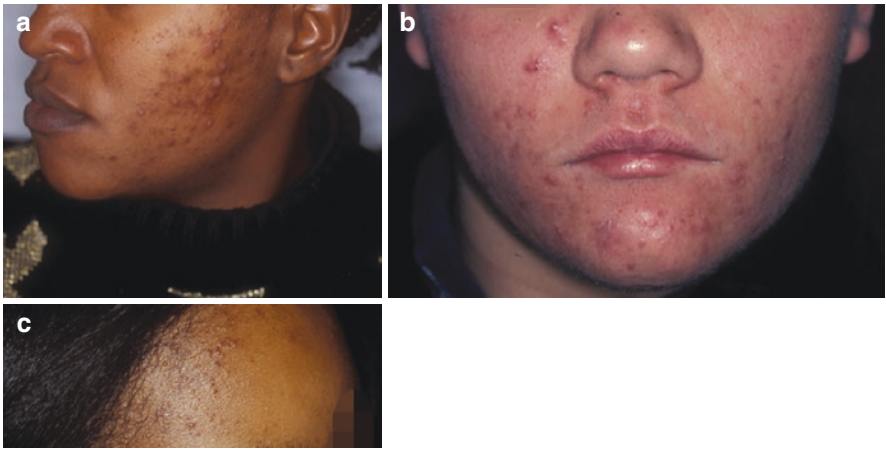


Fig. 20 (**a**, **b**) Acne papules, pustules. (**c**) Acne comedones. (Courtesy of Charles E. Crutchfield III, MD's Dermatology Image Library Archive)

possible dietary associations with high milk consumption or high glycemic load. Treatment includes topical retinoids, benzoyl peroxide, topical/oral antibiotics, oral isotretinoin, and/or hormonal agents (spironolactone, OCPs) [1, 2].

23 Folliculitis

Follicular papules and pustules are MC due to *S. aureus* (Fig. 21a). Gram-negative infections often follow prolonged courses of antibiotics for acne. Hot tub folliculitis (Fig. 21b) is due to pseudomonas infection after poor chlorination. Treatment includes chlorhexidine washes or topical antibiotics for superficial disease. If more widespread, use PO beta-lactamase-resistant penicillins or first generation cephalosporins. If pseudomonal, folliculitis usually self-resolves or treat with ciprofloxacin. Consider staphylococcal decolonization if recurrent [1, 2].

24 Pityriasis Rosea

Pityriasis rosea presents with an enlarging "herald patch" often on the trunk, followed by widespread oval macules/papules with fine, central, trailing scale along skin tension lines (Fig. 22, "Christmas tree" pattern) +/− pruritus. It is common in spring/fall and typically self-resolving by 8 weeks. Etiology is possibly viral related to HHV-6/7 or can be drug-induced (NSAIDS, ACE inhibitors, beta-blockers). Often reassurance is all that is needed due to its self-limited course; antipruritic lotion or topical CS can be given if symptomatic [1, 2].

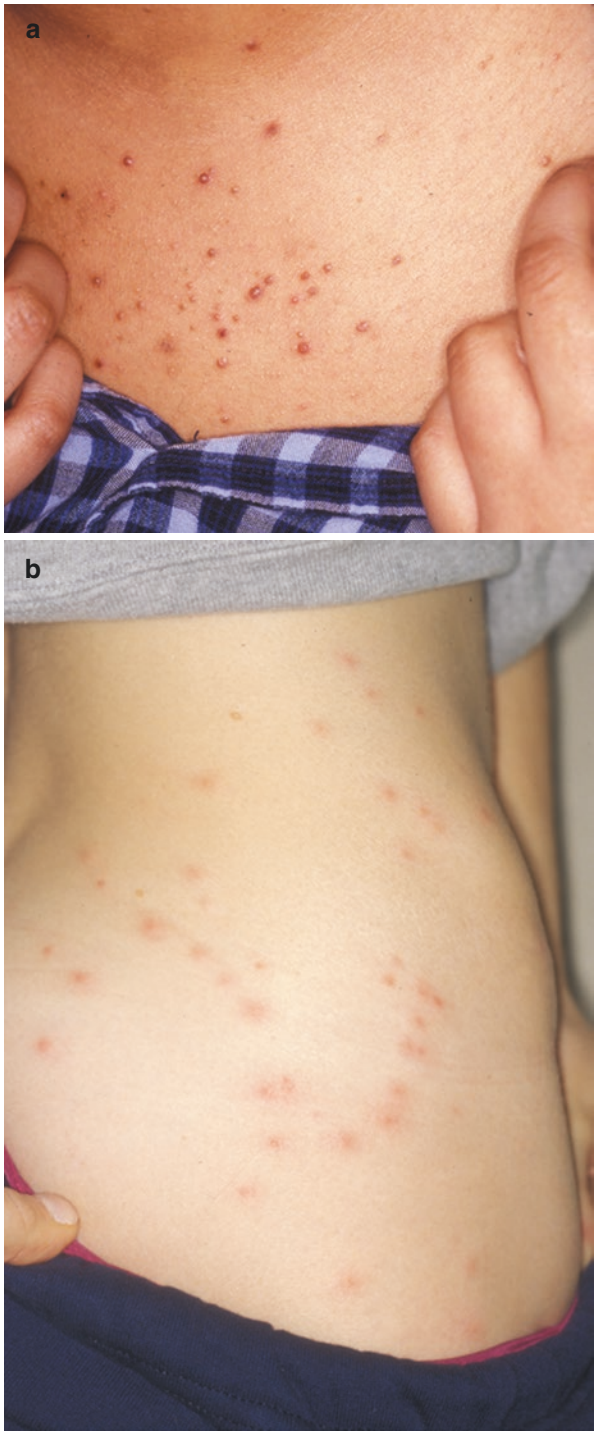


Fig. 21 (**a**) Follicular papules/pustules. (**b**) Follicular papules due to hot tub folliculitis. (Courtesy of Charles E. Crutchfield III, MD's Dermatology Image Library Archive)

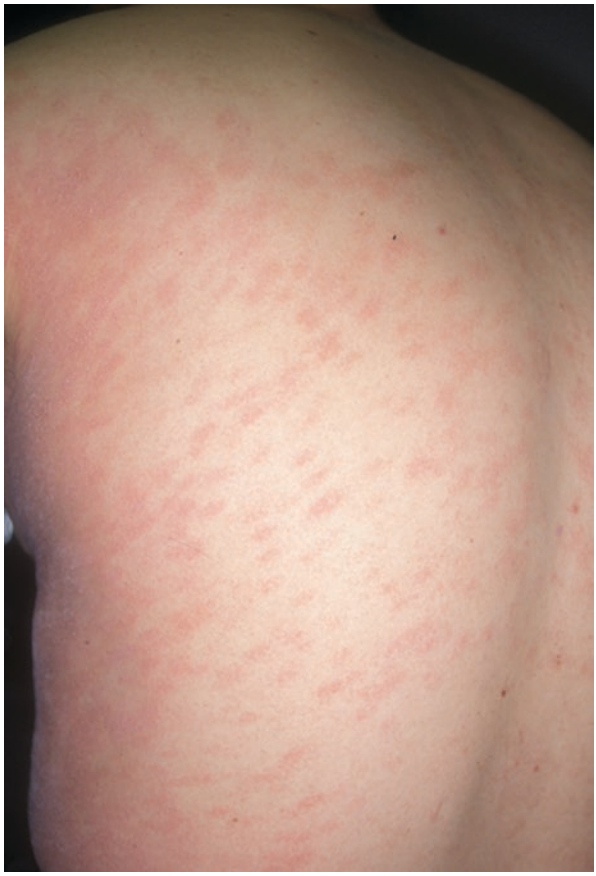


Fig. 22 Pink macules/patches with trailing scale follow skin tension lines in a "Christmas tree" pattern in pityriasis rosea. (Courtesy of Charles E. Crutchfield III, MD's Dermatology Image Library Archive)

25 Rosacea

Rosacea is typically central facial (Fig. 23a–c). There is an erythematotelangiectatic/vascular type with flushing that can become permanent. The papulopustular type is similar to acne *without comedones* (Fig. 23a). The phymatous type shows overgrowth of oil glands and thickening of skin often on the nose (Fig. 23d). In the ocular type, dryness, sensitivity, and inflammation requires oral treatment (minocycline, doxycycline). It is more common in females>males, between 30 and 50 years. Multiple factors influence the pathogenesis of this chronic inflammatory disorder including vascular reactivity, skin sensitivity, heat, sun damage, and *Demodex*. *Triggers* for flares can include hot/cold, spices, alcohol, sun, stress/emotion. Therapy in mild disease includes trigger avoidance and sun protection. In moderate disease, treatment includes oral doxycycline/minocycline, also ophthalmology referral for ocular involvement. Topicals are more effective in the papulopustular subtype (metronidazole, azelaic acid, sodium sulfacetamide-sulfur, ivermectin). Severe papulopustular or rosacea fulminans can require isotretinoin +/− oral CS. Vascular lesions

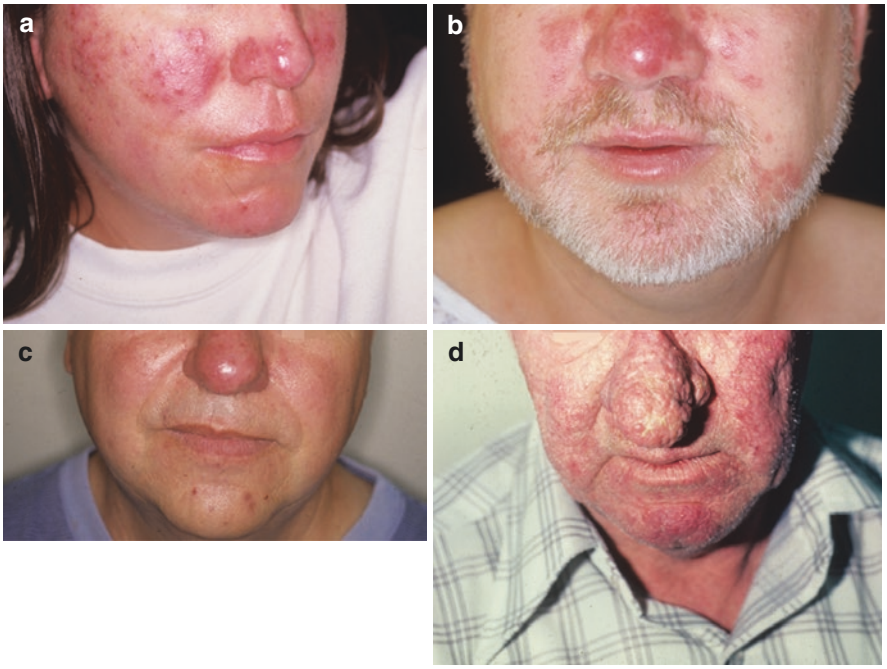


Fig. 23 (**a**, **b**) Central facial papules/pustules/erythema without comedones in classic rosacea. (**c**) Central facial flushing, few papules/pustules, and subtle rhinophyma of the nose. (**d**) Extreme facial edema, erythema, and rhinophyma. (Courtesy of Charles E. Crutchfield III, MD's Dermatology Image Library Archive)

can be treated with laser, intense pulsed light, or topical brimonidine. Rhinophyma typically requires surgery [1, 2].

26 Senile Purpura

Senile purpura are benign recurrent ecchymoses on the extensor upper extremities with minimal trauma, common after age 50, in photodamaged skin, with corticosteroid use or blood thinners (Fig. 24a, b). Treatment is sun protection and reassurance [1, 2].

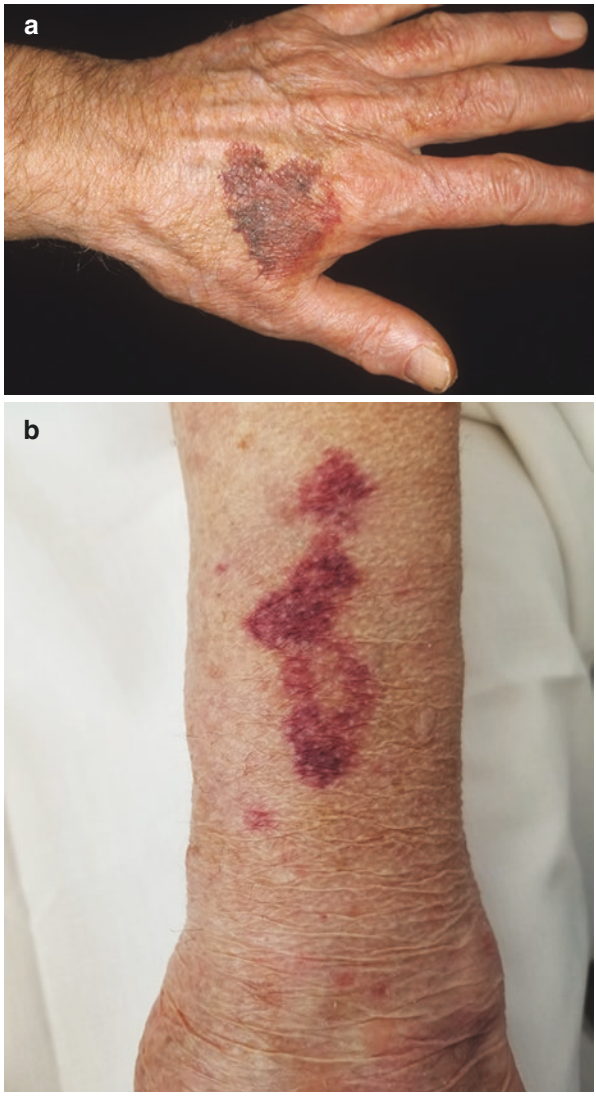


Fig. 24 (**a**, **b**) Ecchymoses on the dorsal extremities in thin, sun-damaged skin. (Courtesy of Charles E. Crutchfield III, MD's Dermatology Image Library Archive)

27 Stevens-Johnson Syndrome (SJS)/Toxic Epidermal Necrolysis (TEN)

There is epidermal detachment characterized by % BSA involved: if <10% = SJS, 10–30% = SJS-TEN overlap, >30% = TEN. SJS/TEN typically presents 7–21 days after exposure with the appearance of confluent dusky macules, targetoid lesions, tenderness, and erythema that progress to full-thickness necrosis/*blistering of skin and erosions of mucosa* (oral, ocular, respiratory, GI, genital) in association with fever, lymphadenopathy, hepatitis, and cytopenias (Fig. 25). Nikolsky and Asboe-Hansen signs will be positive. Mortality is ~5% in SJS and ~30% in TEN, worse with increasing age and involved BSA. The MC cause of death is secondary infection, and the MC sequelae are ocular. Diagnosis can be confirmed with a biopsy showing full-thickness epidermal necrosis. Serologic granulysin levels are elevated. Increased risk comes with certain HLA types, slow acetylator genotypes, the elderly, and *AIDS (1000× risk due to loss of regulatory T cells)*. Common culprit drugs include antibiotics (especially penicillins, sulfonamides), anticonvulsants, allopurinol, NSAIDs, and clobazam. They cause apoptosis of keratinocytes mediated by Fas-Fas ligand, death receptor ligand, perforin, granzyme B, and granulysin [1, 2].

Medication-related cases can be predicted by HLA types. HLA-B*1502 carries >200x risk in Asian, East Indian exposed to carbamazepine. HLA-B-*3101 increased risk among Europeans, exposed to carbamazepine. HLA-B*5801 increases risk among the Han Chinese with allopurinol. HLA-B*5701 are at increased risk with exposure to abacavir. *Immediately withdraw the offending medication/agent* and emergently initiate supportive care for compromised skin barrier +/−ICU. Minimize the medication list, emergently consult dermatology for diagnosis, and consult ophthalmology +/−urology or gynecology depending on areas involved. Treatment includes IVIG, possibly other immunosuppressives (cyclosporine). SCORTEN criteria predicts mortality (age, malignancy, heart rate, BUN, BSA, glucose; *bicarb < 20 mmol/L* is the most important factor). Preventative measures include HLA screening for all abacavir patients and for carbamazepine patients among East Asians [1, 2].

28 Pemphigus Vulgaris

Pemphigus vulgaris is a blistering disease characterized by skin and mucosal (ocular, oral) flaccid blisters/crusting and oral erosions (Fig. 26); Nikolsky and Asboe-Hansen signs are positive. IgG antibodies form to desmosomes (cadherins: transmembrane proteins functioning in cell-cell adherence) disrupting connections between mucocutaneous cells. In the mucosal type, desmoglein 3 is

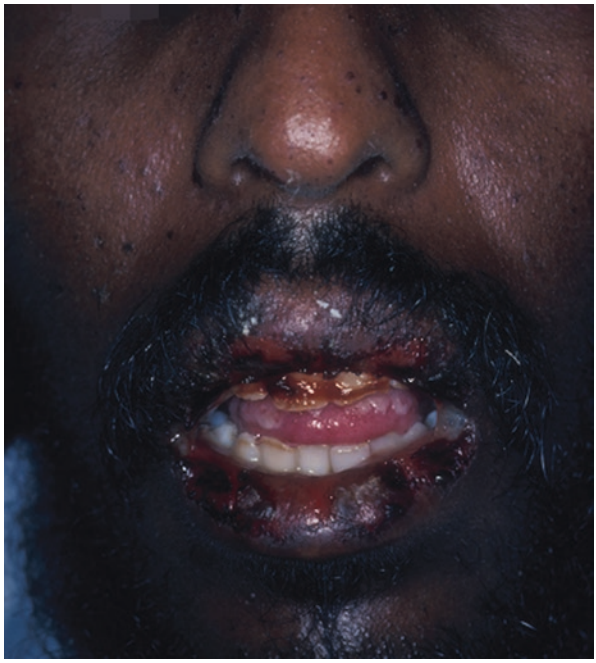


Fig. 25 Full thickness necrosis and hemorrhagic crusting of the lips/mucosa in SJS/TEN. (Courtesy of Charles E. Crutchfield III, MD's Dermatology Image Library Archive)

affected, and in the mucocutaneous type, desmogleins 1 and 3 are affected. It presents in M = F, ages 50–60 years, and can be associated with other autoimmune thyroid and neuromuscular disease. Diagnosis is confirmed by a skin biopsy with DIF showing "chicken-wire" staining of intracellular IgG in the epidermis. IIF or ELISA for IgG Dsg 1/3 antibodies correlates with disease activity. High potency topical steroids treat mild disease. If more severe, treat with oral steroids starting at 1 mg/kg/day + adjunctive rituximab, IVIG, azathioprine, plasmapheresis. Consult with dermatology, ophthalmology, and ENT depending on involved areas [1, 2].

29 Hidradenitis Suppurativa (HS) "Acne Inversa"

HS is characterized by recurrent painful nodules/sterile abscesses/*double comedones*/sinus tracts/scar formation in axillary, inguinal, inframammary, and/or anogenital areas beginning after puberty (Fig. 27a, b). There is risk of secondary infection and squamous cell carcinoma due to chronic scarring. HS is due to dysregulation/chronic destructive inflammation in an apocrine distribution. Therapies include decreasing moisture or friction, weight loss, smoking cessation, topical/oral

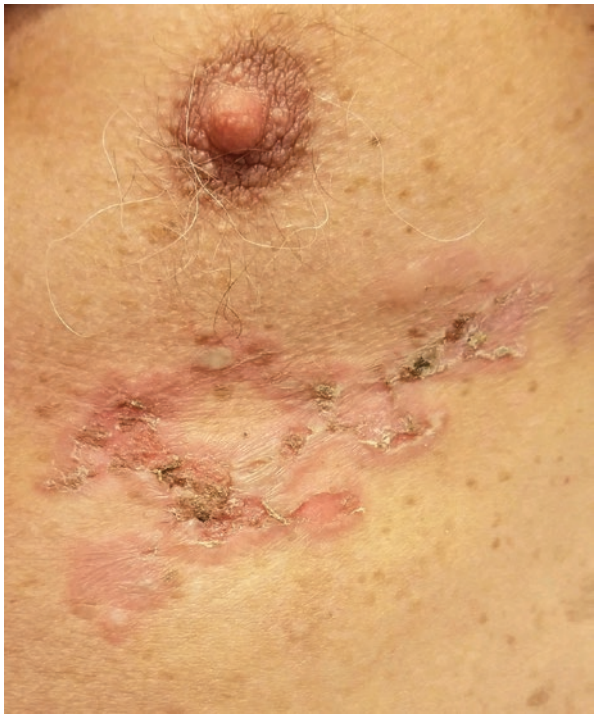


Fig. 26 Fragile, ruptured, and crusted blisters on the trunk in pemphigus vulgaris. (Courtesy of Charles E. Crutchfield III, MD's Dermatology Image Library Archive)

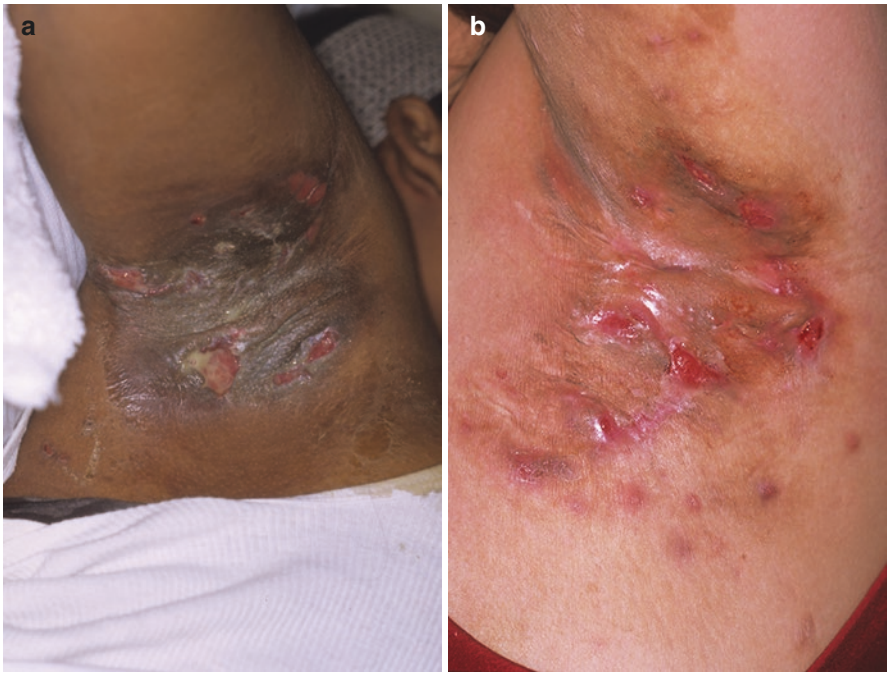


Fig. 27 (**a**, **b**) Nodules, sterile abscesses, scarring, and sinus tract formation in the axillae in HS. (Courtesy of Charles E. Crutchfield III, MD's Dermatology Image Library Archive)

antibiotics, intralesional/oral steroids, excision, lesion marsupialization, CO2 laser ablation, anti-androgens (spironolactone, finasteride, OCPs), isotretinoin, TNF-alpha inhibitors, and cyclosporine [1, 2].

30 Stasis Dermatitis

Stasis dermatitis causes erythema, scale, possible oozing and crusting, itching, and tenderness of the lower extremities (Fig. 28a). It often begins near the medial ankle and is often *bilateral* in a patient with a history of chronic lower extremity edema. The patient may present with a long history of treatment for what was called "chronic cellulitis" (when bilateral and no systemic signs of infection = stasis dermatitis). End-stage disease can lead to elephantiasis with induration and cobblestoning (Fig. 28b). The etiology is venous insufficiency, capillary leak, inflammation, edema, hemosiderin deposition, fibrosis, and ulceration. There is increased risk of superinfection and sensitivity to topicals/irritants. Therapy includes compression stockings and leg elevation, along with topical CS/emollients for dermatitis [1, 2].

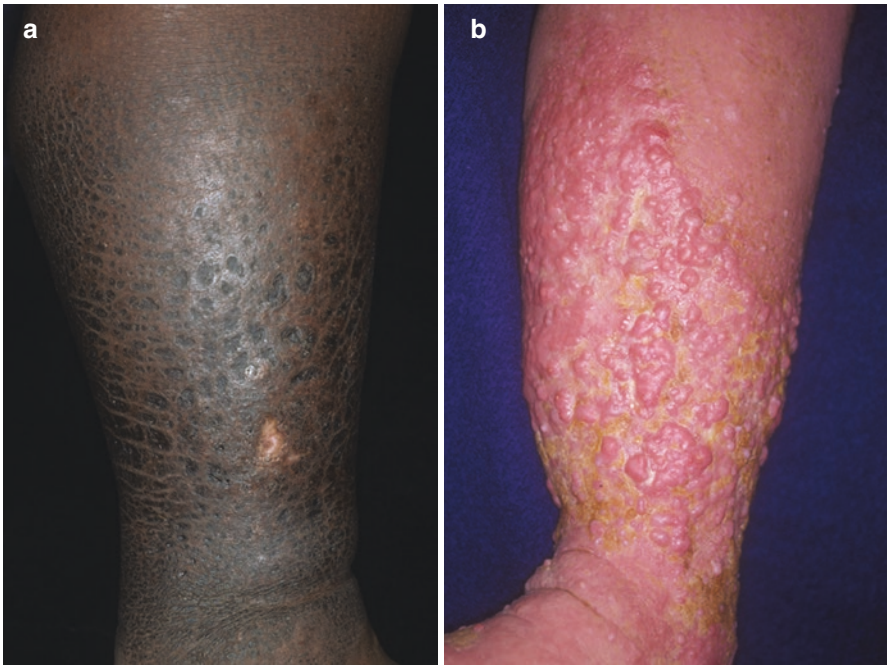


Fig. 28 (**a**) Xerotic, crusted hyperpigmented plaques of an edematous lower extremity due to stasis dermatitis. (**b**) Elephantiasis with grossly edematous lower extremities and thick, verrucous cobblestoned plaques. (Courtesy of Charles E. Crutchfield III, MD's Dermatology Image Library Archive)

31 Cellulitis

Cellulitis leads to red, tender, warm, plaques with fever, chills, lymphangitis, and sometimes bullae or necrosis. It is MC on the head and neck in children, lower extremities in adults, and arms in the setting of IVDA. Cellulitis is a deeper infection of the skin, most commonly due to group A B-hemolytic strep>*S. aureus* (*S. aureus* MC in children). Suspect MRSA if necrosis or abscess. It can also be due to H. flu. Polymicrobial infections are associated with diabetes or decubitus ulcers. The risk increases with previous cellulitis or lymph node dissection due to damaged lymphatics. Blood cultures are negative in healthy adults. Treat with PO dicloxacillin, cephalexin, or clindamycin. If severe, treat with IV antibiotics. If diabetic or decubitus ulcers, treat with piperacillin/tazobactam or ciprofloxacin plus metronidazole. If MRSA, treat with TMP/ZMX, tetracyclines, and clindamycin [1, 2].

32 Erysipelas

Erysipelas ("St. Anthony's fire") presents with an extraordinarily red, warm, burning or painful, sharply demarcated lesion, lymphatic involvement/streaking, and often lymphadenopathy (Fig. 29). It affects the lower extremity > face. Risk factors include lymphedema, extremes of age, venous stasis, portal of entry, ETOH, diabetes, and immunocompromise. It is a superficial version of cellulitis (upper mid dermis) caused by group A beta-hemolytic strep, and often associated with pain, fever, and left shift in leukocyte count. There are elevated ASO and DNase B titers. Cultures are not recommended in the immunocompetent. Treatment is penicillin (if allergic, erythromycin). If severe, extremes of age, or immunocompromised, hospitalization and IV antibiotics may be necessary [1, 2].

33 Hyperpigmentation Disorders

Melasma is typically symmetric facial irregular brown/gray patches and more common in darker skin types (Fig. 30). There is increased melanin (hyperfunctioning melanocytes) in response to UV/visible light, estrogenic states/medications. Therapy is aimed at strict sun/visible light protection, topical retinoids, steroids, hydroquinone, and laser resurfacing [1, 2].

Generalized hyperpigmentation can be related to endocrine disorders leading to increased ACTH/melanocyte stimulating hormone (Addison's, acromegaly, Cushing, hyperthyroidism) [1, 2].

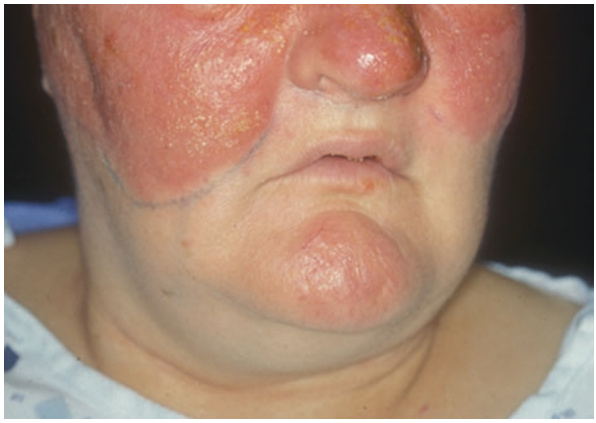


Fig. 29 Erysipelas, a superficial form of cellulitis, forms sharply demarcated bright red plaques; the skin may dimple and even blister. (Courtesy of Charles E. Crutchfield III, MD's Dermatology Image Library Archive)

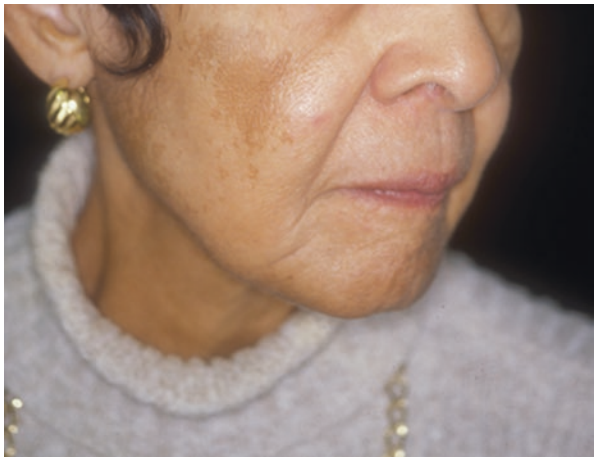


Fig. 30 Irregular brown/gray hyperpigmented macules and patches form across the cheeks in melasma. (Courtesy of Charles E. Crutchfield III, MD's Dermatology Image Library Archive)

Localized hyperpigmentation is more often due to certain drugs. Amiodarone causes slate gray discoloration of sun-exposed areas. Minocycline pigment causes multiple presentations: Type I is blue discoloration of scars, Type II is pretibial bruise-like blue-grey patches, and Type III is muddy-brown discoloration in sun-exposed areas. Hydroxychloroquine/chloroquine can cause pretibial gray to blue-black discoloration [1, 2].

34 Skin Neoplasms

34.1 Melanoma

Melanoma should be suspected for any asymmetric, irregular bordered, multiply colored or black, new or changing lesions (Fig. 31a, b). The superficial spreading subtype is MC; others include nodular, lentigo maligna (in chronically sun damaged skin), acral lentiginous melanoma (less common, mostly in darker skin types), mucosal melanoma, desmoplastic melanoma, ocular melanoma, and melanoma in situ. Melanoma risk factors include the inheritable CDKN2A mutation, higher UV exposure or sunburns, red hair/freckling, family history of melanoma, lighter skin type, and large number of nevi [1, 2].

In Stage IA melanoma, there is >95% 10-year survival; in Stage IIIC, there is <50% 10-year survival. There is a poorer prognosis in the elderly, males, with trunk, head, or neck locations and lymph node or visceral metastasis. For the most accurate diagnosis, refer to a dermatologist; total excisional biopsy is optimal for accurate staging. Pathologic staging includes measurement of primary tumor thickness (Breslow method), which is the single most important prognostic factor. Ulceration, Breslow depth over 1 mm, increased mitoses, and positive lymph nodes portend a worse prognosis. Treatment is with wide local excision or Mohs micrographic surgery (for head and neck) +/− sentinel lymph node biopsy. Melanoma in situ are removed with at least 0.5 cm excision margins. In malignant melanoma the lateral excision margins depend on Breslow depth, and the excision is taken down to fascia.

