Obliterating Hepatic Vein Endophlebitis

►Budd–Chiari Syndrome

Obstetric Conjugate

Distance on a midsagittal section from the sacral promontory to the top of the symphysis.

►Magnetic Resonance Pelvimetry

Obstruction of the Biliary Tree

►Occlusion, Bile Ducts

Obstruction of the Inferior Venacava

►Thrombosis, Caval Vein, Inferior

Obstruction of the Superior Venacava

►Thrombosis, Caval Vein, Superior

Obstructive Emphysema

Obstructive emphysema is identified by over inflation of the lung distal of an airway obstruction.

►Foreign Bodies, Aspiration, Children (Chest View)

Obstructive Uropathy in Childhood

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Synonyms

Obstructive uropathy; Urinary tract obstruction

Definition

Obstructive uropathy is any kind of hindered urine drainage from the renal calyces or pelvis, the ureter or the bladder. However, clinically relevant obstruction means impairment to a degree that in the past has already damaged or in the future will deteriorate renal function of the affected system. Consequences of obstruction initially are dilatation, thickening of the (bladder-, ureteral-, pelviceal- and calyceal-) wall and eventually thinning of renal parenchyma with consecutive diminishing renal function. This may ultimately lead to a decrease in dilatation; therefore, the generally used assessment of dilatation for grading of obstruction may mimic improvement in cases with significant functional deterioration. Depending on the various underlying concepts different definitions are used to define the degree of obstruction as considered relevant for treatment decisions in different parts of the world, with Europe tending to quantify the drainage dynamics, and America focusing on renal function.

Embryology and Pathogenesis

There are several causes for obstruction, with consecutively different embryologic and pathogenetic pathways. They can be divided into congenital anomalies and anatomic variants or secondary and acquired obstruction (Table 1).

Obstructive Uropathy in Childhood. Table 1 Causes of obstructive uropathy

Congenital causes:
• Idiopathic megaureter, with relative narrowing at the uretero-vesical junction (Fig. 1a),
• Ureterocele with megaureter, mostly with a duplex system (Fig. 1b),
• Rare other ureteral variations such as ureteral valves,
• Congenital uretero-pelvic junction obstruction, with narrowing by a fibrotic or dysplastic segment at the uretero-pelvic junction (Fig. 1c),
• Posterior urethral valves and other urethral anomalies, as well as rare ureteric flaps,
• Anatomic variations
• Caliceal neck stenosis or ureteral obstruction caused by prominent or accessory renal vessels (Fig. 1d),
Retrocaval ureter leading to impaired ureteral drainage,
Horse shoe kidney, malrotation and ectopic kidneys, with an unusual coarse of the ureter that may impair drainage,
Secondary obstruction
• Urethral- and bladder polyps,
• Ureteral and urethral fibrosis or stenosis, such as (post-)traumatic urethral stricture leading to bladder outlet obstruction with consecutive bladder pathology and associated obstructive or refluxing uropathy (see entry 'Hydronephrosis'),
• (Post-)infectious causes,
• Motion and peristalsis impairment by dysplasia or dysgenesis and palsy,
• Retroperitoneal processes (tumour, fibrosis, haematoma, …),
• Acute urinary tract obstruction by an ureteral calculus, a haematoma or blood clot, or fungus ball,
• Post-/peritraumatic obstruction (haematoma, compression, rupture, …).

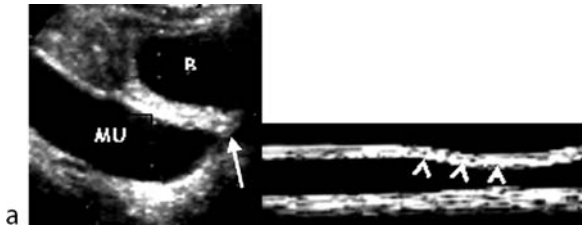
Clinical Presentation

Typical clinical presentation of obstructive uropathy includes abdominal or flank pain, renal colic, abdominal distension or palpable abdominal mass (in gross HN) and (micro-)haematuria. Lower urinary tract obstruction (urethral obstruction) may additionally exhibit anuria or oliguria, e.g. in a neonate with posterior urethral valves (PUV).

Imaging

The purpose of imaging is not only to detect urinary tract dilatation and obstruction, but to provide a detailed anatomic display of the patient's individual situation, to describe the obstruction anatomically (high-, middle-, low/ureteral obstruction? obstruction at the uretero-vesical- or at the uretero-pelvic junction? obstruction of the urethra?) and finally to grade obstruction and—in particular—to differentiate dilative or non-obstructive uropathy from (partial) obstruction, since obstruction may need treatment and follow-up, as well as to assess renal function.

Imaging methods: Imaging is primarily performed using *ultrasound* (US), which can only be successfully applied in sufficiently hydrated children and should always include an assessment at full and post-void bladder. Furthermore, (colour) Doppler sonography (CDS) as well as less common approaches (such as perineal US of the urethra) and application of modern methods (e.g. 3DUS) may offer additional valuable information. For anatomic assessment of the urethra in suspected lower urinary tract obstruction or for differentiating refluxing units from obstructive dilation, an *urethrography*, a *cystogram* or a VCUG is indicated. For anatomic assessment of the upper urinary tract (ureter, renal collecting system and kidney) IVU has been the major imaging tool in the past, in addition to US; however, increasingly *MR-urography* (MRU) is taking this role not only offering an anatomic assessment but also allowing for functional evaluation and MR-angiography in the same investigation. CT is rarely used in children due to its radiation burden—in complicated stone disease a low dose protocol multi-detector CT may become indicated. For grading of obstruction and functional evaluation diuretic renal scintigraphy still is the most commonly used tool (Fig. 2). It allows differentiating dilative from (partially) obstructive systems using various protocols (depending on the time of administration of diuretics) that are applied not only for initial assessment, but also during follow-up, particularly of equivocal findings. Increasingly this functional assessment can also be performed by functional diuretic MRU, however, availability and technical aspects as well as sedation needs have yet hindered a wide spread use of this approach. Retrograde or antegrade *ureterography* and pyelography are rarely performed; in rare cases or in complicated malformations they may be



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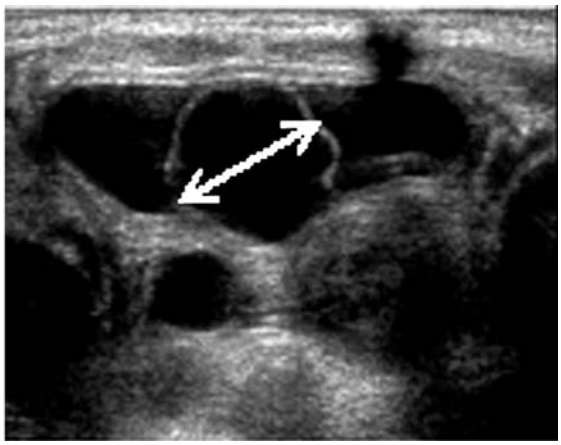

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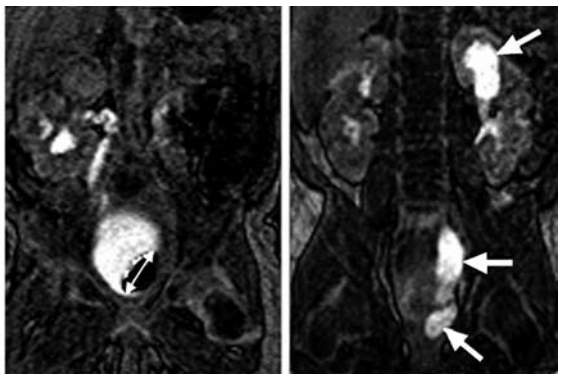

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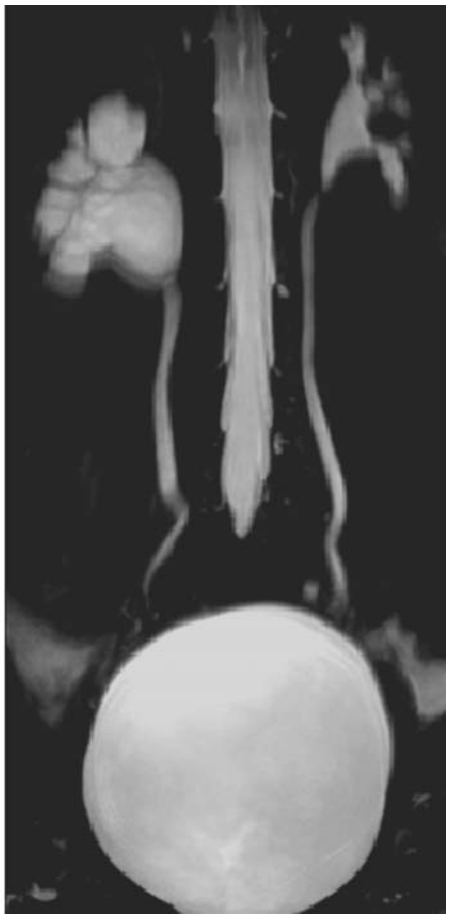

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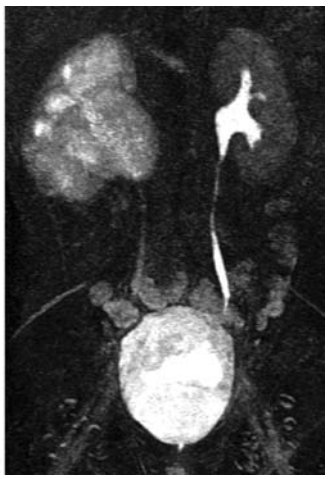

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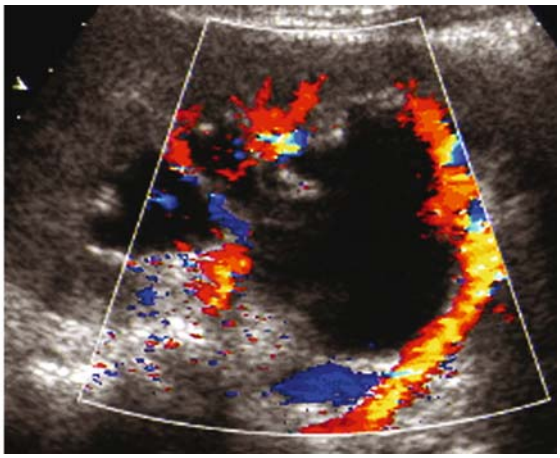

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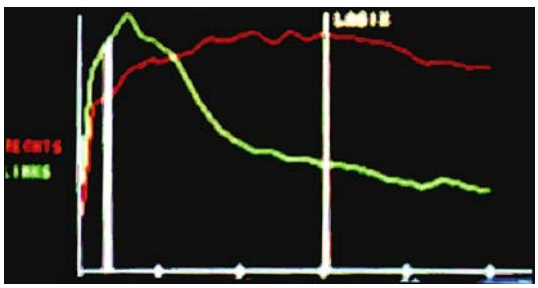
Obstructive Uropathy in Childhood. Figure 1 (continued)

O

used as well as peri-operatively, and in acute events with indication for *percutaneous nephrostomy. Whitaker test* (percutaneous nephrostomy with a flow pressure curve derived from intrapelvic fluid infusion *via* the nephrostomy tube with simultaneous pressure measurement) has been the gold standard for assessment of upper urinary tract obstruction. Due to its invasiveness and the reliable results from diuretic scintigraphy or functional MRU this method is today restricted for complicated cases with equivocal results of standard imaging, potentially in a peri-operative setting, as well as for research purposes.

Imaging indications and algorithms: Basically obstructive uropathy is primarily diagnosed using US. Further imaging depends on the initial findings (Table 2).



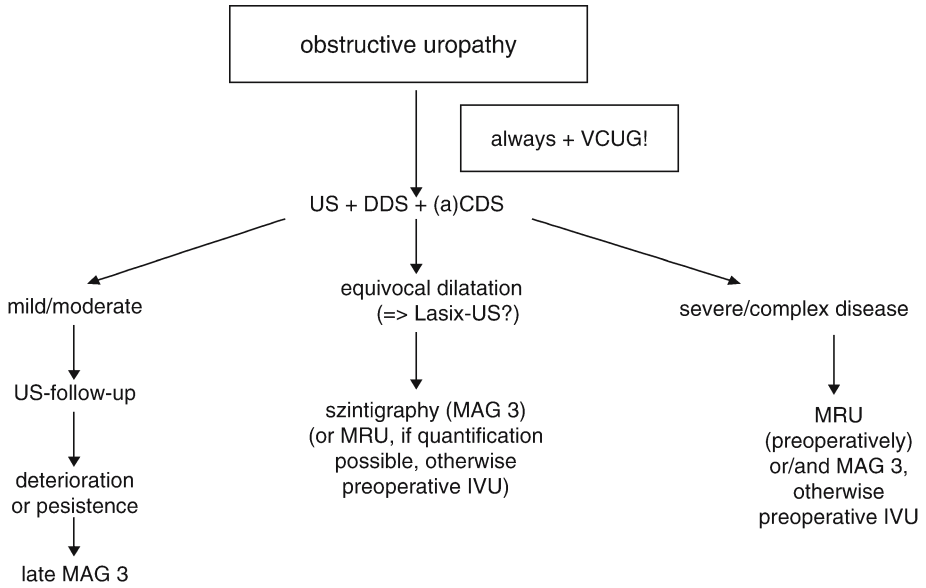
Obstructive Uropathy in Childhood. Figure 2 Diuretic scintigraphy in a patient with severe uretero-pelvic junction obstruction. Note the different perfusion, excretion and drainage curves on this diuretic Tc ^{99m} MAG3 renography, with delayed and reduced renal transit indicating diminished function and causing a flattening of the early activity curve (red (dark) line) and poor drainage in the right obstructed kidney, with only poor response on diuretic stimulation (yellow (light) line). Normal renography curve of the normal left kidney (green (gray) line).

- Low degree hydronephrosis (HN) is followed up by US without any other imaging,
- High degree HN is assessed by diuretic renal scintigraphy and MRU (or IVU), the latter pre-operatively or in case of complex malformations,
- VCUG for differentiation of refluxing from obstructive uropathy
- In acute renal colic (note that acute urinary tract obstruction often only exhibits mild dilatation!) US can often sufficiently diagnose the condition. US is supplemented by an abdominal plain film of the kidney, ureter and bladder ('KUB') for assessment of additional calculi or positioning of a sonographically diagnosed calculus for lithotripsy. In unclear cases an adapted IVU or even an un-enhanced (spiral multi-detector) CT may still become indicated. In case of renal parenchymal findings that indicate an underlying disease such as nephrocalcinosis or medullary sponge kidney adequate imaging of these entities is performed (see entry: 'Nephrocalcinosis', 'Cystic Renal Disease'),
- In complex cases, particularly in combined infection and obstruction, CT- or MR-urography may become indicated. Percutaneous nephrostomy offers an image guided treatment option, and in PUV with lower urinary tract obstruction a suprapubic catheterism may become necessary,
- For follow-up, regular (diuretic) US examinations at 1–3–6 month intervals and diuretic scintigraphy or MRU are recommended, with some variation of timing and protocol in different centres.

Note that imaging indication and method depends on the indication for treatment, which varies between the continents; whereas in Europe usually diuresis impairment indicates surgical action, loss of kidney function expressed by relative renal function (usually assessed by

Obstructive Uropathy in Childhood. Figure 1 Imaging findings in typical obstructive uropathies. (a) US of a primary megaureter. This longitudinal section of the retrovesical dilated ureter (MU) with a short tight uretero-vesical junction (⇒) into the urinary bladder (B) documents the existing ureteral peristalsis (>) using an m-mode track recorded at the site of the dotted line. (b) Ureterocele. Transverse US image of the urinary bladder demonstrating a large intravesical ureterocele (⇔) causing obstructed urinary drainage from the ipsilateral system, seen as a dilated retrovesical ureter on the right side. (c) Serial dynamic MRU images (Gadolinium enhanced, T1-weighted, 3D gradient-echo sequence) in a patient with left sided ureterocele and duplex system show the non-enhanced, fluid filled ureterocele in the bladder visualized during the early phase (⇔), that gradually fills with contrast during late phases that also demonstrate contrast excretion into the dilated upper system with the corresponding megaureter (⇐). (D) MRI in UPJO. Note the dilated collecting system with a narrow uretero-pelvic junction at fully extended urinary bladder after Furosemid application in this non-enhanced, one shot, 3D, T2-weighted HASTE image of MR-urography of a right sided UPJO with a normal appearance of the left system. (E) Three-dimensional volume rendered image of a late phase (after diuretic stimulation using Furosemid), T1-weighted, gradient-echo acquisition of a dynamic MR-urography in an infant with severe hydronephrosis of the right kidney—due to uretero-pelvic junction obstruction—and a normal left kidney with diuresis-induced slight distension of the renal pelvis. (F) Accessory renal vessel causing upper urinary tract obstruction. Additional renal artery (coded in red) coursing to the lower pole of the left kidney causing proximal ureteral obstruction and hydronephrosis, demonstrated by a longitudinal

Obstructive Uropathy in Childhood. Table 2 Flowchart 'imaging algorithm in obstructive uropathy'



Suggestions for an imaging algorithm in pediatric obstructive uropathy.
Note: In case of acute colic with suspected urolithiasis, US usually is supplemented by an abdominal plain film (KUB) and—in still equivocal cases—potentially an adapted IVU; in rare complicated cases a focused or adapted (un-enhanced) spiral CT may become necessary.
Abbreviations: US, ultrasound; DDS, duplex Doppler sonography; (a) CDS, (amplitude coded) colour Doppler sonography; IVU, intravenous urography; MRU, MR-urography; MAG3, dynamic renography; VCUG, voiding cystourethrography.
Source: Adapted from Riccabona M, Fotter R (2004) Reorientation and future trends in paediatric uroradiology: minutes of a symposium. Pediatr Radiol 34:295–301.

scintigraphy) is considered the indication for surgery in the US, were as some other areas relay on the degree of dilatation and basically operated all grossly dilated systems.

Diagnosis

Imaging allows diagnosis of the underlying anatomy and pathology, enables deciding on the level of obstruction, and allows for functional evaluation particularly necessary for treatment decisions. Imaging criteria are different for the various modalities, the underlying pathology and the different treatment paradigms.

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Occlusion and Subocclusion, Small Bowel in Adults

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Synonyms

Small intestinal obstruction

Definition

►Small bowel obstruction (SBO) is the impedance of the progression of intestinal content due to a mechanical obstacle (1).

Pathology/Histopathology

The small bowel extends from the duodeno-jejunal junction to the ileocaecal valve. It is approximately 6.5 m long. The

jejunum represents the proximal two-fifths and the ileum, the distal two-fifths. The small bowel gradually changes from the beginning of the jejunum to the end of the ileum. The jejunum (2.5 cm diameter) is wider than the ileum (2 cm diameter). The mucosa is arranged in circular folds which are larger and more numerous in the jejunum. These folds are a distinguishing feature from large bowel on an adult plain film of abdomen (PFA).

Secreted fluid and swallowed air contribute to bowel distension in SBO. Ischaemia is a serious complication, which may be venous, arterial or both and may lead to infarction. Ischaemia is more likely in ▶closed-loop obstruction (CLO). In CLO, a loop of bowel is occluded at two points by a single constrictive lesion. The mesentery and intestine are occluded. This is usually due to adhesions or hernias.

The causes of SBO are outlined in the Table 1.

The commonest causes are adhesions (50–80%), hernias (10–15%) and neoplasms (10–15%). Other causes are uncommon in the adult population.

Clinical Presentation

SBO accounts for 20% of all acute surgical admissions (1). Classic symptoms are abdominal pain, distension, vomiting and constipation. Clinical presentation depends on the severity of SBO and the presence or absence of ischaemia. In early, uncomplicated SBO, abdominal pain may be crampy and intermittent, but when ischaemia occurs, pain becomes more severe and continuous.

On examination, high frequency bowel sounds and abdominal distension are observed. Dehydration and electrolyte imbalance can occur.

Imaging

The goals of imaging in SBO are

1. Confirm the diagnosis
2. Determine the level of SBO
3. Diagnose the cause
4. Assess for evidence of ▶strangulation or mesenteric ischaemia
5. Determine need for surgery

PFA

A PFA is usually the first radiological test in SBO. The PFA has a sensitivity of 64% for SBO diagnosis but does not reliably allow diagnosis of cause, level of SBO, or ischaemia. Some advocate that the PFA should be performed erect to assess for air-fluid levels (1). If an erect PFA is not possible, a horizontal beam lateral film can also be used to evaluate for air-fluid levels. An erect chest X-ray is the plain radiograph of choice to assess for perforation.