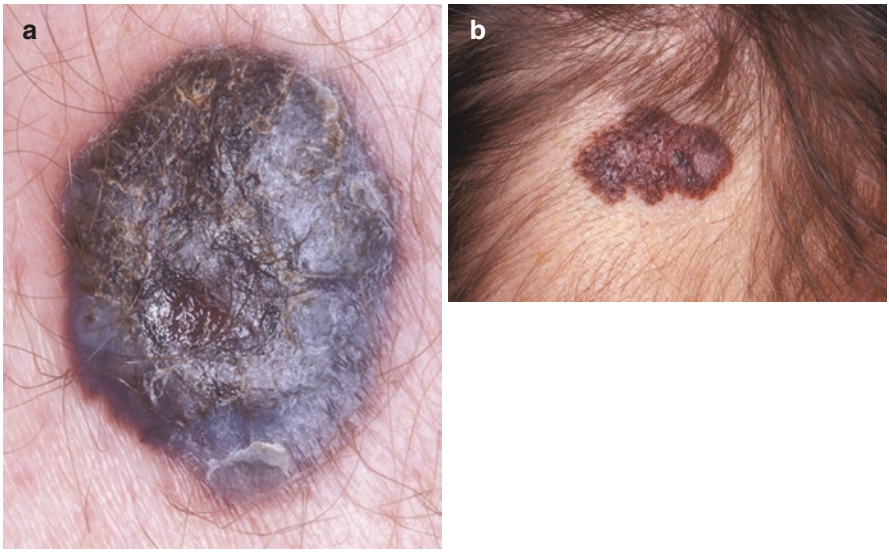


Fig. 31 (**a**) Asymmetric, brown/black/blue/gray eroded plaque of melanoma. (**b**) Irregularly bordered, asymmetrical, multiple colored brown, blue/gray, white plaque of melanoma. (Courtesy of Charles E. Crutchfield III, MD's Dermatology Image Library Archive)

In advanced disease, refer for multidisciplinary management of adjuvant therapy with anti-PD1/PD-L1 agents (pembrolizumab, nivolumab), BRAF inhibitors, MEK inhibitors, CTLA-4 therapies, and/or interferon [1, 2, 4, 5].

Melanoma patients are recommended to have at least annual skin exams for life and as often as q 3–6 months for the first 2–5 years after diagnosis. Imaging is only necessary if dictated by clinical symptoms for Stage I–IIA. In Stage IIB–IV, consider cross-sectional/brain imaging q 3–12 months for 2 years then q 6–12 months for 3 years [1, 2, 4].

34.2 Basal Cell Carcinoma (BCC)

BCCs are often pearly papules or plaques, with telangiectasia, rolled edges, or appearing eczematous (Fig. 32a, b). BCCs results from UV exposure or inherited mutations in the patched 1 gene/hedgehog signaling pathway. BCCs become common in the 6th–7th decades, are indolently growing, and have locally destructive potential. Treatment modalities include destruction, topical chemotherapy (5-FU), immune response modifiers (imiquimod), excision, or Mohs for more aggressive/higher risk lesions. Vismodegib (hedgehog pathway inhibitor) is an option for extensive, inoperable,, or metastatic disease [1, 2].

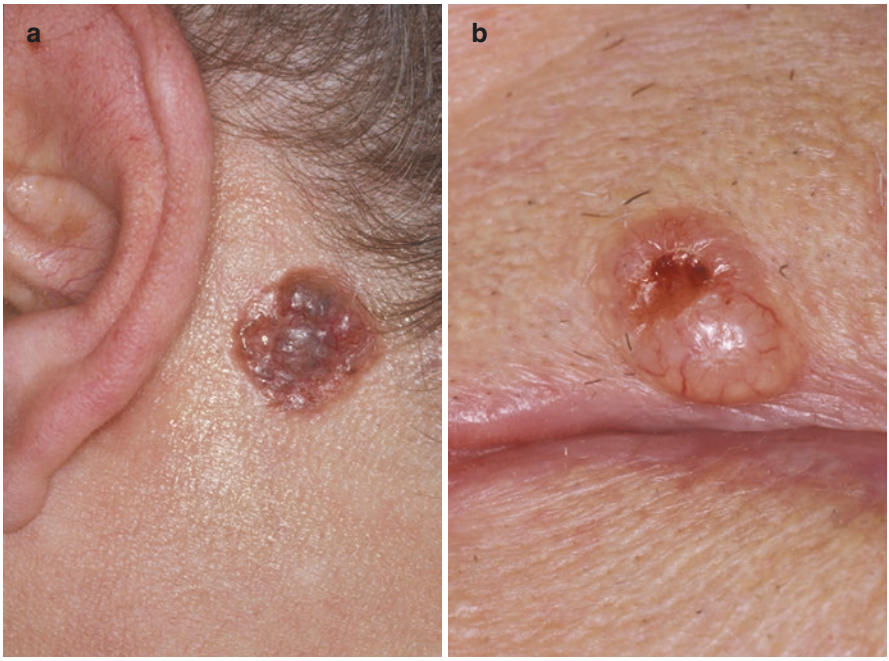


Fig. 32 (**a**) Pearly, pink/pigmented plaque with rolled borders and telangiectasia in BCC. (**b**) Pearly, centrally eroded, plaque with telangiectasia and rolled borders in BCC of the lip. (Courtesy of Charles E. Crutchfield III, MD's Dermatology Image Library Archive)

34.3 Squamous Cell Carcinoma (SCC)

SCCs appear as scaly, erythematous nodules or plaques most commonly on the head, neck, and dorsal hands in M > F (Fig. 33a–c). The keratoacanthoma type often shows rapid growth of pink, domed, centrally hyperkeratotic nodules (Fig. 33c). SCCs arise due to UV exposure, after radiation, immune suppression, in chronic inflammation/scars/ulcers, from precursor actinic keratoses, HPV, industrial carcinogens, or arsenic exposure. There is an increased risk of metastasis with lip or ear location, immunosuppression, diameter over 2 cm, Breslow depth over 2 mm, origin in a scar, and with poorly differentiated tumors. Treatment is excision or Mohs micrographic surgery [1, 2].

34.4 Actinic Keratosis (AK)

AKs are precancerous gritty/scaly red papules (Fig. 34) appearing in a sun-exposed distribution due to chronic UV exposure. They are more common in males, the elderly, and lighter skin types. Due to the risk of transformation to invasive SCC, AKs are treated with destructive methods including cryotherapy, photodynamic therapy, imiquimod, 5-FU, TCA peels, ingenol mebutate, and topical diclofenac [1, 2].

34.5 Seborrheic Keratosis (SK)

SKs are waxy, stuck-on, verrucous appearing papules or plaques in hair-bearing areas (Fig. 35a, b). They are common harmless skin neoplasms, appearing in the fourth decade of life, and may have familial inheritance. The rare sign of Leser-Trelat is eruptive SKs heralding an underlying adenocarcinoma, most commonly GI [1, 2].

34.6 Lentigines

A solar lentigo is a pigmented macule in sun-exposed areas (Fig. 36), increasingly common after the sixth decade of life, more so in lighter skin types [1, 2].

Lentigo simplex describes evenly pigmented brown macules at any age, in any area, sometimes in association with genetic conditions (LEOPARD syndrome, Peutz-Jeghers with oral lesions) [1, 2].

35 Varicella Zoster Virus (Human Herpes Virus-3)

The varicella (chicken pox) virus prodrome is followed by an eruption of “dew drops on a rose petal” vesicles on a red base, beginning on the head and progressing down. Lesions are present in various stages (pustules, crusts). Reactivation is called shingles/herpes zoster (Fig. 37). The eruption is dermatomal, occurs in 1/5 of adults,

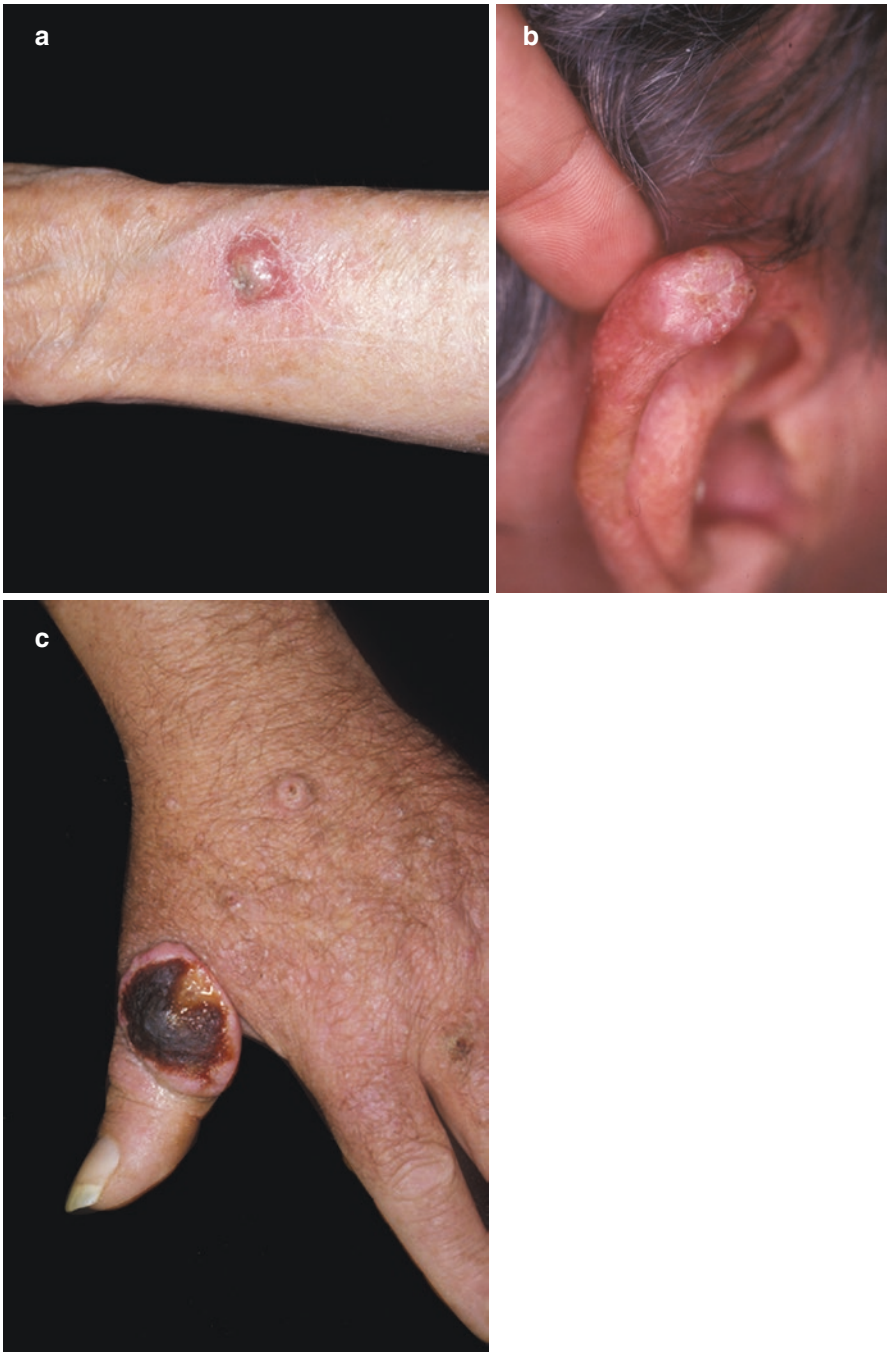


Fig. 33 (**a**) Pink keratotic plaque of SCC on the forearm. (**b**) Pink keratotic plaque/nodule of the helical rim. (**c**) Rapidly growing, crateriform, centrally hyperkeratotic pink tumor of the thumb in keratoacanthoma-type SCC. (Courtesy of Charles E. Crutchfield III, MD's Dermatology Image Library Archive)

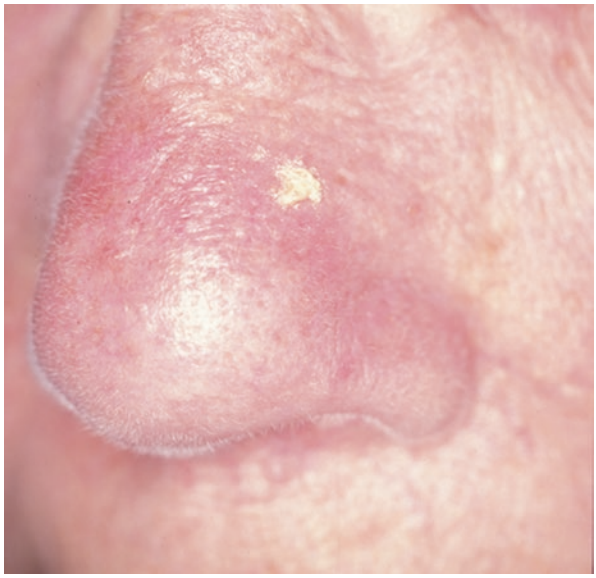


Fig. 34 A gritty, scaly papule, often with an erythematous base on sun damaged skin in this actinic keratosis of the nose. (Courtesy of Charles E. Crutchfield III, MD's Dermatology Image Library Archive)

½ of immunosuppressed, and carries risk of post-herpetic neuralgia. Varicella zoster virus is a double-stranded DNA virus that establishes latent infection in the dorsal root ganglion. It is transmitted by respiratory droplets/lesional fluid; patients are contagious a few days prior to lesion development and until all lesions have crusted. Zoster reactivation risk goes up with immunosuppression, the elderly, stress, fever, and injury. Dissemination can be deadly due to SIADH. Treatment includes systemic acyclovir/valacyclovir, by IV if immunocompromised [1, 2].

36 Human Papillomavirus (HPV)

HPV causes verrucous, hyperkeratotic papules (Fig. 38a–d), cervical cancer, and SCC. It is a double-stranded DNA virus spread by skin-to-skin or fomite contact. Early proteins E1-E7 are involved in DNA replication and late proteins L1-L2 are involved in forming virions. Oncoproteins E6 (involved in p53 destruction) and E7

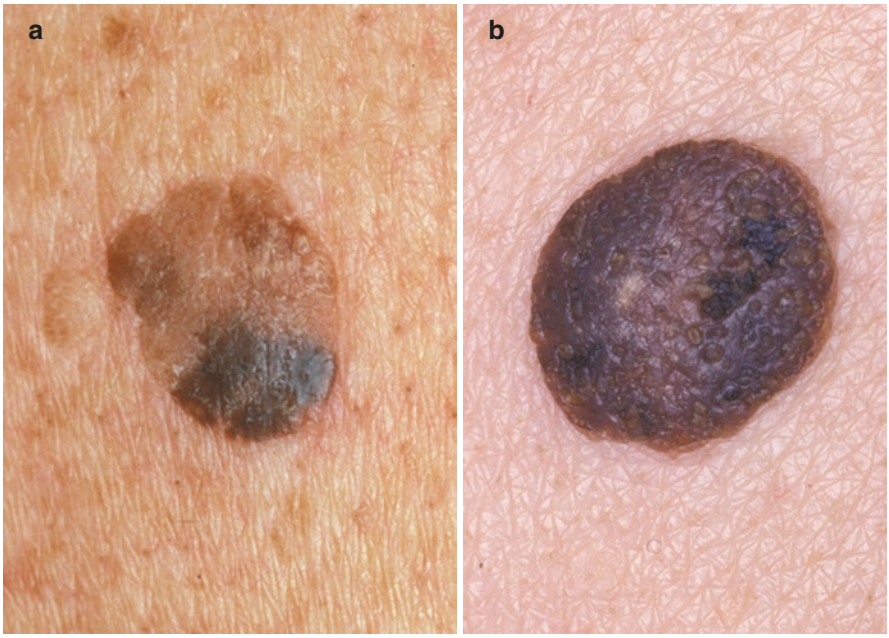


Fig. 35 (**a**, **b**) Waxy or wart-like, stuck-on, variably colored plaques with keratin plugs in seborrheic keratoses. (Courtesy of Charles E. Crutchfield III, MD's Dermatology Image Library Archive)

(involved in loss of inhibition of transcription) decrease host immune responses. HPV- 3, 10 cause flat warts. Genital warts (Fig. 38d, condyloma acuminata) are the most common STD (HPV- 6, 11). Cervical cancer results from high risk types HPV 16, 18, 31, 33, 45. Recurrent respiratory papillomatosis is also associated with HPV 6, 11; this is the MC benign tumor of the larynx but can progress to SCC. Multiple, sometimes combined, modalities of destruction are tried including curettage, shave removal, freezing with liquid nitrogen, cantharidin, salicylic acid, trichloroacetic acid, lasers, topical/intralesional 5-fluorouracil, topical imiquimod, podophyllin, and intralesional bleomycin. Vaccination is best before sexual activity begins and approved from ages 9–45 years. The vaccine contains the L1 major capsid protein without DNA (quadrivalent HPV-6, 11, 16, 18; bivalent HPV-16, 18; 9-valent HPV-6, 11, 16, 18, 31, 33, 45, 52, 58) [1, 2].

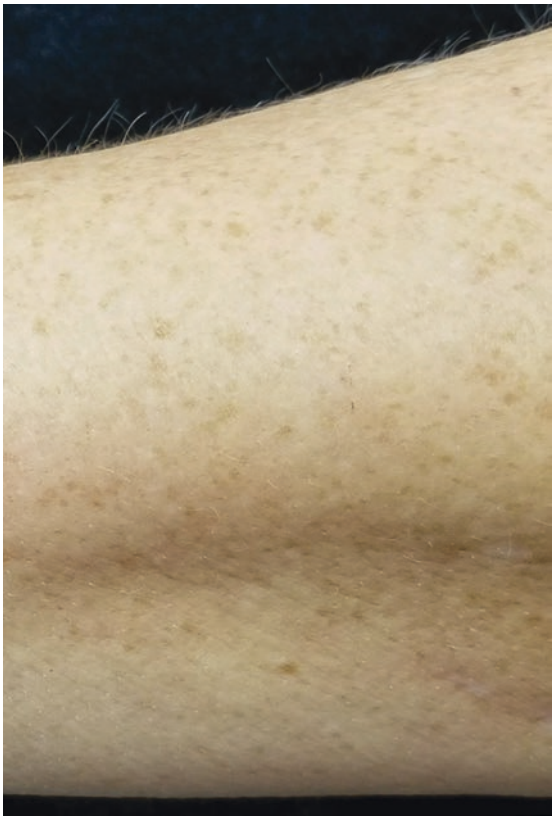


Fig. 36 Lentigines present as brown macules on sun-exposed areas of skin. (Courtesy of Charles E. Crutchfield III, MD's Dermatology Image Library Archive)

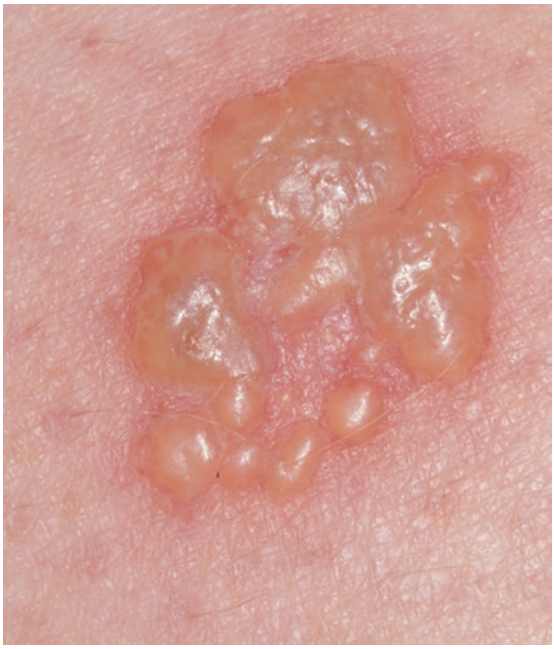


Fig. 37 Herpes zoster shows grouped vesicles/pustules on an erythematous base with scalloped borders. (Courtesy of Charles E. Crutchfield III, MD's Dermatology Image Library Archive)

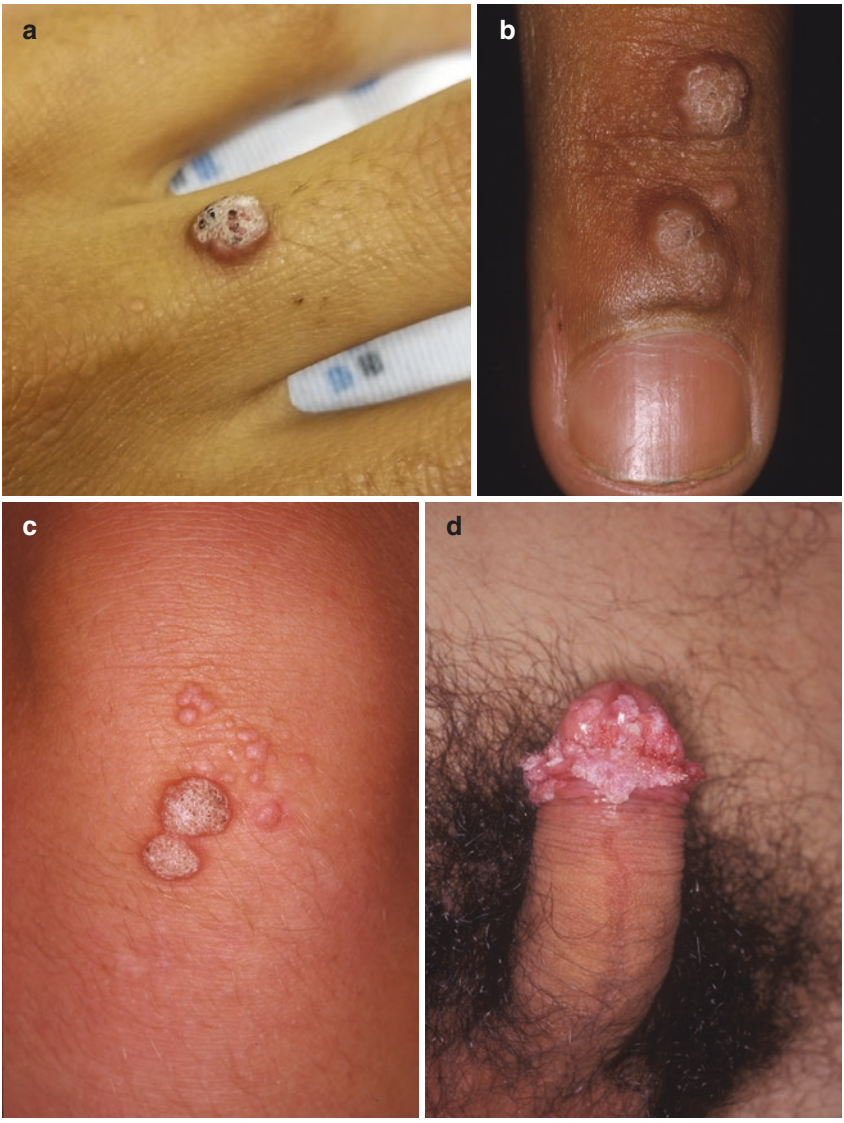


Fig. 38 (**a–c**) Verrucous papules of the extremities and knee. (**d**) Verrucous papules in condyloma acuminata. (Courtesy of Charles E. Crutchfield III, MD's Dermatology Image Library Archive)

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Helpful Resources

https://dermnetnz.org/
https://www.visualdx.com/

Clinical Rotation Tips/Practical Tips for Ordering Imaging Tests: Plain X-Ray, Ultrasound, CT, MRI

Francesca Patella

Contents

It is a truth universally acknowledged that getting along with colleagues is critical to efficient multi-disciplinary team clinical care and job satisfaction. Night or after-hours on-call shifts can present unique challenges in that all parties are fatigued and some overworked, and mistakes and vulnerabilities may be more common after hours. Many radiologists, payors, and healthcare systems will demand adherence to specific indications for imaging tests, or nerves get frayed or tests may not be reimbursed. Also some practical tips may help to order imaging tests, avoid conflicts, and preserve peace.

The Hippocratic Oath originated with students being trained to: *primum non nocere* (first, do no harm). Any improper imaging test is potentially a harm to the healthcare system, a disturbance to the radiologist or professor, but most importantly, it may delay or misguide care, or even harm the patient who may be subject to unjustified radiation exposure or stress. Some considerations and questions for consideration that might guide the proper imaging choice include:

1. *How old is the patient?*
   The implicit question is should the patient be exposed to x-ray radiation?

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As a general rule, x-ray radiation-based imaging tests (plain x-ray and CT) should be avoided in young patients unless they are strictly necessary. When possible and indicated, US and MRI should be preferred [1, 2].

However, diverse x-ray-based imaging techniques implicate very different radiation doses for the patient: a chest CT is responsible of a radiation dose ranging from 3 to 7 mSv, corresponding to 1–3 years of exposure to natural radiation, while a chest x-ray dose is 0.02 mSv, which is equivalent to just 3 days of exposure to natural radiation (yes, there is a natural background radiation on the Earth, since the atmosphere does not protect from radiation from the Sun, a phenomenon that increases on airplane rides) [1] .

Moreover, US and MR accuracy for lung diseases is pretty low. Therefore, chest x-ray, with its low radiation dose, might be considered the right screening option when a lung disease is suspected, regardless of the age of the patient [3]. Chest CT is more sensitive and specific but uses more resources and is more dose and expense.

2. *How is the patient?*

Before ordering a test, try to answer these questions:

- Is the patient hemodynamically stable?

  CT and MRI should be avoided in certain unstable patients, where immediate surgical treatment is indicated without delay. However, in a more stable trauma patient, CT may also clear low suspicion injury to C-spine, head, chest, bones, spleen, liver, kidneys, and aorta. The answer to some questions of CT vs surgery disposition will depend upon the specific acuity, vital signs, and history [4].
- Is the patient compliant?

  Compliance is needed for most imaging tests. Blurred images may be non-diagnostic. Figure 1 shows two pictures of Milan cathedral in Italy. Looking

Fig. 1 The beautiful Milan cathedral. ("Duomo di Milano" by liakada-web is licensed under CC BY 2.0 (https://search.creativecommons.org/photos/71436b7f-4036-416e-93cc-f2faa866d1d1))

at the left one, you can barely argue that it is a church, while on the right one you can see every detail of the facade and admire the splendid example of neogothic style. Radiology images should be like the second picture: a perfect definition is required to capture every tiny detail and to make the right diagnosis. Holding breath may help avoid motion artifact and blur. Sedating a patient may help in certain situations as well.

In photography, the longer the exposure, then the higher the risk of blurry pictures. In radiology the longer the acquisition, the higher the risk of poor-quality images. This means that an MRI characterized by a long acquisition time (a sequence can last several minutes) is the most demanding imaging technique [5]. So, if the patient is unable to stay still, don't order an MRI test, even if it is considered the gold standard for diagnosis. Remember: a good CT is always better than a bad MRI (Fig. 2).

Otherwise, if MRI is crucial for the management of the patient, consider the feasibility of sedation. Sedation is advised when young or uncooperative children have to undergo an MR examination, whereas older patients (5–10 years old) are often perfectly compliant, if they can enjoy a movie during the exam [6].

Compliance does not entail only the capability to stay still but also to hold the breath. Breath holding is essential to get good-quality images with almost every thoracic and abdominal imaging test. Indeed, breathing is diaphragmatic motion; therefore, holding the breath is mandatory to prevent blurry images. Once again, because of the long acquisition time, MRI is especially degraded by the lack of compliance in breathing (Fig. 2) [5]. In addition, holding the breath is crucial for US abdominal scans, since during inspiration, the diaphragm pulls down the organs, facilitating visualization [7].

Finally, remember that a good-quality US scan requires that the patient can turn on the side in order to see the kidneys. Everyone is bothered by gas

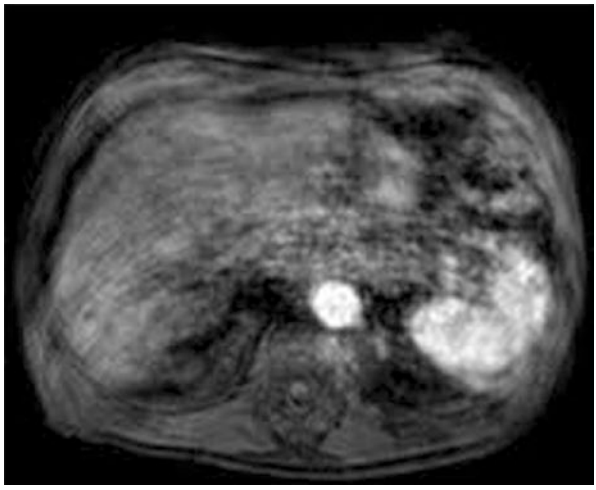

Fig. 2 A very blurred MR abdominal image due to motion artifact

bloating; to the sonographer, gas is the enemy and precludes evaluation of the pancreas body and tail by ultrasound. Indeed, colonic gas is a barrier for ultrasound, impeding organ visualization. One of the possible solutions is to ask the patient to turn on the side to see the kidney from the side/back [7]. US has lower sensitivity and specificity in the non-compliant patient.

- Is the patient overweight?

  Adipose tissue reflects ultrasound, impairing the visualization of abdominal organs (Fig. 3) [7]. On the contrary, abdominal fat may be an advantage on CT images, separating organs and facilitating their identification (especially concerning ureters or intestinal loops) (Fig. 4). Be conscious that there are some technical weight limits for tables in CT, MR, and IR.

3. Are there *patient-specific contraindications to imaging tests?*

   Possible contraindications for radiology examinations:

   - X-ray: pregnancy
   - CT: pregnancy
   - MRI:
     - Pacemaker: this is an absolute contraindication since MR field may interfere with this electronic device, causing the death of the patient; new pacemakers are MRI compatible, but they need to be stopped by a cardiologist, before the patient enters in the MR scan. This takes time and a consult.
     - Other medical devices: minor contraindication device-specific.
     - Metals in the body (e.g., bullets) or metal in eye.
     - Pregnancy (uncertain risk but more in first trimester) [8].

Fig. 3 Foggy morning on the Thames. (Source: mikerolls, licensed under CC BY-SA 2.0); adipose tissue is just like fog on sonographic images: it is white because it reflects ultrasounds, and it surrounds organs, impeding their visualization

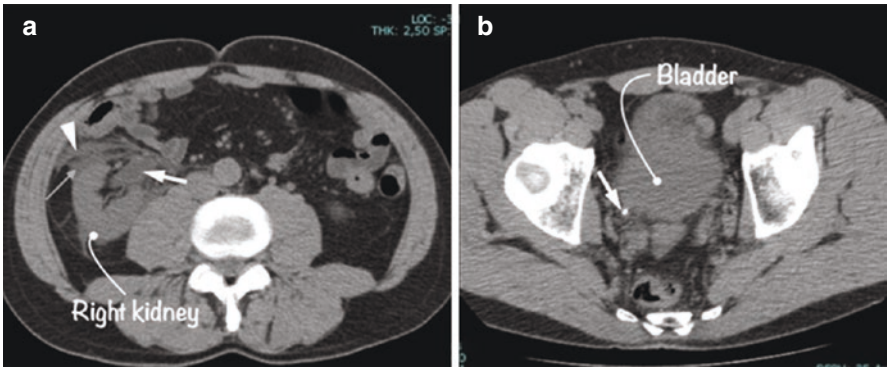

Fig. 4 44 y.o. patient with renal colic undergoing non-contrast abdominal CT. (**a**) Upper abdominal scan showing right hydronephrosis (= dilatation of renal cavities; white arrow), liquid around the homolateral kidney (gray arrow), and thickening of anterior renal fascia (arrow head); (**b**) lower abdominal scan demonstrating the cause of hydronephrosis: a calcific image (a stone) in the right ureter (the white dot indicated by the arrow); in very thin patient, it can be hard to understand whether a calcific image is in the ureter (and therefore it is a real stone) or outside it (and thus it is just a pelvic vascular calcification), but in this patient, the adipose tissue (all you see in dark gray) creates a cleavage that enables following the ureter throughout the entire length

- Contrast-CT/MR:
  - Allergy to contrast material.
  - Renal insufficiency: intravenous contrast materials are excreted by kidneys in urine. In the case of impaired kidney function, the contrast cannot be properly removed from the blood flow [9, 10].

  Hospitals usually have specific guidelines for the management of patients with contraindications to CT/MR contrast material. Check the ones in your hospital, if you (and the radiologist of course) think contrast medium is strictly necessary in such patients. In patients with renal failure or insufficiency, NSF is a risk of gadolinium (related to chelation) and may be a risk when the GFR drops below 60 or 30, depending upon local guidelines.
- US: no contraindications. Consider time limits for extended Doppler exams in early pregnancy (due to heat).
- Contrast US:
  - Cardiac or respiratory insufficiency: the small bubbles that compose contrast medium material are partly eliminated with breathing, a condition that cannot properly occur, in case of heart or lung function compromise.
  - Allergy to US contrast material (very rare) [11].

4. *Which is the pathology to study?*

   The most frequent clinical history shared on imaging tests is "dyspnea," "fever," and (the best) "abdominal pain" (not otherwise specified). Reporting symptoms by typing on a test order does not make a good physician; rather, good communication is necessary for good medicine. Communicate all the history to the radiologist (best in person) or even integrate clinical and laboratory findings.

This section provides an explanation of why it is so important for the radiologist that clinicians filter diagnostic hypotheses better than Google does.

Claude Bernard (the inventor of scientific physiology) used to say to his fellows that "the experimenter who does not know what he is looking for will never see what he finds." This can be perfectly applied to radiology: Look at Fig. 5 for 30 seconds, what can you see? What about if you are told that there is a snake between the plants?

Probably when you looked at the picture for the first time, you were not able to distinguish the snake, but when you knew what pattern or easily identified thing or expectation to search, you found it (solutions at the end of the chapter).

Are you convinced? Let us try again. Look at Fig. 6 for 30 seconds. Can you see something in the sea? Yes, for sure, if you are told there is an octopus (the solution is at the end of the chapter) (Fig. 16).

Patterns are learned and the power of suggestion means the clinician will look for imaging diagnoses or findings that match the clinical or lab picture, like when you learn what to look for. In radiology, imaging tests are a collection of information that may be difficult to interpret or capture without a specific suggestion. This is especially true when the hidden pathological finding is not in the center of the field of view or it is 5 a.m. and you are on-call and have not slept.

Neuroscience has an explanation for this phenomenon. Nobel Prize Eric R. Kandel in his beautiful essay, "The age of the insight" [12], showed that the

Fig. 5 Challenge 1

Fig. 6 Challenge 2

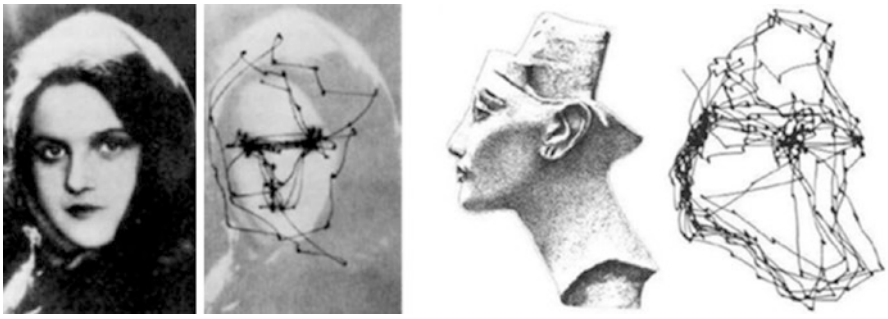

Fig. 7 Famous Yarbus experiment about eye saccadic movements showing that regardless of the position of the subject's head; our gaze (and thus our attention) is mostly fixed on the eyes and the mouth of the person at whom we are looking

human mind is programmed to pay attention only to a few things at the same time. For instance, looking at a person, the gaze will focus on the hands and the face and specifically on the eyes and the mouth Fig. 7 [12].

Maybe one day artificial intelligence (AI) will overcome human limitations and will enable us to capture the entire information that is inside a radiology image. However, deep learning and AI will likely for the near future rely upon human guidance and interpretation from human radiologists. Think of the

radiologist as a part of the imaging test: to increase the sensitivity and the specificity of the test, you need to provide the radiologist with all useful information that may help to focus on the right details.

As part of clinical hypothesis formation, and before ordering an image, it is imperative the clinician asks his/herself whether it is possible to see the suspected pathology with imaging. If yes, then is radiology the best way to confirm the diagnosis?

This is crucial. Pathologies are usually associated with some morphological changes – that is why pathologists can make the diagnosis by looking at tissue. However, most imaging is not able to capture across multiple scales. Indeed, radiological tests can show only macroscopic alterations of organs. PET scan is an example of metabolic or molecular imaging, however.

Generally speaking, certain inflammatory diagnoses in the upper abdominal organs are hard to see with radiological signs: hepatitis may not be visible at all, pancreatitis may be detectable on CT only after 24 h, and nephritis is demonstrable with imaging only when it causes renal perfusion modifications [13].

The most common causes of abdominal pain may not immediately require radiology tests. This may be the case in gastroesophageal reflux, peptic ulcers, food intolerance, overeating, infectious gastroenteritis, or irritable bowel syndrome, where morphological changes may not be detected with standard current imaging techniques.

Notably, there also some tumors that do not entail a tumefaction: some blood tumors (e.g., leukemia) are not mass forming, so they cannot be diagnosed with imaging.

Moreover, some pathologies may involve specific radiological signs, but this fact does not necessarily imply that imaging tests are the best way to confirm the diagnosis. For instance, asthma and other forms of COPD or bronchitis are associated with "air trapping," which can be seen on CT images as more hypodense (darker) areas of lung parenchyma (Fig. 8). However, the diagnosis of asthma

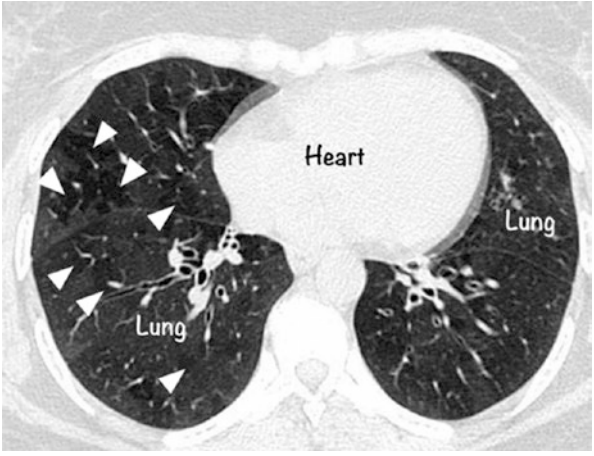

Fig. 8 38 y.o. patient with asthma undergoing non-contrast CT to exclude pneumonia; air-trapping areas can be seen as hypodense (darker) areas in the lung (arrow heads)

remains clinical, and an imaging test should be used only to evaluate complications (e.g. pneumonia).

Although imaging has incredibly enhanced diagnostic accuracy, it is not always indicated or needed. Moreover, some pathologies would be completely missed, if searched for with imaging tests. You can always find the most recent guidelines for the diagnosis of every pathology in textbooks, PubMed, or even on an online search. Students and interns may learn the most from an eager radiologist mentor or teacher. Ask: is this test indicated? Will it change management?

Finally, once assessed that the pathology needs an imaging test, the next question is, is it an acute or chronic disease? This may inform when the imaging result is needed.

In case of immediate life-threatening disease, the right choice is a quick imaging test in order to get a rapid response: plain x-rays, US, and CT are all good options. As said before, MR has a long acquisition time, which may preclude its role in emergency.

On the contrary, in case of chronic disease or not immediate life-threatening acute disease, MRI may be contemplated. For instance, MRCP (= MRI cholangiopancreatography) may be a good option to evaluate patients with cholecystitis, before they undergo cholecystectomy. An ultrasound, however, may be the best way to diagnose acute cholecystitis.

5. *Which is the body region to study?*

Spoiler alert: this section will sum up some indications that will be detailed in the next chapters. As general advice, try always to figure out what you specifically are looking for with the test, and how it might change your management.

- Head and neck:

  Plain x-rays are not very useful in the head and can show only the skull. Moreover, because of the complexity of the skull anatomy and the overlap of bony structures, x-rays have a very poor accuracy in depicting skull alterations, such as fractures (Fig. 9) [14].

  So, forget skull x-rays.

  CT and MRI can both show the soft tissues, skull, and brain.

  However, there are some important distinctions between these techniques. Look at Figs. 10 and 11 that compare CT and MR images of the brain, skull, and soft tissues. Do you find some differences?

  Featured by higher contrast resolution, MRI is the best technique to enhance the brain's as well as head and neck soft tissues' anatomy and, therefore, underlying diseases.

  Nevertheless, since MRI signal depends from the excitation of hydrogen atoms, whose number is pretty low in the skull base (which is water-poor), CT remains the gold standard for the study of the skull and its bony structures (such as the cranial nerves canals and the ear) [14].

  In addition, CT is always the technique of choice in ER, both because of its rapid acquisition time and its high accuracy in detection of acute

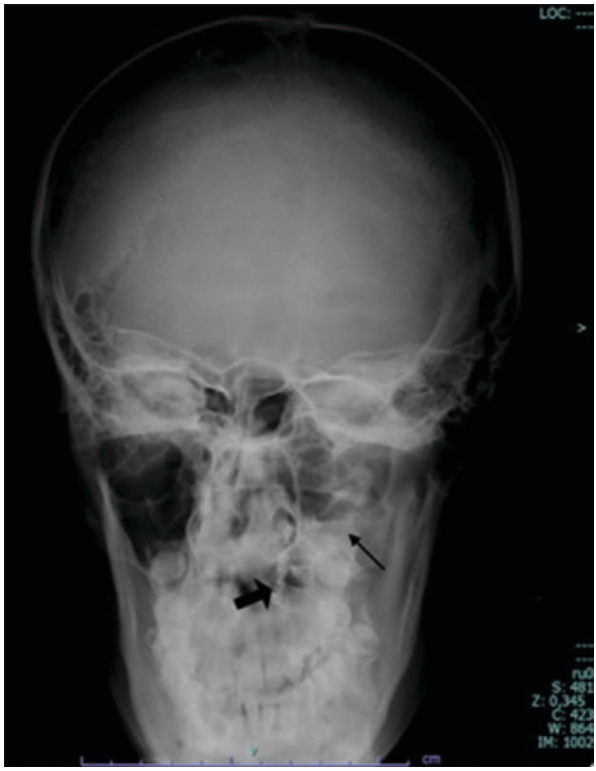

Fig. 9 16 y.o. patient undergoing plain x-rays after surgery to treat maxillary bone fracture (see the metal device indicated by the thick arrow); actually, this patient had multiple maxillary wall fractures on the left side, but the only sign of them is the blood in the sinus that you can see as the presence of intra-sinusal white material (thin arrow) instead of the normal black air (look at the other side)

hemorrhages – which used to be the Achilles heel of MRI [4, 14], although this may no longer be entirely true.

Finally, remember the thyroid, which is better studied with US [14].

- Chest:

  Filled with air, lungs are the perfect object of x-rays exams (both plain x-ray and CT). Indeed, most lung pathologies are associated with an increase in the x-ray attenuation either in the alveoli (parenchymal consolidations) or in the interstitium, creating the perfect contrast resolution with the healthy parenchyma (Fig. 12).

  As said before, the presence of protons (and therefore water molecules) is a prerequisite for MRI. The corollary is that MRI finds many fewer indications for lung studies (air = no water = no protons > no signal > no image).

- Mediastinum:

  Therefore, MRI could theoretically play a role in the study of the mediastinum (Fig. 13). However, in practice, it is rare that the object of the examination is just the mediastinum. Thus, CT remains the gold standard for chest evaluation, since it allows optimal visualization of the lung and the mediastinum (by enhancing vessels with contrast medium) at the same time (Fig. 13).

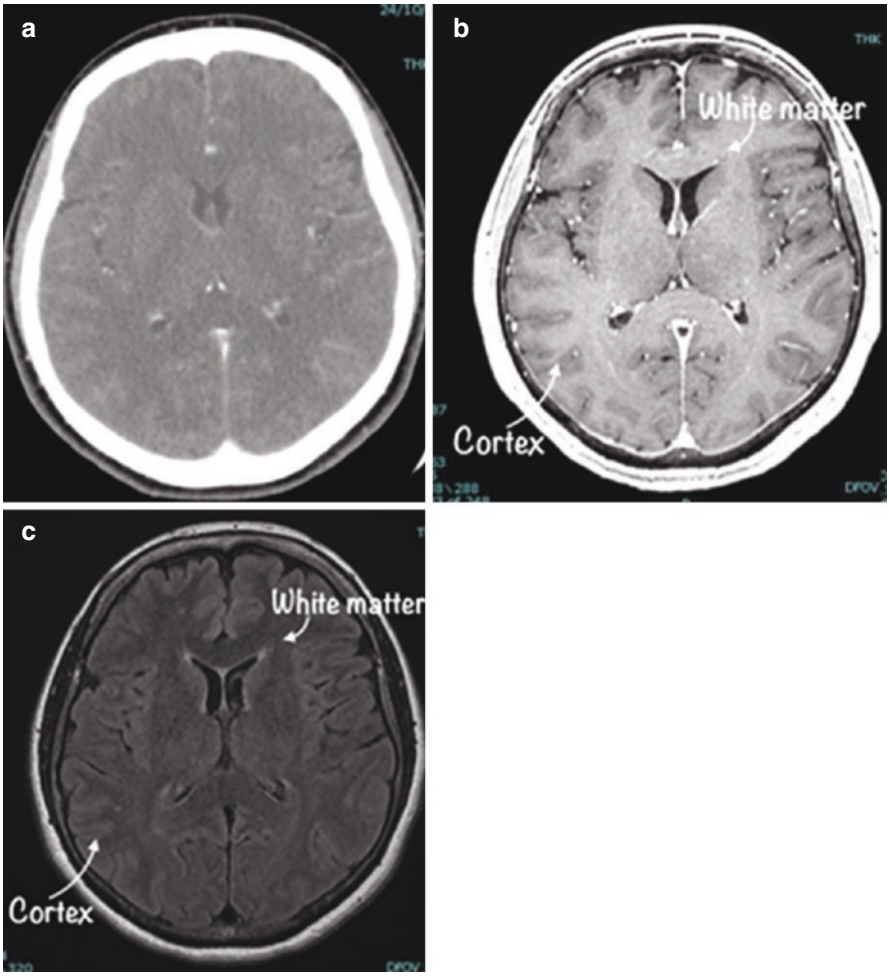

Fig. 10 62 y.o. patient affected by left diplopia and homolateral eye pain; (**a**), (**b**), and (**c**) show the normal brain with post-contrast-enhanced CT (**a**), post-contrast-enhanced T1-weighted MRI (**b**), and non-contrast FLAIR MRI. Notice how MRI highlights brain anatomic structures, regardless of the injection of contrast medium, while CT gives a less detailed image

There is only one mediastinal organ that is commonly studied with multiple techniques in clinical practice. Clue: It beats. Correct, it is the heart: according to the indication, it can be evaluated either with US, CT, MR, or even coronary angiography (coronary arteries that feed the heart) [15].

- Abdomen:

  Here are some general tips considering different techniques (specific indications for each abdominal organ will be reported in the abdominal imaging chapter):

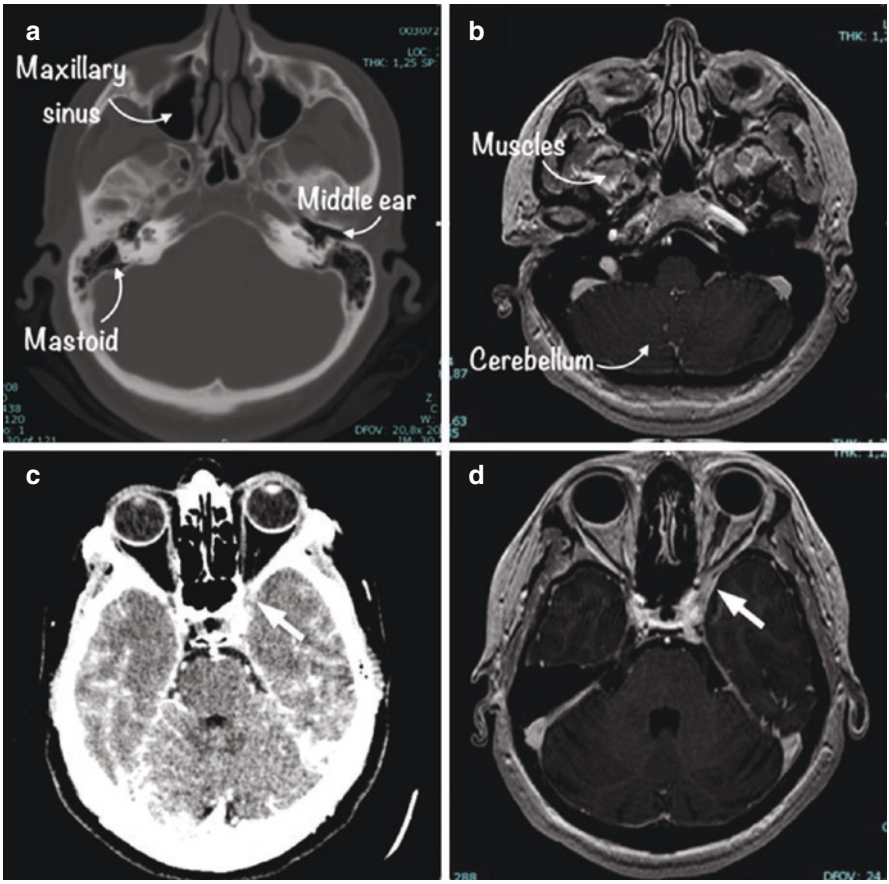

Fig. 11 Same subject that in Fig. 10; on the left side (**a** and **c**), you can see CT images, while on the right side (**b** and **d**), you can see the corresponding MR images; (**a** and **b**) show the patient (normal) skull base. (**a**) is a non-contrast CT image visualized with bony window: the bone is white (hyperdense), the air is black (hypodense), while soft tissues (both brain and facial muscles) have the same shade of grey. (**b**) is a T1 contrast-enhanced MR image: in this case both the skull base (except for the clivus) and the air in the maxillary sinus, in the mastoid and in the medium ear, are black (hypointense due to no signal), but you can easily distinguish the cerebellum (inside the neuroskull), from the masticatory muscles (outside the neuroskull, behind the maxillary sinus). (**c**) and (**d**) are, respectively, a post-contrast CT and MR showing the cause of the symptoms: an inflammation of the nerves inside the superior orbital fissure (arrows); as you can see the enhancement of the nerves is much more evident on MR images

- Plain x-rays: look at Fig. 14. Can you see anything besides bowel gas? Do not be afraid if you cannot: no one can. Abdominal parenchymal organs have similar x-rays attenuation, so you just can see their shadow (only sometimes) and nothing more. In radiology, this situation is defined as poor contrast-resolution, which is the opposite of what is needed to detect pathologies with imaging.

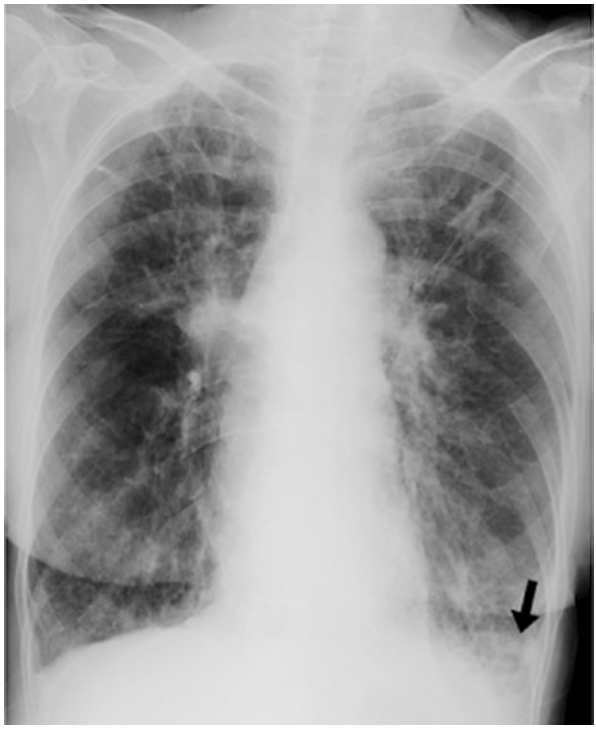

Fig. 12 Chest x-rays of a 52, y.o. patient with suspected pneumonia; the base of the left lung is definitely whiter than the contralateral one: this is an alveolar consolidation of the left lower lobe associated with pleural effusion (blunting of diaphragm), expected to be exudative

Therefore, plain radiography enables the identification of just a few pathological conditions featured by a completely different x-rays attenuation (and thus, a different density or brightness) when compared to normal organs. The most important ones are the presence of air in the bowel wall itself, too much bowel gas from obstruction, or the presence of free abdominal air (pneumoperitoneum) – see the abdominal radiology chapter for some examples. Remember that in normal conditions, plain x-rays show just a small amount of air in the colon and no air in the small bowel (Fig. 14).

Another plain x-rays detectable condition are stones, particularly gallstones or kidney stones.

- CT: Without contrast, in non-contrast CT, all the organs (and their diseases) are gray (Fig. 4). Thus, like plain x-rays, abdominal non-contrast CT has limited contrast resolution. Only a few pathological conditions have the proper CT numbers (i.e., the right shade of grey) to be detected by our eyes. Namely, the main indication for non-contrast CT is kidney stones (Fig. 4) [16].

Technically non-contrast CT allows also the detection of other conditions, such as intestinal occlusions or free abdominal air or liquid, but in this case it is also important to identify the medical cause for specific imaging findings [16]. To do it, it is necessary to create the appropriate contrast resolution (the differences in intensity that your eyes can capture) and therefore to inject IV

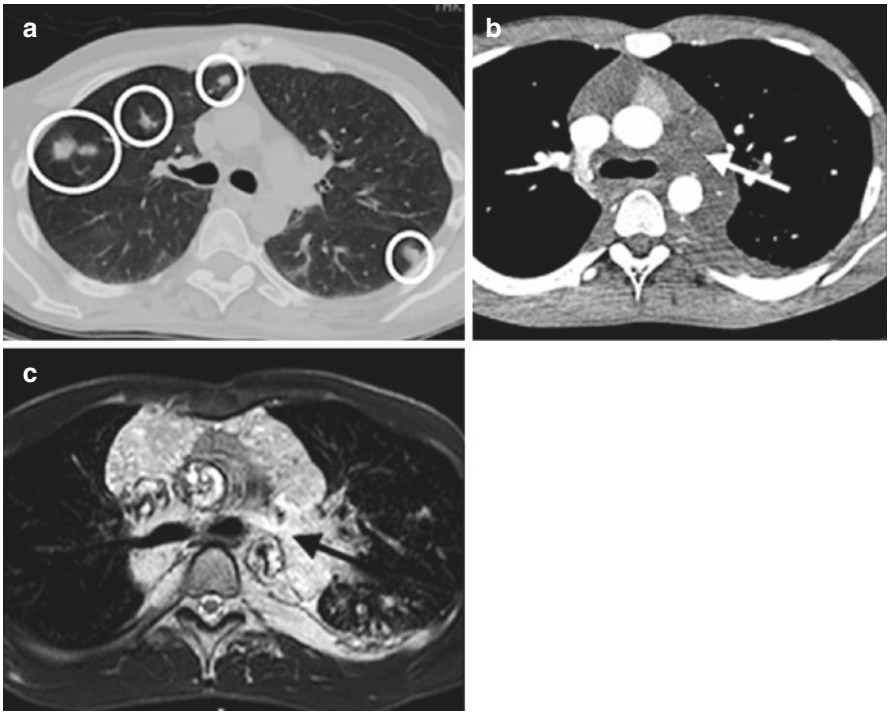

Fig. 13 (**a**) Chest contrast-enhanced CT (visualized with lung window) of a 54 y.o. patient with suspected pneumonia. Multiple bilateral alveolar consolidations confirm the diagnosis; (**b**) and (**c**), respectively, chest contrast-enhanced CT (visualized with soft tissue window) and non-contrast STIR chest MRI of a 15 y.o. patient with mediastinal lymphangioma (a lymphatic vascular malformation indicated by arrows): observe the enlargement of the mediastinum when compared with the normal mediastinum of the patient in (**a**) and appreciate how the abnormal tissue as well as mediastinal structures are evaluable on both CT and MRI. Why in your opinion is lymphangioma gray on CT and bright (hyperintense) on STIR (=T2 fat saturated) MR images? What is bright on T2 fat saturated images? [Answer: it contains lymph which is very similar to water]. Easy to remember this with T**2** = H**2**O (or water is bright on T2)!

contrast. The principle at the base of intravenous contrast medium is that pathologies are featured by abnormal vascularization that would translate in different contrast enhancement.

So generally speaking, to define an abdominal pathology with CT, ask for contrast-enhanced CT (Fig. 15).

- MRI: this is the technique with the highest contrast resolution and therefore the gold standard for the study of some abdominal organs such as the liver, the biliary tree, or the rectum (Fig. 15) [17]. However, MR is an expensive exam and requires high compliance from the patient. It is not uncommon for MR to be a second-level investigation, aimed to characterize or follow-up pathological conditions that were identified in other ways (e.g., liver lesions detected with screening US).

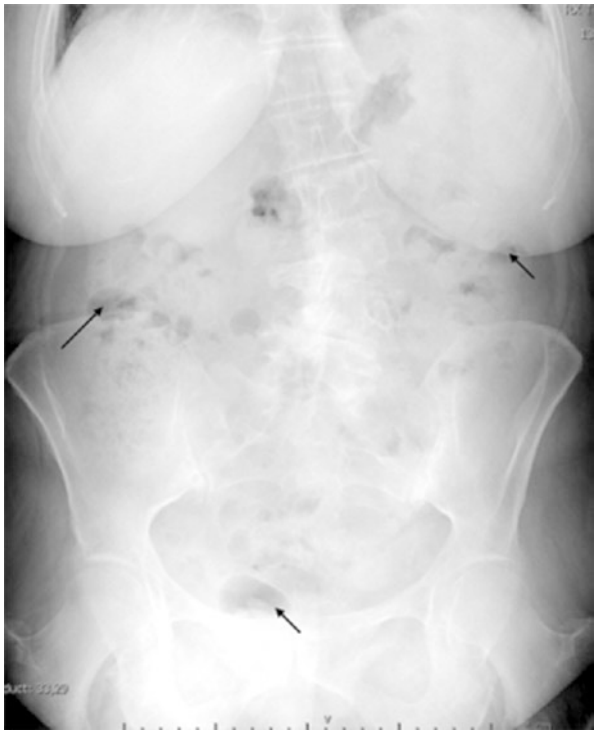

Fig. 14 Abdominal x-ray showing scoliosis, degenerative disc disease, and (age-related) osteoarthritis of the hip joint, with sclerosis of the acetabular surface and joint space narrowing. Arrows indicate unobstructed colon gas

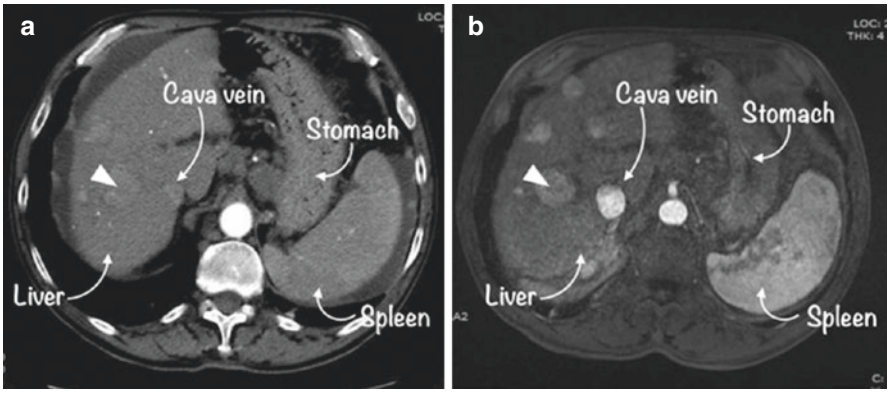

Fig. 15 70 y.o. patient with multiple HCC nodules in the liver; (**a**) post-contrast arterial phase CT; (**b**) post-contrast arterial phase T1 MRI, acquired almost at the same time as the CT scan; compare (**a**) and (**b**) and notice how MRI enables the visualization of many more nodules (all the bright dots in the liver – the largest is indicated by the arrow) because of the higher contrast resolution. HCC parasitizes blood supply from the hepatic artery and reverses the 75% portal vein to 25% hepatic arterial inflow to the normal liver, so it shows up earlier in the dynamic contrast process

- US: it represents an optimal first-level test to verify the presence of parenchymal organ diseases or other pathological conditions such as stones (both gallstones and kidney stones), free liquid, or aortic aneurysm [17].

Fig. 16 Solution of the image games. Eyes can be trained to look for certain things they have seen before or toward which they are directed. Artificial intelligence and computer-aided detection may also assist the radiologist

Gas is the number one enemy of US. So, US in general should not be employed o evaluate intestinal diseases or free abdominal air. Besides, the presence of colic air often precludes the correct visualization of the pancreas.

However, every rule has an exception. Indeed, in children (especially in little ones), US may be successfully used (by an experienced radiologist, of course) as a better choice than CT for some intestinal acute diseases, such as appendicitis or intussusception. In addition, US after adequate preparation can be employed to follow up inflammatory bowel diseases.

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Question bank

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The following questions are presented for use in one of two modes: (1) "Practice question mode" with unknown answers, to test your skills, or (2) "Information gathering mode" for efficient digestion of case data; simply read the correct answer and learn why it is correct and why the incorrect answers are incorrect. The answers are presented immediately after each question, making it easy to read in either mode. Cover up the answers to use in "practice question mode."

1 Emergencies

Question 1

CTA was performed for a hemodynamically stable 50-year-old man with a history of uncontrolled hypertension who presents with sudden-onset sharp chest pain (Fig. 1).

What is the most appropriate next step in managing this patient?

A. Transthoracic echocardiography
B. Transesophageal echocardiography
C. Magnetic resonance angiography
D. Medical therapy with close observation
E. Emergent surgical repair

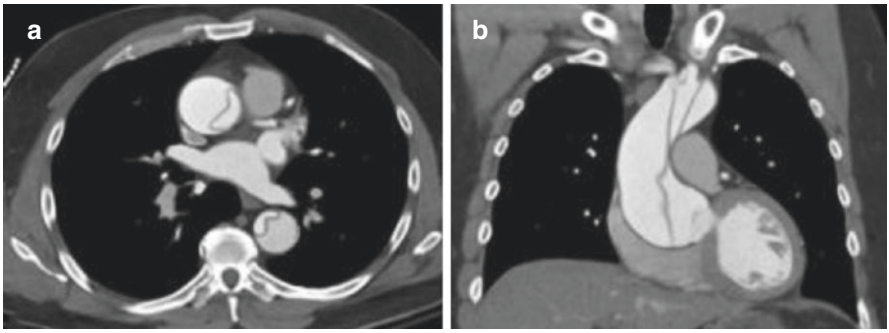


Fig. 1 For Question 1. CT angiography of the chest, axial (**a**) and coronal (**b**) slices

Answer: E. Emergent surgical repair

A Stanford type A aortic dissection extends from the ascending aorta (Fig. 2a, black arrowhead; b, arrows) to the descending aorta (Fig. 2a, white arrowhead). The Stanford system classifies type A dissections as those involving the ascending aorta (but can also have components in the arch and/or descending aorta) and type B dissections as those involving the descending aorta only. Type A dissections are surgical emergencies even for hemodynamically stable patients because these patients are at high risk of death from cardiac tamponade, acute aortic regurgitation, and myocardial infarction. Type B dissections in hemodynamically stable patients are initially managed with antihypertensives and vasodilators. CTA is the imaging modality of choice due to high diagnostic accuracy, wide availability, and short acquisition time. In this case where CTA clearly demonstrates an aortic dissection flap and its extent to establish the diagnosis of type A dissection, further imaging such as with echocardiography or magnetic resonance angiography is not indicated and would delay emergent surgical repair.

Question 2

A 20-year-old otherwise healthy man presents to the emergency department with several hours of severe right scrotal pain. Scrotal ultrasound is performed.

What is the diagnosis?