Contrast Studies

There are limitations to the use of contrast studies in acute SBO. Barium is contraindicated if perforation is suspected. Water-soluble contrast can be used, but is diluted by the excess intra-luminal fluid. If hyper-osmolar agents are used, these can worsen dehydration by osmotic effect on the bowel wall, encouraging further fluid loss into the lumen. Enteroclysis reduces dilution by intra-luminal fluid, facilitates propulsion toward the level of obstruction and also allows a means for drainage of some of the administered fluid.

Since computed tomography (CT) became available, enteric contrast studies have been of limited value in SBO. There are two situations in which contrast studies are useful.

1. Enteroclysis can be used in the investigation of intermittent or low-grade obstruction for which the sensitivity of CT is only 48%.
2. Water-soluble follow through (WSFT) can be used to distinguish which clinically stable patients are likely to have spontaneous resolution of SBO (2). About 100 mL of water-soluble contrast is administered via a nasogastric tube and a PFA is taken 4 h later. If the contrast column has reached the caecum by this time, then the obstruction is very likely to resolve. If not, SBO is unlikely to resolve spontaneously.

Ultrasound

US is not used as a first line investigation in SBO if CT is available. It confirms dilated fluid-filled in SBO in up to 95.3% of cases. Ultrasound is not as good as CT for ruling in SBO but it is better for ruling out SBO, which is unlikely in the absence of dilated, fluid-filled loops (3).

Extrinsic	Intrinsic	Intra-luminal
Adhesions (most common)	Neoplasm (common)	Gallstones
Hernias (common)	Inflammation	Intussusception
Neoplasm (common)	Ischaemia	Meconium ▶ileus equivalent (in cystic fibrosis)
Inflammation	Haematoma	

(*continued*)

Dilated small bowel loops are also seen in ▶paralytic ileus. In SBO versus paralytic ileus, hyper-dynamic intestinal motility can be observed in real time.

Computed Tomography

CT has high accuracy in the diagnosis of SBO and in determining the cause. Enteric contrast is optional depending on clinical severity of obstruction. In high-grade SBO, there is usually sufficient fluid within the bowel to allow accurate evaluation of obstructed loops. Intravenous contrast (150 mL contrast bolus at 3 mL per second) is used to demonstrate bowel wall enhancement, which is important in the diagnosis of ischaemia.

In practice, most patients are initially evaluated with PFA and subsequently with CT.

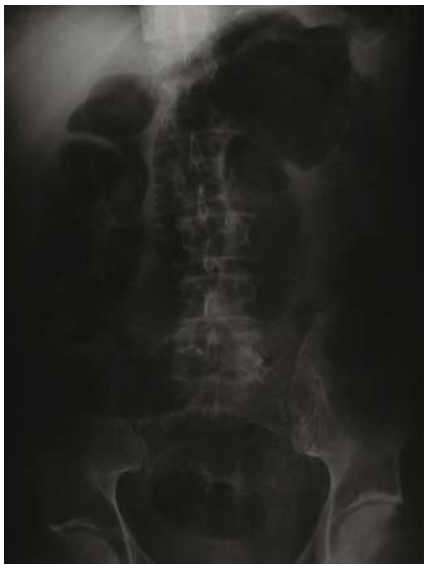
Nuclear Medicine

Scintigraphy does not currently play an important part in the diagnosis of SBO.

Diagnosis

Confirming the Diagnosis of Obstruction

The PFA is diagnostic of SBO in up to 64% of cases. Dilated loops of bowel are seen, usually within 5 h of the onset of obstruction (Fig. 1). If the dilated loops are entirely fluid-filled, then the diagnosis may be occult on PFA. Dilated bowel loops can also be seen in paralytic ileus.



Obstructive Uropathy in Childhood.

Figure 1 PFA of SBO. Multiple dilated loops of small bowel shown.

CT is more than 95% accurate in the diagnosis of SBO (4). The diagnosis depends on demonstrating dilated small bowel loops (>3 cm) with a focal discrepancy in the calibre of the bowel at the transition between obstructed and non-obstructed bowel (Fig. 2). In paralytic ileus, the small bowel is also dilated, but the right colon is usually also dilated, often tapering to normal at, or distal to, the hepatic flexure (4).

Determining the Level of Obstruction

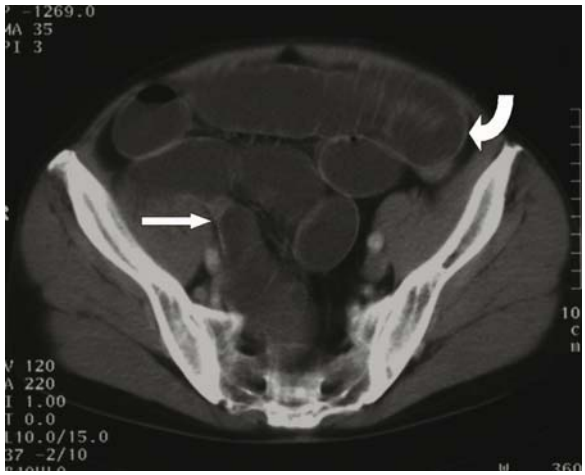
On PFA, the presence of dilated small bowel superiorly in the abdomen may indicate proximal SBO. Dilated pelvic loops more frequently imply distal SBO. In SBO, dilated small bowel loops align along the long axis of the mesentery and jejunal loops can be found in the pelvis, with ileal loops in the upper abdomen.

In order to distinguish large bowel obstruction from SBO on PFA, the following features are helpful:

Megibow (4) describes the systematic assessment of the bowel on CT in bowel obstruction. The bowel should be systematically evaluated in a retrograde way, beginning at the rectum, and proceeding to the caecum. After

Small bowel obstruction	Large bowel obstruction
Haustra absent	Haustra present
Valvulae in jejunum	No valvulae
Many loops	Few loops
Central distribution	Peripheral distribution

(*continued*)



Obstructive Uropathy in Childhood.

Figure 2 SBO due to adhesions. ▶Zone of transition in distal small bowel (arrow). Note normal bowel wall enhancement (curved arrow).

'clearing' the colon, the distal ileal loops should be identified and assessment should proceed proximally to the level at which the calibre of the small bowel is increased (Fig. 2). Review of the images on cine-mode and multiplanar reconstructions [particularly with multidetector-row CT (MDCT)] are helpful in this evaluation.

Diagnosis of the Cause of Obstruction

Adhesions, hernias and neoplasms account for over 80% of cases. CT is the primary diagnostic tool in determining aetiology of SBO, with accuracy of 70–95% (1).

In developed countries, adhesions account for 50–80% of causes. A history of prior abdominal surgery is usual, although previous peritoneal inflammation may also cause adhesions. Even in patients with known intra-abdominal malignancy, SBO is due to adhesions in 21–38% (4). On CT, diagnosis of adhesions is based on an abrupt change in small bowel calibre without another cause of obstruction at the transition point. A beak-like narrowing can be seen. Although the adhesions cannot be visualised with CT, it is important to make the diagnosis of obstruction secondary to adhesions when no other cause is seen (4).

Hernias account for 10–15% of cases (1). External hernias are more common than internal and include inguinal, femoral, paraumbilical, obturator, Richter, Spigelian and incisional hernias. Internal hernias include paraduodenal hernias, transmesenteric hernias, and herniation through the foramen of Winslow. These can be recognised by an abnormally positioned segment of small bowel often with protrusion of mesentery through the internal defect, with proximal bowel dilatation. With increasing use of laparoscopic surgery, port-site hernias are a cause of SBO to recognise. Laparoscopic port sites vary in diameter, generally from 5 to 10 mm. Herniation through laparoscopic fascial defects as small as 5 mm has been described. SBO due to port-site hernias is more common in the early post-operative period (5).

Neoplasia is the third most common cause of SBO. Infiltration of the bowel wall may be caused by adenocarcinoma, carcinoid or metastatic carcinoma. Extrinsic masses such as peritoneal carcinomatosis can also cause obstruction. In these cases a mass is seen at the transition point. Mural mass lesions may also lead to obstruction by causing intussusception.

Assessing for Evidence of Strangulation or Mesenteric Ischaemia

The presence of ischaemia in SBO increases mortality from between 5 and 8% to between 20 and 37% (4). It requires urgent surgical management. CT is 83% sensitive and 93% specific for ischaemia in SBO.

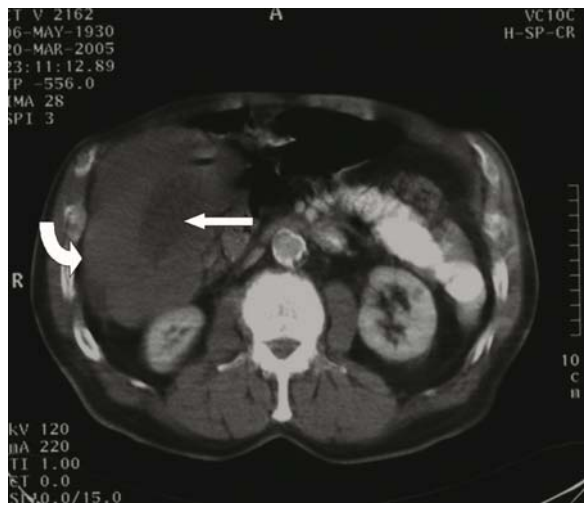
Strangulation is defined as CLO associated with intestinal ischaemia. In CLO (Fig. 3), a loop of bowel is occluded at two adjacent points along its course. CLO usually occurs due to constriction by adhesions or a hernia. The CT signs of CLO are

- Radial distribution of small bowel loops with mesenteric vessels converging toward apex
- U or C-shaped dilated bowel loop
- Two adjacent collapsed or triangular loops at the site of obstruction

CLO is prone to twisting of the affected bowel and mesentery leading to venous congestion with subsequent arterial ischaemia and infarction. This is termed strangulation. The CT signs of strangulation are

- Circumferential mural thickening
- 'Target' sign—concentric mural rings of different attenuation, post-intravenous contrast, a more specific form of mural thickening
- 'Whirl' sign—corkscrew configuration of mesenteric vessels due to twisting
- Increased attenuation of bowel wall
- Lack of bowel wall enhancement
- Air in bowel wall (intestinal pneumatosis)
- Mesenteric haemorrhage
- Ascites

In SBO complicated by ischaemia, the bowel wall appearance depends on the balance between venous obstruction, reduced arterial perfusion and reperfusion. If reduced arterial perfusion predominates, in the absence



Occlusion and Subocclusion, Small Bowel in Adults. Figure 3 Closed-loop obstruction. C-shaped loop of bowel. Note congested mesentery (arrow) and poor enhancement of bowel wall (curved arrow).

of significant reperfusion, then the bowel wall will be of normal thickness and many of the above signs will be absent. Therefore, CT is good at ruling in ischaemic SBO, but poor at ruling out ischaemic SBO (3).

Determining the Need for Surgery

The impact of imaging on management and indications for surgery have been summarised by Taourel et al. (1).

1. Strangulation and ischaemia (emergency)
2. Obstruction due to a tumour
3. Obstruction due to irreducible hernia (urgent)
4. Obstruction due to adhesions, without ischaemia, should be managed conservatively, where possible

As stated above, if SBO is slow to resolve with conservative management, WSFT is a useful test to determine the likelihood of successful non-operative treatment (2).

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Occlusion, Artery, Femoral

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Synonyms

Femoral arterial obstruction; Femoral arterial occlusion; Infrainguinal arterial obstruction; Infrainguinal arterial occlusion; Popliteal arterial obstruction; Popliteal arterial occlusion

Definition

An arterial obstruction of the infrainguinal arteries is an occlusion or narrowing of an arterial segment between the groin and the lower limbs.

Pathology/Histopathology

Occlusions are complete obstructions of the infrainguinal arterial lumen and are due to thrombus formation, mainly on preexisting atherosclerotic plaque. In stenoses, the lumen is still patent but narrowed, with a diameter reduction of more than 50% causing symptoms. Alternatively, acute occlusions occur because of thrombotic emboli from the heart.

Clinical Presentation

Clinical indications for treatment include claudication, pain at rest, and nonhealing ulceration. The severity of the symptoms depends on the acuteness of the occlusion, the condition of collateral pathways, the lesion's location, and concomitant disease such as diabetes or renal insufficiency.

Imaging

Many imaging modalities allow the physician to diagnose infrainguinal artery obstruction. Color-coded duplex sonography as well as magnetic resonance angiography (MRA), computed tomography (CT), computed tomography angiography (CTA), and intra-arterial angiography are useful tools for detecting the location and extent of an iliac obstruction.

Nuclear Medicine

Nuclear medicine plays no particular role in the diagnosis of aortic stenosis.

Diagnosis

Diagnosis is reliably achieved by angiography or MRA. Detection by duplex sonography is a reliable tool for the femoropopliteal segment but is sometimes limited for detection and exact lesion description in the lower limbs. In stage IV (Fontaine) disease, MRA may be limited because of low arterial flow, movement artifacts, or artifacts due to superimposed venous flow.

Interventional Radiological Treatment

Endovascular Versus Surgical Treatment

Endovascular therapy is known to be of low invasiveness with good technical success, achieving fair overall patency. In data taken from eight publications reporting on 1,469 procedures, the weighted average technical success of femoropopliteal endovascular interventions was 90%, the complication rate was 4.3%, and the 3-year patency rate was 51%. Stents did not improve patency, showing a patency of 58% after 3 years (1).

Surgery offers acceptable results for distal reconstruction: An average 5-year patency of 80% for vein bypasses and 65–75% for expanded polytetrafluoroethylene (ePTFE) bypasses has been reported. The combined mortality and amputation risk was calculated to be about 2.2% for aortobifemoral reconstructions and 1.4% for femoropopliteal reconstructions (1).

Location of Lesion

Claudication is mainly related to lesions in the aortoiliac and femoropopliteal regions. It is unlikely to be due to infrapopliteal lesions, and there is general agreement that treatment below the knee should be strictly limited to patients with critical limb ischemia, that is, stages III and IV (Fontaine) or categories 4–6 (Rutherford).

Type of Lesion

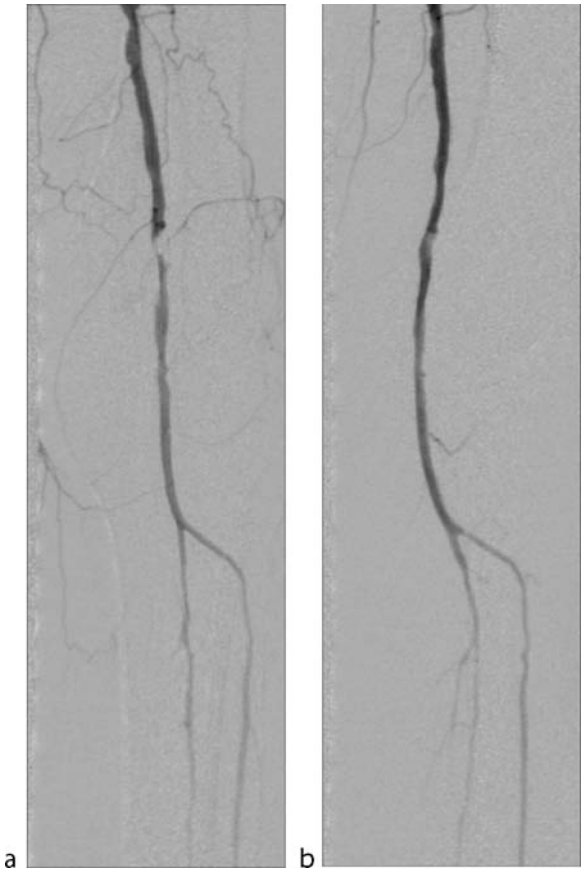
The morphology of a treated lesion influences the technical outcome, follow-up results, and also the risk of treatment. Therefore, the TransAtlantic Inter-Society Consensus (TASC) document introduced a classification system that tries to categorize lesions with regard to their accessibility to either percutaneous treatment or surgery: type A lesions, which are ideal for a percutaneous approach; type B lesions, in which the percutaneous approach is still the preferred technique; type C lesions, in which a surgical approach should be preferred; and type D lesions, in which surgery is the option of choice. The TASC classification overrides older classifications because it takes into account all of the available and published techniques (including stent technology), which offer a much wider variation of treatment as well as effective tools to deal with acute complications of balloon angioplasty, such as occluding dissection and vascular rupture.

If we consider percutaneous therapy as the preferred method to treat those patients presenting with mild or moderate claudication, treatment might be offered to those presenting with type A and B lesions but should be discussed in depth with patients with type C lesions, as the risk and the potential benefit of treatment are related to the underlying morphology.

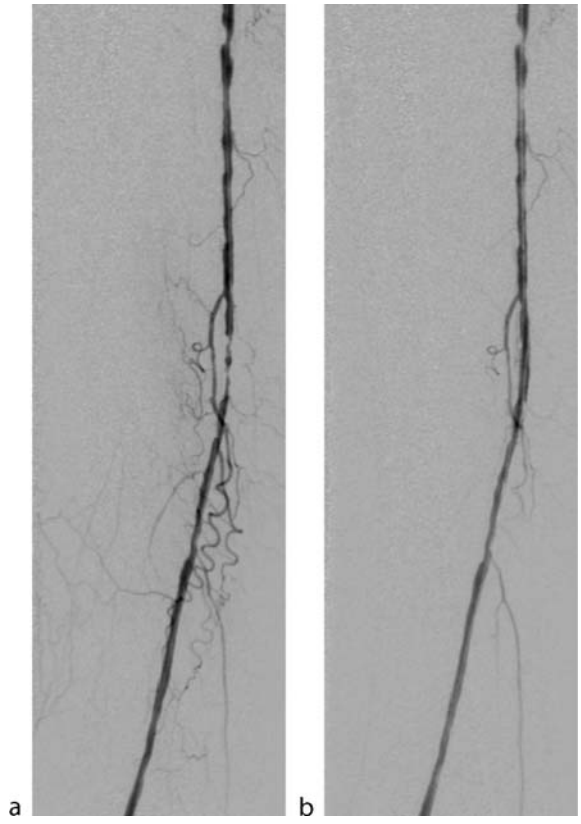
In the femoropopliteal field, type A lesions are single stenoses up to 3 cm in length not involving the very proximal superficial femoral and the distal popliteal artery. Type B lesions include stenoses 3–5 cm in length, heavily calcified stenoses, multiple lesions (each up to 3 cm), and lesions with no sufficient tibial run-off (the latter are unlikely to meet the criteria of mild or moderate claudication). Type C lesions are classified as stenoses or occlusions longer than 5 cm and multiple midsize lesions (3–5 cm). Total common femoral, superficial femoral, and popliteal occlusions are classified as type D lesions. There was some dissenting discussion on the definition of type B lesions: Interventional radiologists represented by the Cardiovascular and Interventional Radiological Society of Europe wished to express their assumption that even longer lesions of up to 10 cm may be justified as being classified type B instead of type C; they claimed that the reported results were mainly due to underdeveloped techniques and instruments that have substantially improved and that no data exists comparing the efficacy of percutaneous transluminal angioplasty (PTA) versus bypass surgery for lesions between 4 cm and 10 cm. (See Figs 1 and 2)

Other than in the iliac area, few femoral lesions meet the criteria for types A and B lesions, especially if limited to 5 cm in length. Thus, few patients with mild or moderate claudication due to femoropopliteal lesions will be ideal candidates for percutaneous treatment. Moreover—without limiting the importance of the TASC document, which certainly means a step forward in the joint approach to peripheral vascular disease—the morphological classification does not take into account some technical considerations that depend on the age and composition of a lesion. Particularly in femoral occlusions, the degree of organization of the occluding thrombus or the composition of the lesion with the original stenosis at the proximal and distal ends or in the middle are factors that are not very predictable but may influence the technical outcome of the intervention or its complication rate. (For instance, distal embolization might aggravate symptoms.) Other than in the iliac arteries, the liberal use of stents and stent grafts may help overcome a failed balloon angioplasty and resolve the technical outcome, but it does not achieve an improved long-term efficacy, and it may start a lifetime dependency on recurrent interventional or surgical procedures. These associated potential drawbacks have to be carefully balanced against the potential benefits and need to be discussed in depth with the patient before treatment is performed, especially in association with mild or moderate claudication.