A. Left testicular torsion
B. Right testicular torsion
C. Left orchitis
D. Right orchitis

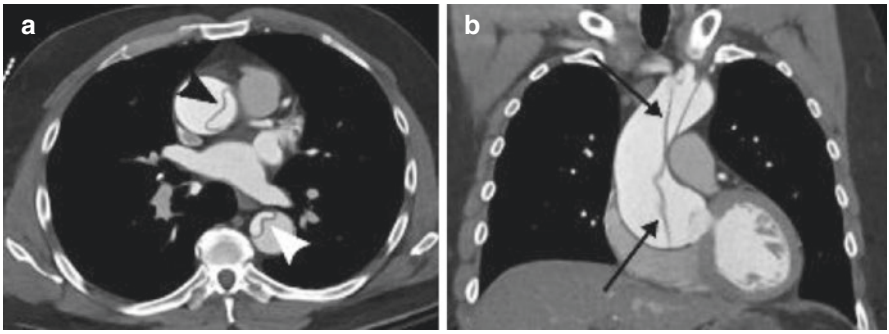


Fig. 2 Aortic dissection seen on axial CT (**a**) and coronal CT (**b**). Annotated Fig. 1, for explanations to Question 1

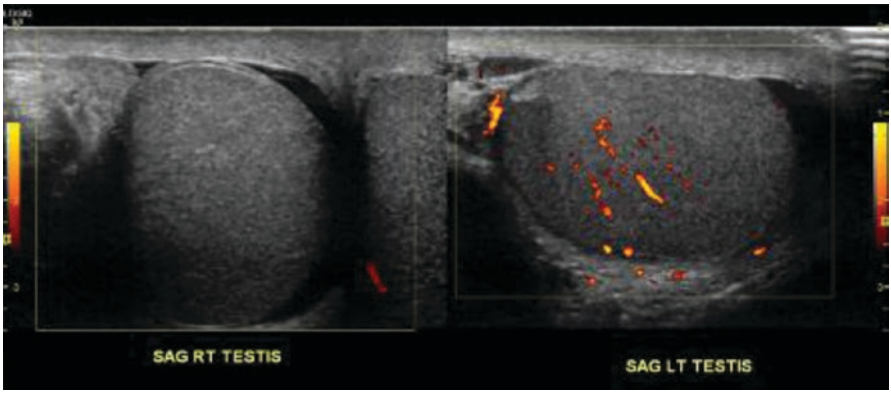


Fig. 3 For Question 2. Power Doppler images of the bilateral testes, right (RT) and left (LT). Images are acquired with identical Doppler settings

Answer: B. Right testicular torsion

The Doppler ultrasound images (Fig. 3) illustrate asymmetrical blood flow in the testes; in fact, no blood flow can be appreciated in the right testis. Absent Doppler flow is pathognomonic for testicular torsion. Urgent surgical de-torsion is indicated.

In orchitis, there is abnormally increased blood flow in the affected testis, which on Doppler ultrasound would also demonstrate asymmetric flow signal. Note that it is possible to set the flow velocity thresholds too high to make the images appear as if there is no blood flow on the normal side. The laterality of the patient's symptoms

is crucial in determining if there is abnormally *increased* blood flow in one testis or abnormally *decreased* blood flow in the other testis. Less reliably, secondary imaging signs on the affected side such as reactive hydrocele, skin thickening, or testicular enlargement may be present, but none of them are readily apparent in this case.

Power Doppler is a sonographic technique that visualizes flow. Compared to color Doppler, which can measure flow velocity and direction, power Doppler can do neither and is not meant to be quantitative. However, power Doppler is more sensitive than color Doppler, making it well suited for evaluation of testicular blood flow when torsion is suspected.

Question 3

A 55-year-old man with dialysis-dependent end-stage renal disease presents to the emergency department with several hours of diffuse abdominal pain that began shortly after hemodialysis, during which he had an episode of hypotension. Upon arrival he is hypotensive. His laboratory studies are notable for serum lactate of 2.4 mmol/L. CT of the abdomen and pelvis is performed (Fig. 4).

What is the most likely diagnosis?

A. Adynamic ileus
B. Pneumobilia
C. Small bowel ischemia
D. Hepatic abscesses

Answer: C. Small bowel ischemia

Peripheral branching gas pattern in the liver is consistent with portal venous gas (arrows). There is pneumatosis intestinalis of the small bowel in the left upper quadrant (arrowheads). These findings in the setting of compatible symptoms and elevated serum lactate are diagnostic of small bowel ischemia. Surgical consultation is required for consideration of small bowel resection. In this patient, it is postulated that his hypotensive episode during hemodialysis may have led to small bowel ischemia (Fig. 5).

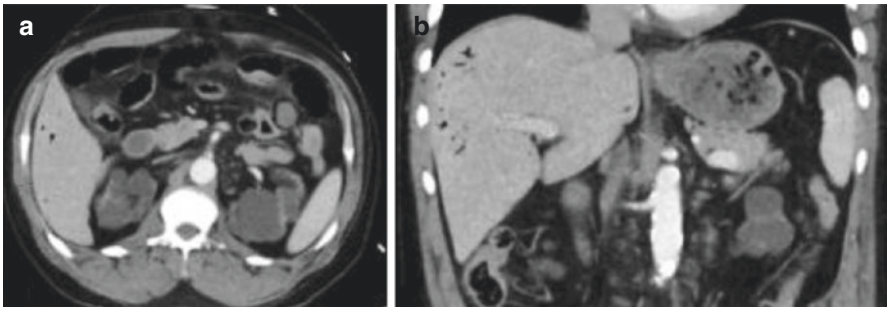


Fig. 4 For Question 3. CT of the abdomen with intravenous contrast, axial (**a**) and coronal (**b**) slices.

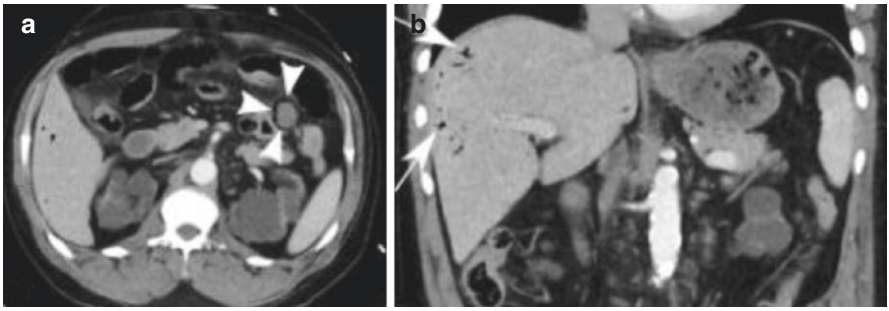


Fig. 5 Axial (**a**) and coronal (**b**) abdominal CT. Annotated Fig. 4, for explanations to Question 3

Distinguishing pneumobilia from portal venous gas is important when gas is seen in the liver. Biliary tree gas is typically more central, involving the common bile duct and common hepatic duct. Because most cases of pneumobilia are due to an incompetent sphincter of Oddi, the patient would usually have a history of endoscopic retrograde cholangiopancreatography (ERCP), sphincterotomy, or biliary surgery.

An adynamic ileus would manifest as diffuse small bowel dilation, which is absent in this case. In addition, there is no history of recent abdominal surgery or gastrointestinal instrumentation that typically precipitates ileus.

Hepatic abscesses would appear as hypodense, peripherally enhancing lesions, which are absent here. Gas is sometimes found in hepatic abscesses but is not the predominant finding.

Question 4

A 25-year-old intoxicated male with no past medical history was found unresponsive after a 6 feet fall from a tree and was intubated on the scene. On arrival, the patient is tachycardic and hypertensive. After holding sedation, the patient remains unresponsive with no eye opening and verbal responses. He shows abnormal flexion of all extremities upon stimulation. Pupils are nonreactive with absent corneal reflex. Trauma survey reveals right scalp swelling and left arm and chest wall bruising. Chest and pelvic radiographs are negative for fractures. eFAST exam is negative.

Part A

Which of the following imaging study is the most appropriate next step?

A. CT head without contrast
B. CT abdomen and pelvis with contrast
C. MRI of the brain without and with contrast
D. MRI of the cervical spine without contrast

Answer: A. CT head without contrast

Presentation following trauma with GCS 5 and absent brainstem reflexes is indicative of severe head injury. According to the American College of Radiology Appropriateness Criteria, non-contrast CT head is the imaging modality of choice

for the initial evaluation of moderate to severe closed head injury (GCS <13). Based on the mechanism of injury, cervical spine injury is a possibility, but the preferred imaging modality would be CT, not MRI. CT abdomen and pelvis would identify concurrent blunt abdominal trauma and may be obtained, but the suspected head injury is likely the more emergent injury in this patient given the negative eFAST exam.

Part B

An emergent non-contrast CT of the head is obtained. Two representative images are shown Fig. 6.

Which of the following findings is present?

A. Subdural hematoma
B. Subarachnoid hemorrhage
C. Intraparenchymal hematoma
D. Epidural hematoma

Answer: D. Epidural hematoma

The large biconvex shaped hyperdensity along the right frontoparietal convexity (Fig. 7a, black arrow) represents an epidural hematoma, a collection of blood between the skull and the dura, causing significant mass effect on adjacent brain parenchyma. This hematoma likely resulted from arterial injury due to the overlying non-displaced right parietal bone fracture (Fig. 7b, white arrow).

Epidural hematomas are typically biconvex in shape and do not cross suture lines, while crescent-shaped subdural hematomas can cross suture lines.

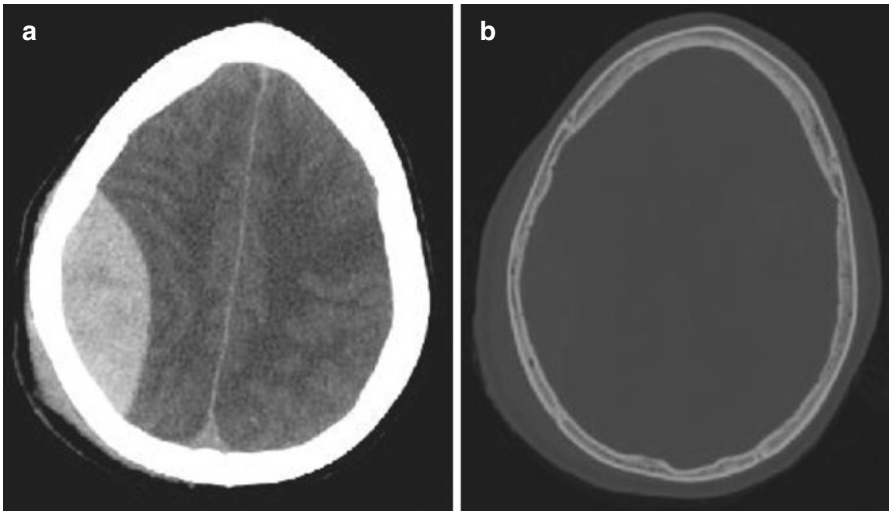


Fig. 6 For Question 4, Part B. Axial images of CT of the head without intravenous contrast, brain (**a**) and bone (**b**) windows

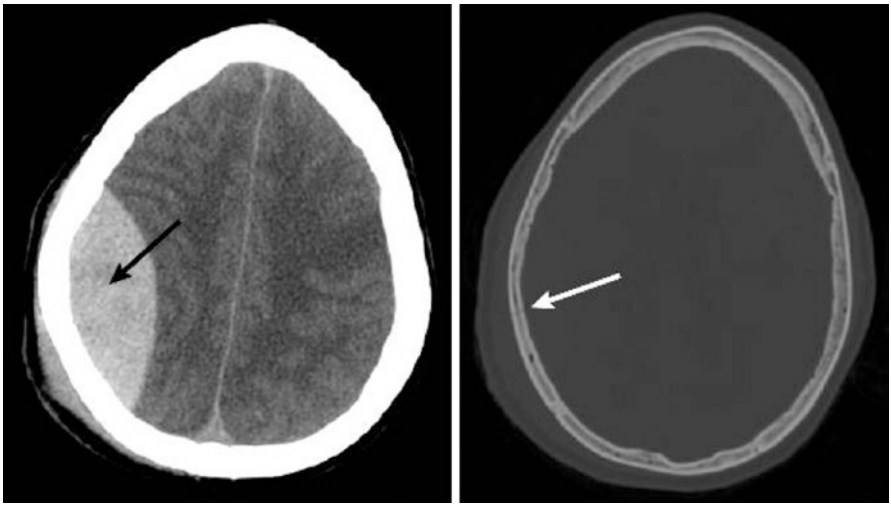


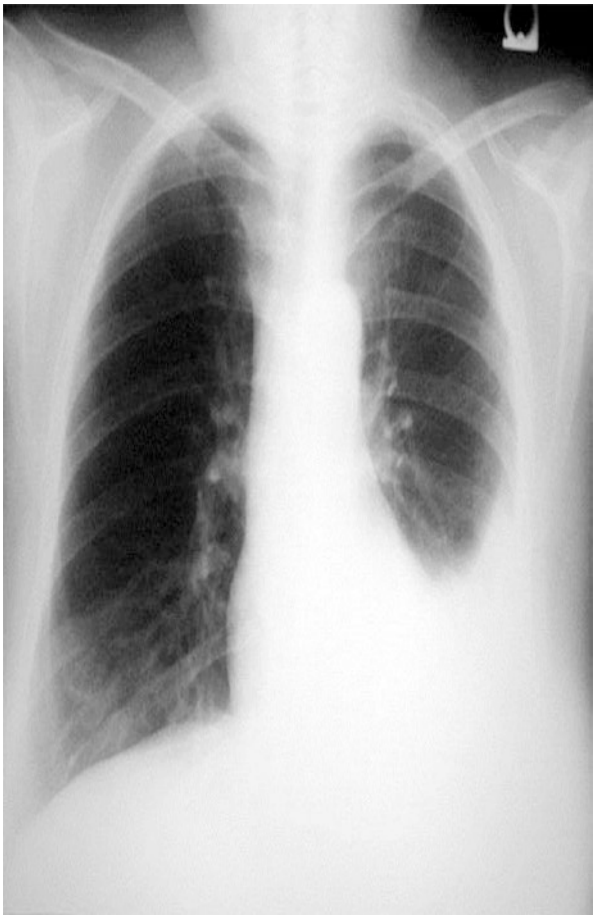
Fig. 7 Brain (**a**) and bone (**b**) windows for same head CT. Annotated Fig. 6, for Question 4, Part B

Subarachnoid hemorrhage extends centrally into the cerebral sulci, while intraparenchymal hematomas would be centered within the brain parenchyma.

2 Chest

1. A 42-year-old female comes to the emergency department complaining of dyspnea. She reports that over the past weeks, she has become more easily "winded" and feels generally fatigued with activities that she previously performed without question. Her temperature is 98.9 degrees Fahrenheit, blood pressure is 128/82, pulse is 84/minute, and respirations are 20/minute with an oxygen saturation of 96% O2 on room air. A chest radiograph is obtained, as shown below. Which of the following would you expect to find upon a physical exam?
   A. Kussmaul breathing
   B. Decreased tactile fremitus
   C. Increased tympanic percussion
   D. Crepitus
   E. Increased pectoriloquy

   Answer: B. Decreased tactile fremitus



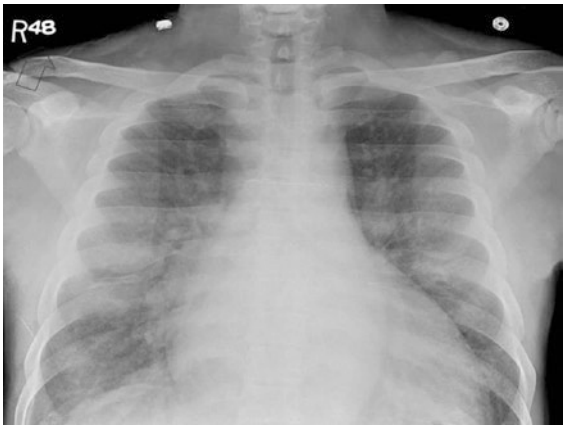
2. A thoracentesis was performed with the following fluid analysis:
   - Volume: 100 cc of fluid
   - Pleural fluid protein: 4.9 g/dL
   - Serum protein ratio: 7.0 g/dL
   - Pleural fluid lactate dehydrogenase (LDH): 266 U/L

   Which of the following conditions may be associated with the patient's condition?

A. Hypoalbuminemia
B. Hepatic hydrothorax
C. Heart failure
D. Nephrotic syndrome
E. Systemic lupus erythematosus

Answer: E. Systemic lupus erythematosus

3. A 67-year-old male with a past medical history of hypertension and hyperlipidemia comes to the emergency department for progressive dyspnea on exertion that began two days ago. Review of systems is notable for orthopnea, productive cough, and bilateral lower extremity swelling. His temperature is 99.3 degrees Fahrenheit, blood pressure is 150/94, pulse is 82/minute, and respirations are 18/minute with an oxygen saturation of 92% O2 on room air. Physical exam demonstrates crackles in the bilateral lung fields and a displaced PMI. The chest x-ray taken in the ED is below:

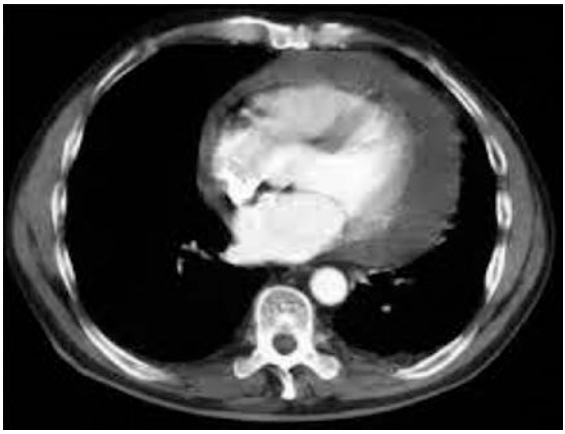


Which of the following findings do you see on chest x-ray?

A. Lobar consolidation with hilar lymphadenopathy
B. Multiple bilateral pulmonary nodules
C. Increased cardiothoracic ratio, peribronchial cuffing, and Kerley B lines
D. Pneumothorax without mediastinal deviation

Answer: C. Increased cardiothoracic ratio, peribronchial cuffing, and Kerley B lines

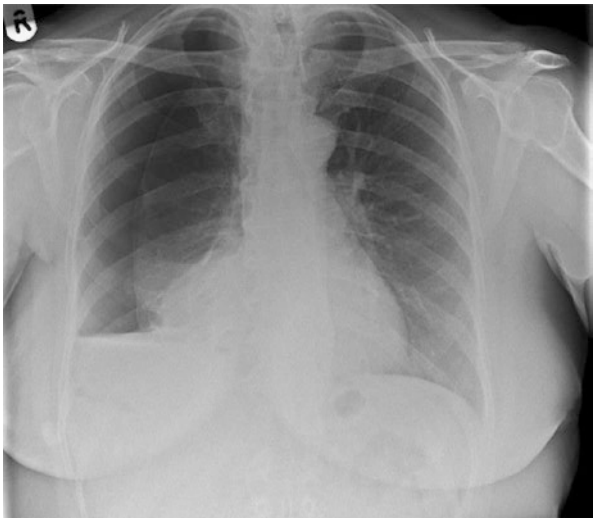
4. Where is the abnormality on this axial contrast-enhanced chest CT?



A. Left atrium
B. Pericardium
C. Vertebral body
D. Descending aorta
E. Myocardium
F. Left ventricle

Answer: B. Pericardium

5. Air (and/or fluid) can enter the pleura space with each breath but cannot escape. This increases the intrapleural pressure. This process is depicted in the accompanying image below.



What does the increased pressure do to the diaphragm?

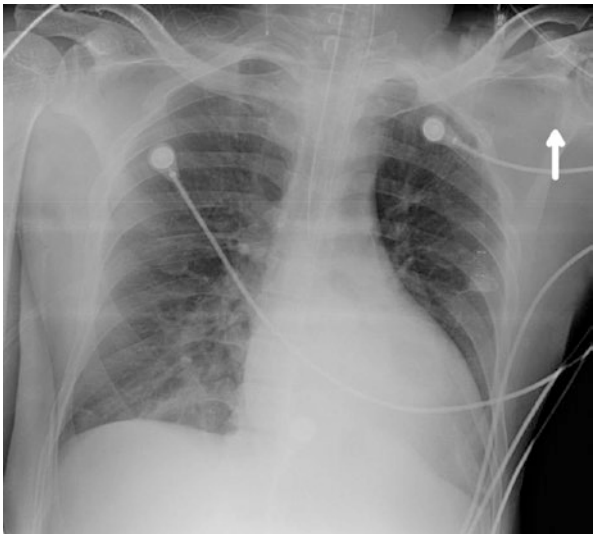
A. Elevates the diaphragm
B. Depresses the diaphragm

Answer: B. Depresses the diaphragm

6. In relation to the above process, what does the increased pressure do to the mediastinum?
   A. Shifts the mediastinum toward the hydropneumothorax
   B. Shifts the mediastinum away from the hydropneumothorax

Answer: B. Shifts the mediastinum away from the hydropneumothorax

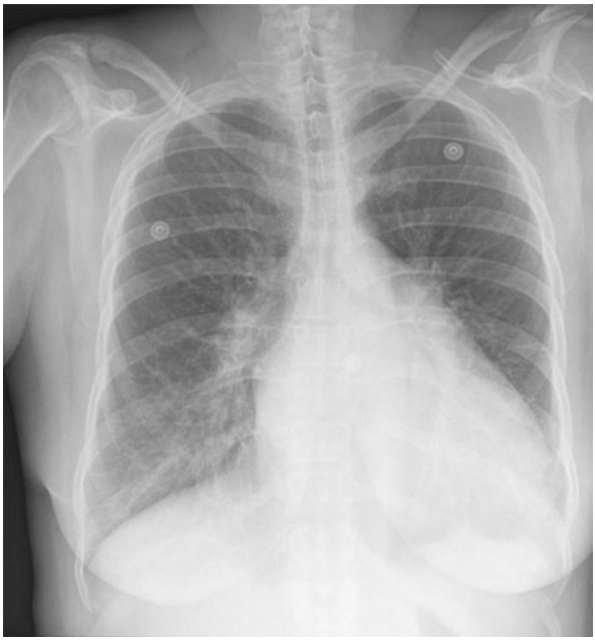
7. You are on the night call shift in radiology and a referring physician calls to ask for an interpretation of their patient's chest x-ray in the ICU. What do you tell her?

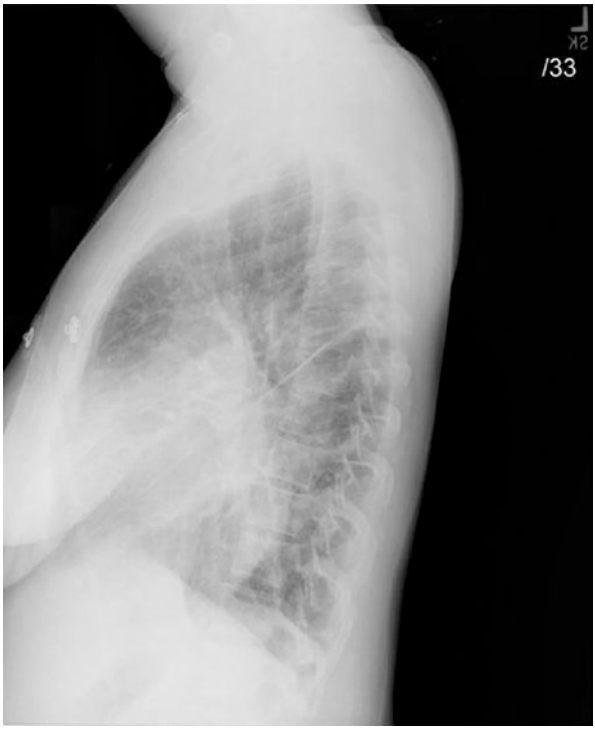


A. The endotracheal tube is too low, intermittently descending in the left mainstem bronchus causing left lower lobe collapse. Suggest retraction by a few centimeters.
B. The patient has a right lower lobe consolidation consistent with pneumonia.
C. There is pulmonary alveolar edema, an enlarged heart, and bilateral pleural effusions. Suggest diuresis.
D. There is a left-sided tension pneumothorax with deviation of the mediastinum to the right. Recommend urgent chest tube placement.

Answer: A. The endotracheal tube is too low, intermittently descending in the left mainstem bronchus causing left lower lobe collapse. Suggest retraction (pull tube back) by a few centimeters. 2 to 5 cm above the carina is ideal.

8. A 55-year-old female presents to the clinic for a preoperative evaluation for total knee arthroplasty. She has no complaints at this time. Her temperature is 97.9 degrees Fahrenheit, blood pressure is 132/78, pulse is 76/minute and, respirations are 15/minute with an oxygen saturation of 98% O2 on room air. Physical exam is notable for an early diastolic murmur with an opening snap. Her lungs are clear to auscultation, and she does not have lower extremity swelling. Her preoperative chest x-ray is seen below:



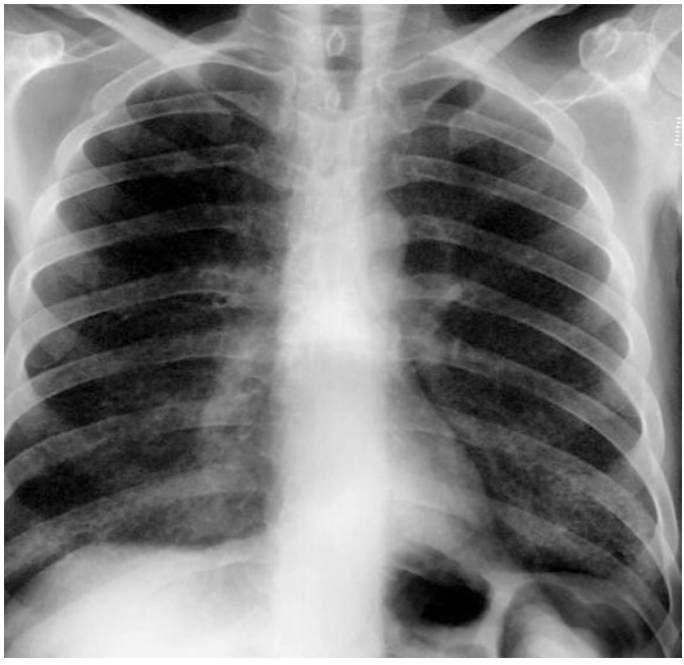

/33

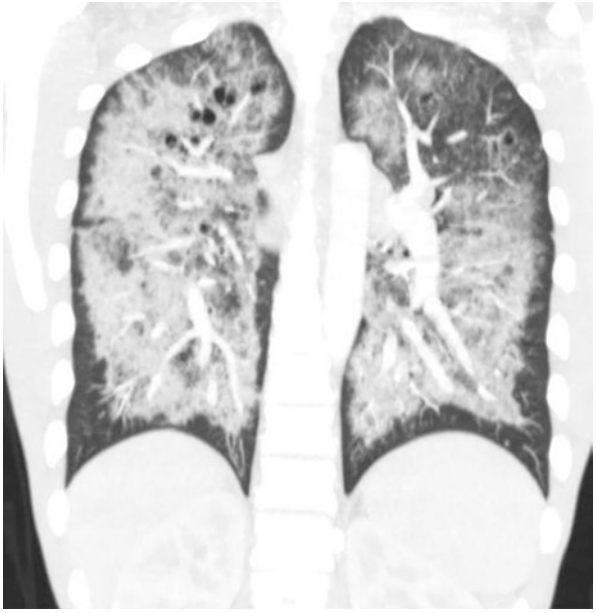
What is the radiographic finding?

A. Right atrial enlargement
B. Anterior mediastinal mass
C. **Left atrial enlargement**
D. Left ventricular enlargement

Answer: C. Left atrial enlargement

9. A 45-year-old male with HIV complains of one month of low-grade fever, cough, and dyspnea. He denies recent travel abroad and spelunking. He is unsure of his latest CD4 count. His temperature is 100.6 degrees Fahrenheit, blood pressure is 110/78, pulse is 76/minute, and respirations are 17/minute with an oxygen saturation of 90% O2 on room air. On physical exam, he has diffuse dullness to percussion and rhonchi bilaterally. The chest x-ray and follow-up CT (coronal view) are seen below:



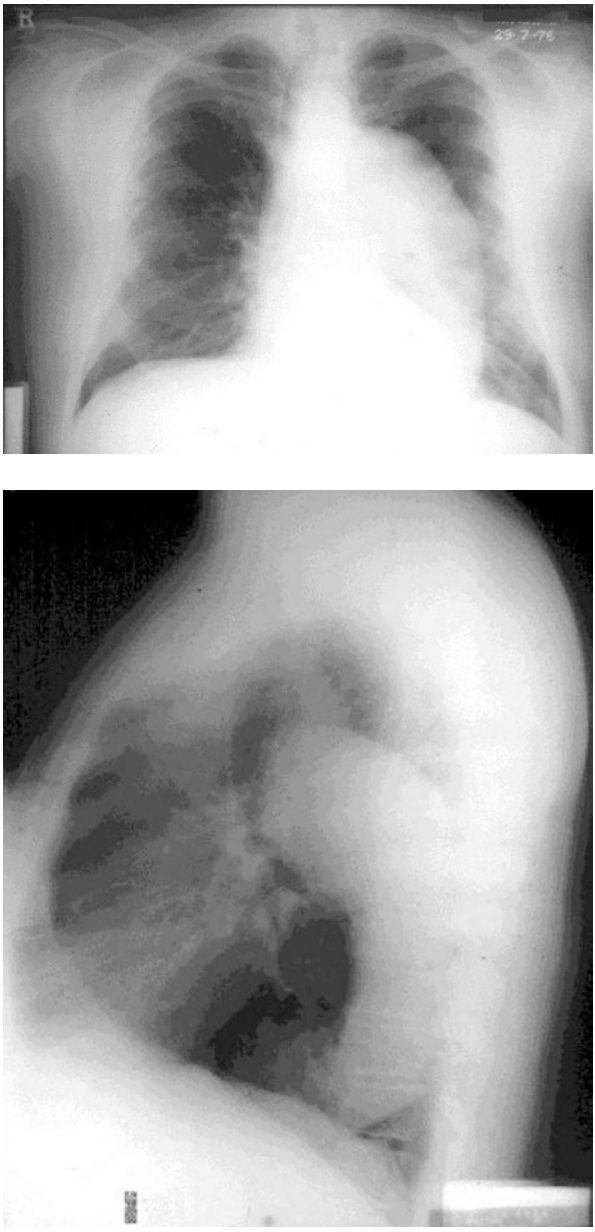


Results from bronchoalveolar lavage have returned and are as you suspected. What is the treatment?

A. Isoniazid, rifampin, ethambutol, and pyrazinamide
B. Itraconazole
C. Oncology consult and chemotherapy
D. Trimethoprim-sulfamethoxazole

Answer: D. Trimethoprim-sulfamethoxazole

10. A 65-year-old male with a past medical history of hypertension presents to his primary care provider for a routine follow-up visit. During this visit, he mentions that he has been having episodes of fleeting chest pain. His temperature is 100.6 degrees Fahrenheit, blood pressure is 110/78, pulse is 76/minute, and respirations are 17/minute with an oxygen saturation of 90% O2 on room air. Physical exam is within normal limits. His chest x-ray is seen below:



What condition should you rule out, and with which appropriate imaging modality?

A. Normal chest x-ray; no further imaging necessary
B. Hilar lymphadenopathy; CT chest with contrast

C. Aortic aneurysm or dissection; CT angiogram of the chest
D. Benign versus malignant posterior mediastinal mass; CT chest without contrast
E. Lobar collapse; CT chest with contrast

Answer: C. Aortic aneurysm or dissection; CT angiogram of the chest

3 Neuroradiology/Brain/CNS

1. A 20-year-old baseball player is hit on the side of the head by a baseball. He is stunned but seems otherwise fine until he loses consciousness 30 minutes later. What study should you order first?
   A. CT head
   B. MRI brain
   C. MRA head and neck
   D. Lumbar puncture

   Answer: A. CT head

2. A 59-year-old woman is found unresponsive. EMT achieves return of spontaneous circulation (ROSC), but she has a GCS of 3 and is intubated in the field. A CT head is ordered. What might you see in anoxic brain injury?
   A. No abnormality
   B. Intraparenchymal hemorrhage
   C. An acute MCA territory infarct
   D. Global cerebral edema

   Answer: D. Global cerebral edema

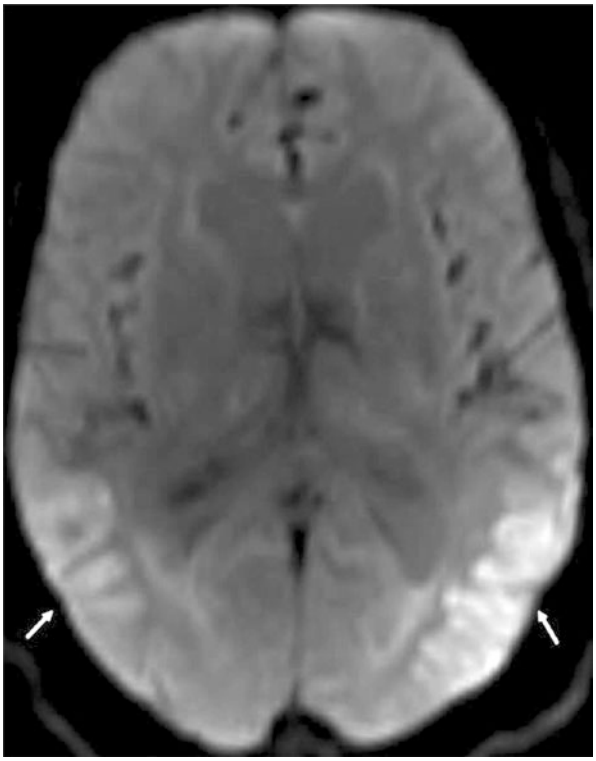
3. A 63-year-old man presents to his ophthalmologist for double vision. The ophthalmologist identifies a pupil-involving cranial nerve III palsy on physical exam and sends him directly to the ED. What pathology must be excluded emergently?
   A. A brain tumor
   B. A posterior communicating artery aneurysm
   C. Optic neuritis
   D. Subarachnoid hemorrhage

   Answer: B. A posterior communicating artery aneurysm

4. A 29-year-old man has a history of intravenous drug use and presents with back pain and fever. What diagnosis must be excluded first?
   A. Cord compression
   B. Urinary tract infection
   C. Osteomyelitis-discitis
   D. Transverse myelitis

   Answer: C. Osteomyelitis-discitis

5. A 67-year-old man presents after three months of rapid decline in metal status. What diagnosis is important not to miss with this history and pattern on DWI (Fig. 1)?



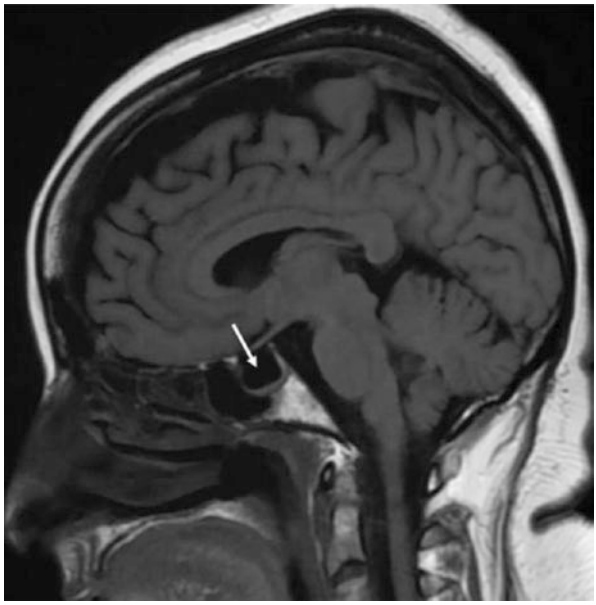
A. Creutzfeldt-Jakob disease
B. Alzheimer disease
C. Glioblastoma
D. Communicating hydrocephalus

Answer: A. Creutzfeldt-Jakob disease

6. A 32-year-old man from Ecuador presents after a first-time seizure. What infection etiology should be considered in the differential diagnosis?
   A. Brain abscess
   B. Neurocysticercosis
   C. Mesial temporal sclerosis
   D. Prion disease

Answer: B. Neurocysticercosis

7. A 33-year-old obese woman presents with headache and blurry vision. There was no mass on her MRI, but there was this sagittal midline image that showed a pituitary abnormality (Fig. 2).



What procedure would confirm her diagnosis?

A. Lumbar puncture with opening pressure
B. Craniotomy for biopsy
C. Myelogram
D. Intra-operative MRI

Answer: A. Lumbar puncture with opening pressure

8. A 27-year-old woman presents with new blurry vision and right arm weakness. What diagnosis should you consider in this patient population?
   A. Oligodendroglioma
   B. Meningioma
   C. Multiple sclerosis
   D. Mesial temporal sclerosis

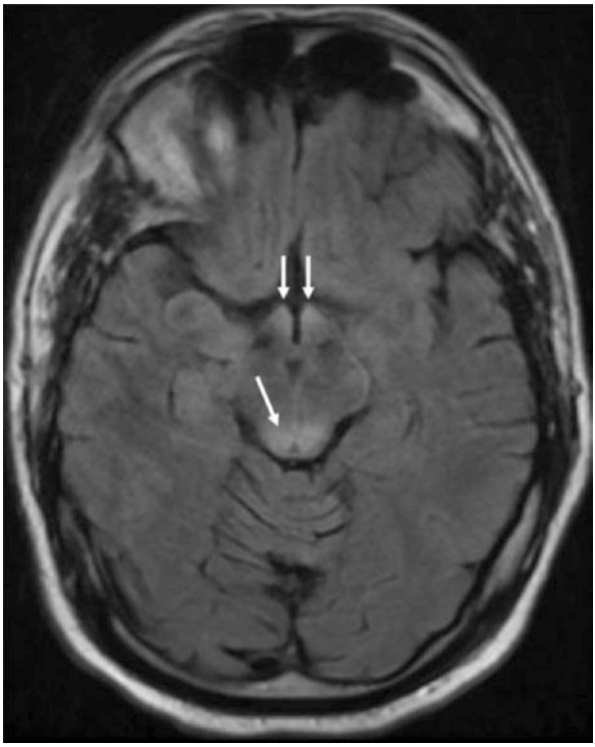
Answer: C. Multiple sclerosis

9. A 30-year-old man presents after a trip to Thailand where he experienced vision loss after a night of partying. The outside MRI report describes FLAIR hyperintense signal in the putamina. What toxin was in his drink?

A. Benzodiazepine
B. Barbiturate
C. Heroin
D. Methanol

Answer: D. Methanol

10. A 78-year-old homeless man was brought in by the police because he was walking strangely and was not making sense. Based on the FLAIR hyperintense signal in the mammillary bodies and periaqueductal gray matter on this MRI image, what medication does the patient need immediately (Fig. 3)?



A. Intravenous tissue plasminogen activator (tPA)
B. Thiamine
C. Vitamin B12
D. Normal saline

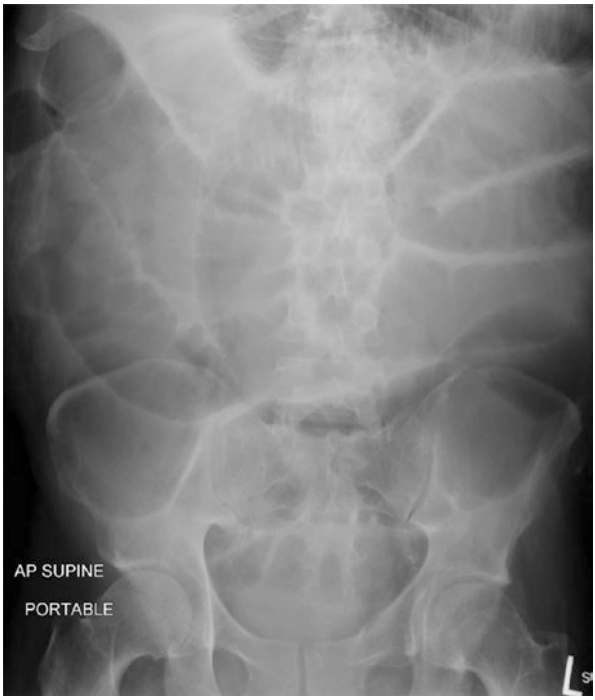
Answer: B. Thiamine

4 Abdomen

1. A 70-year-old woman undergoes a screening colonoscopy, which discovers a mass at the splenic flexure. Following biopsy, the diagnosis of adenocarcinoma is made. A CT of the chest, abdomen, and pelvis is ordered for staging. In addition to the splenic flexure mass and surrounding fat stranding, a few adjacent lymph nodes are identified. Which lymph node station would most likely be involved first?
   A. Left colic
   B. Middle colic
   C. Right colic
   D. Inferior mesenteric
   E. Superior mesenteric

Answer: A Left colic. Staging is an important aspect of everyday work of a radiologist. Patterns of spread are different for all types of cancer and are always related to anatomy. Lymphatic drainage usually follows venous drainage, as is the case for the splenic flexure which is preferentially drained by the left colic vein and nodes.

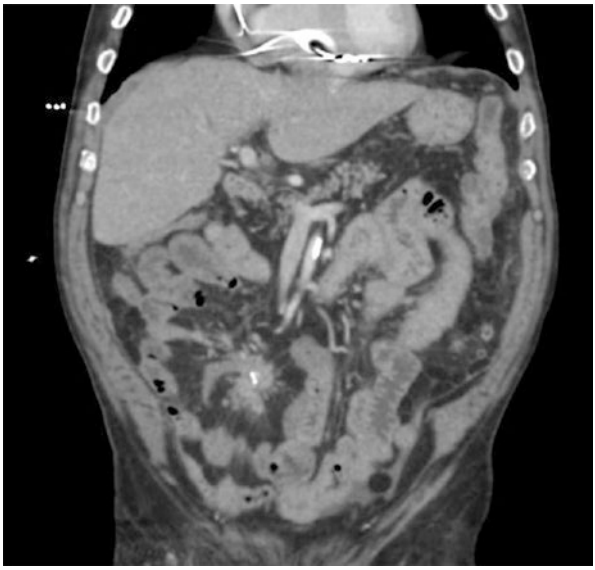
2. A 25-year-old male with a history of ulcerative colitis comes to the emergency complaining of abdominal pain and bloating. An abdominal radiograph is performed. What is the most likely diagnosis?



A. Ulcerative colitis flare
B. Small bowel obstruction
C. Large bowel obstruction
D. Toxic megacolon
E. Sigmoid volvulus

Answer: D. Toxic megacolon. In patients with inflammatory bowel disease, a diffuse dilation of the large bowel with thickened bowel walls ("thumbprinting") is caused by toxic megacolon.

3. A 50-year-old female presents with unexplained abdominal pain and weight loss for the past year. A non-contrast CT is performed, which reveals the following finding:

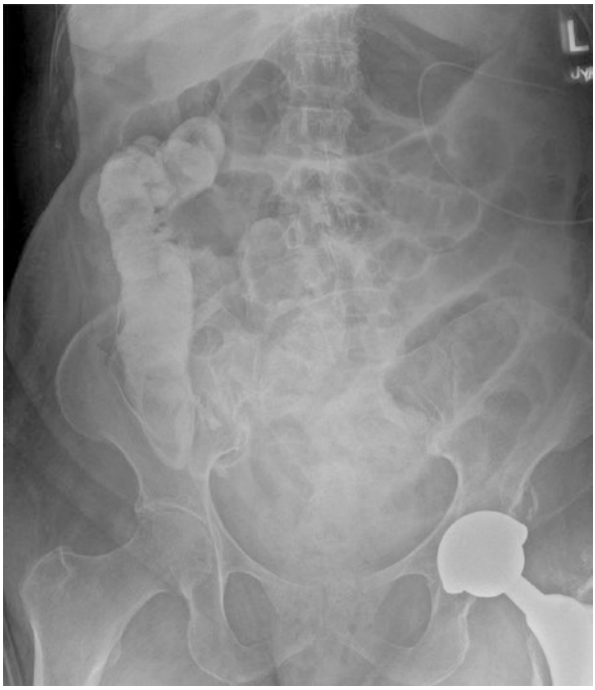


Which blood test would most likely be elevated?

A. Chromogranin A
B. Serum carcinoembryonic antigen
C. Synaptophysin
D. Serum CA-125
E. Ki-67

Answer: A. Chromogranin A. CT reveals a mesenteric mass with calcifications and a spoke-wheel pattern. These findings are suggestive of carcinoid. Initial testing can be performed by blood tests, which may reveal an elevated Chromogranin A. Synaptophysin would also be positive, but the analysis is performed on tissue. Obtained tissue is also evaluated for Ki-67, which is a cellular proliferation marker elevated in carcinoid. Serum CA-125 and CEA are classically elevated in ovarian and colon cancers, respectively.

4. A 75-year-old female with a history of recurrent diverticulitis arrives at the emergency room with severe abdominal pain. In addition to lab tests, an abdominal radiograph is ordered. The radiologist is not picking up the telephone, and your attending asks you about the radiograph below. What is your diagnosis?



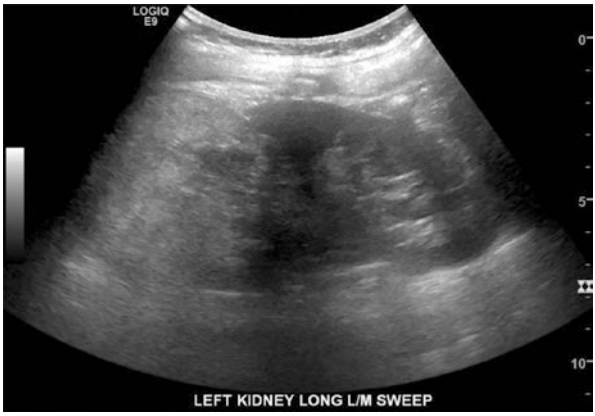
A. Small bowel obstruction
B. Large bowel obstruction
C. Acute diverticulitis
D. Complicated diverticulitis with abscess formation
E. Perforation

Answer: E. Perforation. This is an important diagnosis that should be made by anyone, not only radiologists. In this image, you can see the faint double wall (Rigler) sign, which shows air that encompasses a bowel loop from the inside and outside.

5. A 25-year-old male is admitted with abdominal pain. Due to a high clinical suspicion for appendicitis, a targeted ultrasound is ordered. As the radiology resident, you look at the images to prepare for your dictation. What is the best way to verify that you are looking at the appendix?
   A. Presence of appendicolith
   B. Periappendiceal fluid
   C. Target appearance in the transverse plane
   D. Blind-ended tubular structure
   E. Thickened wall

Answer: D. Blind-ended pouch**.** Identifying appendicitis on ultrasound is difficult and requires anatomical localization of the cecum, with a small blind-ended pouch. In practice, this is not always possible and therefore limits the use of the exam. The remaining options can be used as supportive evidence but cannot be used by themselves for definite identification.

6. A 65-year-old female came for a follow-up examination from the primary care provider due to known 6 cm fat-containing renal mass found on ultrasound below. The patient has not seen any specialists and would like to know what to do next. What should your recommendation be to the referring physician?

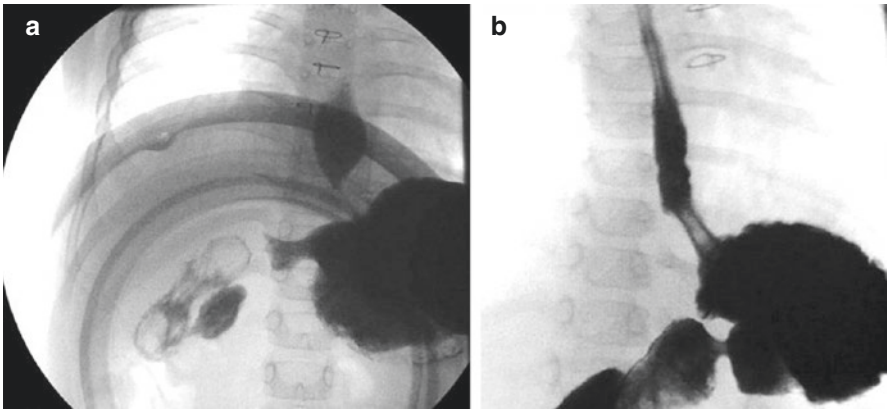


A. Benign. Further routine follow-up recommended
B. Benign. Urology and IR consultation recommended
C. Malignant. Urology consultation recommended
D. Unknown. Re-biopsy recommended
E. Unknown. CT or MRI recommended

Answer B. Urology and IR consultation are recommended. Given the fat content by history, the lesion is likely an angiomyolipoma (AML). Although a benign tumor, the size of the lesion in this case should lead to definitive treatment due to their propensity to cause hemorrhage. Coordination of management with a multidisciplinary team is usually the best choice. In this case, the urology consultation will commonly lead to a tumor board discussion, and possible embolization of the AML to reduce spontaneous or traumatic bleeding risk. Devascularization is faster than shrinkage, which may take years, but most shrinkage occurs in the first year. More angioid or myoid solid components mean more shrinkage, and pseudoaneurysms above 5mm or so equate to increased risk for bleeding. AML is usually sporadic but may be associated with tuberous sclerosis. By the way, in general terms, hematuria and a solid renal mass in a 65-year-old means that renal cell carcinoma must always be ruled out by imaging (CT or MRI). Biopsy may be required if no fat is found in the tumor on CT or MRI.

7. A 35-year-old female presents with repetitive vomiting and abdominal distention that was especially aggravated after the introduction of solid and semisolid

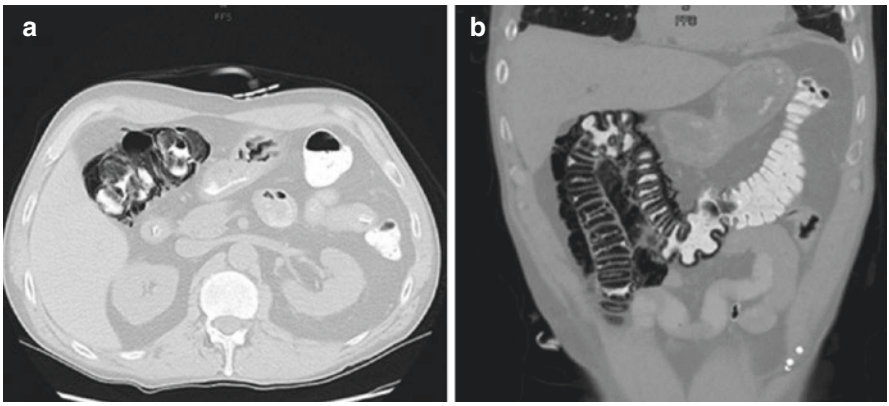
foods. At 5 months of age, the patient underwent surgery for ventricular septal defect, atrial septal defect, and patent ductus arteriosus and had subsequently been frequently hospitalized for respiratory infections and other viral infectious diseases. Findings of gastrointestinal barium study are shown below. What genetic abnormality is mostly likely present in this patient?



A. Trisomy of chromosome 15
B. Monosomy of the X chromosome
C. Trisomy of chromosome 21
D. Trisomy of chromosome 18
E. Genetic mutation of the CFTR protein

Answer: C. Trisomy of chromosome 21.

8. A 54-year-old man who had undergone bilateral sequential lung transplant for idiopathic pulmonary fibrosis was admitted to the hospital for further evaluation of an abnormal abdominal CT scan below. Three months previously, a gastrojejunostomy tube had been placed after he was found to have evidence of silent aspiration with oral intake. At a recent clinic visit, he denied abdominal pain or problems with the feeding tube. He described frequent diarrhea since placement of the feeding tube. What is the diagnosis?

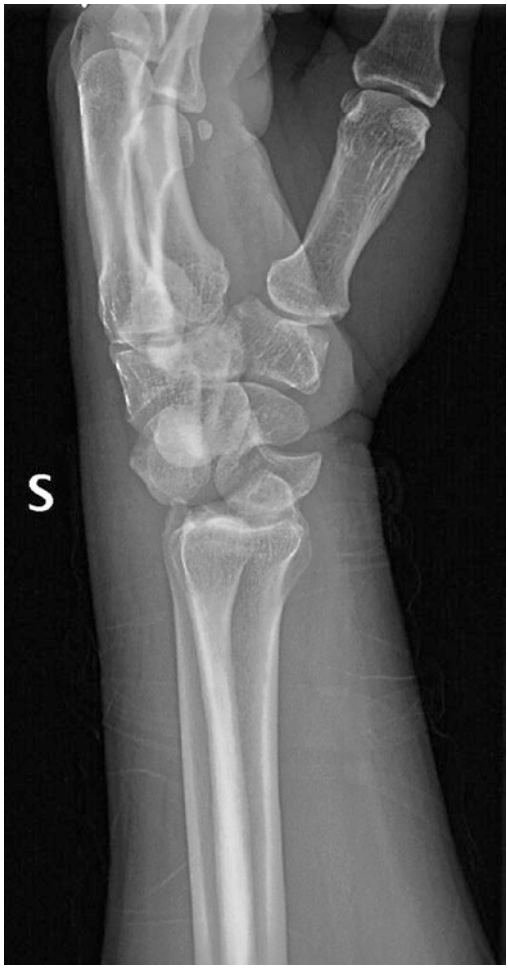


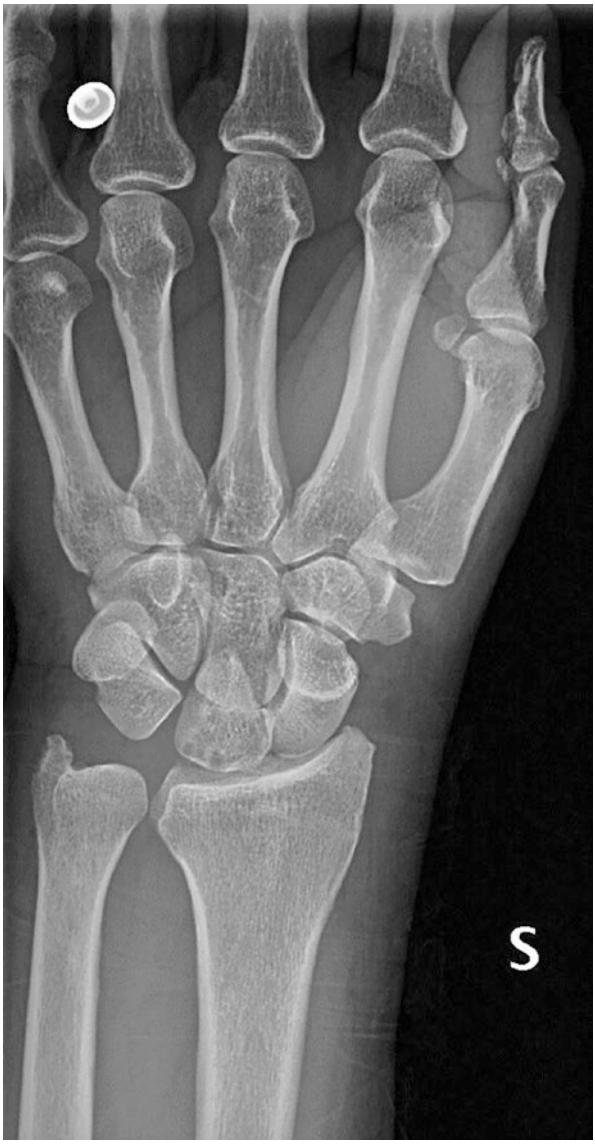
Mistrot, Daniel P., Vincent A. Gemma, Ronald A. Gagliano Jr, Ashraf Omar, and Tanmay S. Panchabhai. "A 54-Year-Old Man Presenting With an Abnormal Abdominal CT Scan 8 Months After Double Lung Transplant." Chest 149, no. 5 (2016): e151-e155

A. Adenomas
B. Colitis cystica profunda
C. Lipoma
D. Leiomyoma
E. Pneumatosis intestinalis

Answer: E. Pneumatosis intestinalis. Gas is clearly seen in or surrounding the bowel wall. This can be an innocuous finding (COPD, steroids) however may also indicate a surgical emergency (intestinal ischemia / infarction).

5 Bone and Musculoskeletal

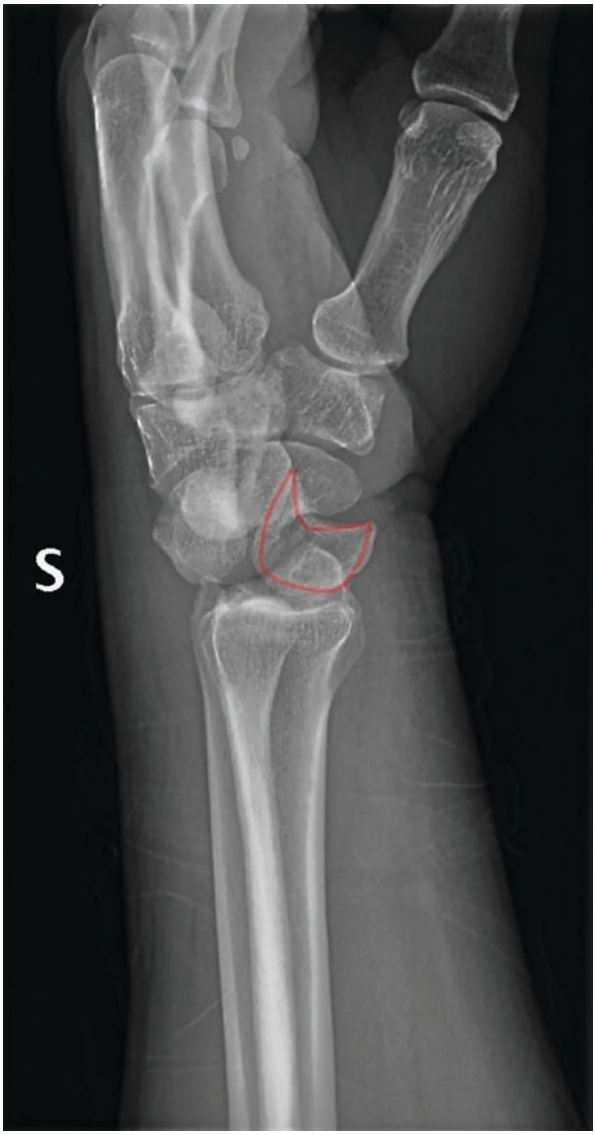


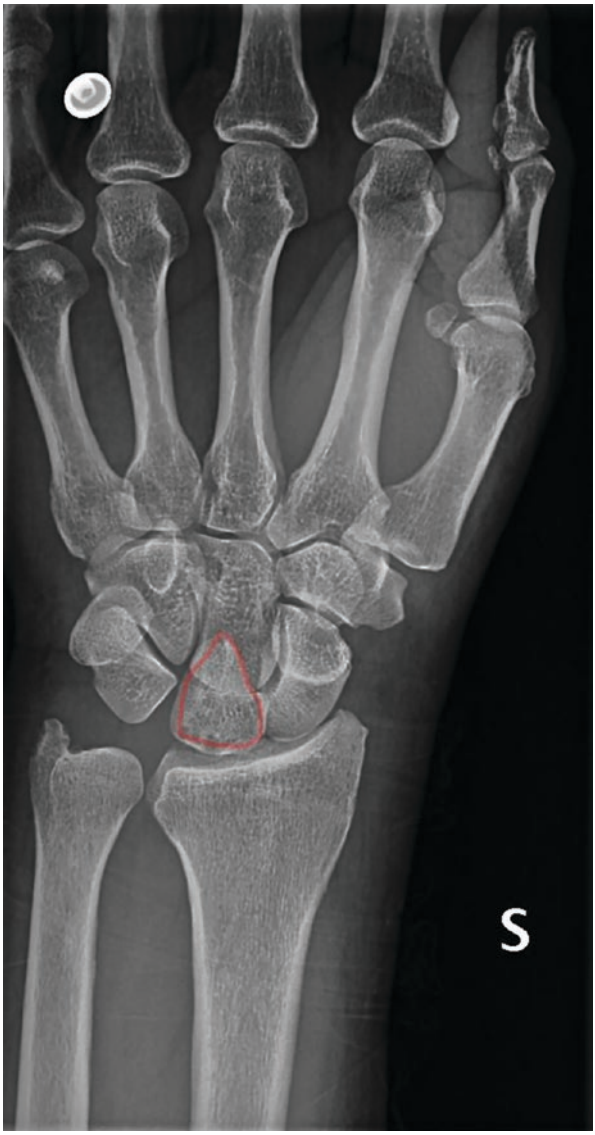


A 49-year-old presented to the emergency department because of a with wrist pain following a fall from a 10-feet high ladder. Physical examination revealed prominent volar wrist swelling and partial flexion of the fingers. Tactile sensibility was mildly diminished in the left thumb and the index and long fingers. An x-ray of the wrist in antero-posterior (AP) and lateral (LL) view are shown. Which of the following is the correct diagnosis?

A. Perilunate dislocation
B. Lunate dislocation

C. Midcarpal dislocation
D. Scaphoid fracture
E. Scapholunate dissociation





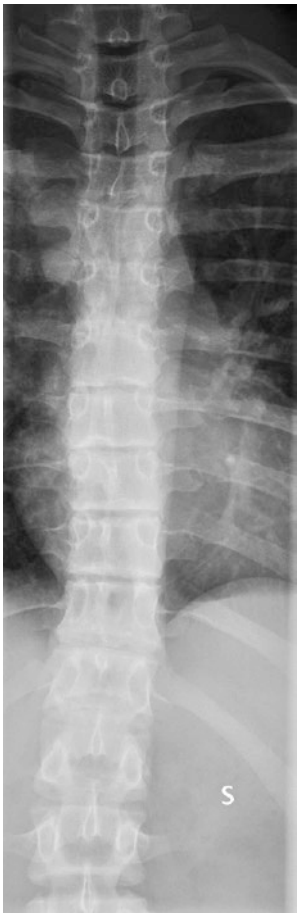
Answer: B Lunate dislocation

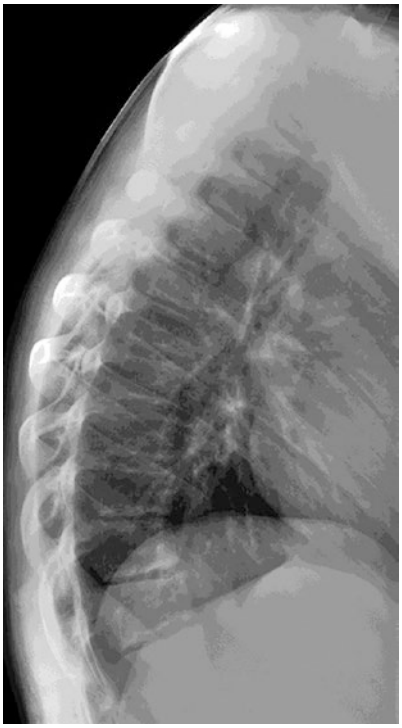
Mechanism – Typically occurs in young adults with high energy trauma (e.g., motor-vehicle injury) resulting in loading of a dorsiflexed wrist.

Clinical presentation - Volar wrist swelling is typically prominent. Patients often hold their fingers in partial flexion because of the pain on extension. The swelling often causes a decrease in 2-point discrimination in the median nerve distribution caused by acute carpal tunnel syndrome.

Classification - Mayfield classification of carpal ligamentous injuries and carpal dislocations consists of four stages.

Radiographic features - In lunate dislocations (stage IV), the lunate's articulations with both the radius and capitate are disrupted displacing and angulating the lunate volarly creating a so-called "spilled teacup" sign, seen on the lateral view radiograph. On AP view of the wrist, lunate overlaps the capitate and appears triangular producing the "piece-of-pie" sign.





A 16-year-old boy was brought to the office because of a 4-year history of kyphotic deformity in the middle back and subacute mid-low back pain. His pain aggravated by activity and improved with rest. He also complained of limited thoracolumbar activity. No neurological deficit was observed. There was a family history of similar characteristics in the patient's father and sister. An x-ray of the thoracic spine in antero-posterior (AP) and lateral (LL) view are shown above. Which of the following is the most likely diagnosis?

A. Postural kyphosis
B. Ankylosing spondylitis
C. Scheuermann disease
D. Paget's disease
E. Psoriatic arthritis

Answer: C. Scheuermann disease (SD) – juvenile kyphosis or juvenile discogenic disease or vertebral epiphysitis. It is a condition of rigid hyperkyphosis that involves the vertebral bodies and discs of the thoracic or thoracolumbar spine.

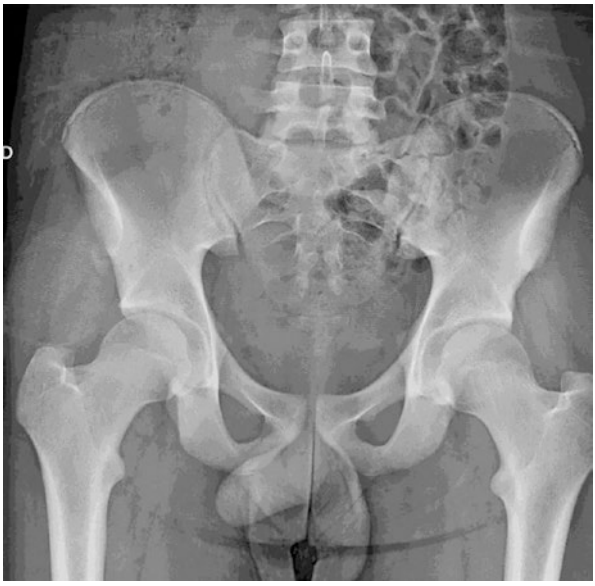
Mechanism – a proposed mechanism is the avascular necrosis of the vertebral ring apophysis. However, a hereditary component is understood to contribute to this condition's development.