These considerations restrict the use of endovascular treatment in femoropopliteal lesions to mainly stages IIb and IIa patients presenting with type A or less pronounced type B lesions.



Occlusion, Artery, Femoral. Figure 1 Popliteal artery stenosis. (a) Short-segment stenosis at the level of the joint. The stenosis is eccentric, which sometimes indicates insuffient reaction to percutaneous transluminal angioplasty (PTA). Stenting in that region is problematic because it is in the flexure zone of the popliteal artery. If needed as a bail-out treatment, a very flexible stent is preferred. (b) After PTA, sufficient opening has been achieved, and no further treatment is necessary.



Occlusion, Artery, Femoral. Figure 2 Superficial femoral artery lesion. (a) Short stenosis of a small superficial femoral artery close to the adductor's canal in a patient with stage IV disease and diabetes. (b) After percutaneous transluminal angioplasty, the arterial lumen is wider, but a dissection remains. No stent was used due to good flow, absence of collateral filling, and small arterial lumen.

Assisting Forms of Treatment

It is widely accepted that well-conducted physical exercise should precede any type of interventional treatment and that cessation of smoking is mandatory. Nevertheless, it is also true that in many institutions it is very difficult to find an infrastructure that allows instruction of state-of-the-art physical exercise for claudicants. And as far as smoking is concerned, there is a major difference between wanting to stop and actually stopping.

Moreover, even with state-of-the-art exercise, young patients will not recover completely from claudications in all activities, including sports. The process will be long and will compromise their abilities in their professional lives. Therefore, it should perhaps be discussed whether young and active patients, especially, should be held to the axiom of "physical exercise first" or whether invasive treatment might be offered even as a first approach to this group of patients.

Treatment Options with Relation to Location and Lesion

Treatment of femoropopliteal lesions in claudicants has to be seen as more critical and less liberal compared with the iliac region. The main reasons are less favorable technical success, a higher complication rate, and poorer long-term success. There are many more lesions in the femoropopliteal arteries that do not meet the criteria for suitability for endovascular treatment. On the other hand, the versatility of endoluminal techniques opens treatment options in many particular lesions, and taking clinical symptoms as the only criteria to indicate or exclude treatment is not justified because—depending on the type of lesion—a simple and limited intervention can mean considerable improvement for the patient.

Additional Morphological Factors (not Included in the TASC Classfication)

Femoropopliteal occlusions may especially become a source of complications, particularly if they occurred recently. Simple PTA may result in downward embolization of occlusion material that may aggravate the symptoms or may turn the condition into a limb-threatening situation. Even in short occlusions, PTA may be insufficient to reopen the vessel, necessitating additional treatment such as stent placement (which does not result in better patency compared with balloon angioplasty alone). Reobstruction of stents, however, is more difficult to treat compared with simple restenosis. Eccentric calcified stenosis is frequently insufficiently treated. Because stenting is a technical but not necessarily a long-standing solution to such lesions, alternative techniques such as atherectomy may be considered if available. Unfortunately, these niche techniques are difficult to place in the market because of the costs involved, and some of the well-advanced devices such as the Simpson atherectomy catheter have been withdrawn from the market. (See Fig. 1)

Techniques

Balloon Angioplasty

Balloon angioplasty remains the working horse in femoropopliteal lesions. Modern angiographic units allow a built-in, fairly exact measurement of the true arterial diameter, and with the use of semicompliant balloons, adaptation to the diameter is well performed. We prefer not to grossly overdilate the artery in order to avoid dissection. Dilation times of 1–3 min are preferable by using pressure gauges. Balloon lengths of 2–4 cm are mainly used. In cases of major dissection, the first step should be an additional attempt to improve the result by prolonged balloon dilatation over 4–5 min; in many cases, the result will be improved by this cost-effective and simple approach (Fig. 1).

Stent Placement

The use of all kinds of stents should be limited to those cases in which balloon angioplasty in all its variations did not achieve a sufficient result. This is particularly true for occluding dissections. Other than in the iliac field, stents should not be used liberally.

The stented segment should be as short as possible. Balloon-expandable stents normally allow coverage of only short segments and might therefore be preferred for those lesions. In longer segments or in parts where bending of the artery is an issue, a self-expanding stent is advantageous if a stent cannot be avoided at all.

The overall results of femoral stenting are disappointing. New developments with drug-coated stents are on the way that allow elusion of drugs, such as rapamycin (Sirolimus) or taxol, from the stent surface. Especially for rapamycin, the first results from the coronary arteries are very promising, but no valid data yet exist on their use in the femorals. Radiation in stents, primarily at the time of insertion, did not show improved patency but was followed by an increased risk of thrombosis. Afterloading might therefore be a potential tool in the treatment of stent reobstruction.

Stent Grafts

Stent grafts still play a limited role in the femoropopliteal field. ePTFE-covered self-expanding stent grafts such as the Hemobahn device (Gore, Flagstaff, AZ, USA) yielded promising results in a multicenter trial even in the femoropopliteal field and stimulated the hope of offering a percutaneous alternative, especially for those patients presenting with long femoropopliteal occlusions. But there is also a risk of midterm or late rethrombosis.

Below the inguinal ligament, an ePTFE covering should be used exclusively because in animal experiments it has shown much less tendency to induce neointimal growth compared with Dacron covering. Other than in extraluminal bypasses, transcovering growth of tissue has been demonstrated, probably due to the long-segment wall contact between the stent graft and the original vascular lumen (2, 3).

A considerable disadvantage of stent grafts is that important collaterals frequently have to be covered by the full body of the stent graft. In the event of reocclusion, these collaterals will not be available anymore, which might cause aggravation of symptoms. This is particularly true for the popliteal artery, for which development of compensating collaterals is limited. Therefore, we favor limiting the use of stent grafts to the proximal two-thirds of the superficial femoral artery, especially in claudicants.

Results

Balloon Angioplasty

In femoropopliteal endovascular interventions (data taken from eight publications reporting on 1,469 procedures), the weighted average technical success was 90%, the complication rate was 4.3%, and the 3-year patency rate was 51% (1).

Long-term patency was positively influenced by a good outflow tract (two or three lower limb arteries), absence of diabetes, and absence of residual stenosis. The latter would

favor the use of stents, but unfortunately, there is no proof that stenting will improve overall patency.

Analyzing subgroups after femoral PTA, Huninck et al found different patencies for patients with stenotic and occlusive femoral lesions and good run-off (62% vs. 48% after 5 years) as well as those with poor run-off (stenoses: 62% vs. 43% after 5 years; occlusions: 43% vs. 27% after 5 years) (4).

Stents

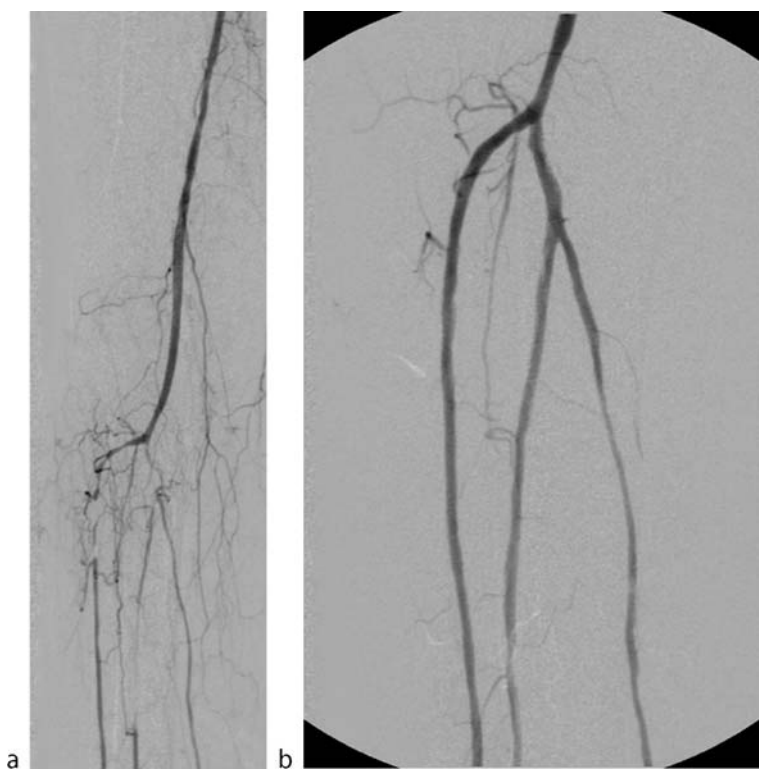
Follow-up results from stent implantation into the femoropopliteal arteries have not yielded improved results compared with balloon angioplasty alone. In a meta-analysis, Muradin et al found a 3-year patency of 63–66% after 3 years for stents, compared with 61% for PTA of stenoses. They also found, however, that in patients with more severe disease and more severe lesions, the patients achieved a higher benefit from stenting compared with those with less severe disease (5). In a randomized trial, Cejna and colleagues found no significant difference between patients who received PTA alone and those who received stenting (6).

Endoluminal radiation therapy with afterloading or beta irradiation as well as drug-eluding stents may change the overall results in the future. To date, stenting in the femoral arteries should be used as a bail-out therapy in the case of PTA failure. Failure, however, needs to be defined strictly as severe dissection refractory to prolonged balloon dilatation, antegrade dissection with increasing obstruction, or severe residual obstruction. Minor irregularities of the wall are not enough to justify stenting because treatment of restenosis is more difficult compared with treatment after PTA alone. (See Figs 3 and 4).

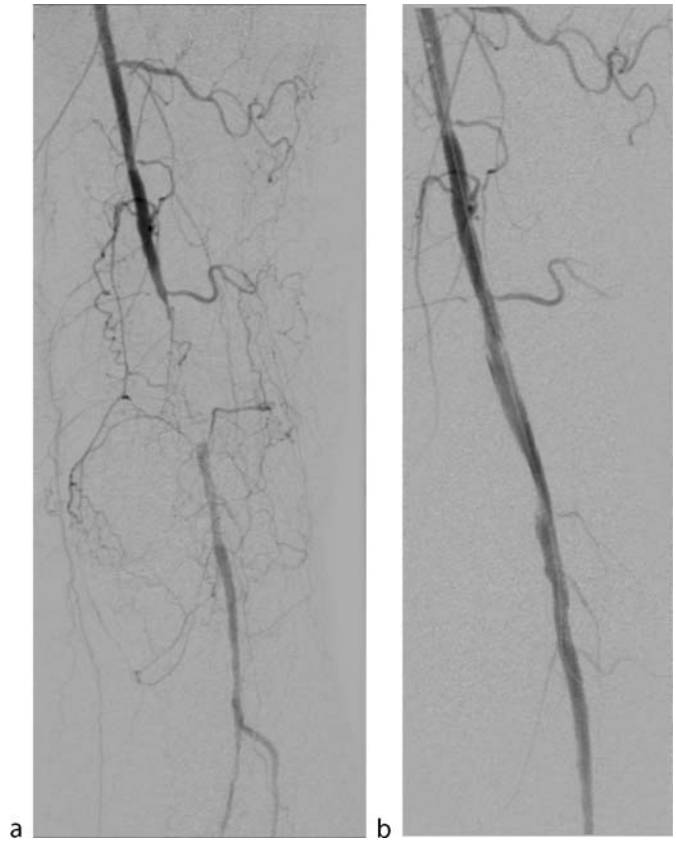
Stent Grafts

Little data exist on the usefulness of stent grafts in the femoropopliteal arteries. In a multicenter trial using the Hemobahn endoprosthesis, Lammer and colleagues achieved a primary patency of 90% after 6 months and 79% after 12 months with 80 limbs treated. Secondary patency was 93% at 12 months after treatment (7).

These encouraging results are in contrast to many single-center experiences in which endografts showed a high rate of thrombosis that was frequently due to development of stenoses adjacent to the stent graft.



Occlusion, Artery, Femoral. Figure 3 Infrapopliteal occlusion. (a) Occlusion of all lower limb arteries in their proximal segments in a patient with stage IV disease. (b) After mechanical passage and percutaneous transluminal angioplasty, all three arteries have been sufficiently recanalized. No peripheral embolization has occurred.



Occlusion, Artery, Femoral. Figure 4 (a) A 7-cm-long occlusion of the popliteal artery in a patient with stage IV disease. (b) After subintimal recanalization and percutaneous transluminal angioplasty, sufficient reopening with stent placement.

Complications

The nature and quality of complications in the femoropopliteal arteries do not differ principally from those in the aortoiliac area: dissection, perforation, and embolization of occluding material. With stents, the risk of early thrombosis was a problem in the very beginning but has since become rare with combined treatment that includes modern antiplatelet drugs.

In occlusions, the risk of embolization of the occluding material is the most potentially dramatic complication. Aspiration embolectomy in combination with selective thrombolysis is the treatment option of choice. Especially in claudicants, the risk therefore needs to be well balanced with the potential benefit.

Adjunctive Drug Regimen

In iliac and femoral PTA in claudicants, heparinization during the intervention and for 24 h after it—either by low-molecular-weight heparin or conventional heparin—is usually sufficient. A dose of 100 mg of aspirin daily is usually prescribed. After femoral stent placement or in patients with marked irregularities after PTA, heparinization may be prolonged up to 72 h, and an additional platelet inhibitor such as Clopidogrel is recommended for 4–6 weeks.

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Occlusion, Artery, Iliac

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Synonyms

Iliac artery occlusion; Iliac artery stenosis artery

Definition

An arterial obstruction of the iliac arteries is an occlusion or narrowing of an arterial segment between the aortic bifurcation and the groin, not including the common femoral artery (CFA).

Pathology/Histopathology

Occlusions are complete obstructions of the iliac arterial lumen due to thrombus formation, mainly onto a preexisting atherosclerotic plaque formation. In stenoses, the lumen is still patent but narrowed, with symptoms occurring with a diameter reduction of more than 50%.

Clinical Presentation

Unilateral claudication is the leading clinical symptom and has a predilection for the upper thigh. Depending on the collateral pathways, the walking distance may vary between a few meters and a few hundred meters. Severe ischemic syndromes are rare unless there is no additional involvement of the infrainguinal arteries.

Imaging

Many imaging modalities allow clinicians to diagnose iliac artery obstruction. Color-coded duplex sonography as well as magnetic resonance (MR) angiography, computed tomography (CT), CT angiography, and intra-arterial angiography are useful tools for detecting the location and extent of an iliac obstruction. Because of access limitations, angiography is preferably performed *via* transbrachial or contralateral access if the clinical findings suggest iliac artery obstruction.

Nuclear Medicine

Nuclear medicine plays no particular role in the diagnosis of aortic stenosis.

Diagnosis

Diagnosis is reliably achieved by angiography or MR angiography. Detection by duplex sonography is sometimes difficult due to unfavorable anatomic conditions.

Interventional Radiological Treatment

In the iliac segment, many lesions are amenable to percutaneous treatment with an acceptable outcome. Thus, a lesion's location and type must be taken into consideration before treatment is recommended. Whereas most lesions in the aortoiliac segment will be conducive to an endovascular approach, this is not generally true for femoropopliteal lesions. In addition, the risk of treatment is related to its location and has to be addressed before an endovascular approach is recommended.

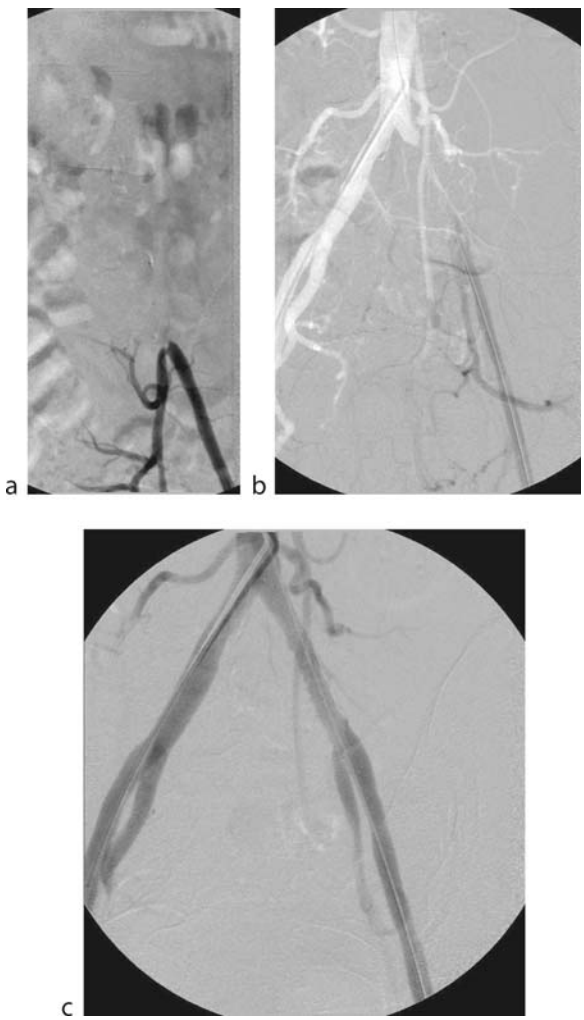
Type of Lesion

The morphology of the treated lesion will influence the technical outcome, follow-up results, and also the risk of treatment. The Transatlantic Inter-Society Consensus (TASC) document therefore introduced a classification system that tries to categorize lesions with regard to their accessibility to either percutaneous treatment or surgery: Type A lesions, for which the percutaneous approach is ideal, type B lesions, for which the percutaneous approach is still the preferred technique; type C lesions, for which a surgical approach should be preferred; and type D lesions, for which surgery is the option of choice. The TASC classification overrules older classifications because it takes into account all available and published techniques, including stent technology, which offer a much wider variation of treatment and are also effective tools for dealing with acute complications of balloon angioplasty such as occluding dissection and vascular rupture.

If we consider percutaneous therapy as the preferred method to deal with patients presenting with mild or

moderate claudications, treatment might be offered to those presenting with type A and B lesions but should be discussed in depth with patients with type C lesions, since the risk and the potential benefit of treatment will be related to the underlying morphology.

For iliac lesions, single stenoses up to 3 cm in length both in the common iliac artery (CIA) and external iliac artery (EIA) are classified as type A lesions, while single stenoses of 3–10 cm (not involving the CFA), double stenoses not longer than 5 cm each, and unilateral occlusions of the CIA are classified as type B lesions (Fig. 1).



Occlusion, Artery, Iliac . Figure 1 Occlusion of the left common iliac artery. (a) Occlusion of the left common iliac artery imaged *via* a retrograde access. The internal iliac artery is open. (b) After passage of the occlusion, the complete occlusion is visible with a stump at the orifice. (c) After primary stenting followed by careful balloon dilation, patency is fully restored.

Type C lesions include bilateral long stenoses (5–10 cm in length), unilateral EIA occlusions not extending into the CFA, and unilateral EIA stenoses extending into the CFA. More advanced lesions are classified as type D.

Using this classification, many iliac lesions will fall into the A and B groups, opening a potentially growing field for endovascular procedures performed on mild to moderate claudicants. Even in some type C lesions, percutaneous treatment has no major technical concerns, complication risks, or compromised outcomes. This is particularly the case for EIA occlusions not extending into the CFA. However, published data are lacking to back up this experience.

Multilevel Disease

Even mild symptoms may be associated with multilevel disease (e.g., iliac stenosis and a well-collateralized femoropopliteal lesion). There is some chance that exclusive intervention in the iliac region may be sufficient to improve the clinical situation. If present, multilevel disease does not preclude treatment in these patients.

Assisting Forms of Treatment

It is widely accepted that well-conducted physical exercise should precede any type of interventional treatment and that cessation of smoking is mandatory. Nevertheless, it is also true that in many institutions it is very difficult to find an infrastructure that allows teaching of state-of-the-art physical exercise to claudicants, and as far as smoking is concerned, there is a major difference between willing and doing.

Moreover, even with state-of-the-art exercise, young patients will not recover completely from claudications in all their activities, including sports. The process will be longer and compromise their abilities in their professional lives. Therefore, it might be discussed whether young and active patients should be vigorously kept to the axiom of "physical exercise first" or whether invasive treatment might be offered to this group of patients even as a first approach.