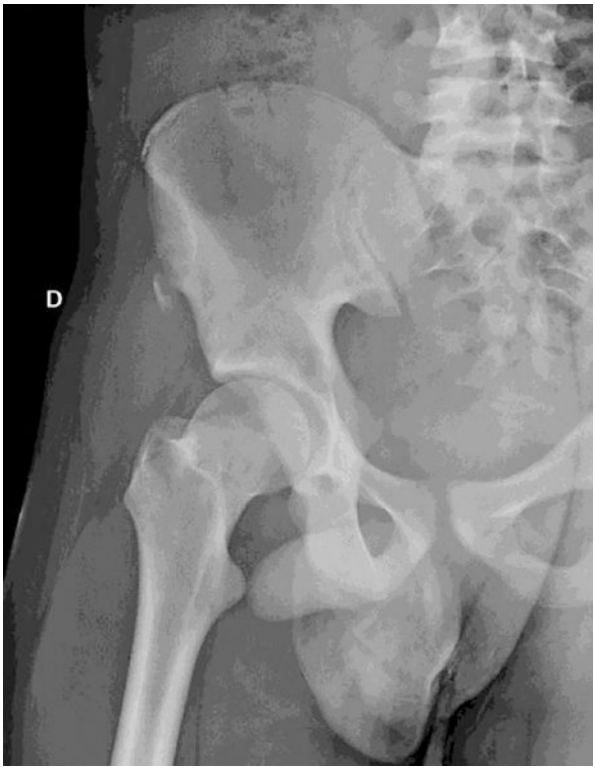
Clinical presentation – most commonly diagnosed in adolescents (range 12–17 years). The adolescent will present with postural deformity ("hunchbacked" appearance) and/or subacute thoracic pain. The deformity is usually recognized in the teenage years by child or parents or on a school screening exam. The pain is subacute with no clear history of trauma or other inciting event. The pain gets worse with activity and improves with rest.

Classification – Sørensen diagnostic criteria for SD including wedging over an angle of 5° in three or more vertebrae, kyphosis over 40° in sagittal plane, and irregularities in vertebral endplates were considered typical.

Radiographic features – Antero-posterior (AP) and lateral radiographs. Diagnostic criteria include determining:

1. Degree of kyphosis on lateral imaging (Cobb angle)
2. Degree of anterior wedging on lateral imaging (wedge angle)
3. Associated findings noted on AP/lateral radiographs (irregular vertebral endplates; Schmorl nodes; loss of disc space height)





A 15-year-old male patient is brought to the emergency department because of instant pain in his anterior right thigh and anterior pelvic region after an injury sustained during a football match. He was about to kick a football, but he missed the shot. He was unable to walk, and he had to crawl off the football pitch. On examination his gait was affected by pain in the right thigh, and there was tenderness to the anterior superior iliac spine on palpation. An x-ray of the hip is shown. Which of the following structures is attached to the fractured anterior superior iliac spine?

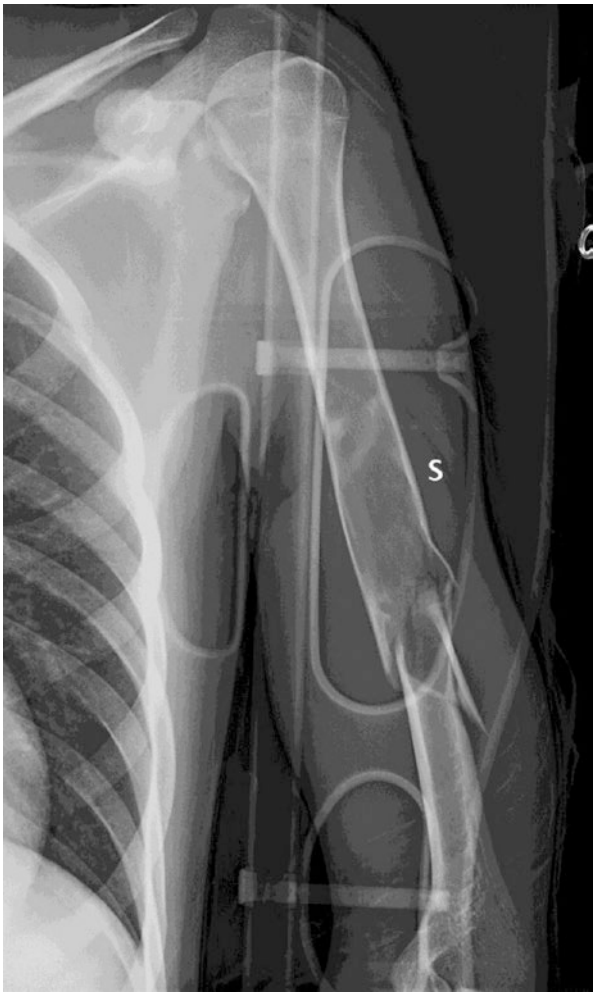
A. Rectus femoris muscle
B. Abdominal external oblique muscle
C. Iliopsoas muscle
D. Sartorius muscle and tensor fasciae latae
E. Gluteus minimus muscle
F. Inguinal ligament

Answer: D. Sartorius muscle and tensor fasciae latae
***Diagnosis* – Avulsion fracture of the anterior superior iliac spine**

Mechanism – indirect trauma caused by sudden vigorous contraction or repetitive contraction of the sartorius and tensor fasciae latae muscles resulting in avulsion fracture of the growing apophyses in adolescents. Forceful contraction or sudden repetitive actions of these muscles occur when running or kicking a ball.

Clinical presentation – complain of pain and weakness; may be confused or misdiagnosed as an acute muscle strain in case of poor physical examination. All hip movements are usually limited by pain.

Radiographic features – displaced fractures usually can be seen on radiographs. They may be missed due to location and small size of bony fragments. In some cases, CT or MRI can be obtained to confirm the diagnosis.



A 20-year-old male patient is brought to the emergency department because of shoulder pain, deformity, and swelling after a minor trauma. An x-ray of the

shoulder shows oblique fracture of the middle third of humeral diaphysis with smaller comminuted fragments of thin cortical bone. There is also a well-defined lesion arising centrally in the medullary cavity which extends from the proximal humeral physis into the distal third of the humerus. Cortical thinning and areas of varying density are appreciated. There is no associated periosteal reaction or soft-tissue mass. Which of the following is the most appropriate additional imaging study that provides a road map for intervention?

A. MRI
B. PET scan
C. CT scan
D. Ultrasonography
E. Radionuclide bone scan

Answer: C. CT scan

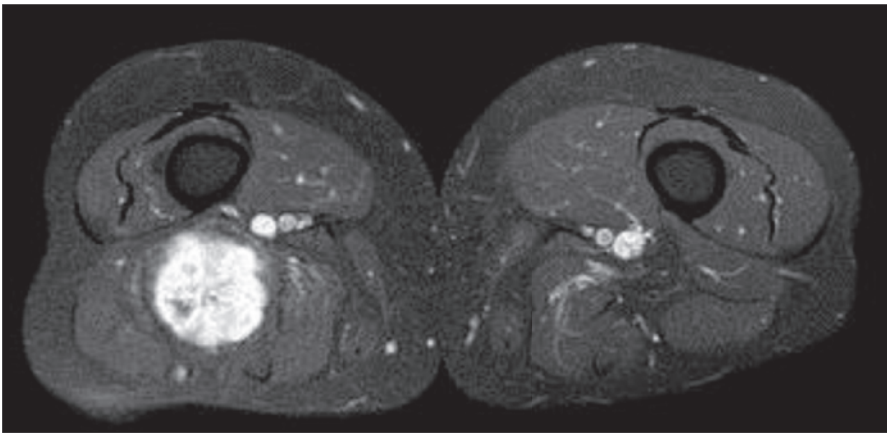
***Diagnosis* – Unicameral bone cyst**

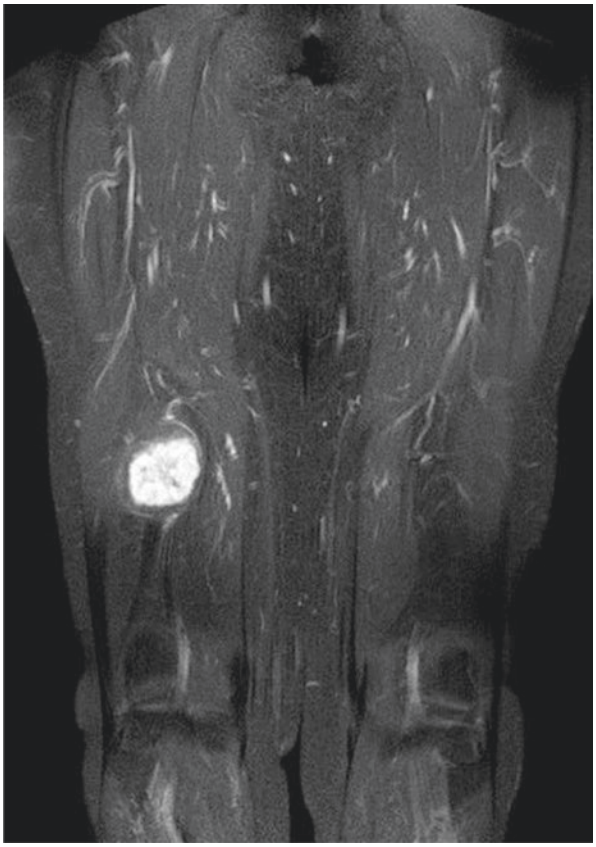
Mechanism – simple or solitary bone cysts are benign fluid-filled cavities that enlarge over time, resulting in thinning of the bone. The proximal humerus and femur account for almost 90% of these cases.

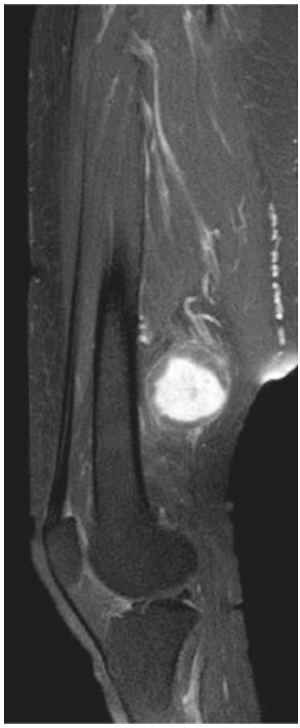
Clinical presentation – the majority of the patients will not have any symptoms unless a pathologic gross fracture or nondisplaced stress fracture occurs. In patients with no symptoms, the lesion may be an incidental finding. These lesions are classified as active when they are within 1 cm of the physis and become latent as they advance to a diaphyseal location.

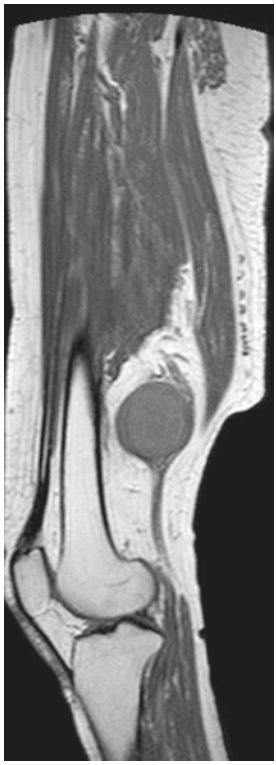
Imaging features

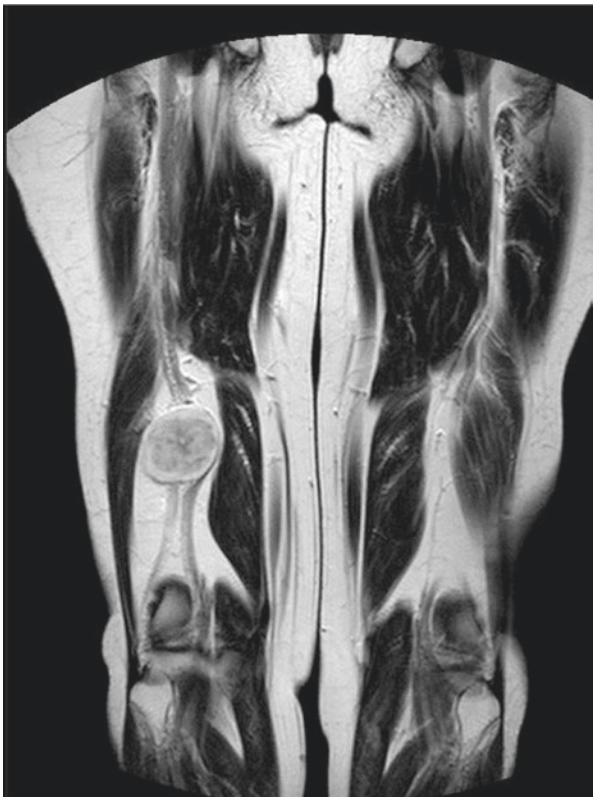
1. **Plain radiography** – is modality of choice and has a high diagnostic accuracy. In the presence of fracture, the "fallen fragment "sign is noted. The fractured fragment moves in the dependent position and changes its position with change in the patient's posture. Likewise, "rising bubble sign" (gas bubbles upward migration) has been described for unicameral cyst. This sign is considered as pathognomonic and is also recommended to follow the case post-treatment.
2. **CT –** to evaluate cyst present in areas difficult to assess on plain x-ray. It demonstrates more accurately the extent of a cyst. CT also helps to differentiate cyst from a lipoma which is difficult to assess on plain x-ray. Fluid to fluid level can be observed. "Fallen fragment" and "rising bubble sign" are also seen on CT. CT also provides a road map for intervention.
3. **MRI –** is the modality to further elaborate on the aggressive features, such as fracture, local expansion, cortical thinning, erosions, and significant damage.
4. **Bone scintigraphy/PET** – they have no significant role in diagnostic workup.

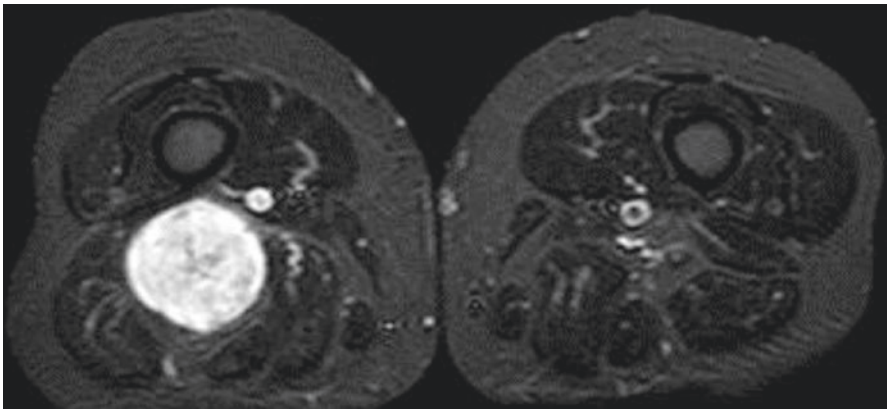


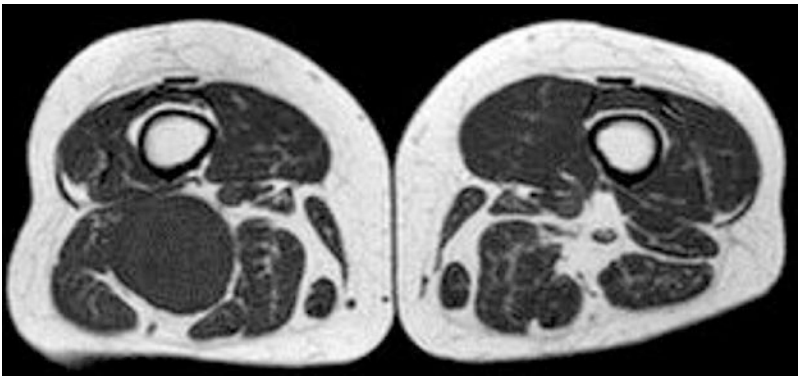






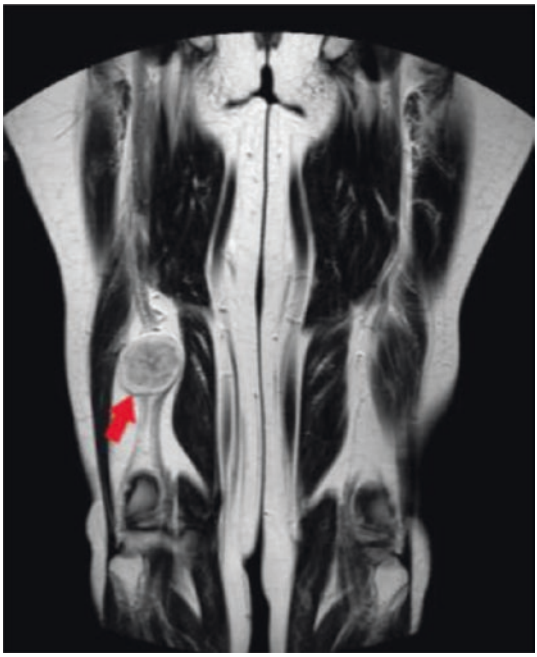


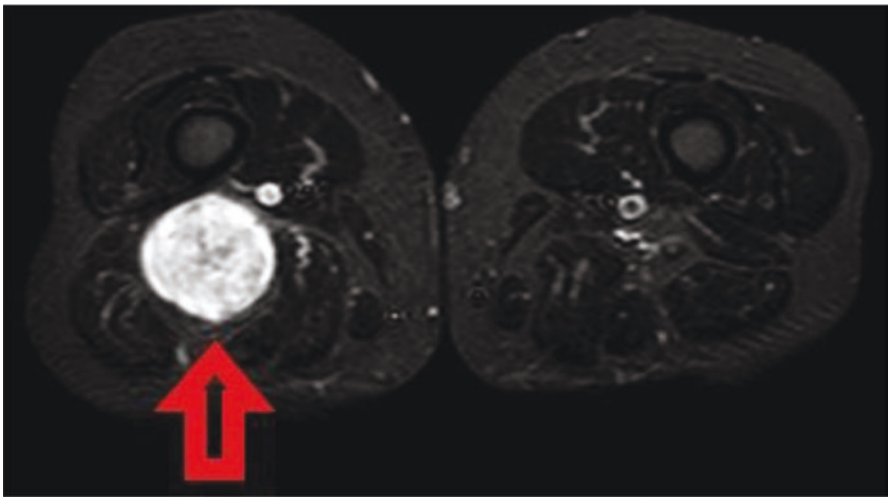


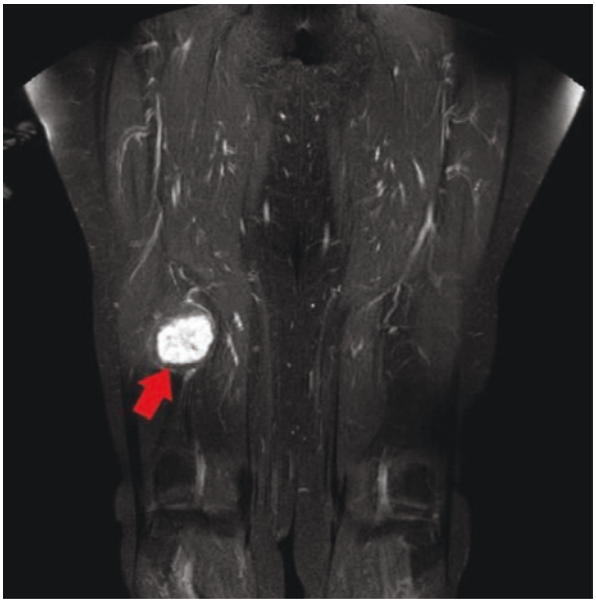


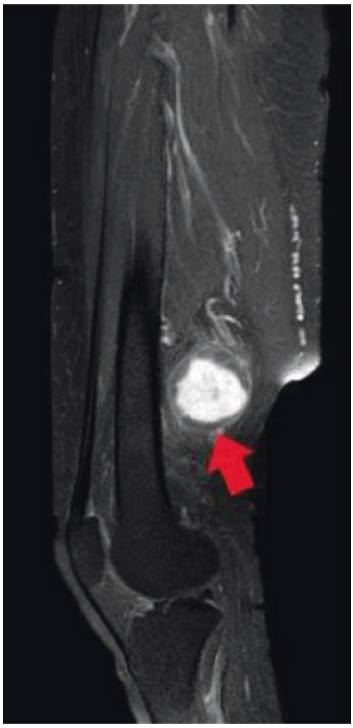
A 63-year-old woman comes to the office because she had a 2-year history of a slow-growing painful palpable mass at the medial right posterior thigh. On physical examination, a painful isolated soft-tissue mass was detected which also elicited a shooting pain down the leg. No motor deficit was detected on neurological examination. Tinel sign was positive on the posterior aspect of the right thigh. MRI of the right thigh in coronal (A), axial, (B) and sagittal (C) planes are shown. Which of the following is the most likely diagnosis?

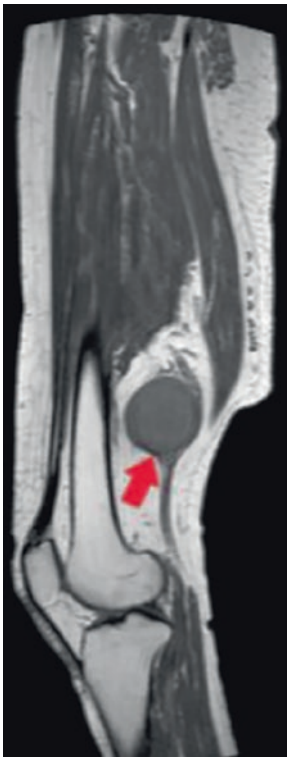
A. Neurofibroma
B. Rhabdomyosarcoma
C. Femoral artery aneurysm
D. Lymphoma
E. Sciatic nerve schwannoma

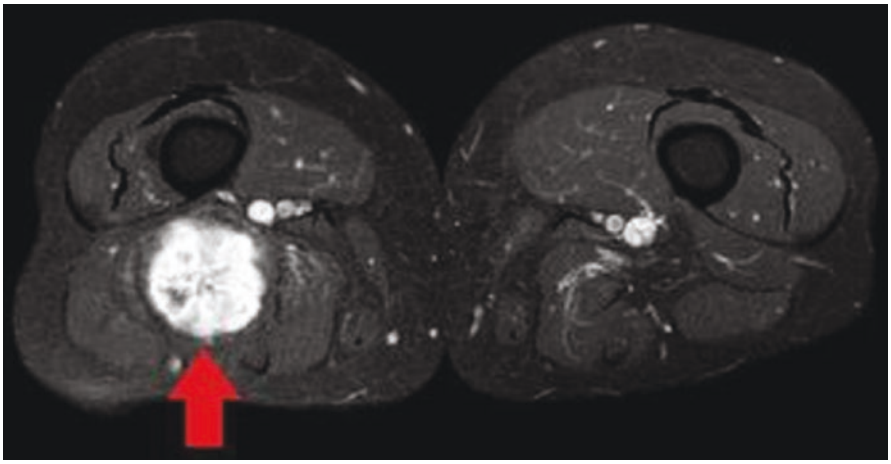


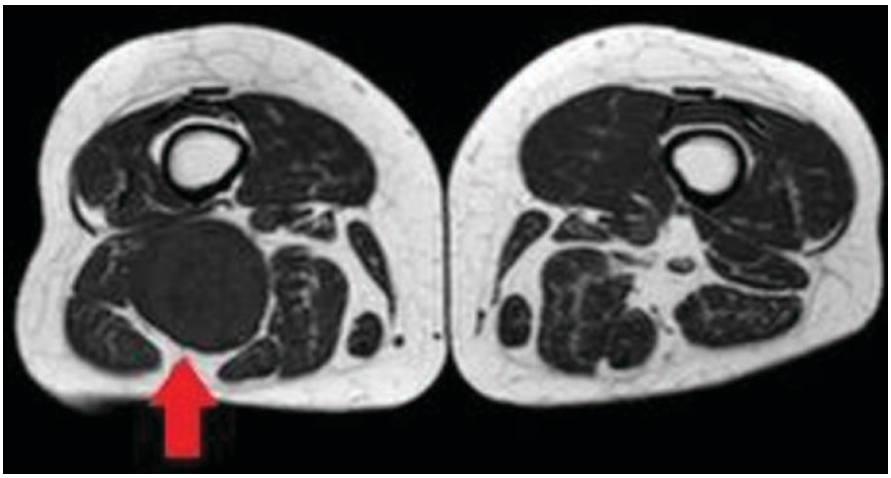












Answer: E. Sciatic nerve schwannoma

This is the most common benign peripheral nerve sheath tumor composed of Schwann cells. However, schwannomas in the sciatic nerve are rare and typically present as a painful palpable mass in the thigh. Schwannomas are slow-growing tumors and can achieve large size in time. They are usually solitary, well circumscribed, encapsulated, and eccentrically located on nerve roots.

Clinical presentation – direct compression from large lesions (>40 mm) can present with symptoms similar to those of sciatica.

Imaging features –

MRI – the diagnoses of sciatic nerve-associated schwannomas are typically made with MRI, often subsequent to a normal lumbar spine MRI showing no signs of disc herniation. MRI demonstrates a well-defined, homogeneous fusiform mass arising from the sciatic nerve, which is isointense to muscle on T1, hyperintense on T2, and enhancing after gadolinium administration.

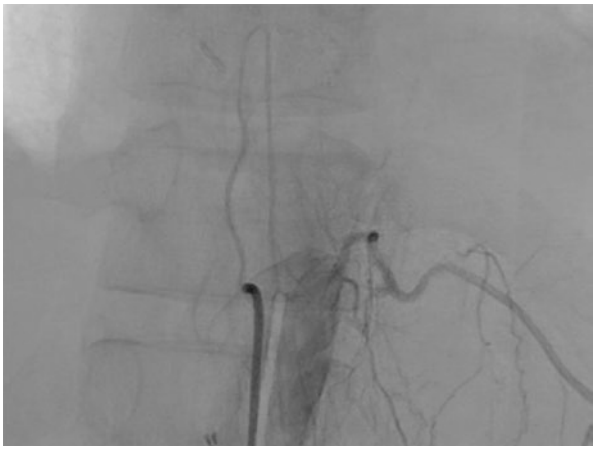
6 Vascular/Interventional Radiology

1. Which of these techniques does not use a predominantly thermal source as an ablative method?
   A. Cryoablation
   B. Microwave ablation
   C. Irreversible electroporation
   D. Radiofrequency ablation
   E. All the above

   Answer: C

2. Conventional TACE is:
   A. Injection of a chemotherapeutic drug combined with an embolic agent (oil, gelfoam, or embolic microsphere/bead) in the hepatic artery
   B. Injection of a chemotherapeutic drug alone in the portal vein
   C. Injection of a chemotherapeutic drug alone in the right hepatic vein
   D. Injection of radioactive particles in the portal vein
   E. Injection of embolic particles in the hepatic artery

   Answer: A



3. This midline vessel seen on this angiogram (spinal artery of Adamkiewicz) takes a "hairpin" turn, and one should identify its location in:
   A. Bronchial artery embolization
   B. Upper lumbar artery embolization
   C. Lower thoracic subcostal artery embolization
   D. Typically originates at T9–T12 on the left
   E. Occlusion or thrombus can cause anterior cord syndrome/corticospinal and spinothalamic tracts
   F. All of above

   Answer: F

4. Signs of pulmonary embolism include:
   A. Elevated right heart pressure on echocardiogram
   B. S1Q3T3 on ECG
   C. Elevated D-dimer
   D. Ventilation perfusion scan mismatch with perfusion defect
   E. Westermarck sign
   F. Hampton's Hump
   G. All are correct

   Answer: G

5. IR is the newest approved independent training residency in the USA and uses spatial awareness, hand to eye coordination, and novel navigation and drug plus device applications (true or false).

   Answer: True. It also allows you to apply your video game skills to good use.

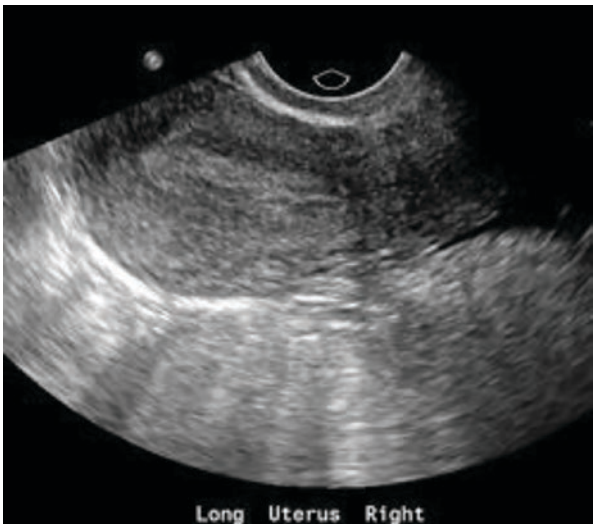
7 Women's Imaging

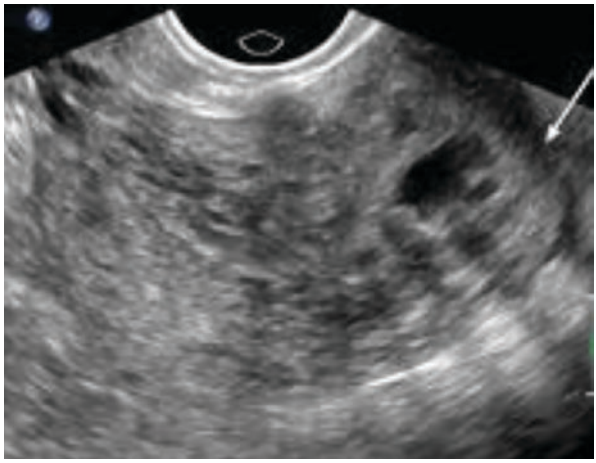
7.1 Case 1

A 25-year-old female presents with pelvic pain and abnormal vaginal bleeding. Urine pregnancy test was positive.

1. What should be the first imaging test?
   A. CT abdomen/pelvis
   B. US pelvis transabdominal
   C. US pelvis transabdominal and transvaginal
   D. MRI pelvis

   Answer: C
   Pelvic ultrasound demonstrates the following:



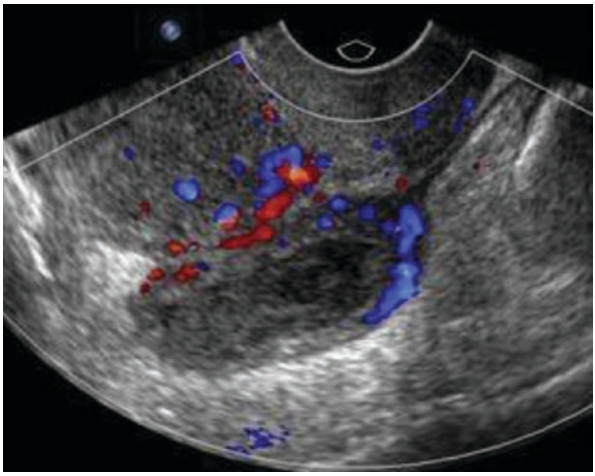


2. Which of the following would be the most likely diagnosis?
   A. Trophoblastic gestation (molar pregnancy)
   B. Ectopic pregnancy
   C. Ovarian torsion
   D. False-positive urine pregnancy test

Answer: B

7.2 Case 2

A 23-year-old female with pelvic pain, fever, dyspareunia, and vaginal discharge. Pelvic ultrasound demonstrates the following finding.



Which of the following would be most likely diagnosis?

A. PID/tubo-ovarian abscess
B. Ovarian torsion
C. Normal ovary
D. Hemorrhagic cyst

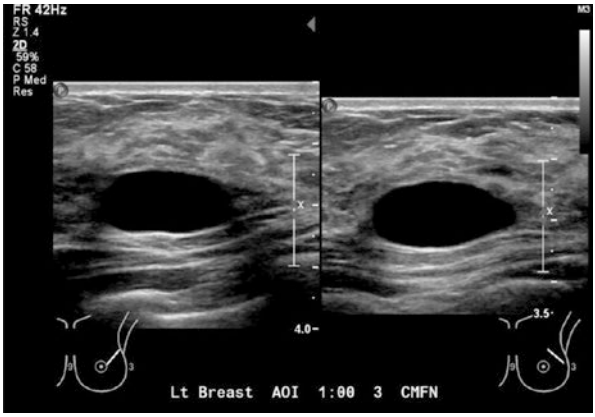
Answer: A

7.3 Case 3

A 25-year-old female presents with a palpable abnormality in the right breast for the past 2 months.

1. What is the initial imaging study of choice?
   A. Screening mammogram
   B. Diagnostic mammogram
   C. Diagnostic ultrasound
   D. Screening ultrasound

   Answer: C
   Ultrasound shows the following:

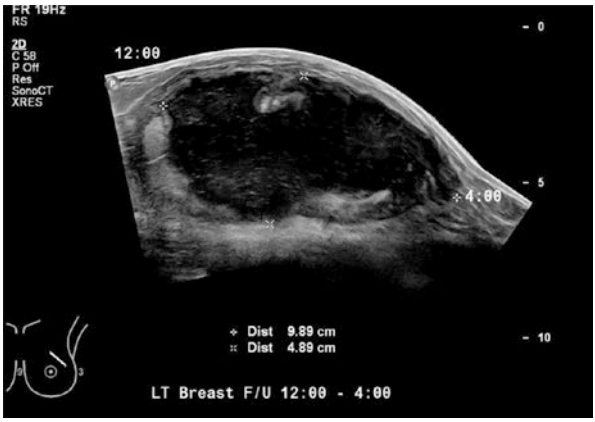


2. What is the most likely diagnosis?
   A. Simple cyst
   B. Hematoma
   C. Fibroadenoma
   D. Abscess

   Answer: A

7.4 Case 4

A 45-year-old female with recent history of mastitis presents with a palpable lump in the left breast. Ultrasound shows the following:



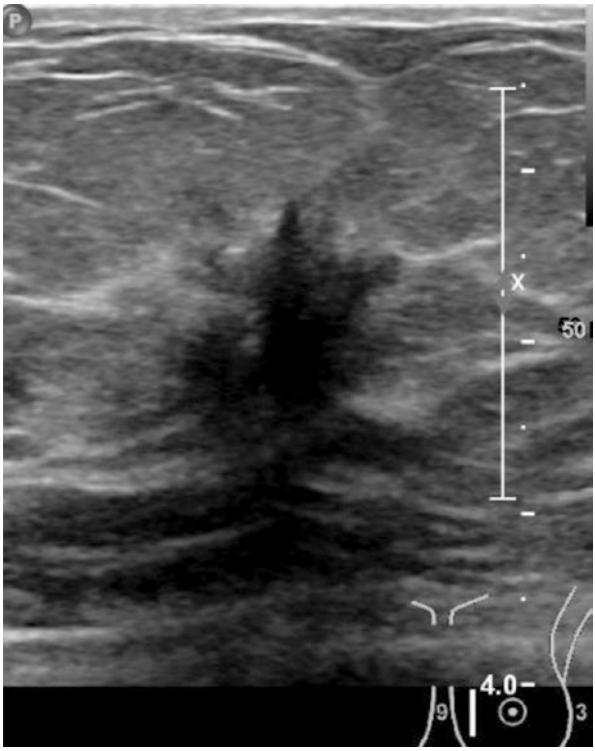
What is the most likely diagnosis?

A. Simple cyst
B. Hematoma
C. Fibroadenoma
D. Abscess

Answer: D

7.5 Case 5

A 55-year-old female is called back for a left breast mass seen on a screening mammogram. Diagnostic ultrasound shows the following finding in the left breast, at 5 o'clock 5 centimeters from the nipple:



What is the BI-RADS category and management of this finding?

A. BI-RADS 0, needs additional imaging evaluation
B. BI-RADS 1, return to routine annual screening
C. BI-RADS 2, return to routine annual screening
D. BI-RADS 4, biopsy

Answer: D

8 Clinical Tips

1. What's the gold standard imaging test for a child with suspected pneumonia:
   A. None: no imaging test is needed to confirm pneumonia.
   B. None: chest x-rays would be the best examination, but children cannot be irradiated.
   C. Chest MRI because it enables optimal lung visualization.
   D. Chest x-rays in one projection since it is associated with a very low radiation dose.
   E. Chest CT because it enables optimal lung and mediastinum visualization.

Answer: D

2. Which imaging tests are more demanding with respect to patient collaboration?
   A. MR and ultrasound.
   B. MR and CT.
   C. CT and X-ray.
   D. Ultrasound and x-ray.
   E. None: imaging tests don't require compliance from the patient.

   Answer: A

3. Which imaging test would you order in an overweight patient with a suspicion of pancreatitis?
   A. Sonography: pancreas can be perfectly seen with ultrasounds, and adipose tissue doesn't interfere.
   B. Contrast-enhanced CT, but only within 5 hours from the onset of symptoms.
   C. Abdominal MRI because it has the best contrast resolution.
   D. Abdominal plain x-ray.
   E. Contrast-enhanced CT, but only after >12 hours from the onset of symptoms**.**

   Answer: E

4. How should you formulate your clinical question:
   A. I should write all the patient's symptoms using his/her own words.
   B. I should report all the patient's symptoms, but using only medical terms.
   C. I have to select the most important symptom.
   D. It is helpful to suggest one/two clinical hypotheses (diseases).
   E. I don't have to: images should be interpreted without clinical information.

   Answer: D

5. Which kind of pathologies can be detected with imaging tests?
   A. Every sort of pathology
   B. Only those that involve macroscopic morphological changes of a certain magnitude
   C. Only malignant tumors
   D. Only tumors
   E. Only inflammation and tumors

   Answer: B

6. Which imaging test would you order after a cranial trauma?
   A. Ultrasound to verify the presence of intracranial hemorrhage
   B. MRI because it is fast and accurate
   C. CT because it is fast and has the best accuracy for early bleeding and fracture
   D. X-rays to see fractures
   E. Both x-rays and US

   Answer: C

7. Comparing contrast-enhanced CT and MRI in the abdomen:
   A. CT is faster and has higher spatial resolution, but lower contrast resolution.
   B. There is no difference.
   C. MRI is faster and has higher spatial resolution, but lower contrast resolution.
   D. MRI involves a higher radiation dose.
   E. CT is the best option in children.

Answer: A

8. What is the rationale for using contrast injection in CT or MRI?
   A. It is not helpful.
   B. Contrast medium enhances pathologic tissue because of higher metabolic activity when compared to normal tissue.
   C. Contrast medium highlights pathologic tissue because it has different vascular properties when compared to normal tissue.
   D. Contrast medium enhances only tumors because neoplasms are always more vascularized than normal tissues.
   E. MRI rarely needs contrast medium injection.

Answer: C

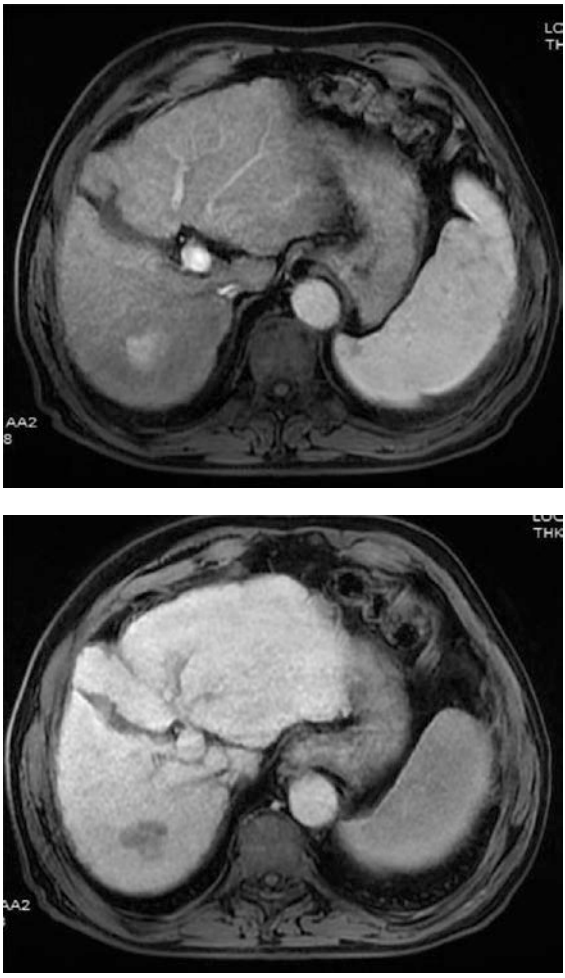
9. What is the imaging gold standard in an adult with a pulmonary disease?
   A. MRI enables an optimal lung visualization, even with motion.
   B. Ultrasound is the imaging test with best spatial resolution.
   C. Chest x-ray allows detection of the smallest lung pathology early.
   D. MRI and CT are equally accurate.
   E. CT is the test with the highest accuracy in the chest and lung.

Answer: E

10. What is the most risky contraindication to an MR examination?
    A. 1-year-old tattoo
    B. 1-year-old orthopedic metal prosthesis
    C. 39 weeks pregnancy
    D. 1-year-old pacemaker
    E. None

Answer: D

11. 67 y.o. with HCC on a cirrhotic background (Figs. 1a and 1b)



This is a liver imaging test showing a typical HCC lesion in Segments 6/7, with a non-rim enhancement in the arterial phase (bright when aorta is bright) and "wash-out" on delayed phase (gets darker fast). Aorta is bright in arterial phase, and portal vein is bright in portal venous phase. If both are bright, then the image is in between these phases.

12. Which imaging test is depicted in Fig. 1 from question 11? (look at color of the vertebra before answering)? [Needs figure]
    A. X-rays
    B. Post-contrast MRI
    C. Post-contrast CT
    D. US

Answer: B

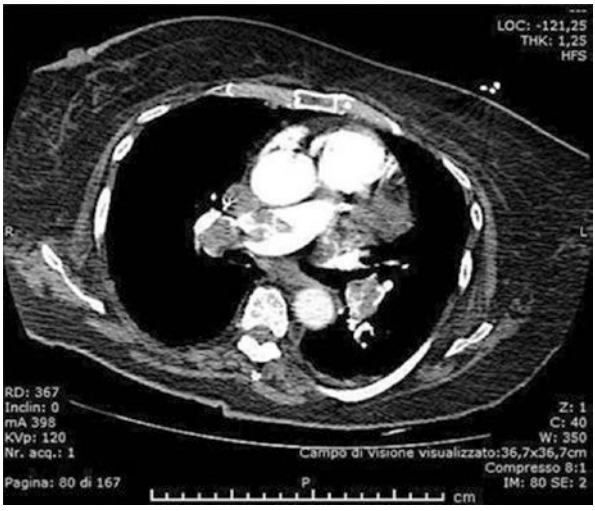
13. What is the indication for liver MRI in a cirrhotic patient?
    A. MRI should be done every 3 months in every cirrhotic patient.
    B. MRI is a first-level examination and should be done every month in a patient who has cirrhosis.
    C. MRI is a first level examination and cirrhotic, patients should undergo MRI every time they feel abdominal pain.
    D. MRI should be ordered if the patient presents with a high alpha-fetoprotein level or/and a suspicious US or CT in the face of hepatitis or alcoholic liver disease or fatty liver.

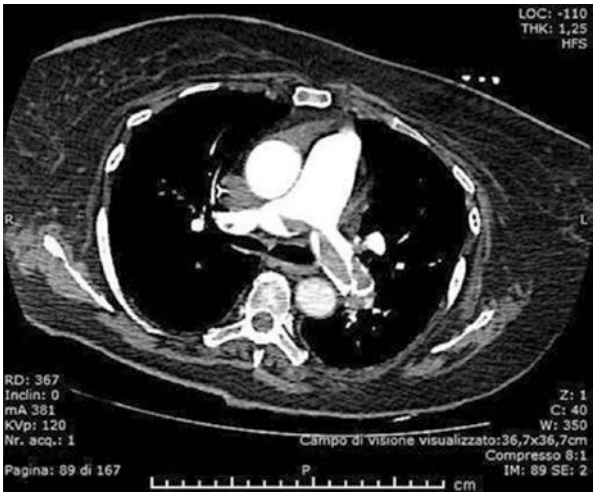
Answer: D

14. Why after contrast injection does the HCC nodule look different from the background liver parenchyma?
    A. Because the patient is cirrhotic
    B. Because HCC nodules have different texture from the background liver parenchyma
    C. Because HCC nodules parasitize arterial blood supply, flipping the normal 1:3 ratio of hepatic artery to portal venous blood supply
    D. Because HCC nodules are malignant tumors and tumors are always detectable with MRI

Answer: C

15. A 70-year-old patient with diabetes was hospitalized after falling a month prior to presentation at which time she underwent a chest x-ray that was negative, but with blood labs showing high values of CPK, consistent with rhabdomyolysis. After a month, she came to the ER for acute respiratory failure. The arterial blood gas test revealed p02 = 67 and a low pCO2, and D-Dimer value was markedly elevated. (Fig. 2a and b)





16. Looking at the images Fig. 2a and b, which radiology test was performed and why?
    A. The patient underwent a non-contrast CT scan because the most likely diagnosis was pulmonary embolism.
    B. The patient underwent a post-contrast CT Pulmonary arteriogram scan because the most likely diagnosis was pulmonary embolism,
    C. The patient underwent a post-contrast MR scan because the most likely diagnosis was pulmonary embolism.
    D. The patient underwent a non-contrast CT scan because the most likely diagnosis was pneumonia.

Answer: B

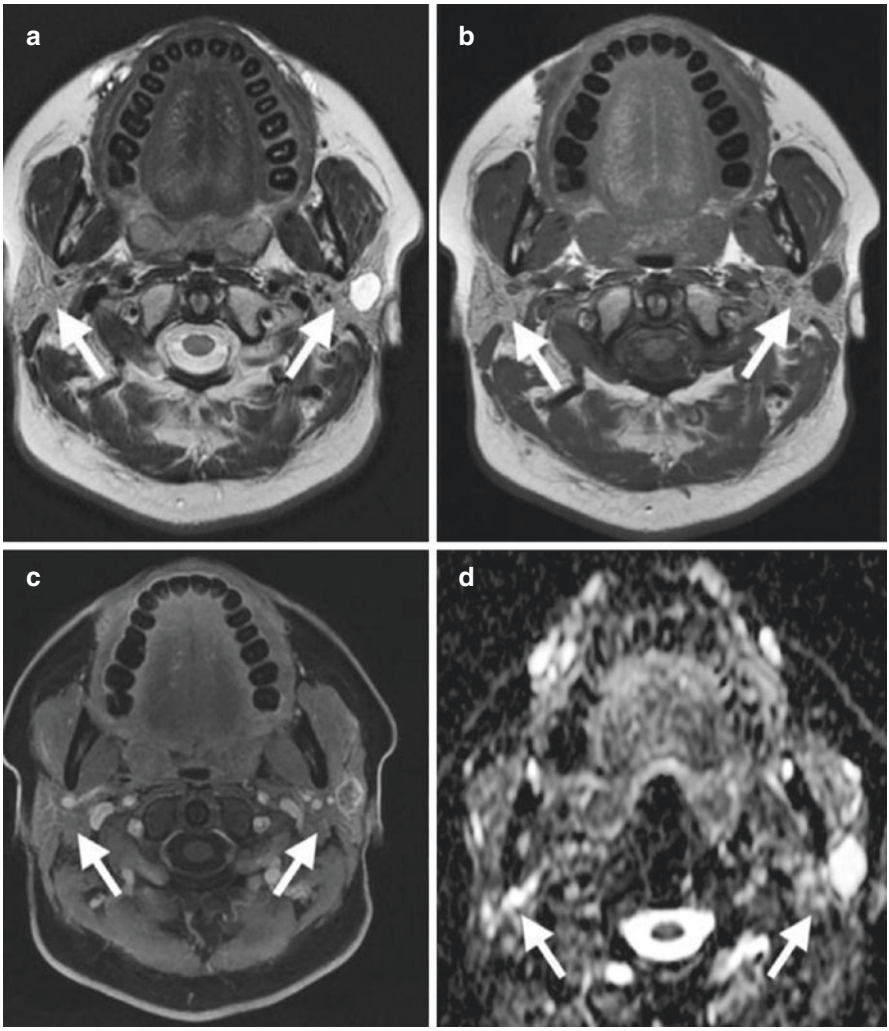
17. Bilateral filling defects (thrombi) are present in the pulmonary artery main branches. Why was contrast injection necessary in this case?
    A. Because it is the only way to highlight the presence of filling defects inside the vessels corresponding to thrombi.
    B. Because thrombi have different vascularization.
    C. It wasn't necessary: contrast injection was a mistake.
    D. Because contrast is needed for interstitial lung disease.

Answer: A

18. Looking at the images Fig. 2a and b, could you identify the CT window level?
    A. These CT images are visualized with a bone window.
    B. These CT images are visualized with a lung window.
    C. These CT images are visualized with a soft tissue window emphasizing the vessels.
    D. CT images can be visualized with just one window level.

Answer: C

19. The image below shows an MRI in a 48 y.o. patient with a parotid gland pleomorphic adenoma (a benign tumor representing the most common salivary gland neoplasm). (3A) T2-weighted image; (3B) T1-weighted image; (3C) post-contrast fat-saturated T1-weighted image; (3D) ADC map (Fig. 3).



Considering that the arrows indicate the parotid glands, where is the left side tumor, and why did the surgeon order an MRI test and not a CT test for this patient?

A. Because he was wrong.
B. There was not a rationale to it: CT and MRI are equally accurate in the head and neck.

C. Because MRI is the gold standard in the head and neck district since it is characterized by high-contrast resolution, enhancing anatomic structures and potential pathologies.
D. Because MRI can be performed using contrast.

Answer: C

20. What is the rationale of using multiple acquisitions with different sequences or series in MRI?
    A. It is beautiful.
    B. It helps the detection of the lesion.
    C. It helps measuring the lesion.
    D. The signal (brightness) of the lesion in different weighted sequences is related to its structure; therefore, the use of multiple sequences provides better characterization of the lesion.

Answer: D

21. Considering the very superficial localization of this pleomorphic adenoma, which other imaging investigation could have enabled the detection of the lesion? [HINT: Correct answer modality is also the best tool for the detection of solid versus cystic structures.]
    A. X-ray
    B. Ultrasound

Answer: B

9 Ophthalmology/Eye

1. A 50-year-old man presents to the emergency room, complaining of increasing left eye pain and swelling for 2 days. He denies any headache, neck stiffness, or recent trauma. Review of systems is positive for a recent sinus infection, for which he was prescribed a course of amoxicillin-clavulanate. He took the medication for 2 days but stopped once his symptoms decreased. On examination, the patient has a temperature of 100.4 F. He has significant upper and lower lid edema and erythema of the left eye, along with proptosis and conjunctival swelling (Fig. 33, Chapter 12). Ocular motility is restricted by pain. What is the next best step in evaluation and treatment?
    A. Tell the patient to finish his course of amoxicillin-clavulanate.
    B. Get a CT scan of the head and prescribe antibiotics.
    C. Get a CT scan of the head and admit to the hospital for IV broad-spectrum antibiotics.
    D. Obtain nasal cultures.

Answer: C. Get a CT scan of the head and admit to the hospital for IV broad-spectrum antibiotics

2. A 70-year-old man with a history of hypertension, type II diabetes, and hypothyroidism presents with sudden, painless visual loss in his right eye. He denies any preceding trauma or photopsia. Fundus examination of the right eye is performed (Fig. 34, Chapter 12). Which of the following is a risk factor for his disease?
   A. Age
   B. Hypertension
   C. Diabetes
   D. All of the above

   Answer: D. All of the above

3. A 65-year-old woman presents with progressive visual loss in the left eye for the past 6 months. She denies any ocular pain, headache, photopsia, or recent trauma. Past medical history is significant for hypertension and two Mohs surgeries for basal cell carcinoma of the left cheek. Ultrasound of the left eye is performed in the office (Fig.12.35). What is the next best step in evaluation and treatment of this patient?
   A. Admit to the hospital for IV antibiotics.
   B. Referral to an ophthalmologist.
   C. Biopsy.
   D. CT scan of the head.

   Answer: B. Referral to an ophthalmologist

4. A 28-year-old woman with type II diabetes presents with a 4-month history of episodic dizziness and blurred vision. The episodes last 30–60 seconds and are associated with nausea and a ringing sensation in the ears. Review of systems is negative for any other symptoms. The patient takes insulin for diabetes and oral contraceptive pills. Direct ophthalmoscopy is performed to show similar findings in both eyes (Fig. 36, Chapter 12). What is the next best step?
   A. Prescribe carbonic anhydrase inhibitors.
   B. MR and MRV of the head to rule out tumor or dural sinus thrombosis.
   C. Surgery.
   D. Perform lumbar puncture.

   Answer: B. MRI of the head to rule out tumor

5. A 35-year old woman with no past medical history presents to the emergency room after a car accident. She is complaining of a headache with associated neck pain. Physical examination shows a drooping left eyelid and a constricted left pupil. What imaging should you order?
   A. CT spine
   B. CT head
   C. MRI head
   D. CT angiography of the head and neck

   Answer: D. CT angiography of the head and neck

6. A new mother calls your office to ask about her newborn's eyes. The baby is four days old and has copious, yellow-green mucus streaming from both eyes. The mother thinks that the baby feels warm but is not sure if she has a temperature. Of note, the mother refused any ointments or vaccinations during and after delivery in the hospital. What is the next best step for treatment of the patient?
   A. Prescribe topical antibiotic drops
   B. Ask the mother to bring the baby to the ER for IM ceftriaxone
   C. Irrigation of the eyes
   D. Answer choices A and C
   E. Answer choices B and C

   Answer: E. Answer choices B and C

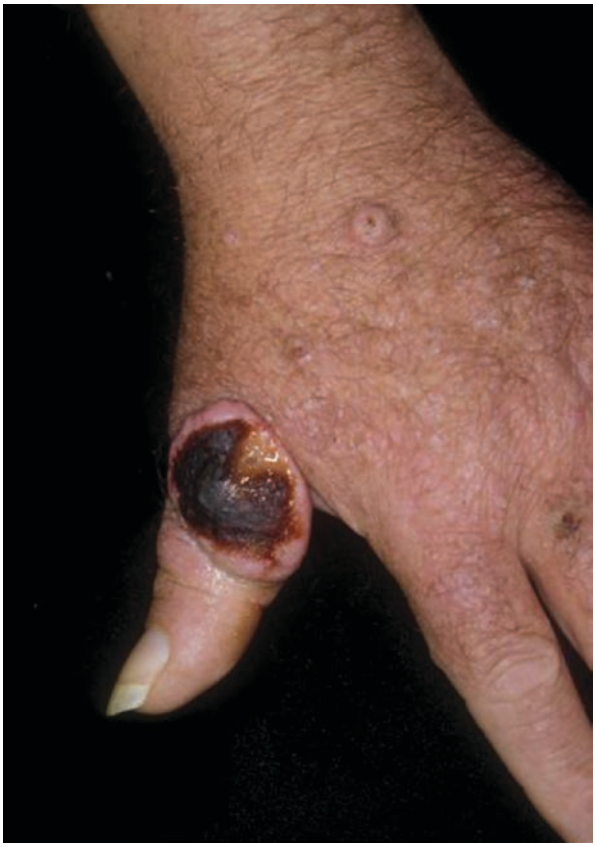
7. A 35-year old woman presents with gradual visual loss over the past year. An MRI reveals a suprasellar mass (Fig. 37, Chapter 12). What type of visual loss would a visual field examination reveal?
   A. Bitemporal hemianopsia
   B. Right homonymous hemianopsia
   C. Left homonymous hemianopsia
   D. Right homonymous inferior quadrantanopia

   Answer: A. Bitemporal hemianopsia

10 Dermatology/Skin

10.1 Scenario 1

A 45-year-old Caucasian male presents with a rapidly growing lesion on the left thumb. He lives in Arizona, works as a farmer, and has a history of kidney transplantation (on oral prednisone, tacrolimus). He reports that the lesion appeared at a site where his cat scratched the skin one month ago, and afterwards it would not heal. The site occasionally bleeds. Vital signs are within normal limits. On initial physical exam, there is a three centimeter, pink, centrally ulcerated, and crusted tumor at the base of the left thumb. CBC with differential is unremarkable.



1. What is the correct diagnosis?
   A. Human Papilloma Virus
   B. Cat-scratch disease
   C. Coccidioidomycosis
   D. Graft-versus-host disease
   E. Pyoderma gangrenosum
   F. Pyogenic granuloma
   G. Squamous cell carcinoma
   H. Sporotrichosis

   Answer: G. Squamous cell carcinoma

2. Which of the following are risk factors for this condition? (Select all that apply)
   A. Living in Arizona
   B. History of transplantation
   C. Male sex
   D. Cat-scratch history

E. Chronic injury
F. Radiation
G. Arsenic exposure
H. HPV infection
I. Outdoor occupation

Answer: B. History of transplantation, C. Male sex, E. Chronic injury, F. Radiation, G. Arsenic exposure, H. HPV infection, I. Outdoor occupation

3. What is the next most important system to evaluate on the physical exam?
   A. Lungs
   B. Musculoskeletal
   C. Lymph nodes
   D. Cardiac
   E. Mucosa
   F. GI
   G. Neurologic

   Answer: C. Lymph nodes

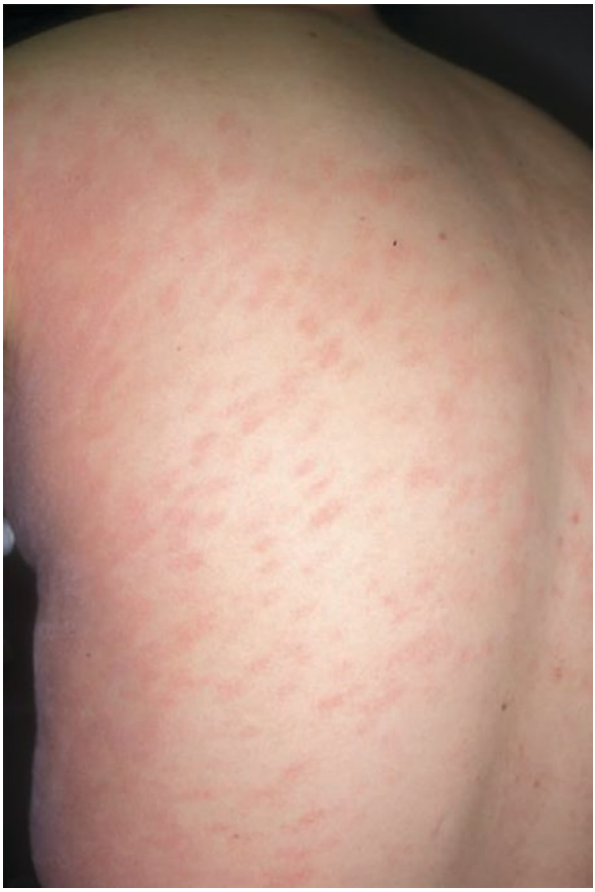
4. If no further abnormalities are noted on physical exam, what is the most appropriate next step in management?
   A. IV antibiotics
   B. IV antifungals
   C. Surgical debridement
   D. MRI
   E. Biopsy
   F. Radiation
   G. Referral to general surgery

   Answer: E. Biopsy

Explanation: Squamous cell carcinoma is depicted, and the best course of action is a skin biopsy for definitive diagnosis followed by Mohs micrographic surgery in this high-risk patient.

10.2 Scenario 2

A 17-year-old male presents to the outpatient clinic with a chief complaint of an itchy eruption that has been spreading over the past few weeks. Over-the-counter lotions have been unhelpful. His younger brother had a viral illness with a cough, fever, and rash one month prior. There is a family history of lupus. Upon examination, the patient is afebrile; there is no lymphadenopathy. A photograph of the eruption is shown below: the lesions are flat macules and patches with fine central scale favoring the trunk and extremities, sparing the hands and feet.



1. What is the most likely diagnosis?
   A. Systemic lupus erythematosus
   B. Syphilis
   C. Pityriasis rosea
   D. Tinea versicolor
   E. Tinea corporis
   F. Psoriasis
   G. Nummular eczema
   H. Seborrheic dermatitis
   I. Zoster
   J. Varicella
   K. Drug Eruption
   L. Erythema multiforme
   M. Urticaria

Answer: C. Pityriasis rosea

2. What is the best treatment?
   A. PO antibiotics
   B. PO valacyclovir
   C. IM penicillin
   D. Hydroxychloroquine
   E. Topical ketoconazole
   F. Topical corticosteroids
   G. Observation
   H. Topical antivirals
   I. PO ivermectin
   J. Phototherapy
   K. Shingles vaccine
   L. Gabapentin

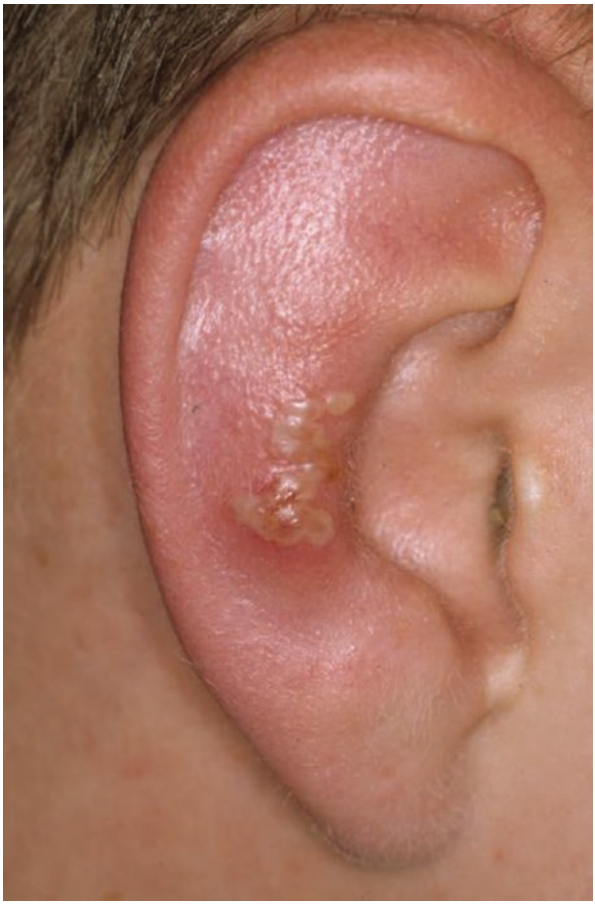
Answer: G. Observation

3. What is the next best test?
   A. No further testing
   B. KOH scrape
   C. Biopsy
   D. Blood cultures
   E. RPR
   F. ANA
   G. CBC
   H. Viral PCR
   I. Skin prick test
   J. Patch testing

Answers: A. No further testing

Explanation: Pityriasis rosea is described/depicted with lesions following skin tension lines in a "Christmas tree" pattern. It is typically self-limiting, and the best course of management is reassurance and observation.

Image-Based 3: Answer the following questions based on the image below.



1. The patient depicted is a ten-year-old boy. What is the most likely risk factor for this condition?
   A. Exotic pet
   B. Lack of vaccination
   C. Immunosuppressed status
   D. On the wrestling team
   E. History of cave spelunking
   F. History of autoimmune disease
   G. History of atopy
   H. Spider bite
   I. MRSA carrier status
   J. Anticonvulsant use
   K. UV exposure

Answer: D. On the wrestling team

2. What are possible sequelae? (Select all that apply)
   A. MRSA carrier status
   B. Recurrence
   C. Neuralgia
   D. Atypical rash on the hands and feet
   E. Inflammatory bowel disease
   F. Hearing loss
   G. Pneumonia
   H. Encephalitis

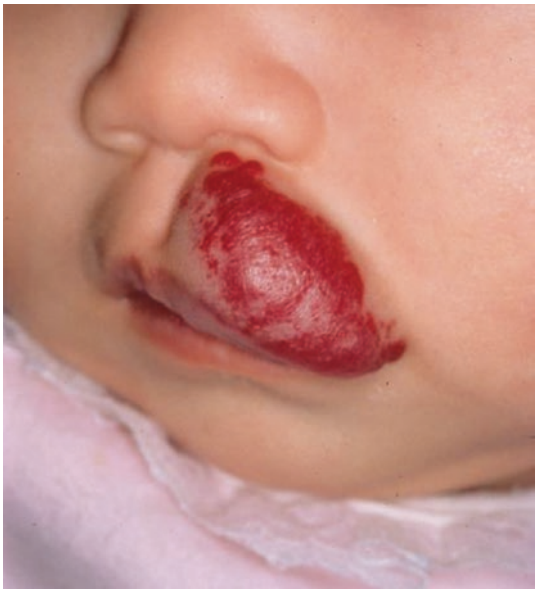
   Bonus: Would your answers change if the patient were a 65-year-old woman?

Answers: B. Recurrence, E. Inflammatory bowel disease

Explanation: A classic lesion of grouped herpetic vesicles/pustules is depicted. Herpes gladiatorum refers to HSV transmission between athletes with high skin-skin contact such as wrestlers.

Bonus: Varicella zoster virus is more likely with a herpetic outbreak in an older adult; risk factors include immunosuppression and lack of shingles vaccination. Sequelae include post-herpetic neuralgia, and when located on the ear, there is a risk of Ramsay Hunt Syndrome with unilateral facial paralysis and hearing loss.