Treatment Options with Relation to Location and Lesion

The aorta and the iliac arteries have been a primary field for percutaneous interventions for a long time. Easy access to the lesion, the relatively large diameter of the target vessels, and the comparably benign outcomes even with major complications contribute to the wide acceptance of percutaneous interventions in that area. Over the years, the indications have been extended and now include not only stenotic but also occlusive disease and treatment of

aneurysms. The introduction of vascular stents has especially been very helpful for overcoming major problems and offers a tool to treat major complications that otherwise remained a domain for surgical repair.

Iliac occlusive disease accounts for approximately one-third of occlusive arterial disease, while two-thirds is located subinguinally. However, iliac percutaneous transluminal angioplastry (PTA) plays a major role in interventional radiological routines because it is well-established in most institutions and by most surgeons, many lesions are amenable to percutaneous treatment, and technical as well as clinical results are satisfying.

Clinically, intermittent claudication starting in the upper thigh in combination with lower limb claudication is the leading symptom. Erectile dysfunction in men may also be present. In isolated iliac lesions, critical ischemia is rare if not combined with additional subinguinal disease. Rarely, blue-toe syndrome might be present if cholesterol embolization has occurred from an ulcerated plaque in the iliac axis.

Weakened femoral pulses and a reduced ankle-arm index are simple clinical signs that can indicate iliac obstruction and that can be verified by direct or poststenotic color-coded or duplex studies. For planning a percutaneous intervention, angiography is still the most helpful procedure.

Clinical indications for treatment depend on the severity of symptoms and how they limit the daily life of an individual patient.

Stent placement in stenotic lesions should be indicated from a technical point of view if angioplasty remains insufficient, as defined by visibly poor outflow or major pressure gradients. Because follow-up data are now available showing that iliac stent placement is safe, a liberal approach is justified, although primary stenting of stenoses does not seem to be generally recommended because of socioeconomic reasons and potential follow-up problems.

Furthermore, primary stenting is not beneficial over successful balloon dilation (8). Thus, primary stenting is recommended only if PTA fails or if technical requirements compromise the success of simple PTA, such as with iliac occlusions.

Iliac Artery Stenoses

Although balloon angioplasty has proved to be an effective procedure in treating iliac stenoses in particular, the indication for stent placement should be restricted to lesions that are not primarily amenable to PTA alone. An inadequate postangioplasty result has been suggested as a general indication for stent placement, although the term "inadequate" remains ill-defined. Residual pressure gradients are certainly a useful way to assess the angioplasty result, but it is still unclear what the borderline gradient ultimately requiring additional intervention is. Moreover, the decision should not be made without referring both to morphologic criteria and visibly reduced flow.

Long-segment stenoses with irregular surfaces, aneurysm formation, or markedly ulcerated plaques may be included in the group of complex lesions. Eccentric stenoses and ostial lesions with extension to the aortic bifurcation are known to not respond well to balloon angioplasty.

A stenotic lesion may respond well to balloon inflation but may collapse after the balloon is deflated.

Complications of balloon angioplasty can be treated well by stent placement. These include intramural hematoma and flow-obstructing dissections complicating PTA, which may be an acute indication for stent placement in order to maintain the vascular lumen, obviating emergency surgery.

Iliac restenosis after previous PTA does not require stent placement in general since there is no proof that stenting prevents restenosis under those circumstances. However, stenting may be considered from a technical point of view in cases in which the result of balloon angioplasty remains compromised.

An inadequate result after state-of-the-art balloon dilatation of the iliac stenoses is a prerequisite for secondary stent placement in stenotic lesions.

Iliac Artery Occlusions

Percutaneous treatment of iliac occlusions is technically feasible. In cases of acute thrombosis, thrombolysis as an alternative to surgical thrombectomy might precede PTA of an underlying lesion. Mechanical thrombectomy *via* percutaneous access is still in its infancy and cannot be recommended as a routine approach because potential risks such as downward or cross-over embolization are possible, and no data are yet available to determine the overall complications of such an approach.

In chronic occlusions with an occlusion time exceeding 3 months, balloon angioplasty alone, thrombolysis with subsequent balloon angioplasty, and elective stenting or mechanical passage of the occlusion followed by primary stent implantation have been described as alternative techniques.

Metallic stents—and self-expandable endoprostheses in particular—offer a new concept of percutaneous revascularization in chronic iliac occlusion, which we believe is a primary indication (9). Self-expandable stents are used to cover the occluding thrombotic material, thereby preventing peripheral dislodgement, a well-known complication of percutaneous recanalization of occlusions.

Indications for the use of metallic stents into arteries should always be technical. The type and morphology of

the lesion, the outcome of balloon angioplasty, and complicated situations are important considerations, although stenting has been tested and shown to be a safe procedure.

This is particularly true for treatment of restenosis. There is currently no proof that stent placement is more effective in preventing restenosis than balloon angioplasty with good technical success. Furthermore, there is no proof that in a restenosed vessel, the use of a stent would be beneficial to prevent recurrent stenosis.

Technical Considerations in the Iliac Arteries

Balloon Dilatation

Balloon dilatation of the iliac arteries is relatively simple to perform. A retrograde transfemoral approach provides the easiest access to that type of stenotic lesions. Crossover dilatation may be performed in special indications such as double-sided stenoses in the event that both lesions should be dilated in one session, or if an external iliac stenosis extends far down into the CFA. After the diseased segment is carefully traversed, a suitable balloon is placed across the lesion, and dilatation is performed either manually or by using a pressure-monitoring syringe. The size of the balloon can be depicted either by film measurements or in digital subtraction angiography images by use of graduated catheters that allow fairly exact measurement of vessel size.

There is wide agreement that both the hemodynamic relevance of a lesion as well as post-PTA success can be accurately monitored by measuring the pressure gradient across the lesion. However, there is some dispute about the level of the pressure. A mean pressure gradient of 10 mmHg and less after peripheral drug-induced vasodilation is accepted by most authors to indicate successful PTA even if the morphological result is not excellent. Some authors use a systolic gradient of 10 mmHg.

There is no uniform agreement on whether PTA of simple iliac lesions requires any additional anticoagulation afterward. We regularly keep our patients on full heparinization (500–1,000 IU/h) for 12–24 h and recommend lifetime aspirin therapy (100 mg/day).

Stent Implantation

If balloon angioplasty fails by morphologic and functional criteria, stent implantation can be considered in stenotic lesions. The technique depends on the type of stent used. There is no stent preference because most clinical series have shown similar results. The length and location of the lesion, the experience of the investigator, and the availability of appropriately sized stents are important details that may lead to the preference of one or another type.

Exact placement is mandatory to avoid major complications, especially if the stent has to placed close to the aortic bifurcation. While self-expanding Wallstents can be corrected during placement to a limited extent, balloon-expandable stents and self-expanding nitinol stents cannot undergo correction of their localization once inflation of the balloon has begun.

Chronic iliac artery occlusions are primarily indicated for stent placement. We avoid predilatation and place the stent directly into the occluded segment. After stenting, careful balloon dilatation is performed to avoid dislodgement of occluding material.

Atherectomy

Directional atherectomy does not play a major role in treating iliac arterial disease. This is because the ratio of introduction and working diameters in most atherectomy systems is relatively low, which requires a considerably large puncture to achieve atherectomy in larger iliac diameters of 8–12 mm. The Simpson atherectomy catheter—which is no longer available commercially—required an 11F sheath to sufficiently treat iliac lesions. Atherectomy plays a more important role in the recanalization of stent reobstruction in order to debulk stents from reobstructing neointimal tissue.

Stent Reobstruction

In cases of in-stent stenosis, directional atheterectomy or balloon dilation are both recommended. If PTA is used, a balloon size according to the outer diameter of the stent in place is recommended in order to compress the neointimal intima to a maximum, especially if occurring within a self-expanding stent that does not allow overexpansion. If a balloon-expandable stent has been used, slight overdilation of the stent is recommended to gain a larger diameter despite neointimal tissue. Some authors prefer atherectomy to debulk the stent. This is achievable in smaller stents such as in the femorals, but it may require very large instruments in iliac stents.

In stent occlusion, treatment is more difficult. In acute occlusions early after placement, technical problems are mainly responsible, and it is mandatory to overcome these problems to maintain long-term success. Recent thrombosis should be treated by thrombolysis followed by PTA and/or additional stent placement.

Late occlusion is mainly due to reobstruction by neointima within or adjacent to the stent. There is not much published experience about how to treat complete stent occlusion at a chronic stage. Thrombolysis, atherectomy, and mechanical aspiration followed by balloon

angioplasty are possible techniques. One of the simpler techniques is the stent-in-stent technique: After traversal of the occluded stent (which is usually easy to accomplish), a stent is placed within the occluded segment, bridging it at both ends. It is then carefully dilated with a tendency to underdilation of 1–2 mm. This is considered safe in order to avoid embolization of occlusion material.

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Occlusion, Artery, Popliteal

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Synonyms

Infrainguinal arterial obstruction

Definition

An arterial obstruction of the infrainguinal arteries is an occlusion or a narrowing of an arterial segment between the groin and the lower limbs.

Pathology/Histopathology

Occlusions are complete obstructions of the infrainguinal arterial lumen and are due to thrombus formation, mainly on preexisting atherosclerotic plaque. In stenoses, the lumen is still patent but narrowed, with a diameter reduction of more than 50% causing symptoms. Alternatively, acute occlusions occur because of thrombotic emboli from the heart.

Clinical Presentation

Clinical indications for treatment include claudication, pain at rest, and nonhealing ulceration. Severity of the symptoms depends on the acuteness of the occlusion, the condition of collateral pathways, the lesion's location, and concomitant disease such as diabetes or renal insufficiency.

Imaging

Many imaging modalities allow the physician to diagnose infrainguinal artery obstruction. Color-coded duplex sonography as well as magnetic resonance angiography (MRA), computed tomography, computed tomography angiography (CTA), and intraarterial angiography are useful tools to detect the location and extent of an iliac obstruction.

Nuclear Medicine

Nuclear medicine plays no particular role in the diagnosis of aortic stenosis.

Diagnosis

Diagnosis is reliably achieved by angiography or MRA. Detection by duplex sonography is a reliable tool for the femoropopliteal segment but is sometimes limited for detection and exact lesion description in the lower limbs. In stage IV (Fontaine) disease, MRA may be limited because of low arterial flow, movement artifacts, or artifacts due to superimposed venous flow.

Interventional Radiological Treatment

Endovascular versus Surgical Treatment

Endovascular therapy is known to be of low invasiveness with good technical success, achieving fair overall patency. In femoropopliteal endovascular interventions (data

taken from eight publications reporting on 1,469 procedures), the weighted average technical success was 90%, the complication rate was 4.3%, and the 3-year patency rate was 51%. Stents did not improve patency, showing a patency of 58% after 3 years (1).

Surgery offers acceptable results for distal reconstruction; an average 5-year patency of 80% for vein bypasses and 65–75% for expanded polytetrafluoroethylene (ePTFE) bypasses has been reported. The combined mortality and amputation risk was calculated to be about 2.2% for aortobifemoral reconstructions and 1.4% for femoropopliteal reconstructions (1).

Location of Lesion

Claudication is mainly related to lesions in the aortoiliac and femoropopliteal regions. It is unlikely to be due to infrapopliteal lesions, and there is general agreement that treatment below the knee should be strictly limited to patients with critical limb ischemia, i.e., stages III and IV (Fontaine) or categories 4–6 (Rutherford).

Type of Lesion

The morphology of a treated lesion influences the technical outcome, follow-up results, and also the risk of treatment. Therefore, the TransAtlantic Inter-Society Consensus (TASC) document introduced a classification system that tries to categorize lesions with regard to their accessability to either percutaneous treatment or surgery: type A lesions, which are ideal for a percutaneous approach; type B lesions, in which the percutaneous approach is still the preferred technique; type C lesions, in which a surgical approach should be preferred; and type D lesions, in which surgery is the option of choice. The TASC classification overrides older classifications because it takes into account all of the available and published techniques (including stent technology), which offer a much wider variation of treatment as well as effective tools to deal with acute complications of balloon angioplasty, such as occluding dissection and vascular rupture.

If we consider percutaneous therapy as the preferred method to treat those patients presenting with mild or moderate claudication, treatment might be offered to those presenting with type A and B lesions, but should be discussed in depth with patients with type C lesions, as the risk and the potential benefit of treatment are related to the underlying morphology.

In the femoropopliteal field, type A lesions are single stenoses up to 3 cm in length not involving the very proximal superficial femoral and the distal popliteal artery. Type B lesions include stenoses 3–5 cm in length, heavily calcified stenoses, multiple lesions (each up to 3 cm), and lesions with no sufficient tibial run-off (the latter are unlikely to meet the criteria of mild or moderate claudication). Type C lesions are classified as stenoses or occlusions longer than 5 cm and multiple midsize lesions (3–5 cm). Total common femoral, superficial femoral, and popliteal occlusions are classified as type D lesions. There was some dissenting discussion on the definition of type B lesions: Interventional radiologists represented by the Cardiovascular and Interventional Radiological Society of Europe wished to express their assumption that even longer lesions of up to 10 cm may be justified as being classified type B instead of type C; they claimed that the reported results were mainly due to underdeveloped techniques and instruments that have substantially improved and that no data exists comparing the efficacy of percutaneous transluminal angioplasty (PTA) versus bypass surgery for lesions between 4 and 10 cm.

Other than in the iliac area, few femoral lesions meet the criteria for types A and B lesions, especially if limited to 5 cm in length. Thus, few patients with mild or moderate claudication due to femoropopliteal lesions will be ideal candidates for percutaneous treatment. Moreover—without limiting the importance of the TASC document, which certainly means a step forward in the joint approach to peripheral vascular disease—the morphological classification does not take into account some technical considerations that depend on the age and composition of a lesion. Particularly in femoral occlusions, the degree of organization of the occluding thrombus or the composition of the lesion with the original stenosis at the proximal and distal ends or in the middle are factors that are not very predictable but may influence the technical outcome of the intervention or its complication rate (for instance, distal embolization might aggravate symptoms). Other than in the iliac arteries, the liberal use of stents and stent grafts may help overcome a failed balloon angioplasty and resolve the technical outcome, but it does not achieve an improved long-term efficacy, and it may start a lifetime dependency on recurrent interventional or surgical procedures. These associated potential drawbacks have to be carefully balanced against the potential benefits and need to be discussed in depth with the patient before treatment is performed, especially in association with mild or moderate claudication.

These considerations mainly restrict the use of endovascular treatment in femoropopliteal lesions to stages IIb and IIa patients presenting with type A or less-pronounced type B lesions.

Assisting Forms of Treatment

It is widely accepted that well-conducted physical exercise should precede any type of interventional treatment and that cessation of smoking is mandatory. Nevertheless, it is also true that in many institutions it is very difficult to

find an infrastructure that allows instruction of state-of-the-art physical exercise for claudicants. And as far as smoking is concerned, there is a major difference between wanting to stop and actually stopping.

Moreover, even with state-of-the-art exercise, young patients will not recover completely from claudications in all activities, including sports. The process will be long and will compromise their abilities in their professional lives. Therefore, it should perhaps be discussed whether young and active patients, especially, should be held to the axiom of "physical exercise first" or whether invasive treatment might be offered even as a first approach in this group of patients.

Treatment Options with Relation to Location and Lesion

Treatment of femoropopliteal lesions in claudicants has to be seen as more critical and less liberal compared with the iliac region. The main reasons are less favorable technical success, a higher complication rate, and poorer long-term success. There are many more lesions in the femoropopliteal arteries that do not meet the criteria for suitability for endovascular treatment. On the other hand, the versatility of endoluminal techniques opens treatment options in many particular lesions, and taking clinical symptoms as the only criteria to indicate or exclude treatment is not justified because depending on the type of lesion, a simple and limited intervention can mean considerable improvement for the patient.

Additional Morphological factors (not Included in the TASC Classfication)

Femoropopliteal occlusions may especially become a source of complications, particularly if they occurred recently. Simple PTA may result in downward embolization of occlusion material that may aggravate the symptoms or may turn the condition into a limb-threatening situation. Even in short occlusions, PTA may be insufficient to reopen the vessel, necessitating additional treatment such as stent placement (which does not result in better patency compared with balloon angioplasty alone). Reobstruction of stents, however, is more difficult to treat compared with simple restenosis. Eccentric calcified stenosis is frequently insufficiently treated. Because stenting is a technical but not necessarily a long-standing solution to such lesions, alternative techniques such as atherectomy may be considered if available. Unfortunately, these niche techniques are difficult to place in the market because of the costs involved, and some of the well-advanced devices such as the Simpson atherectomy catheter have been withdrawn from the market.

Techniques

Balloon Angioplasty

Balloon angioplasty remains the working horse in femoropopliteal lesions. Modern angiographic units allow a built-in, fairly exact measurement of the true arterial diameter and by use of semicompliant balloons, adaptation to the diameter is well performed. We prefer not to grossly overdilate the artery in order to avoid dissection. Dilation times of 1–3 min are preferable by using pressure gauges. Balloon lengths of 2–4 cm are mainly used. In cases of major dissection, the first step should be an additional attempt to improve the result by prolonged balloon dilatation over 4–5 min; in many cases, the result will be improved by this cost-effective and simple approach (Fig. 1).

Stent Placement

The use of all kinds of stents should be limited to those cases in which balloon angioplasty in all its variations did not achieve a sufficient result. This is particularly true for occluding dissections. Other than in the iliac field, stents should not be used liberally.

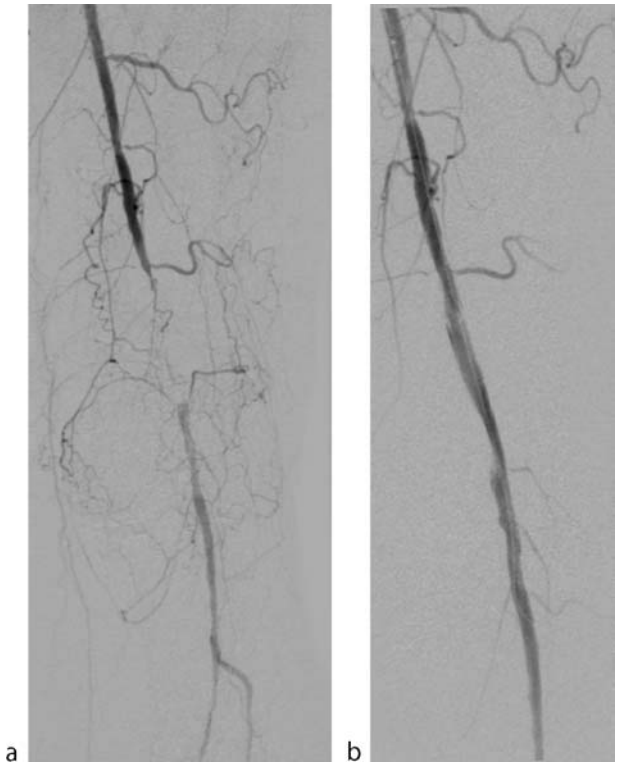
The stented segment should be as short as possible. Balloon-expandable stents normally allow coverage of only short segments and might therefore be preferred for those lesions. In longer segments or in parts where bending of the artery is an issue, a self-expanding stent is advantageous if a stent cannot be avoided at all.

The overall results of femoral stenting are disappointing. There are new developments with drug-coated stents on the way that allow elusion of drugs, such as rapamycin (Sirolimus) or taxol, from the stent surface. Especially for rapamycin, the first results from the coronary arteries are very promising, but no valid data yet exist on their use in the femorals. Radiation in stents, primarily at the time of insertion, did not show improved patency but was followed by an increased risk of thrombosis. Afterloading might therefore be a potential tool in the treatment of stent reobstruction.

Stent Grafts

Stent grafts still play a limited role in the femoropopliteal field. ePTFE-covered self-expanding stent grafts such as the Hemobahn device (Gore, Flagstaff, AZ, USA) yielded promising results in a multicenter trial even in the femoropopliteal field and stimulated the hope of offering a percutaneous alternative, especially for those patients presenting with long femoropopliteal occlusions. But there is also a risk of midterm or late rethrombosis.