Image-Based 4: This infant has multiple (>5) smaller lesions on the trunk. What are the greatest risks? Select all that apply.

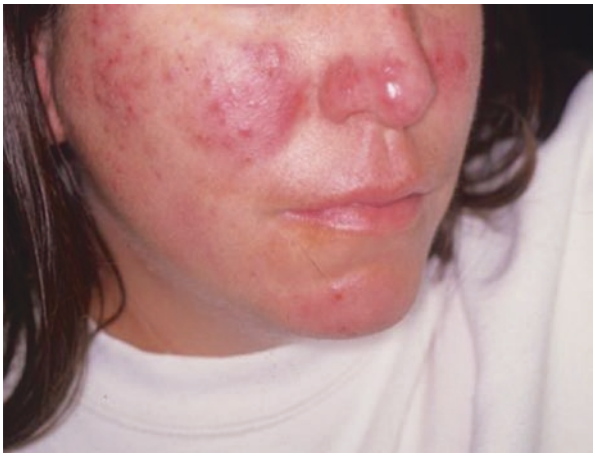


A. Airway compromise
B. Hypothyroidism
C. Disfigurement
D. Visual disruption
E. PHACES syndrome
F. LUMBAR syndrome
G. High output cardiac failure

Answers: B. Hypothyroidism, C. Disfigurement, G. High output cardiac failure

Explanation: There is a risk of visceral hepatic involvement with >5 hemangiomas, which in turn is associated with the potential for high-output cardiac failure and hypothyroidism. (There is a risk of airway hemangioma/airway compromise with lower face/beard area infantile hemangiomas.)

Image-Based 5: What is the best treatment?



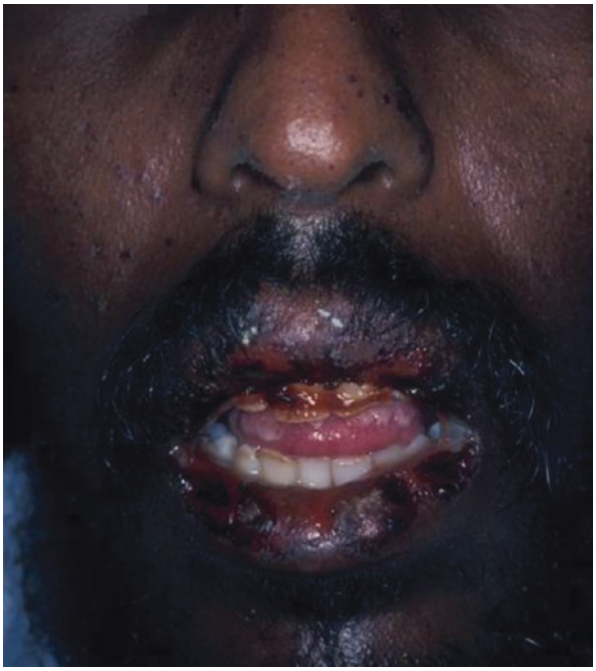
A. Topical ketoconazole cream
B. Oral antibiotics, topical metronidazole
C. Topical corticosteroids
D. Hydroxychloroquine
E. Topical tretinoin, clindamycin, and benzoyl peroxide
F. Topical permethrin
G. PO prednisone
H. Isotretinoin

Answer: B. Oral antibiotics, topical metronidazole

Explanation: The best initial treatment for papulopustular rosacea (note the lack of comedones) is a combination that often comprises of oral doxycycline/

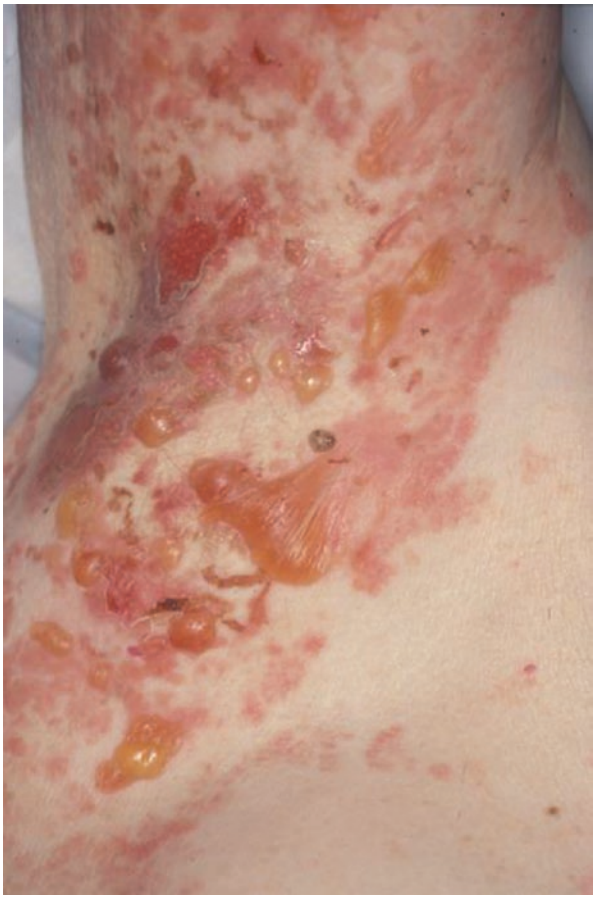
minocycline plus topicals including metronidazole, sulfacetamide sulfur, azelaic acid, and/or ivermectin.

Image-Based 6: What is the most important prognostic factor?



A. Age
B. Serum bicarbonate
C. Immunosuppression
D. HIV
E. BSA involved
F. Heart rate
G. Serum glucose
H. BUN level

Answer: B. Serum bicarbonate
Explanation: SCORTEN criteria for SJS/TEN
Image Based 7: Nikolsky sign is negative. Where is the level of split in the skin?



A. Stratum corneum
B. Stratum lucidum
C. Stratum spinosum
D. Intra-epidermal
E. Subepidermal
F. Deep dermis

Answer: E. Subepidermal

Explanation: Bullous pemphigoid is pictured; the split is subepidermal and Nikolsky sign is negative.

Suggested Reading

1. Alikhan A, Hocker T. Review of dermatology. 1st ed. Elsevier; 2016.
2. Bolognia J, Schaffer JV, Duncan KO, Ko CJ. Dermatology essentials [E-book]. Elsevier; 2014. p. 1040. Available from: http://www.clinicalkey.com/dura/browse/bookChapter/3-s2.0-C20090416568.
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4. National Comprehensive Cancer Network. Clinical practice guidelines in oncology: cutaneous melanoma. 2020. . Available from: https://www.nccn.org/professionals/physician_gls/pdf/cutaneous_melanoma.pdf.
5. Rice PL, Orgill, DP. Assessment and classification of burn injury [Internet]. 2019 [updated 2019 Jun 17]. Available from: https://www.uptodate.com/contents/assessment-and-classification-of-burn-injury.

Helpful Resources

https://dermnetnz.org/
https://www.visualdx.com/

Google Image Search Terms

Patrick H. Andrews and Amel Amalou

Contents

1 Perspectives from a Medical Student on Using This Resource

Google search terms (under google image) are an easy and new way to study medical imaging in a rapid-fire pictorial superficial fashion so you can learn pattern recognition. It is important to do this for common and critical images like "free air" and "pneumothorax" and "pneumonia" for example. If you are like most medical students, you might feel the stress of the question: "Am I studying right?" This is common. However, no two medical students will study in exactly the same way, use exactly the same materials, or spend exactly the same amount of time on any subject, and this is how it should be! We each have unique strong suits as well as those things we need to work on, and this means that our individual processes will be our own. It's been said here before and it will be said again before you're done reading

All search terms supplied by book authors and faculty

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H. Amalou et al. (eds.), *The Radiology Survival Kit*,
https://doi.org/10.1007/978-3-030-84365-6_15

this book: don't compare yourself to others and don't judge your progress by the apparent progress of your peers!

Our advice is much like what we all had to do when we entered our respective schools: spend a couple days playing around with different study techniques, a quick review platform (i.e., flashcards through any online program or software, such as Anki or Quizlet), and your study schedule (i.e., study for 50 minutes, take a 10 minute break to drink some water, call your mom, and then resume). Then once you find one that feels right, stick with it! Do not second guess your studying process and start from scratch—I had to learn this the hard way at the start of medical school and made life way more stressful for myself than it needed to be (and probably ended up with exactly the same results). Just as studying can take a number of different trajectories throughout the months that you dedicate toward USMLE, there are a number of different ways that we can study and still be successful so long as we stick with it and have a little faith in ourselves.

This doesn't mean we shouldn't periodically check in and assess our performance—if you're a runner, you know that you have to do this to assess your pace and how your body feels every couple of miles to see if you should slow down or if you can speed up. Every time you do some practice questions or a mock exam, ask yourself: for the ones I got wrong, why did I get these wrong? How did I feel when I was answering these questions? Did I eliminate answers through reasoning, or did I know what I was looking for after reading the question stems and clinical scenarios? Using your answers to these questions to inform how you continue studying will be important for being efficient and effective so long as you build on the study process and techniques you have already been employing. Make little adjustments and track how they impact your performance.

Run out of practice questions or gone through all of the Google Image search terms in this book? This is a great opportunity to test your knowledge with higher order thinking. During my pre-clerkships, I consolidated the most important and most pertinent information about specific topics into practice questions that I created myself and then could also share with others. It didn't take long, and it helped with remembering high-yield topics because it placed information into condensed clinical pictures that often mirrored the kinds of questions or patient scenarios we would encounter on exams or in our clinical reasoning practice sessions. My process for this was pretty straightforward and can easily be applied to the content in this book. *Going chapter by chapter, highlight the important topics and diagnoses, utilize the condensed information that is available in this book, and then create a clinical picture as follows:*

1. Create a relatively broad chief complaint. *Ex: 55 y.o. male with right sided abdominal pain.*
2. Make a differential list. This can be long or short depending on how specific your chief complaint is. *Ex: This DDX could include, among others, appendicitis, cholecystitis, cholelithiasis, acute cholangitis, hepatitis, liver abscess, diverticulitis, nephrolithiasis, Crohn's disease, infectious colitis, malignancy, obstruction, perforation.*

3. For each differential, name some of the features that would make it more likely. This is a great time to use the Google Image Search Terms as well as relevant histology/pathology images from Google Image since rad/path connections are easily testable items.
4. Choose an answer and design the question stem to include the relevant risk factors, symptoms, medical imaging, histology, and other pertinent information. Create an image and a story of a person who is dealing with these symptoms! *Ex: More specifically, acute right lower quadrant (RLQ) pain. Sharp pain upon palpation of right lower quadrant midway between umbilicus and anterior superior iliac spine. Pain in RLQ when LLQ is palpated. Fever. CT imaging reveals thickened, hyperenhancing wall and fat stranding in a structure in the RLQ (use Google Images to find a picture so you have to find an image that includes these findings!)*
5. List a couple answer options for the question. In the answer key, give an explanation as to why the other options were not correct given the imaging and presentation. *Ex: Appendicitis is the answer for this example, but you could include diverticulitis, cholecystitis, malignancy, and perforation as other answers and include the relevant imaging for each in the answer key so you can easily spot the different key findings.*
6. Try switching one or two aspects of the chief complaint and create another question. This will help you differentiate similar presentations and the necessary diagnostic imaging. *Ex. 24 y.o. female with subacute right-sided abdominal pain. This changes your imaging workup (CT contraindicated until pregnancy ruled out because she is of child-bearing age) and your differential (age, time course, and sex of the patient would provide a somewhat different DDX even though the core symptom is the same).*
7. Alternatively, give the radiologic and histology/pathology findings and ask for which clinical intervention is indicated in order to apply some higher-level thinking.

This can be a powerful, condensed way to quickly think about and review many different disease presentations, their relevant radiographic findings, and common clinical scenarios that one might encounter in the hospital or on tests. Place these questions into a slideshow so you can quickly go through these prior to test day—good luck!

2 Tips on How to Study and Search Google Image

Each chapter's authors supplied lists of Google Image search terms for common boards scenarios and settings which provide a unique method to prepare for USMLE topics with major imaging components (Radiology, Interventional Radiology, Dermatology, Ophthalmology).

Type one term only for each list into Google Images (go to: https://images.google.com), and review the top 10, 20, or 50 images quickly, just to get a general idea of what the entity commonly looks like, as well as a general sense of the variety

of presentations and imaging appearances. Do not get bogged down in any one image here! If you need a quick reminder of what a certain term is referring to, click on the far left "All" tab at the top of the search page. Note: you might have to add the word "medical to the end of the search for phrases that might have other meanings. Certain chapters are divided into everyday, must-know search terms that commonly appear on USMLE boards, followed by less common esoteric or uncommon boards question terms. These sample terms are in no way meant to represent actual cases, actual boards questions, or reproduce in any way, shape or form the USMLE examination, or similar preparatory coursework from a review course, book, medical school classwork, or medical school institution itself (Figs. 1 and 2).

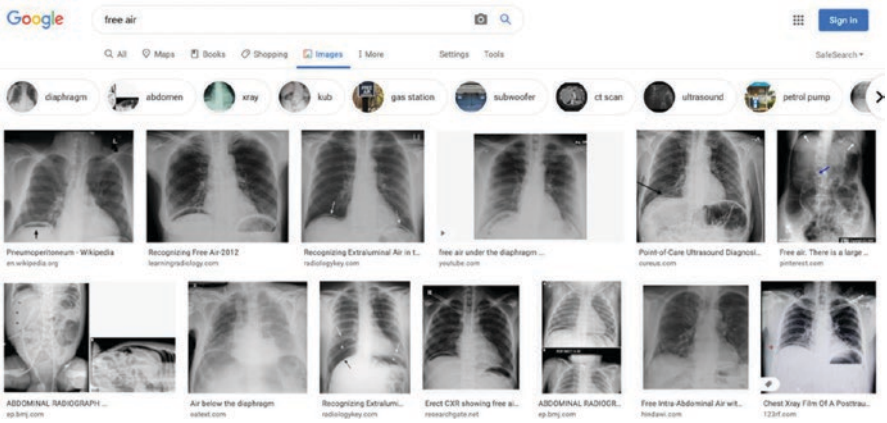

Fig. 1 Google Image search tool as a rapid and efficient visual review for USMLE. Example case on Apple/Mac with search term: "free air"

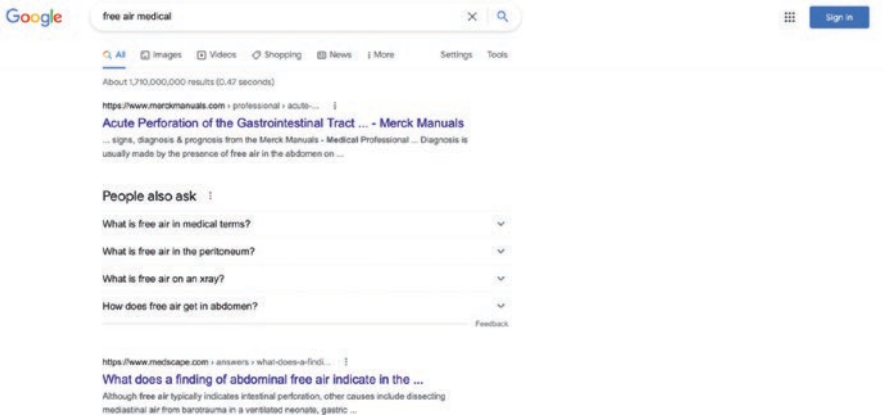

Fig. 2 Google search "All" tab for more information on topics that need very brief reviewing or keyword reminders. Example case on Apple/Mac with search term: "free air medical." This is an example of a search term with different meanings that require the addition of "medical" to the search criteria

Study Tips:

- *Keep Track:* For terms that you anticipate needing to review even quicker prior to test day, compile a personalized document or slideshow of images as you search them so you can scroll through later on, without searching again.
- *Search Comparisons:* Many items may look very similar, or it may be difficult to ascertain what the key differences are between two related topics, for example, cardiogenic and noncardiogenic pulmonary edema. Searching these terms alone is helpful if you know what to look for, but searching "cardiogenic edema **vs** noncardiogenic edema" reveals the key differences to look for in potential question images.

3 Google Image Search Terms

Clinical Rotation Tips:

1. Imaging planes
2. Medical imaging orientation
3. T1 vs T2 MRI
4. Chest MRI
5. Chest x-rays
6. AP vs PA chest x-ray
7. Chest CT
8. Brain MRI
9. Brain CT
10. Contrast-enhanced CT
11. Non-contrast CT
12. Contrast-enhanced MRI
13. Skull-base MRI
14. Skull-base CT
15. Abdominal x-rays
16. Pneumonia
17. Pneumonia imaging
18. Cranial nerves
19. Pleural effusion
20. Liver CT
21. Liver MRI
22. Hepatocellular carcinoma (HCC)
23. Abdominal CT
24. Abdominal MRI
25. Renal colic CT
26. Hydronephrosis
27. Hydronephrosis imaging

Emergencies:

1. Pneumothorax vs tension pneumothorax
2. Pneumothorax chest radiograph
3. Tension pneumothorax chest radiograph

4. Impending aortic aneurysm rupture
5. Aortic dissection chest radiograph
6. Aortic dissection CTA
7. Hyperattenuating crescent sign
8. Draped aorta sign
9. Pneumoperitoneum radiograph
10. Pneumoperitoneum CT
11. Trauma colon perforation CT
12. Pneumatosis intestinalis CT
13. Portal venous gas CT
14. Pneumobilia CT
15. Appendicitis ultrasound
16. Acute uncomplicated appendicitis CT
17. Appendicitis abscess CT
18. Acute cholecystitis ultrasound
19. Acute cholecystitis CT
20. Stroke hyperdense vessel sign
21. Stroke CTA cutoff
22. Hemorrhagic stroke CT
23. Acute subarachnoid hemorrhage CT
24. Acute subdural hemorrhage CT
25. Acute epidural hemorrhage CT
26. C1 burst fracture
27. Hangman fracture cervical spine
28. Jumped facets cervical spine
29. Odontoid fracture types
30. Atlantoaxial dissociation CT
31. Atlanto-occipital dissociation CT
32. Pulmonary embolism CTA
33. Pulmonary embolism right heart strain CT
34. S1Q3T3 (ECG OR EKG)
35. Ectopic pregnancy ultrasound
36. Ectopic pregnancy ring of fire

Chest:

1. Chest x-ray anatomy
2. Lateral chest x-ray anatomy
3. Chest CT anatomy
4. Systematic approach to the chest x-ray
5. Central venous catheter
6. San-Ganz catheter
7. Enteric tube
8. Nasogastric tube
9. Orogastric tube
10. Esophageal pH probe
11. Thoracostomy (chest) tube

12. Endotracheal tube
13. Atelectasis
14. Round atelectasis
15. Right lower lobe collapse
16. Left lower lobe collapse
17. Right middle lobe collapse
18. Left upper lobe collapse
19. Right upper lobe collapse
20. Golden S sign
21. Alveolar airspace disease
22. Ground-glass opacity
23. Lung consolidation
24. Lobar pneumonia
25. Silhouette sign
26. Spine sign
27. Air bronchogram sign
28. Rounded pneumonia
29. Atypical "walking" pneumonia
30. Aspiration pneumonia
31. Lung abscess
32. Air-fluid level
33. Empyema with bronchopleural fistula
34. Empyema
35. Split pleura sign
36. *Pneumocystis* (PCP/PJP) pneumonia
37. Crazy paving
38. COVID-19
39. SARS-CoV-2
40. Pulmonary hemorrhage
41. Cardiogenic pulmonary edema
42. Non-cardiogenic pulmonary edema
43. Cephalization
44. Peri-bronchial cuffing
45. Doughnut sign
46. Kerley B lines
47. Bat wing sign
48. Cardiothoracic ratio
49. Diffuse alveolar damage
50. Acute respiratory distress syndrome
51. Type II pneumocyte
52. Alveolar endothelial cell
53. Lung adenocarcinoma
54. Squamous cell carcinoma of the lung
55. Small cell carcinoma of the lung
56. Large cell carcinoma of the lung

57. Lung metastasis
58. Pulmonary cavitation
59. Septic emboli
60. Grape-skin sign
61. Inhomogeneous enhancement sign
62. Pulmonary cavity wall thickness
63. Tuberculosis
64. Miliary tuberculosis
65. Aspergilloma
66. Fungus ball
67. Monod sign
68. Angioinvasive aspergillosis
69. Air crescent sign
70. Halo sign
71. Solitary pulmonary nodule
72. Centrilobular emphysema
73. Panlobular emphysema
74. Paraseptal emphysema
75. Transudative pleural effusion
76. Exudative pleural effusion
77. Meniscus sign
78. Hemithorax white out
79. Pleural plaques
80. Incomplete border sign
81. Mesothelioma
82. Pulmonary embolism
83. Well's criteria
84. Westermark sign
85. Hampton's hump
86. Saddle embolus
87. Polo mint sign
88. Railway track sign
89. Right heart strain
90. Pacemaker
91. Implantable cardioverter defibrillator
92. Left atrial enlargement
93. Ballerina sign
94. Double density sign
95. Walking man sign
96. Right atrial enlargement
97. Left ventricular enlargement
98. Right ventricular enlargement
99. Pericardial effusion
100. Water bottle sign
101. Oreo cookie sign

102. Cardiac tamponade
103. Widened mediastinum
104. Aortic coarctation
105. Figure 3 configuration
106. Rib notching
107. Pneumomediastinum
108. Pneumopericardium
109. Continuous diaphragm sign
110. Ring around the artery sign
111. Tubular artery sign
112. Anterior mediastinal mass
113. Middle mediastinal mass
114. Posterior mediastinal mass
115. Hilar lymphadenopathy
116. Pneumoperitoneum
117. Rib fracture
118. Flail chest

Less Common:

1. Luftsichel sign
2. Ghon focus
3. Ghon complex
4. Monod sign

Neuroradiology and CNS:

1. Anterior cerebral angiogram
2. Posterior cerebral angiogram
3. Epidural hematoma on CT
4. Middle meningeal artery
5. Swirl sign and spot sign
6. Subdural hematoma on CT
7. Acute on chronic subdural hematoma
8. Non-accidental trauma acute on chronic hematoma
9. Subarachnoid hemorrhage on CT and star of death sign
10. Pseudo-subarachnoid hemorrhage on CT
11. Posterior communicating artery aneurysm on CTA and MRA
12. Berry aneurysm and oculomotor nerve (CN III) on MRI
13. Brain abscess on MRI
14. Encephalitis
15. Meningitis
16. Dural enhancement and leptomeningeal enhancement
17. Osteomyelitis-discitis on MRI and CT
18. Epidural abscess
19. Pott's disease and gibbus deformity
20. Prion disease on MRI
21. Creutzfeldt-Jakob disease

22. Cortical ribboning and hockey stick sign
23. Neurocysticercosis scolex on MRI and CT and racemose neurocysticercosis
24. Herpes simplex encephalitis on MRI or CT
25. Temporal lobe on MRI and CT and hippocampus on MRI
26. Multiple sclerosis on MRI
27. Demyelinating MS plaques
28. Modified McDonald Criteria
29. Dawson fingers and transverse myelitis
30. Methanol poisoning on MRI
31. Putamen
32. Hemorrhagic putaminal necrosis and basal ganglia
33. Carbon monoxide poisoning on MRI
34. Globus pallidus and globus pallidus necrosis
35. Wernicke encephalopathy on MRI
36. Mamillary bodies and periaqueductal gray matter
37. Subacute combined degeneration on spine MRI and posterior columns of the spinal cord
38. Idiopathic intracranial hypertension on MRI
39. Empty sella on MRI
40. Narrowing of the sinodural angle
41. Low lying cerebellar tonsils and dilation of the optic nerve sheaths
42. Pontine myelinolysis on MRI
43. Glioblastoma on MRI
44. Butterfly lesion and corpus callosum
45. Cord compression from vertebral metastasis
46. Cord compression from an epidural abscess and cauda equina leptomeningeal disease
47. Schwannoma on internal auditory canal MRI and ice cream cone sign
48. Neurofibromatosis type 2 on MRI
49. Bilateral vestibular schwannomas
50. Meningiomas and ependymomas
51. Neurofibromatosis type 1 on MRI
52. Optic pathway pilocytic astrocytoma
53. Sphenoid bone dysplasia
54. Dural ectasia and T2/FLAIR focal areas of signal intensity (FASI)
55. Chiari I malformation on MRI and “peg-shaped” cerebellar tonsils
56. Chiari II Malformation on MRI
57. Myelomeningocele
58. Hydrocephalus
59. Ultrasound lemon sign and ultrasound banana sign
60. Alzheimer’s disease volume loss on MRI, CT, and PET

Abdomen:

1. Abdominal radiograph anatomy
2. Abdominal mass

3. Abdominal pain
4. Cholangiography
5. Cholangiopancreatography
6. Endoscopic retrograde
7. Cholecystography
8. Abdominal aortic aneurysm
9. Abscess
10. Steatosis
11. Hemochromatosis
12. Angioma
13. Pancreas CT
14. Abdominal hemorrhage CT
15. Spleen CT
16. Colon CT
17. Duodenum CT
18. Liver CT
19. Hepatic portal vein CT
20. Inferior vena cava CT
21. Superior mesenteric artery/vein CT
22. Ischemia
23. Hamartomas
24. Lithiasis
25. Cholangiocarcinoma
26. Cholecystitis on US
27. Small bowel obstruction
28. Large bowel obstruction
29. Cecal volvulus x-ray
30. Sigmoid volvulus x-ray
31. Perforated viscus
32. Achalasia
33. Bird beak sign
34. Esophageal stricture
35. Pyloric stenosis
36. Upright vs supine abdominal radiograph
37. Endoscopy
38. Diverticulitis CT
39. Apple core sign
40. String sign

Bone/Musculoskeletal:

1. Imaging modalities MSK
2. Shoulder x-ray anatomy
3. Shoulder MRI anatomy
4. Spine x-ray anatomy
5. Types of bone fractures
6. Pediatric fractures

7. Foot fractures and dislocations
8. Muscle injury imaging
9. Muscle injury classification
10. Tendon injuries x-ray
11. Syndesmosis injury imaging
12. Bone infection radiology
13. Muscle infection radiology
14. Septic arthritis radiology
15. Cellulitis ultrasound
16. Osteoarthritis x-ray
17. Rheumatoid arthritis x-ray
18. Bone tumor
19. Osteosarcoma
20. Ewing sarcoma
21. Multiple myeloma x-ray
22. Periosteal reaction
23. Lytic bone lesions
24. Osteoblastic bone lesions
25. Sclerotic bone lesions
26. Gout x-ray
27. CPPD x-ray
28. Disc herniation
29. Degenerative disc disease MRI
30. DISH x-ray
31. Ankylosing spondylitis x-ray
32. Avulsion fracture
33. Developmental dysplasia of the hip
34. Slipped capital femoral epiphysis
35. Leg-Calve-Perthes disease
36. Avascular necrosis
37. ACL MRI
38. PCL MRI
39. Arthroscopy
40. Bursitis MRI

Vascular and Interventional Radiology

1. Seldinger technique
2. Vascular sheath
3. Balloon angioplasty
4. TACE
5. TARE
6. Hydrophilic guidewire
7. EVAR
8. TEVAR
9. Angiographic catheters

10. Metallic stent
11. Chest port
12. Inferior vena cava filter
13. Vascular plug
14. PVA particles
15. Gelfoam collagen sponge
16. Lipiodol
17. Heat sink effect
18. Thermal ablation
19. Embolization
20. Percutaneous biopsy
21. Paget-Schroetter syndrome
22. May Thurner syndrome
23. Polyarteritis nodosa
24. Subclavian steal
25. Fibromuscular dysplasia
26. Arc of Riolan
27. Arc of Buhler
28. Marginal Artery of Drummond
29. Circle of Willis
30. Renal artery stenosis
31. Stanford classification aortic dissection
32. Thrombolysis
33. TIPS
34. Portal vein thrombosis
35. Portal hypertension
36. Isolated gastric varices
37. Downhill vs uphill esophageal varices
38. Collateral pathways
39. MRA
40. Blood on MRI
41. Bleeding duodenal ulcer angiography
42. Shunt fraction in TARE
43. Acalculous cholecystitis
44. Spinal artery of Adamkiewicz
45. Vertebroplasty
46. Abscess drainage
47. Percutaneous biopsy
48. Biliary drainage
49. Nephrostomy tube placement

Women's Imaging

1. Mammogram breast cancer
2. Microcalcifications mammography
3. Breast MRI

4. Breast US
5. Adnexal mass
6. Molar pregnancy
7. Ectopic pregnancy
8. Ovarian torsion
9. Tubo-ovarian abscess
10. Snow storm uterus
11. Breast abscess US
12. Gynecomastia
13. Hemorrhagic cyst—lacelike
14. Fibroadenoma
15. Pelvic congestion syndrome
16. Hepatic adenoma

Ophthalmology/Eye:

1. Visual axis
2. Contralateral homonymous hemianopsia
3. Bitemporal hemianopsia
4. Horner's syndrome
5. Pancoast tumor
6. Carotid dissection
7. Cranial nerve III palsy
8. Cranial nerve VI palsy
9. Neurofibroma
10. Lisch nodule
11. Proptosis
12. Chemosis
13. Preseptal cellulitis
14. Orbital cellulitis
15. Orbital floor
16. Herpes keratoconjunctivitis vesicles
17. Endophthalmitis
18. Cupping of optic disc
19. Angle closure glaucoma
20. Drusen
21. Dot-blot hemorrhages
22. Flame hemorrhages
23. Cotton-wool spots
24. CMV retinopathy
25. Branch retinal artery occlusion (BRAO)
26. Branch retinal vein occlusion (BRVO)
27. Basal cell carcinoma
28. Squamous cell carcinoma
29. Sturge-Weber
30. Leukocoria

31. Neonatal conjunctivitis
32. Gonococcal conjunctivitis
33. Chlamydial conjunctivitis
34. Allergic conjunctivitis
35. Chemical conjunctivitis

Dermatology/Skin:

1. Skin rash USMLE
2. Lyme disease rash
3. hand foot syndrome
4. Necrotizing Fasciitis
5. Pyoderma Gangrenosum
6. Burns
7. Porphyria cutanea tarda
8. Atopic dermatitis
9. Eczema
10. Herpes simplex
11. Scabies
12. Molluscum contagiosum
13. Impetigo
14. Folliculitis
15. Erysipelas
16. Cellulitis
17. Pyoderma gangrenosum
18. Necrotizing fasciitis
19. Melanoma
20. Basal cell carcinoma
21. Squamous cell carcinoma
22. Actinic keratoses
23. Human papilloma virus
24. Wart
25. Lentigo
26. Seborrheic keratosis
27. Anaphylaxis
28. Angioedema
29. Infantile hemangioma
30. Tinea capitis
31. Tinea versicolor
32. Tinea corporis
33. Bullous pemphigoid
34. Hypertrophic and keloid scars
35. Psoriasis
36. Hypersensitivity reactions
37. Seborrheic dermatitis
38. Vitiligo

39. Stevens-Johnson syndrome/TEN
40. Pemphigus vulgaris
41. Senile purpura
42. Pityriasis rosea
43. Acne vulgaris
44. Rosacea
45. Hyperpigmentation
46. Melasma
47. Hidradenitis suppurativa
48. Contact dermatitis
49. Stasis dermatitis
50. Erythema multiforme
51. Kawasaki disease
52. Measles
53. Chickenpox
54. Shingles
55. Fifth's disease

You can also enter the chapter titles into "google image search", or any term or word plus the word: "USMLE." An alternate method of study is to search regular google with the same search words above, followed by "USMLE." You can also of course type in the search words followed by "USMLE question" or "boards" or "sample USMLE questions" or "NBME" "NBME questions" or "Q bank." In general, when you enter just the word into "google image," this will retrieve mostly radiographs. When you add the word "USMLE" and still enter the search in "google image," then both images and cute little cartoons, and flashcards, and some very simple sources, will appear.

The approach to test-preparations in the era of Google also comes with some pitfalls, however. Avoid the temptation to get stuck in the weeds by digging deeper and deeper and learning more and more about weird little esoteric and rare diseases. Do not obsess on any one topic or any one finding or image. Set a time limit for one topic and for one diagnosis or section. If you find yourself losing time like this, then step away and start fresh. Test prep is about learning "a little about a lot" …just a little about a wide variety of things… so you can make good guesses on tests, or at least rule out a few incorrect answers. In the days and weeks immediately before test time, this is even more important to "float above the details," so you can keep up the fast (and sometimes superficial) pace of prep/review. If you know one thing well, do not review it again. Save repeats for hard to grasp topics or short term memory topics (like numbers). You got this.

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