Below the inguinal ligament, ePTFE covering should be used exclusively because in animal experiments it has



Occlusion, Artery, Popliteal. Figure 1 (a) A 7-cm-long occlusion of the popliteal artery in a patient with stage IV disease. (b) After subintimal recanalization and percutaneous transluminal angioplastyPTA, sufficient reopening with stent placement.

shown much less tendency to induce neointimal growth compared with Dacron covering. Other than in extraluminal bypasses, transcovering growth of tissue has been demonstrated, probably due to the long-segment wall contact between the stent graft and the original vascular lumen (2, 3).

A considerable disadvantage of stent grafts is that important collaterals frequently have to be covered by the full body of the stent graft. In the event of reocclusion, these collaterals will not be available anymore, which might cause aggravation of symptoms. This is particularly true for the popliteal artery, for which development of compensating collaterals is limited. Therefore, we favor limiting the use of stent grafts to the proximal two-thirds of the superficial femoral artery, especially in claudicants.

Results

Balloon Angioplasty

In femoropopliteal endovascular interventions (data taken from eight publications reporting on 1,469 procedures), the weighted average technical success was 90%, the complication rate was 4.3%, and the 3-year patency rate was 51% (1).

Long-term patency is positively influenced by a good outflow tract (two or three lower limb arteries), absence of diabetes, and absence of residual stenosis. The latter would favor the use of stents, but unfortunately, there is no proof that stenting will improve overall patency.

Analyzing subgroups after femoral PTA, Huninck et al. found different patencies for patients with stenotic and occlusive femoral lesions and good run-off (62% versus 48% after 5 years) as well as those with poor run-off (stenoses: 62% versus 43% after 5 years; occlusions: 43% versus 27% after 5 years) (4).

Stents

Follow-up results from stent implantation into the femoropopliteal arteries have not yielded improved results compared with balloon angioplasty alone. In a meta-analysis, Muradin et al. found a 3-year patency of 63–66% after 3 years for stents, compared with 61% for PTA of stenoses. They also found, however, that in patients with more severe disease and more severe lesions, the patients achieved a higher benefit from stenting compared with those with less severe disease (5). Cejna and colleagues found no significant difference between

patients who received PTA alone and those who received stenting in a randomized trial (6).

Endoluminal radiation therapy with afterloading or β irradiation as well as drug-eluding stents may change the overall results in the future. To date, stenting in the femoral arteries should be used as a bail-out therapy in the case of PTA failure. Failure, however, needs to be defined strictly as severe dissection refractory to prolonged balloon dilatation, antegrade dissection with increasing obstruction, or severe residual obstruction. Minor irregularities of the wall are not enough to justify stenting as treatment of restenosis is more difficult when compared with treatment after PTA alone.

Stent Grafts

Little data exist on the usefulness of stent grafts in the femoropopliteal arteries. In a multicenter trial using the Hemobahn endoprosthesis, Lammer and colleagues achieved a primary patency of 90% after 6 months and 79% after 12 months with 80 limbs treated. Secondary patency was 93% at 12 months after treatment (7).

These encouraging results are in contrast to many single-center experiences in which endografts showed a high rate of thrombosis that was frequently due to development of stenoses adjacent to the stent graft.

Complications

The nature and quality of complications in the femoropopliteal arteries do not differ principally from those in the aortoiliac area: dissection, perforation, and embolization of occluding material. With stents, the risk of early thrombosis was a problem in the very beginning but has since become rare with combined treatment that includes modern antiplatelet drugs.

In occlusions, the risk of embolization of the occluding material is the most potentially dramatic complication. Aspiration embolectomy in combination with selective thrombolysis is the treatment option of choice. Especially in claudicants, the risk therefore needs to be well balanced with the potential benefit.

Adjunctive Drug Regimen

In iliac and femoral PTA in claudicants, heparinization during the intervention and for 24 h after it—either by low-molecular-weight heparin or conventional heparin—is usually sufficient. A dose of 100 mg of aspirin daily is usually prescribed. After femoral stent placement or in patients with marked irregularities after PTA, heparinization may be prolonged up to 72 h, and an additional platelet inhibitor such as Clopidogrel is recommended for 4–6 weeks.

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Occlusion, Bile Ducts

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Synonyms

Bile duct obstruction; Cholestasis; Obstruction of the biliary tree

Definition

Any condition in which there is a complete obstacle to the biliary flow.

Pathology and Histopathology

Cholestasis may be related to mechanical, cellular, and metabolic causes.

Mechanical biliary obstruction may be due to intrinsic or extrinsic obstruction of bile flow, which can occur either at the intrahepatic or extrahepatic level (Table 1).

Occlusion, Bile Ducts. Table 1 Mechanical biliary obstruction

Intrahepatic	Intrinsic	Congenital cause • Biliary atresia • Alagille syndrome • Intrahepatic calculi complicating Caroli disease • Cystic fibrosis
		Benign cause • Intrahepatic stones • Sclerosing cholangitis • Biliary parasitosis
		Malignant cause • Cholangiocarcinoma
	extrinsic	Benign and malignant tumors
Extrahepatic	Intrinsic	Congenital anomalies • Choledochal atresia • Choledochal cysts
		Benign conditions • Gallstones • Parasitosis • Cholangitis • Inflammatory strictures • Postinterventional strictures • Vater's papillary stenosis • ▶AIDS-related cholangiopathy • ▶Biliary tuberculosis • ▶Sump syndrome
		Malignant tumors • Cholangiocarcinoma • Vater's papillary carcinoma
	extrinsic	Benign causes • ▶Mirizzi syndrome • Enlarged lymph nodes • Pancreatitis • Local benign tumors and cysts
		Malignant conditions • Gallbladder carcinoma • Malignant lymphadenomegalies • Pancreatic head carcinoma • Stomach and colon cancer

Overall, gallstones are the most common cause of biliary obstruction.

Intrahepatic intrinsic biliary obstruction can be due to: congenital affections (biliary atresia, Alagille syndrome, intrahepatic calculi complicating Caroli disease and cystic fibrosis), benign conditions (intrahepatic ductal stones, sclerosing cholangitis, and biliary parasitosis), and malignant conditions, with cholangiocarcinoma as the most important malignant cause. Extrinsic causes of intrahepatic obstruction include compression by benign and malignant tumors. Extrahepatic intrinsic biliary obstruction can be due to: congenital anomalies (choledochal atresia, choledochal cysts), benign conditions (gallstones representing the first cause, parasitosis, cholangitis, inflammatory and postinterventional strictures, Vater's papillary stenosis, AIDS-related cholangiopathy, biliary tuberculosis, sump syndrome), and malignant tumors (cholangiocarcinoma, and Vater's papillary carcinoma). Extrinsic compression of extrahepatic biliary tract includes benign conditions (Mirizzi syndrome, enlarged lymph nodes, pancreatitis, local benign tumors, and cysts), and malignant conditions (gallbladder carcinoma, malignant lymphadenomegalies, pancreatic head carcinoma, stomach and colon cancers) (1).

Clinical Aspects

Symptoms are determined by abnormal elevation of primary and secondary bile products in blood and the absence of the extrinsic intestinal bile function. Additionally, cholangitis may represent a concomitant complication in biliary obstruction. Common symptoms are jaundice, pale coloured stools, dark urine, itching, fever, and right upper quadrant pain. Blood tests show increase of bilirubin, alkaline phosphatase, and hepatic enzymes.

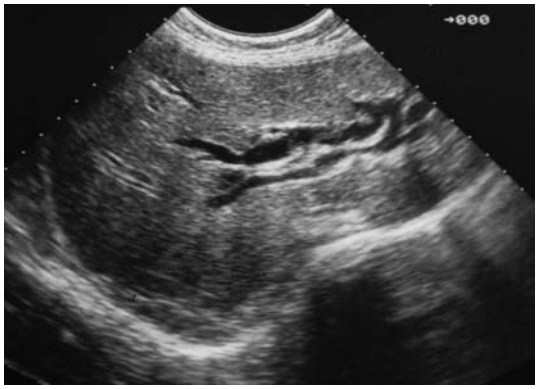
Imaging

Plain radiographs are of limited value to detect abnormalities in the biliary system. Frequently, calculi are not visualized because of their low radiopacity.

Biliary obstruction results in dilatation of bile ducts involving the biliary tree proximally to the obstruction; depending on the site of obstruction, the bile ducts dilatation may involve an isolated lobe or segment or both lobes. Especially in biliary stone disease, mostly both lobes present ductal dilatation. Nevertheless, the absence of bile ducts dilatation does not exclude recent or intermittent obstruction.

Ultrasound (US) is the method of first choice to establish the presence and extent of bile duct dilatation (Fig. 1). While peripheral ductal branches can be visualized only if dilated, the normal common bile duct is easily displayed (2). In general, US is able to establish the level of obstruction and reveal the cause of obstruction, as intrahepatic calculi, gallbladder stones, lymphadenopathy, hepatic and pancreatic masses. The demonstration of common bile duct calculi is strongly dependent on their location within the biliary system; in fact, calculi in the lower part of the common bile duct may be obscured by duodenal gas.

Computed tomography (CT) is more accurate than US in determining the specific cause and level of obstruction, but it is of limited value in detecting radiolucent bile duct stones. On the other hand, US can



Occlusion, Bile Ducts. Figure 1 Biliary dilatation. Ultrasound can easily demonstrate dilatation of both intrahepatic and extrahepatic bile ducts in mechanical biliary obstruction.

be significantly limited in the differentiation of cholangiocarcinoma from sludge accumulation since both appear hypoechoic. In these cases, the different enhancement behaviour (absence of contrast enhancement in case of sludge) allows differential diagnosis on CT.

With the advent of easy applicable magnetic resonance (MR) techniques displaying fluid-filled structures (i.e., heavily T2-weighted, snap-shot techniques, "MR-hydrography"), MR cholangiopancreatography (MRCP) is the reference standard for noninvasive imaging of the biliary tree.

In comparison to invasive endoscopic retrograde cholangiopancreatography (ERCP), MRCP provides noninvasive detailed evaluation of the biliary tree. Since MRCP is generally combined with complementary cross-sectional MR a comprehensive evaluation of in- and extrinsic factors that might affect the biliary tree is possible. With high-resolution techniques in- and extrinsic bile duct, obstructions and dilatations can be visualized by MRCP with a superior high sensitivity and specificity. Since the MR visualization of ducts is strongly correlated to the presence of fluid (bile) within the lumen, severe strictures, sludge, small stones or postoperative scars and clips may appear as short segments of signal void. In general, this condition can be clarified by additional unenhanced and contrast-enhanced T1- and T2-weighted axial (and/or multiplanar) sequences. In characterizing the features of the biliary tree, the same characteristics already known from cholangiography may be applied to MRCP. In general, stone-related obstruction can be displayed easily with the concave transition from fluid-filled ductal lumen to the stone and the usually smooth, regular outlining of the lumen. An irregular tapering of the lumen can often be seen in malignant obstructions due to an extrinsic or infiltrating mass that can be uncovered by cross-sectional imaging. Typical examples for that are tumors of the pancreatic head affecting the common bile duct alone or together with the pancreatic duct (so called "double duct sign," in about 5% of cases of pancreatic cancer). In case of such malignancies, MR can provide a comprehensive evaluation in terms of staging for intrahepatic metastases, lymph node metastasis, peritoneal tumour spread, and vascular encasement. However, comparing MR and multidetector CT there is no significant difference in staging of biliary and pancreatic malignancies regarding recent literature.

Due to the noninvasive, high diagnostic accuracy of MRCP, in most centres percutaneous (PTC) and endoscopic procedures (ERCP) are performed in selected cases for diagnostic purpose (e.g., tissue sampling by brush) and interventional procedures (e.g., balloon dilatation, stone extraction, stent placement) avoiding pure diagnostic interventions which may cause procedure related complications in up to 5% (e.g., acute pancreatitis) (1).

In patients with external biliary drainage (i.e., Kehr, Bracci), trans-catheter cholangiography is performed to evaluate the placement of the catheter, the patency of the common bile duct and the transpapillar drainage.

Nuclear Medicine

In patients with biliary obstruction, cholescintigraphy with ^{99m}Tc IDA (iminodiacetic acid) or analogs demonstrates a "congestion" of the radiotracer mostly associated with a normal hepatic distribution/activity in absence of delayed (>1 h after tracer injection) biliary activity.

Interventional Radiology

Radiologic interventional procedures in biliary obstruction may be performed using either a percutaneous or an endoscopic approach or a combination of both (i.e., "rendevouz procedures"). Possible interventions are catheter placement, biliary stenting, balloon dilatation of biliary strictures, stone removal, and tissue sampling.

Stone removal is the most common biliary intervention and generally performed by endoscopy. Usually a papillotomy is necessary, followed by stone extraction by balloon or basket instruments guided by the endoscope. Larger stones (>2 cm in diameter) are less prone to endoscopical removal and present a higher risk of perforation, bleeding, secondary pancreatitis, and cholangitis (3, 4). In the era of advanced endoscopic techniques, the use of percutaneous transhepatic radiological interventions is restricted to the treatment of intrahepatic stones and cholangitis where an internal–external drainage may be needed to relieve the biliary system. Sequentially, a stone can be removed or an

obstruction can be treated. In cases of unsuccessful or impractical endoscopic maneuvers, stone removal, or stone fragmentation and stent placement are also percutaneously possible.

Balloon dilatation can be necessary both in patients with benign and malignant biliary strictures. Stenoses are safely and minimally invasively treated by balloon dilatation. Among the benign strictures biliary-enteric anastomotic strictures respond best to dilatation whereas a temporarily support by a drainage tube may enhance the result (5). In case of recoiling, redilatation may be necessary. Malignant strictures, due to the high radial force compressing the ductal system or the intruding mass within the duct, often require the combination of balloon dilatation, temporarily drainage and stent placement.

Stenting of the biliary tract is performed both in benign and malignant stenoses. There are three types of stents, which are used in biliary intervention: plastic endoprostheses (polyethylene stents), balloon-expandable metallic stents, and self-expandable metallic stents with and without covering. They can be placed either percutaneously or endoscopically. Metallic stents become strongly adherent to the bile duct and are impossible to remove. In the setting of malignancy, stents are mainly indicated for the palliation of patients with unresectable tumors. In such cases, metallic stents are preferred because they remain patent much longer than polyethylene stents and usually a single session of metal stenting maintain ductal patency for the reminding lifetime of the patients (6). Occlusion of metallic stents usually is determined by tumoral in-growth and can create the need for further interventions.

For treatment of benign biliary strictures, plastic endoprostheses are preferred, since they are needed in most cases only for limited time. Nevertheless, in chronic recurrent biliary stenoses where a long-term treatment is needed, they may be occluded by biliary encrustations, while in metallic stents reactive epithelial hyperplasia may occur.

Diagnosis

The diagnosis of biliary obstruction is suggested by the presence of jaundice, hypocholic stools, dark urine, itching, increase of serum levels of bilirubin, with prevalence of conjugated bilirubin, and alkaline phosphatase.

Imaging has the role to confirm biliary obstruction and to establish the level and the cause of obstruction. US is performed in the initial evaluation to demonstrate bile duct dilatation, the level of obstruction and sometimes the cause of obstruction, such as stones, hepatic masses, pancreatic masses, lymphadenopathies. The main limitations of US are the inconsistent visualization of the distal tract of the choledochal duct and the retroperitoneum. CT provides a more comprehensive examination that permits evaluation of the liver, biliary tree, pancreas, portal and retroperitoneal lymph nodes, and vascular structures, but it has a limited value in case of radiolucent stones of common bile duct. MRCP offers a noninvasive accurate assessment of the biliary tract. Associated cross-sectional images adequately demonstrate extra-ductal findings (1). Invasive techniques include ERCP and PTC. Currently they should be performed only to guide interventional procedures and cannot be recommended as diagnostic imaging techniques (2).

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Occlusion, Bowel in Childhood

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Synonyms

Small bowel obstruction (SBO); Large bowel obstruction

Definition

The lumen of the small or large bowel is occluded. This may be due to extrinsic compression of the bowel, an intrinsic abnormality of the wall or lumen of the bowel, or due to a filling defect in the lumen of the bowel. It may be congenital or acquired. Complete occlusion of the bowel is described as 'obstruction'.

Pathology/Histopathology

Any of the pathologies listed in the tables may give rise to bowel obstruction and if the diagnosis is delayed this may go on to cause bowel ischaemia with necrosis and possible perforation.

Clinical Presentation

The infant or child will usually present with abdominal distension, irritability, pain and vomiting, or high nasogastric aspirates if a tube is in place. The timing of the clinical presentation may be partly determined by the underlying causes: congenital causes will usually present in the first few hours or days of birth and 95% of small bowel obstruction in the perinatal period is due to an atresia of some type. Meconium ileus will present within the first 48h of life and occurs almost exclusively in patients with cystic fibrosis. Fifteen to twenty per cent of cystic fibrosis patients present in this way (2). Small left colon syndrome (also termed meconium plug syndrome and functional immaturity of the colon) presents with failure to pass meconium and an increasingly dilated abdomen. Intussusception is the most common cause of obstruction in infants of 3 to 6 months. Strictures secondary to NEC most commonly occur within 1 to 6 months of the initial episode and are usually in the terminal ileum and the colon. Post-operative adhesions may occur at any time but most frequently in the first 6 months following surgery and in approximately 2% of patients who have had a laparotomy, accounting for 7% of small bowel obstruction overall.

Congenital causes of bowel occlusion	Acquired causes of bowel occlusion
Stenosis (jejunal, ileal, colonic)	Intussusception
Atresias (jejunal, ileal, colonic)	Malrotation with midgut volvulus
▶Meconium ileus (1)	Postoperative adhesions
▶Small left colon syndrome	Ileus
Annular pancreas	Caecal volvulus
Duplication cysts	Necrotising enterocolitis with secondary strictures
Hirschsprung's disease	Incarcerated hernias (inguinal, umbilical, omental

(*continued*)

Acute complete bowel occlusion	Chronic partial bowel occlusion
Intussusception	Hirschsprung's disease
Incarcerated hernias	Caecal volvulus
Malrotation with midgut volvulus	Inflammatory bowel disease including Crohn's disease
Post-operative adhesions	Post-operative adhesions
Annular pancreas	
Meconium ileus	
Necrotising enterocolitis strictures	

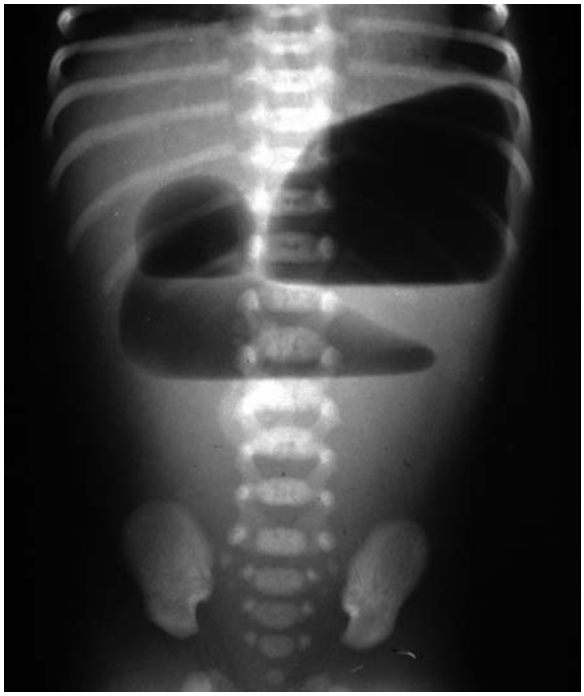
(*continued*)

Imaging

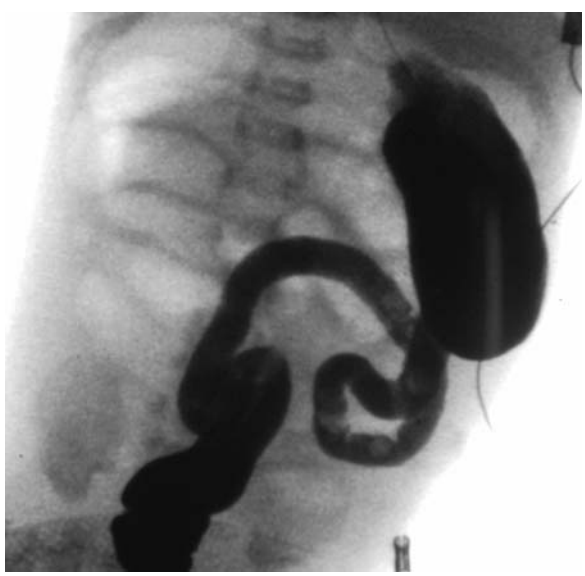
The initial investigation is usually an abdominal radiograph, but this may be supplemented by an upper GI series in the case of a suspected high obstruction or by a contrast enema for a suspected low obstruction. Ultrasound has been used in some centres to examine the large bowel following the introduction of saline per rectum.

The abdominal radiograph will show dilated gas and fluid-filled loops of bowel (Fig. 1). In the neonate and infant it may not be possible to differentiate small and large bowel obstruction and hence the level of obstruction is described as high or low in the GI tract depending on the pattern of distended loops. If only a few high central loops are seen this is termed a 'high' obstruction and the level of obstruction is most likely to be in the small bowel. If distended loops are seen throughout the abdomen it is likely to be a 'low' obstruction with the level of obstruction being in the most distal ileum or in the colon. Further investigation of a high obstruction is by an upper GI series and of a low obstruction by a contrast enema. Water-soluble contrast should be used in both cases due to the increased risk of perforation and the high probability of the patient subsequently undergoing surgery to resolve the obstruction. On a contrast enema a long filling defect of meconium in the left side of the colon is indicative of small left colon syndrome and the colon proximal to this will be dilated (Fig. 2). The neonate will usually clear the meconium plug spontaneously after the contrast enema. However, meconium ileus will demonstrate a micro-colon on contrast enema (Fig. 3). The colon will be thin and long, having not been used and dilated loops of bowel will be confined to the small bowel loops above the level of obstruction by meconium in the distal ileum.

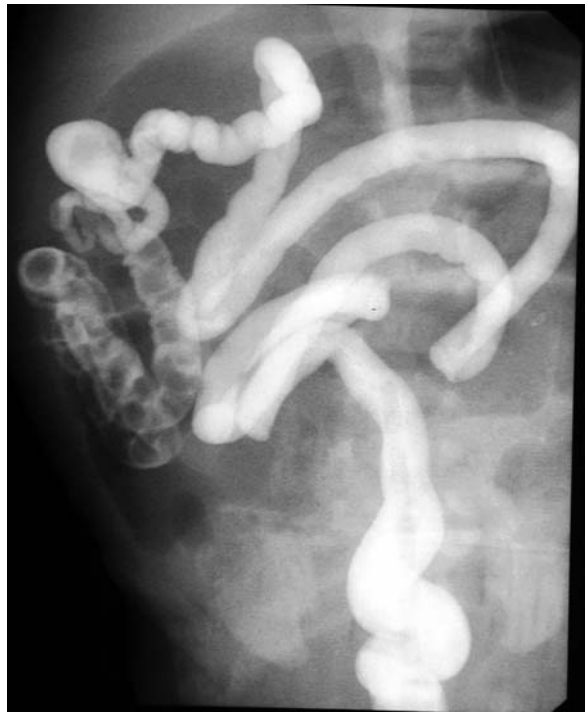
Meconium ileus is the only cause of obstruction that may be treated in the fluoroscopy room. A standard contrast enema is performed first to establish the diagnosis. Having established the diagnosis, ensuring that



Occlusion, Bowel in Childhood. Figure 1 Abdominal X-ray showing typical appearances of a high (upper GI) obstruction. The stomach and two loops of bowel are shown to be dilated and gas filled. Bowel gas is absent below this level indicating complete obstruction. Fluid levels are present as this film was taken erect. The patient was confirmed to have jejunal atresia.



Occlusion, Bowel in Childhood. Figure 2 Contrast enema showing in 'small left colon syndrome' with dilated colon seen proximally and meconium plugs seen in the descending colon.



Occlusion, Bowel in Childhood. Figure 3 Contrast enema showing a micro-colon and meconium causing obstruction in the distal ileum in a neonate with meconium ileus.

standard non-ionic water soluble contrast has reached beyond the obstructing meconium to the dilated bowel loops may be enough to precipitate clearing of the meconium. However, some centres value the use of a gastrografin® enema: if the infant is stable and appropriately fluid resuscitated, a ►gastrografin enema can be performed using diluted gastrografin (for details see also entry 'meconium ileus'). The gastrografin should be instilled until it is observed to have reached and be filling dilated loops of bowel, therefore being above the level of the obstructing meconium. Gastrografin is hyperosmolar and is believed to draw water into the gut thereby loosening the sticky meconium, allowing it to be passed, and relieving the obstruction.

Nuclear Medicine

Has no role to play in this condition.

Diagnosis

The diagnosis of obstruction is made by a combination of the clinical findings and the imaging as earlier. The underlying cause is usually confirmed at surgery.

Interventional Radiological Treatment

Only for meconium ileus as described above (also see entry 'meconium ileus'); note that sonographically guided saline reduction of meconium ileus is performed in some centres at the bedside of babies too ill to be transported to the fluoroscopy suite (3); some also recommend the use of a C-arm for fluoroscopic guidance on such occasions.

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Occlusion, Venous Central, Benign

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Synonyms

Benign central venous obstruction; Benign central venous thrombosis

Definition

Acute or chronic thrombotic uni- or bilateral occlusion of one or more central veins.

Pathology/Histopathology

Central venous occlusion can broadly be divided into two eliciting categories: benign and malignant. Regardless of the underlying malady, early detection and treatment of complications is essential to provide adequate care for patients suffering from central venous obstruction. The most common cause for benign central venous obstruction is hemodialysis related; other benign reasons are rather uncommon, but are picking up due to the increased use of permanent central venous access catheters and implantable cardiac rhythm management devices.

Clinical Presentation

Clinically these patients present with arm swelling and occasionally obvious widespread subcutaneous collateral vessels around the shoulder and thoracic aperture. Additional swelling of the face, neck, and breast may develop.

Imaging

Consequently, the diagnostic and therapeutic regimen of hemodialysis related central venous obstruction will be the centre of attention and discussed herein.

In the hemodialysis patient, chronic swelling of the access arm is the most indicative clinical symptom of central venous obstruction. Striking superficial collaterals veins may be observed accompanied by pain and paresthesia. In such an obvious case of impeded central venous flow, digital subtraction angiography of the fistula or graft and the complete venous outflow tract must be executed, since the central veins cannot be confidently examined with ultrasonography. Direct antegrade puncture of the access is suggested (1).

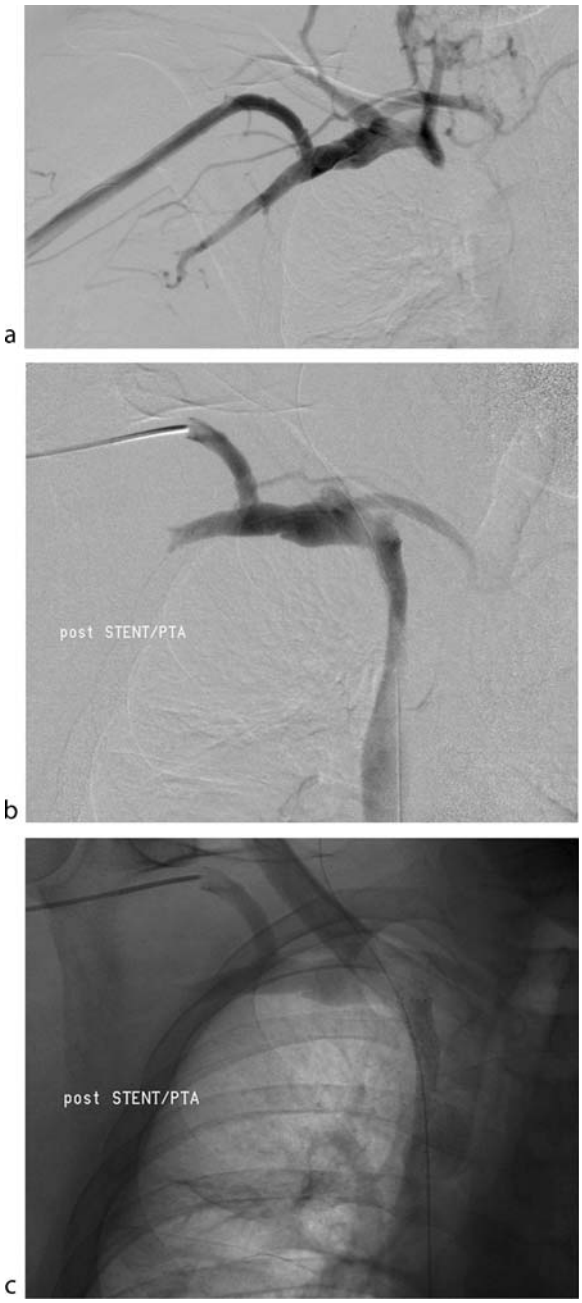
Diagnosis

Since clinical diagnosis is often unreliable, imaging techniques need to be incorporated in the diagnostic process.

Interventional Radiological Treatment

Interventional treatment of central venous lesions is indicated when they are impairing hemodialysis or arm swelling is painful and limiting. Reported primary patency rates in patients treated with PTA alone were 10% or less at one year with frequent restenoses (2). Primary stent implantation has clearly been shown to improve primary one-year patency rates to 56% and more, similar to those reported from surgical intervention (1, 3). Yet, due to the invasiveness of surgery for central venous obstructions, the less invasive percutaneous interventional therapy can be considered primary choice for treatment (1). Regular follow-up and reinterventions are however required to maintain patency and achieve long-term clinical success (4). Reports show that

symptomatic central venous obstruction in dialysis patients can be treated with a high success rate through radiological intervention (5, 6).



Occlusion, Venous Central, Benign. Figure 1 (a) Digital subtraction angiogram demonstrates complete occlusion of the right brachiocephalic vein draining a Brescia–Cimino fistula; (b) & (c) after unsuccessful transbrachial negotiation of the occlusion, angiogram shows restoration of flow and vanishing of collateral veins after placement of a self-expanding stent and subsequent PTA *via* a transfemoral approach.

Regarding the placement technique, stent placement should evade overlapping the ostium of a patent internal jugular vein to achieve a secure and satisfactory result, since this latter vein is important for future placement of central venous catheters. Correspondingly, a stent placed in the innominate vein if possible should not overlap the ostium of the contralateral vein; otherwise contralateral stenosis may come about and prohibit later use of the contralateral arm for access creation (1). A suitable endoprosthesis for central veins should be flexible enough to be used in curved and tortuous vessels. To avoid stent dislocation and proximal embolization, a self-expanding stent is necessary, in view of the fact that venous occlusions may undergo progressive luminal enlargement after stent placement (Fig. 1). Mechanical thrombectomy should not be regularly used as a primary therapy for dialysis-related central venous occlusions, because of the sharp angles and slim vessel walls observed in this vascular region. Furthermore, modest data are available on the application of thrombolytic agents in hemodialysis-related ▶benign central venous thrombosis. It can therefore not be recommended as a primary treatment regimen.

In any case reocclusion is a frequently observed complication and is more likely to occur after thrombosis has occurred for the first time (7). The radiologist should be prepared for repeat interventions.

To summarize, treatment of the hemodialysis patient population is specific due to the unusual underlying pathophysiology in dialysis outflow veins, which are exposed to much higher flow volumes. In the event of hemodialysis-related central venous occlusion, primary stent deployment is very effective in ensuring long-term vascular access for hemodialysis with superior long-term patency rates compared with percutaneous PTA alone or other therapeutic approaches.

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Occlusion, Venous Central, Malignant

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Synonyms

Malignant central venous obstruction; Malignant central venous thrombosis

Definition

Acute or chronic thrombotic uni- or bilateral occlusion of one or more central veins.

Pathology/Histopathology

The central venous vasculature, in particular the superior caval venous system, may be obstructed by two types of lesions. The so called SVC syndrome is in more than 90% of cases caused by malignancy. Bronchogenic carcinoma is the most common causative malignant tumor and often leads to edema of the upper thorax, shortness of breath, cough, dysphagia, hemoptysis, and headaches. Less often, direct extension or compression due to the primary tumor or by invasion of the mediastinal lymph nodes is triggered by lymphoma, extra-thoracic tumors, mesothelioma, and lymph node metastases (1). Benign diseases causing SVC obstruction are often iatrogenic due to central venous catheters and pacemaker leads.

Clinical Presentation

Clinical signs and symptoms of SVC obstruction (SVCO) are scored according to Kishi (2, Table 1). Clinical manifestations of venous obstruction can be extremely serious, requiring prompt treatment (also see ▶Benign central venous occlusion). Although the primary diagnostic suspicion can be rendered clinically, imaging is required for demonstrating the extent of the pathology.

Imaging

Contrast venography for decades has been the standard of reference for benign and malignant central venous obstruction; however this procedure has its shortcomings. Venous puncture can be challenging in a swollen extremity, the procedure may cause thrombophlebitis and there is a low risk of an allergic contrast agent reaction. Ultrasonography is not reliable for detection of central venous pathologies, owing to difficult access to these vessels. In addition enlarged collateral veins and nonocclusive thrombi may cause false negative results. Sensitivity can be improved with the demonstration of normal cardiac pulsatility and respiratory phasicity within the examined vessels (read ▶Thrombosis, vein brachial).

Occlusion, Venous Central, Malignant. Table 1 Kishi scoring system for superior vena cava obstruction with the total score for signs and symptoms calculated as the sum of the highest grades in each category

Signs and symptoms grade	
Neurologic symptoms	
	Stupor, coma, or blackout 4
	Blurry vision, headache, dizziness, or amnesia 3
	Changes in mentation 2
	Uneasiness 1
Laryngopharyngeal or thoracic symptoms	
	Orthopnea or laryngeal edema 3
	Stridor, hoarseness, dysphagia, glossal edema, or shortness of breath 2
	Cough or pleural effusions 1
Nasal and facial signs or symptoms	
	Lip edema, nasal stiffness, epistaxis, or rhinorrhea 2
	Facial swelling 1
Venous dilatation	
	Neck vein or arm vein distension, upper extremity swelling, or upper body plethora 1

Nowadays contrast enhanced spiral or preferably multislice computed tomography is employed to define the site of the obstruction and the presence of possible thrombosis and reveal surrounding soft tissue alterations (3). Alternatively, MRI is of comparative or even higher sensitivity and specificity in evaluating the patency of the central chest veins and may just as well hint at impending SVCO (▶Thrombosis, caval vein inferior).

Another drawback of digital subtraction angiography is that it can only evaluate one single venous drainage system for each injection and other major draining vessels, for instance the internal jugular veins, may remain indeterminate. CT and MR venography more clearly depict the site and extension of the obstruction in clinically relevant venous vessels segments (4).

Both cross-sectional modalities provide a fast, virtually noninvasive evaluation of the central chest veins. If a percutaneous therapy is anticipated, naturally, digital subtraction venography should be carried out immediately prior to, during, and after the intervention.

Diagnosis

Due to the deficiencies of a clinical diagnosis, imaging techniques need to be incorporated in the diagnostic process.

Interventional Radiological Treatment

Especially in acute central venous obstruction, traditional treatment methods in malignancy have been nonoperative, such as steroids, radiation therapy, and chemotherapy. These however may require up to 4 weeks to show an effect and thus often are too time consuming (5). In benign and malignant lesions, anticoagulation alone is not efficient but may be used combined with other treatment modalities (6). Thrombolysis is ineffective in 75% of cases when the event is older than 5 days. In early chronic and chronic occlusions, thrombolysis is outright unsatisfactory (7). If a stenosis is the trigger for thrombosis, sole lysis will also be inefficient (8).

In conclusion, an approach which offers urgent and rapid nonoperative relief should be the preferred treatment of choice.

Admittedly, with balloon angioplasty alone early restenosis can be expected; plus, interventional success is limited because of the well-known fibrous and elastic features of venous lesions (6, 8). It however can be valuable before stenting by allowing the stent to uncomplicatedly cross relatively tight lesions.

For all the above mentioned reasons percutaneous endovascular stenting of obstructive central venous lesions, which are symptomatic and caused by benign or malignant lesions, is an effective therapeutic option with acceptable patency rates and proven efficacy (9).

Stenting results in a rapid and consistent relief and maintains patency throughout the life span of most patients suffering from malignant tumors. Different vascular access sites like the femoral vein, internal jugular vein, subclavian vein, and basilic vein are possible. Recanalization can be attempted with a hydrophilic-coated steerable 0.035 inch guide wire or a straight guide wire with a movable core, combined with a selective catheter.

The obstruction may require predilation after safe passage through the segment, but only if presence of thrombus material can be excluded. To avoid venous rupture, which in the worst case may lead to cardiac tamponade, PTA should be performed carefully and by hand. Next, a stent which is flexible enough to allow implantation even in kinked vessels should be introduced. Coverage of the obstructed segment is advised to be at least 1 cm free at the proximal and distal end to cover beyond the obstruction. Sometimes the placement of an additional stent may be necessary, especially if there is obstruction of both anonymous veins and the superior vena cava. In this case recanalization and revascularization of one anonymous vein lead to good clinical results and are associated with fewer complications provided that sufficient venous collaterals from left to right or vice versa are present (10).

Stent size should be adapted to the diameter of the adjacent nonobstructed vessel segment. Postprocedural balloon dilatation is advised. Stent size should be at least 10% above the venous diameter. To avoid stent dislocation and central embolization, a self-adjusting, self-expanding stent is advantageous because especially chronic venous occlusions may undergo progressive luminal enlargement after stent placement (11). Previously, many interventionalists preferred balloon expandable stents because of their flexibility and their marginal risk of migration. Current self-expanding stents however have overcome these problems of significant foreshortening and migration.

Often a superimposed thrombosis can be observed which can be treated with thromboaspiration or fibrinolytic therapy before stenting (1). The significant bleeding risk in patients with corresponding contraindications must be considered though. The chance of hematoma formation and gastrointestinal and intracerebral hemorrhage must be taken into account. Nevertheless the presence of extensive thrombus may require the use of thrombolytics. The thrombolytic agent should be infused with the tip of the infusion catheter inside the thrombus at a rate of ~0.02 mg tissue plasminogen activator/kg body weight/hr (12). To save time, pulse spray injection can be employed. Active thrombus removal with mechanical thrombectomy devices may be an adjunct or even alternative to intralesional thrombolysis, however it must be handled with care and expertise.

The peri- and postprocedural anticoagulation for stent placement with or without additional thrombolysis is still unclear. Heparinization during stenting and postprocedural intravenous or subcutaneous heparin can be administered. Subsequent antiplatelet therapy, typically aspirin and/or clopidogrel is recommended. Thrombosis may occur after the stent insertion in up to 45% (13). Long-time anticoagulation therefore helps in avoiding clinical deterioration.

To recapitulate, the efficacy and safety of stent placement in central venous occlusion of benign and malignant origin have been proven with rapid relief and less invasiveness for the often extremely ill patients. Stenting is widely accepted now; it provides fast and durable symptomatic relief and can nowadays be favored to radiation and chemotherapy or used in combination

with them. If clinical symptoms are severe or worsen rapidly, stenting is indicated while surgical therapy should be reserved for patients undergoing refractory to percutaneous therapy. Repeated percutaneous intervention can prolong the cumulative patency rate.

Pharmacologic/pharmacomechanical/mechanical thrombolysis may be necessary to improve the final result in case of superimposed central venous thrombosis; they should however not be employed as a single means for revascularization.

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Occupational Lung Diseases

►Pneumoconioses

Oesophageal Atresia

A congenital abnormality in which the upper oesophagus is a blind-ending sac and is not continuous with the lower oesophagus.

►Oesophageal Disease, Childhood
►GI Tract, Paediatric, Congenital Malformations

Oesophageal Cancer

►Neoplasms, Oesophagus

Oesophageal Clearance

This term is used to describe the process by which the oesophagus is cleared of refluxed stomach acid.

►Gastroesophageal Reflux in Adult Patients: Clinical Presentations, Complications, and Imaging

Oesophageal Disease, Childhood

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Synonyms

Achalasia; Gastro-oesophageal reflux; Gastro-oesophageal reflux (GOER); Oesophageal atresia; Oesophageal duplications (and other foregut duplications); Oesophageal foreign body; Oesophageal stenosis (corrosive ingestion); Oesophageal webs; Tracheo-oesophageal fistula

Definition

►*Oesophageal atresia* is the congenital occlusion of the oesophagus. It has an incidence of between 1 in 3,000 to

4,500 live births. Oesophageal atresia is associated with ►tracheo-oesophageal fistula in more than 85% of cases. There are several different anatomic variations of the oesophageal atresia and the insertion of an associated tracheo-oesophageal fistula. Tracheo-oesophageal fistula is the abnormal connection between the trachea and the oesophagus and occurs in isolation only in 8% of cases. The remainder of the time it occurs in conjunction with oesophageal atresia.

Oesophageal webs may be congenital or acquired and are a thin, 2–3 mm, eccentric, smooth extension of the normal oesophageal wall that can occur anywhere along the length of the oesophagus but typically is located in the anterior post-cricoid area of the proximal oesophagus.

►*Oesophageal duplications* are part of a wider group of foregut duplications. Duplications may cystic or tubular and may contain gastric mucosa. They can be associated with sequestrations or congenital stenosis or atresia of the oesophagus. Oesophageal duplications may be separated from the oesophagus or may share a common wall. Duplications of the oesophagus are sometimes associated with vertebral anomalies and intraspinal cysts and often are associated with intra-abdominal intestinal duplications.

►*Oesophageal stenosis* is a narrowing of the oesophagus at any point along its length and over a variable distance. A stenosis my be congenital or may be acquired such as after surgery for oesophageal atresia, secondary to acid or alkali ingestion, or to another disease process such as epidermolysis bullosa.

►*Achalasia*: Abnormal dilatation and motility of the distal oesophagus with failure of relaxation of the lower oesophageal sphincter.

►*Oesophageal foreign body*: An object that is ingested but does not pass freely through the oesophagus. The most common sites for hold-up are at the level of cricopharyngeus, at the aortic knuckle and at the gastro-oesophageal junction.

►*Oesophageal inflammatory change:* Inflammation of the mucosa of the oesophagus in response to an irritant. This in most commonly secondary to reflux or to ingested corrosives but may also be seen in Crohn's disease, epidermolysis bullosa, pemphigoid and other systemic or dermatological diseases.

►*Gastro-oesophageal reflux*: Reflux is the retrograde movement of fluid across a sphincter and in the context of the oesophagus is the movement of stomach contents up into the oesophagus.

Pathology/Histopathology

Oesophageal atresia is due to posterior deviation of the tracheo-oesophageal septum leading to incomplete separation of the oesophagus from the laryngo-tracheal tube. There is failure of recanalisation of the oesophagus in the eighth week of foetal development. There are several variations on how the fistula may connect the trachea and oesophagus. The most common is that of a blind-ending upper pouch with a fistula then connecting the distal trachea to the lower, patent, part of the trachea. A fistula that joins a patent oesophagus to a patent trachea is termed an 'H'-shaped fistula (8%). Oesophageal atresia and tracheo-oesophageal fistula is the most common congenital malformation of the oesophagus (1).

Oesophageal webs may represent incomplete recanalisation of the oesophagus during foetal life. They are made of the normal structures in the wall of the oesophagus.

Oesophageal duplications: It is believed that these anomalies are due to failure of the notochord to detach from the endoderm, resulting in a persisting neurenteric canal. They often contain gastric mucosa.

Oesophageal stenosis: Congenital stenosis is believed due to poor canalisation of the oesophagus in embryonic life. Acquired stenoses are usually secondary to an insult and will therefore show scar tissue.

►*Achalasia:* Aetiology unknown.

Oesophageal foreign body: No specific pathology.

Oesophageal inflammatory change: The inflammatory change may be superficial involving only the mucosal surface or maybe transmural.

Gastro-oesophageal reflux: In GOER the lower oesophageal sphincter (LES) opens inappropriately or is incompetent and there is transient complete relaxation of the sphincter. Due to a pressure difference across the LES (with negative pressure in the oesophagus during inspiration) or a transient rise in intra-abdominal pressure the stomach contents move up into the oesophagus This may result in chronic inflammation in the oesophagus and in severe cases can lead to stricture formation.

Clinical Presentation

Oesophageal atresia and tracheo-oesophageal fistula: Most neonates present at birth with difficulty swallowing secretions, drooling, choking and respiratory distress as secretions and feeds spill over into the airway. In complete OA, the neonate will present with difficulty swallowing secretions and will be unable to tolerate the first feed. If a fistula is present between the trachea and oesophagus, air can still reach the gastrointestinal (GI) tract. An ►H-fistula may present later with blue episodes or repeated chest infections and aspiration as some fluid tracks from the oesophagus through the fistula into the airway. A blind-ending upper pouch is often detected

when there is failure to pass a nasogastric tube and/or this becomes coiled in the upper oesophagus. With the increase in antenatal scanning oesophageal atresia may be detected *in utero*. The mother may present with polyhydramnios and on ultrasound there will be very little if any fluid present in the stomach or GI tract of the foetus.

A complication of repair of oesophageal atresia is a stricture of the oesophagus and the child may present with dysphagia and regurgitation of food.

Oesophageal web: The very young child will present with drooling and aspiration, and may suffer repeated chest infections. The older child will may have dysphagia and regurgitation of solids.

Oesophageal duplication: Completely separated duplications can present as a mediastinal mass and may or may not be associated with symptoms. Incompletely separated duplications may give rise to dysphagia, regurgitation and rarely airway obstruction (2).

Oesophageal stenosis: Dysphagia and regurgitation.

Achalasia: Chronic regurgitation of undigested food, repeated aspiration pneumonia, retrosternal discomfort in the older child.

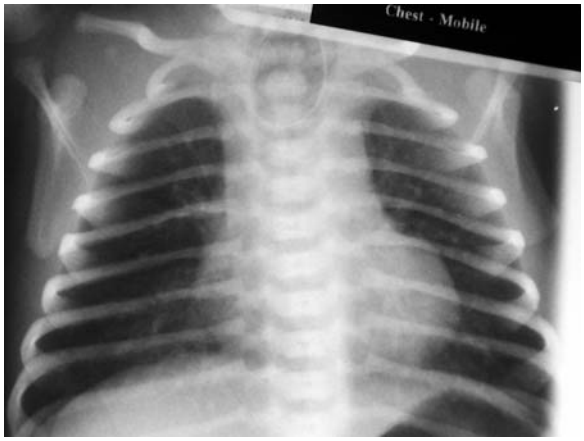
Oesophageal foreign body: The patient will usually be drooling and may be dysphagic.

Oesophageal inflammatory change: Presentation will depend on the underlying cause but the patient will usually have retrosternal pain. With chronic change going on to stricture formation they may present with dysphagia.

Gastro-oesophageal reflux: GOER is most common in infants and young children and a minor degree of reflux in the first few months of life may even be considered within normal levels. The symptomatic infant will present with recurrent regurgitation of feeds and/or pain. In severe cases, the child may fail to gain weight or even lose weight if it is not possible to maintain adequate calorific intake. Older patients may present with retrosternal pain, food aversion or less commonly with a stricture. Alternatively, young children and patients with neurologic deficits may present with recurrent chest infections secondary to aspiration as a result of GOER.

Imaging

Oesophageal atresia and tracheo-oesophageal fistula: A chest X-ray will often show an air-filled, dilated and blind-ending upper oesophageal pouch. If the passing of a nasogastric tube (Fig. 1) has been attempted this is likely to be coiled in the upper pouch (1). A Replogle tube (identified by a dashed radio-opaque marker allowing easy identification on X-ray) may have been placed in the upper oesophagus to allow aspiration of the secretions.



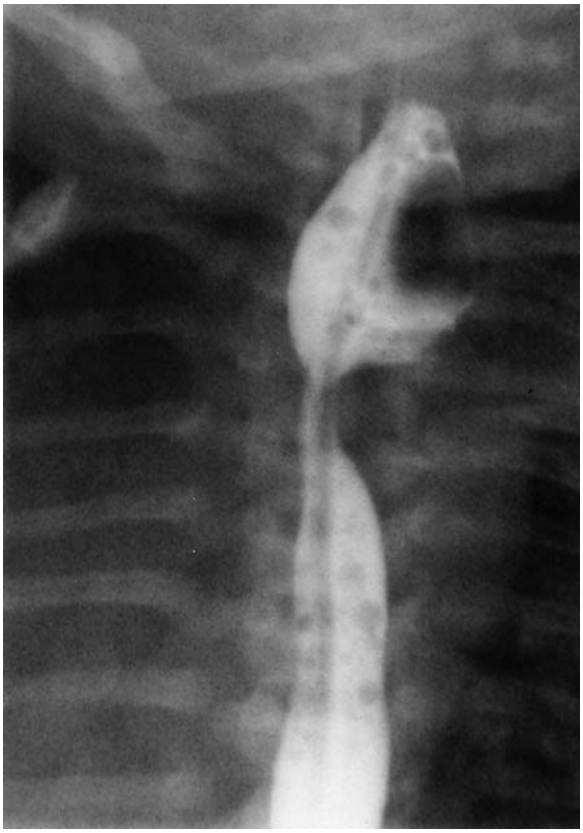
Oesophageal Disease, Childhood. Figure 1 Chest X-ray showing a nasogastric tube coiled in a blind-ending upper pouch in a patient with oesophageal atresia.

This is a dual lumen tube which allows secretions to be continually sucked from the blind-ending upper oesophagus thereby preventing aspiration. It is used almost exclusively in this condition. The abdomen will be gasless unless there is an associated fistula, and indeed the presence of gas in the abdomen will confirm that a fistula is present, this being the only route for air to get from the airway into the GI tract. A contrast study is not required and the immediate management is usually surgical. Imaging for an H-type fistula (a fistula between a patent airway and a patent oesophagus) is by a tube oesophagram. With the patient prone and with lateral screening water-soluble contrast is injected into the oesophagus whilst the nasogastric tube is slowly withdrawn. If a fistula is present, the contrast should flow through the fistula from the oesophagus into the airway. This procedure therefore carries a significant risk of acute respiratory compromise and should only be undertaken if suction, oxygen, nursing support and appropriate resuscitation facilities are available.

Stricture formation as a delayed complication of repair of oesophageal atresia is best demonstrated by upper GI (UGI) series (Fig. 2).

Oesophageal web: Imaging is by upper GI series or direct visualisation on endoscopy.

Oesophageal duplication: If the duplication is not completely separated it may be demonstrated on an upper GI series (Fig. 3). A closed duplication may be best demonstrated on MRI of the mediastinum, although CT and ultrasound may also be used. Ultrasound would generally use an approach through the sternal notch or between the ribs, but in very young patients a transsternal approach can sometimes be used as the sternum has not ossified.



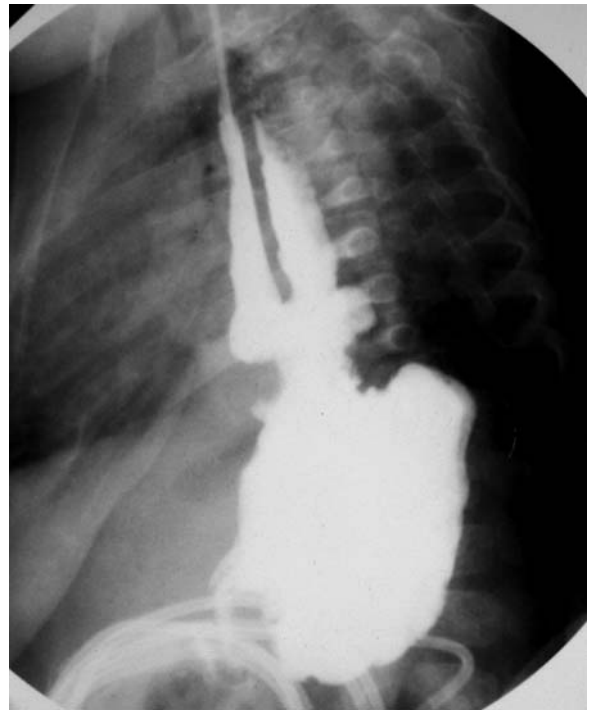
Oesophageal Disease, Childhood. Figure 2 Upper GI series demonstrating a tight oesophageal stenosis at the site of previous surgery for oesophageal atresia with tracheo-oesophageal stenosis.

Oesophageal stenosis: Upper GI series will show the level and extent of the narrowing.

Achalasia: An upper GI series will show the accumulated food debris in the dilated oesophagus and poor opening of the lower oesophageal sphincter with a characteristic fine jet of contrast passing through or an abrupt point ('birds beak') at the gastro-oesophageal junction through which no contrast passes.

Oesophageal foreign body: Chest X-ray (AP and lateral) will show radio-opaque foreign bodies; an upper GI series may demonstrate the filling defect on a radiolucent foreign body. Endoscopy allows direct visualisation and simultaneous removal (see entry Foreign body, ingestion, children).

Oesophageal inflammatory change: Very subtle superficial changes may not be detected by imaging and will only be identified under direct vision with endoscopy. An upper GI series using barium will demonstrate mucosal surface irregularity with an uneven appearance to the barium column (Fig. 4). If a double contrast image is achieved ulceration may be visible. Endoscopic ultrasound will show the degree of intramural thickening.

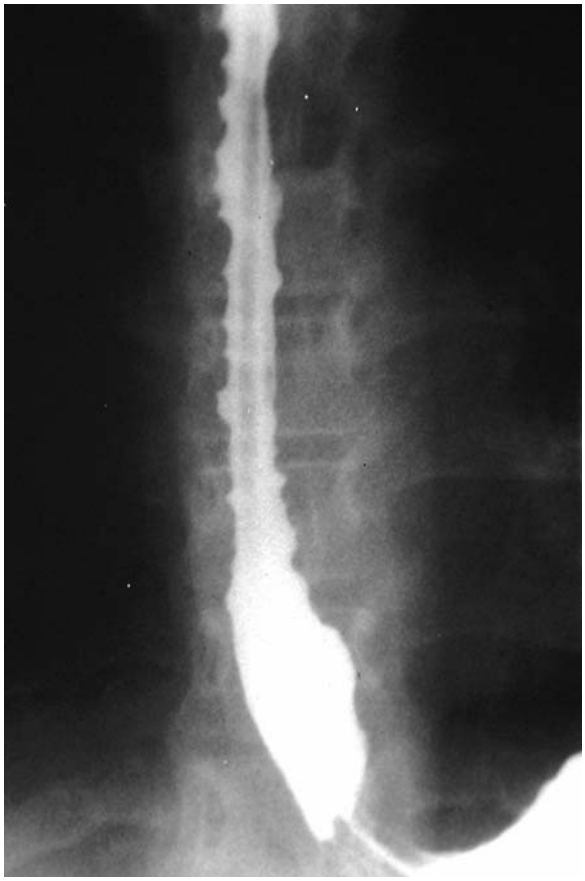


Oesophageal Disease, Childhood. Figure 3 Contrast outlines an oesophageal duplication with two, parallel, fluid-filled cavities opacified.

Gastro-oesophageal reflux: An upper GI series or ultrasound of the lower oesophagus are the modalities of choice. Reflux is usually self-evident in infants by the regurgitation of feeds so much of the value of the UGI is to exclude anatomical causes for the reflux such as hiatus hernia or gastric outlet obstruction and in preparation for surgery if a fundoplication is being considered. GOER is characteristically intermittent and it would be inappropriate to image the patient with prolonged screening to try and demonstrate an occasional episode. Only intermittent screening should be performed and the absence of demonstrable reflux during a UGI does not exclude the diagnosis. Ultrasound is increasingly used to demonstrate relaxation of the lower oesophageal sphincter and fluid tracking into the lower oesophagus. It has the advantage that the sphincter can be observed directly and for prolonged periods but the disadvantage that it cannot show the rest of the stomach and duodenal anatomy. An UGI is also useful for assessment of the complications of GOER such as stricture formation.

Nuclear Medicine

No role to play in this condition.



Oesophageal Disease, Childhood. Figure 4 Upper GI series with contrast outlining an irregular and narrowed oesophagus following ingestion of caustic fluid.

Diagnosis

By imaging as detailed above. GOER may also be diagnosed by monitoring with pH probe.

Interventional Radiology

Image guided balloon dilatation of strictures (3), and sometimes removal of oesophageal foreign bodies may be carried out by the interventional radiologist (see also entry 'foreign body, ingestion, children').

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Oesophageal Duplication

A congenital abnormality in which there has been duplication of part of the foregut in embryological development resulting in either a cyst or second lumen of the oesophagus.

▶Oesophageal Disease, Childhood

Oesophageal Duplications (and other foregut duplications)

▶Oesophageal Disease, Childhood

Oesophageal Dysmotility

This may be either abnormal contractions of the oesophagus, or a diminution or failure of normal peristalsis. It has many causes, of which the most common in industrialized countries is gastro-oesophageal reflux disease.

▶Gastroesophageal Reflux in Adult Patients: Clinical Presentations, Complications, and Imaging

Oesophageal Foreign Body

▶Oesophageal Disease, Childhood
▶GI tract, Pediatric, Foreign Bodies

Oesophageal Malignancy

▶Neoplasms, Oesophagus

Oesophageal Stenosis (corrosive ingestion)

▶Oesophageal Disease, Childhood

Oesophageal Tumor

▶Neoplasms, Oesophagus

Oesophageal Webs

▶Oesophageal Disease, Childhood

Oesophagitis

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Synonyms

Esophagitis; Gastroesophageal reflux disease (GERD); Oesophagitis

Definition

In the literature the term "esophagitis" (oesophagitis) is sometimes used synonymously with "reflux esophagitis" because gastroesophageal reflux is the most common cause of esophagitis. However, numerous etiologic factors may cause esophagitis:

- Infectious esophagitis
  1. Fungal infections
  2. Viral infections
     (a) Herpes simplex virus (HSV)
     (b) Cytomegalovirus (CMV)
     (c) Varicella-zoster virus (VZV)
  3. Bacterial infections
  4. Parasitic infections
- Drug-induced esophagitis
- Radiation-induced esophagitis
- Caustic esophagitis
- Reflux esophagitis
- Miscellaneous
  1. Skin disorders
- Dystrophic epidermolysis bullosa
- Pemphigus vulgaris
- Bullous pemphigoid
- Benign mucous membrane pemphigoid
- Stevens–Johnson syndrome
- Lichen planus
- Acanthosis nigricans
- Darier's disease
- Leukoplakia
  1. Behcet's disease
  2. Graft-versus-host disease (GVHD)
  3. Crohn's disease
  4. Ulcerative colitis
  5. Sarcoidosis
  6. Collagen vascular diseases.

Pathology/Histopathology

Infectious Esophagitis

In Candida esophagitis, plaques consist of heaped-up areas of necrotic epithelial debris or actual colonies of *C. albicans* on the esophageal mucosa; the esophagus itself as an irregular or shaggy appearance. Three viral infections cause ulcerative esophagitis: HSV, CMV, and VZV.

In HSV the earliest esophageal lesions are vesicles, the centers of which slough to form discrete, circumscribed ulcers with raised edges. Routine histologic stains may show epithelial cells with occasional multinucleation, "ground-glass" nuclear staining, and intranuclear inclusions. More specific and sensitive is immunohistologic identification with monoclonal antibodies to HSV antigen and *in situ* hybridization for HSV nucleic acid.

CMV infection occurs only in immunosuppressed patients. CMV may be activated from latency or acquired from blood-product transfusions. CMV lesions first appear as serpiginous ulcers in otherwise normal mucosa but coalesce to form giant ulcers, particularly in the distal esophagus. Infection occurs within submucosal fibroblasts and endothelial cells, not in epithelium, and is usually part of widespread visceral infection. Routine histologic stains show large cells in the submucosa bearing amphophilic intranuclear inclusions and intracytoplasmic inclusions. Immunohistology with monoclonal antibodies to CMV antigens and *in situ* hybridization for CMV DNA are useful for finding infected cells that are neither large nor inclusion bearing.

Drug-induced Esophagitis

In drug-induced esophagitis, affected individuals ingest the medication with little or no water immediately before

going to bed. When such drugs dissolve in the esophagus, they cause mucosal injury either by creating an acid pH (analogous to a form of caustic esophagitis) or by direct irritating the epithelium.

Radiation Esophagitis

A radiation dose of 4,500–6,000 rad over a 6- to 8-week period or more to the mediastinum may cause severe injury to the esophagus. Acute radiation-induced esophagitis usually occurs 2–4 weeks after the initiation of radiation therapy. The mucosa typically has a granular appearance because of edema and inflammation. Ulceration and decreased luminal distensibility are other frequent findings.

Caustic Esophagitis

Caustic esophagitis causes injury similar to that from thermal burns. Liquid lye causes liquefactive necrosis, resulting in the most severe form of caustic injury to the esophagus. Alkaline agents can produce deep coagulation necrosis in minutes. Necrosis from acids tends to be more superficial. The severity and extent of esophageal injury depend on the type, concentration, and volume of the caustic agent. By 2–6 weeks, healing is well in progress, often accompanied by severe fibrosis.

Gastroesophageal Reflux Disease, Esophagitis (Reflux Esophagitis)

This type encompasses reflux esophagitis and is characterized histologically by inflammatory cells and reflux changes consisting of epithelial hyperplasia without inflammation. The multiple determinant factors in the production of reflux disease include the frequency and volume of GER, the volume of gastric contents available to reflux, the potency of the reflux material, the efficacy of esophageal clearance, and the tissue resistance to injury.

Miscellaneous Esophagitis

Eosinophilic esophagitis (IEE) is a chronic inflammatory disease characterized by eosinophilic infiltration of the esophagus with an increased number of intraepithelial eosinophils (more than 20 eosinophils per high-power field) in endoscopic biopsy specimens from the esophagus. Alkaline reflux esophagitis is caused by reflux of bile or pancreatic secretions into the esophagus after partial or total gastrectomy. Pemphigus and pemphigoid are nonhereditary conditions in adults, and epidermolysis bullosa dystrophica occurs as an autosomal recessive condition in children. Pemphigus, pemphigoid, and epidermolysis bullosa can produce esophagitis.

Pemphigus vulgaris affects skin, mouth, and other mucous membranes with weeping bullous lesions. Histology shows acantholysis and intraepithelial bullae, and specific immunohistology should be used. Bullous pemphigoid is a chronic disease of the elderly, in whom tense, pruritic skin bullae arise. Esophageal bullae occur rarely, with sloughing of the epithelium as a cast. The histology shows subepithelial bullae and circulating antibodies to the basement membrane.

Behcet's disease is a multisystem inflammatory process characterized by oral and genital ulcers, ocular inflammation, skin lesions, and vasculitis. Esophageal lesions include ulcerations that can tunnel under the mucosa, strictures, and perforations.

GVHD is an immunologic reaction against host tissues by donor lymphoid cells. The esophagus shows webs, rings, and tight strictures but often fails to show generalized desquamation apparent on endoscopy. Crohn's disease rarely affects the esophagus but may show small aphthous ulcers, inflammatory strictures, filiform polyps, and fistulas. Granulomas can occasionally be found in endoscopic biopsy specimens.

Clinical Presentation

Infectious esophagitis is seen with increasing frequency in debilitated individuals, alcoholics, diabetics, immunocompromised or transplant patients, individuals with impaired T-cell function from acquired immunodeficiency syndrome (AIDS), and patients receiving steroids, antibiotics, radiotherapy, or chemotherapy. Candida esophagitis (and infectious esophagitis generally) is usually characterized by the abrupt onset of odynophagia (painful swallowing), chest pain, or dysphagia. Clinical symptoms can occasionally be absent. Symptoms of tuberculosis esophagitis include dysphagia and chest pain, but esophageal symptoms are commonly absent. Patients with HSV esophagitis present with odynophagia, retrosternal pain, and heartburn. Infection can also start with mild symptomatology of nausea and pyrosis (heartburn) or with very severe symptoms of hematemesis. The clinical presentation of CMV esophagitis is nausea, vomiting, painful swallowing, heartburn, and/or hematemesis.

Symptoms of *drug-induced esophagitis* are usually sudden in onset and consist of chest pain, odynophagia, or dysphagia. A typical location is the aortic arch level, where tablets are delayed because of the aortic indentation on the esophagus and the low contractile force of peristalsis. Bleeding or esophageal perforation may occur.

Radiation esophagitis typically produces mild heartburn or dysphagia several weeks after the onset of treatment. Some patients may have progressive dysphagia

due to strictures 4–8 months after completion of radiation therapy.

In *caustic esophagitis*, the initial clinical symptoms are the rapid onset of chest pain and dysphagia. These symptoms tend to resolve in several days. Acute complications include shock, fever, respiratory distress, mediastinitis, and perforation. Late complications are related primarily to fibrosis and stricture, which may cause dysphagia several weeks after the initial injury.

Symptoms of *GERD esophagitis* (*reflux esophagitis*) can be divided into typical (esophageal) symptoms (heartburn, regurgitation, dysphagia) and atypical (extraesophageal) symptoms (chronic cough, respiratory complaints, laryngeal symptoms). Dysphagia is usually due to esophageal narrowing. Some patients complain of a lump in the throat (globus). In young children the predominant reflux symptoms are regurgitation, repetitive vomiting, and failure to thrive.

IEE appears in the adult population. It typically affects young men (20–40 years of age) with long-standing dysphagia and recurrent food impactions who have not responded to the usual forms of antireflux therapy. Other findings include chest pain and vomiting.

Imaging

Barium examination continues to be the primary radiologic modality for evaluating patients with dysphagia, reflux symptoms, or other clinical findings of esophageal disease. The double-contrast phase optimizes the ability to detect all kinds of esophagitis, particularly reflux disease, whereas the single-contrast phase optimizes the ability to detect hiatal hernia and lower esophageal rings or strictures. Barium contrast studies are useful for evaluating mucosal surface lesions but provide little information about the extramucosal extent of disease. Computed tomography (CT) and magnetic resonance imaging (MRI), on the other hand, permit evaluation of wall thickness, mediastinal involvement, adjacent lymphadenopathy, and distant spread.

Diagnosis

Infectious esophagitis: Candida esophagitis is characterized on esophagrams by plaques or a "shaggy" esophagus. Plaquelike lesions are seen as linear or irregular filling defects that tend to be oriented longitudinally and are separated by normal mucosa. The luminal contour may show fine speculations, irregularity, a cobblestone pattern, or bizarre thickened folds simulating varices. Barium may dissect beneath a pseudomembrane, causing a shaggy contour that gives the appearance of ulceration. The esophagus tends to be atonic. Peristalsis may be feeble or incomplete. Because of muscular hypotonia, the esophagus is generally slightly dilated or normal in caliber but may show areas of moderate narrowing. *Monilia* organisms are a frequent companion of esophageal intramural pseudodiverticulosis.

Double-contrast esophagrams have been found to have a sensitivity of 90% for detecting Candida esophagitis, primarily because of the ability to show these plaques.

CT findings are nonspecific and commonly seen in various kinds of esophagitis (circumferential esophageal wall thickening of >5 mm, with relatively long segmental involvement). Enhanced scans may also depict the target sign (circumferential wall thickening and enhancing internal mucosa). Morphologic abnormalities caused by tuberculosis esophagitis are often eccentric and may show skip areas. The luminal contour may show mild irregularity, large or deep ulcers, and sinus tracts. Fistulas are common. The esophageal wall is generally thickened and the lumen often narrowed. Enlarged mediastinal nodes may displace or compress the esophagus and widen the mediastinum.

In HSV, barium contrast X-ray shows multiple ulcers. Small ulcers surrounded by edema give the appearance of targets or shallow irregularities in the profile view. Diagnosis requires endoscopic brushing and biopsies. The first, presumptive diagnosis can be made in patients with esophageal symptoms and HSV infections of the mouth or nares.

CMV esophagitis is characterized by one or more giant flat ulcers that are several centimeters or more in length. The ulcers may have an ovoid or diamond-shaped configuration and are often surrounded by a thin radiolucent rim of edematous mucosa. Endoscopy with multiple biopsies targeting the center of the ulcer formation is mandatory. Brushing of overlying exudates is seldom useful.

Drug-induced esophagitis: Signs are luminal irregularity, frank ulceration, and luminal narrowing. Tetracycline and doxycycline are associated with the development of small shallow ulcers in the upper or middle part of the esophagus. Other drugs may cause more severe esophageal injury.

Radiation esophagitis: A morphologic abnormality consists of diffuse ulceration. Late findings consist of strictures that are generally smooth with tapering margins and that rarely show irregularity or ulceration.

Caustic esophagitis: In the acute stage, the examination should be initiated with water-soluble contrast medium to exclude esophageal or gastric perforation. Such studies may also reveal marked edema, spasm, and ulceration of the affected esophagus. During the first week, characteristic frank ulcerations are seen. A pseudomembrane may cause intramural trapping of barium. Long, ulcerated strictures may be observed in patients who ingested lye or

other caustic agents, and in severe cases, diffuse esophageal narrowing may reduce the thoracic esophagus to a thin, tight stricture. Cross-sectional images depict narrowing or obliteration of the esophageal lumen, and perifibrotic tissue may be observed. Reformatted CT images may be useful for demonstrating the surrounding fibrotic change to which caustic esophagitis leads.

GERD esophagitis: Reflux esophagitis manifests at esophagography as finely nodular or granular relief with poorly defined radiolucencies that fade peripherally due to edema and inflammation of the mucosa. Endoscopy is the gold standard for diagnosing erosive GERD, and the Los Angeles classification for esophagitis is generally accepted as the endoscopic assessment of GERD. Most patients—approximately 60% referred for endoscopy with typical reflux symptoms—do not have erosive reflux disease. The spectrum of GERD can be subdivided into endoscopy-negative, nonerosive GERD (NERD), reflux esophagitis (GERD), and GERD with esophageal columnar metaplasia (Barrett). Further changes include irregularity of luminal contours, discrete ulcerations, transverse esophageal folds, thickened longitudinal folds, esophageal wall thickening, smooth polypoid protuberance (also known as an inflammatory esophagogastric polyp), and segmental narrowing.

The ulcers can have a punctate, linear, or stellate configuration and are often associated with a surrounding halo of edematous mucosa, radiating folds, or sacculation of the adjacent wall. Predominant involvement of the distal esophagus and the presence of an associated hiatal hernia and gastroesophageal reflux should suggest the correct diagnosis.

Thickened folds wider than 3 mm are best seen on mucosal relief views of the collapsed esophagus. Multiple delicate transverse folds 1–2 mm wide may also be found in patients with gastroesophageal reflux disease. Between 10 and 20% of patients with reflux esophagitis develop peptic strictures as a result of circumferential scarring of the distal esophagus. The classic appearance of a smooth, tapered area of concentric narrowing in the distal esophagus above a sliding hiatal hernia should be virtually pathognomonic of a benign peptic stricture. Most peptic strictures range from 1 to 4 cm in length and from 0.2 to 2.0 cm in width. Between 25 and 50% of patients with reflux esophagitis have abnormal esophageal motility, manifested by feeble or absent primary peristalsis associated with an increased frequency of nonperistaltic contractions.

Miscellaneous esophagitis: Alkaline reflux esophagitis is characteristic by mucosal nodularity or ulceration or, in severe disease, by distal esophageal strictures that often progress rapidly in length and severity over a short period of time. During the acute stage of pemphigus, pemphigoid, and epidermolysis bullosa esophagitis, bullae may cause multiple esophageal filling defects on esophagograms, or ruptured bullae may appear as ulcerations. Repetitive insults often lead to strictures that are generally smooth but sometimes have an irregular contour. IEE manifests at esophagography as segmental esophageal strictures (sometimes ringlike—the so-called ringed esophagus at endoscopy and/or barium studies) and occasionally with diffuse esophageal narrowing, which produces a "small-caliber" esophagus.

Interventional Radiological Treatment

Esophageal dilation is usually indicated for benign stenoses and strictures, which can be caused by esophagitis. The main indications are strictures after caustic and reflux esophagitis. All esophageal strictures should be carefully evaluated with esophagography or endoscopy before dilation.

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Oesophagogram

Radiologic examination where images are obtained of the oesophagus during drinking of either barium sulphate suspension or iodine contrast medium. When an effervescent agent is used a double-contrast effect may be obtained.

▶Diverticulum, Oesophagus

Oil Cyst

Conglomerate of almost entirely pure neutral fat, encapsulated by a thin, smooth, fibrous wall in which calcium deposition may occur.

▶Trauma, Breast

OLF

▶Ossification or Calcification of Ligamenta Flava (OLF)

OLT

▶Transplantation, Liver
▶Orthotopic Liver Transplantation

Omphalocele

Persistence of herniation of the abdominal contents into the base of the umbilical cord at the time of birth and associated with a high incidence of other congenital anomalies.
▶GI Tract, Paediatric, Congenital Malformations

Open and Closed Spinal Dysraphisms

Etymologically, the term "dysraphism" implies defective closure of the neural tube, and should therefore be used to refer to abnormalities of primary neurulation only. However, the term has gained widespread use as a synonym to congenital spinal cord malformation.

Spinal dysraphisms are categorized into open (OSD) and closed (CSD). OSDs are characterized by exposure of nervous tissue to the environment through a congenital defect of the child's back. On the contrary, CSDs are covered by skin, although cutaneous birthmarks, such as angiomas, dimples, overgrowing hair, dyschromia, and dystrophy, are present in greater than 50% of cases. Use of the term "occult spinal dysraphisms" is discouraged as it suggests complete absence of external abnormalities, a condition that occurs only in a minority of CSDs.
▶Congenital Malformations, Spine and Spinal Cord

Ophthalmology or Colposcopy

▶Optical Imaging

OPLL

▶Ossification of Posterior Longitudinal Ligament

Opportunistic Infections

Opportunistic infections are infections caused by a microorganism that normally does not cause disease but becomes pathogenic in persons with impaired immune system.
▶Infection, Opportunistic, Brain

Opportunistic Screening

Screening not performed in an organized or population-based screening program, i.e., resulting from self-referral or from referral as a result of a routine medical consultation, a consultation for an unrelated condition, or on the basis of a possibly increased risk for developing breast cancer (family history or other known risk factor).
▶Screening, Breast Cancer

Optical Contrast Agents

▶Molecular Probes, Optical Probes

Optical Imaging

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Synonyms

Bioluminescence imaging; Bronchoscopy; Diagnostic imaging in endoscopy; Fluorescence imaging; Laparoscopy; Microscopy; Ophthalmology or colposcopy; Optical tomography

Definition

Optical imaging is a broad term which can be used to describe a large range of imaging systems, from tissue microscopy, through macroscopic imaging of tissue with endoscopic, laparoscopic, or telescopic systems. Generally, this wording is used to describe optical imaging, when compared with other imaging systems which do not use optical detection, such as X-ray, ultrasound, or magnetic resonance. Optical imaging is carried out with a light sensitive system for capturing the images. Standard optical imaging systems use a charged coupled device (CCD), for detecting the image, having many pixels and a lens or fiber optic system, which is customized for the application. Analysis of the system performance in terms of resolution and contrast are always specific to the geometry and the tissue being imaged.

Imaging of tissue *in vivo* is largely based upon the effects of absorption and scattering of the light as it interacts with tissue, causing chromatic changes or allowing viewing of morphologic features. Some *in vivo* imaging relies upon tracking temporal changes or changes in response to a stimulus, but this is less common. Preclinical or *ex vivo* imaging, such as in pathology typically relies upon contrast from exogenously introduced agents which are specific to chemical features of the tissue.

Optical imaging of contrast agents *in vivo* is also used in experimental and developmental work as well as a few clinical applications, looking at fluorescence imaging of tissue, where a filter is used on the image detection side to remove the excitation light and only allow the longer wavelength emission light into the imaging camera. Recent developments in biochemistry and experimental biology have introduced a large number of fluorescent proteins and bioluminescent agents that can be transfected into the DNA of cells and animals, thereby allowing optical imaging of organs or specific gene-expression. The use of optical imaging in experimental biomedical research has increased substantially due to these developmental areas.

Fiber Coupled Systems

Fiber optic coupled systems are used throughout routine medical practice in imaging the interior cavities of the body, or imaging organs during surgical intervention or exploratory examination see Endoscopy, Brochoscopy or Laparoscopy. Generally, the optical fibers bundle is used to translate the image from a lens inside the body to a remote camera which is mounted on the exterior end of the device being held by the person doing the procedure. The endoscope is used in this way for imaging the digestive tract, and similarly a bronchoscope is used to image the airways. These are typical flexible fiber bundles which allow movement through these complex structures. The video or CCD camera is usually fed to a television for viewing of the procedure in real time by the endoscopist or bronchoscopist.

Lens Coupled Systems

Lens coupled imaging systems are used routinely in ophthamoscopic imaging, cervix imaging, ear imaging, as well as most experimental biology imaging systems. The commonality in this area is that if broadband light imaging is required, the lenses used must be compound lenses to avoid issues of chromatic aberration in the resulting image at the camera. Thus, significant care is taken to design and optimize the lens system and how it focuses onto the camera. Generally, lower the f-number of the objective lens and the closer the lens is to the tissue, the more light will be captured in the imaging procedure.

Microscopy

Microscopy is by far the most widely used application of optical imaging in medicine, yet it is often considered in a different category because it is so specialized. Pathological analysis of biopsied tissue is the most common application here, where the tissue is fixed and stained for imaging.

Tomographic Imaging

Tomographic imaging of tissue is sometimes called more generically optical imaging, but is better described in the Optical Tomography section or Fluorescence Imaging section. The major difference between tomography and imaging is largely considered to be the acquisition of signals from below the surface, thereby allowing reconstruction or backprojection of the image below the surface.

Surface Topography and Tracking

Optical imaging systems are used in many different applications for surface tomographic mapping, and several commercial systems exist either using patterned light generation to measure surface topography or using stereovision cameras together with computed algorithms to create surface maps. These are used in applications where the three-dimensional topography of a tissue surface is needed to be known.

Optical Probes

▶Molecular Probes, Optical Probes

Optical Tomography

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Synonyms

Diffuse imaging; Diffuse optical tomography; Frequency-domain photon migration; Near-infrared imaging; Near-infrared tomography; Photon density wave imaging; Photon migration; Time-resolved optical tomography

Definition

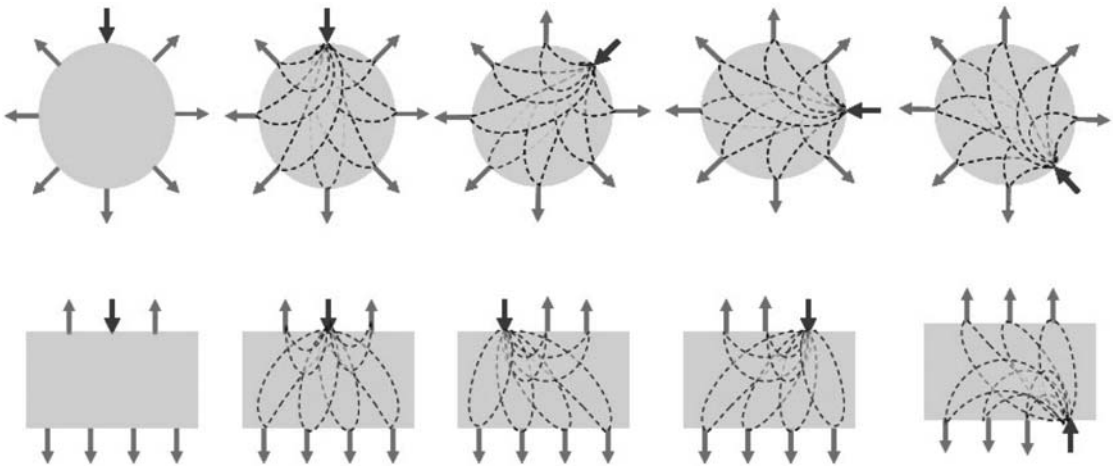
Optical tomography has been an active area of research study since the late 1980s, when both technological and algorithmic breakthroughs led to workable methods to understand light transport in tissue and solve the inverse image reconstruction problem. The exact tools for tomographic imaging vary widely in terms of hardware and software, but the commonality is that the method allows imaging of the interior of tissue, by recovering the interaction coefficients or chromophore and scattering parameter maps. Near-infrared light, in the range of 650 to 950 nm, is most commonly used in optical tomography as it has the lowest scattering and absorption values, thereby providing the best penetration through tissue possible. Imaging through volumes of up to 10–14 cm is possible given sufficiently designed equipment. Tomographic measurements in this wavelength range are most sensitive to the molecules that absorb light, which are hemoglobin, oxyhemoglobin, water, and lipids. Thus imaging tissue function related to blood concentration and oxygen saturation is possible with near-infrared tomography. In addition, injection of absorbing or fluorescent contrast agents is possible, providing further information about the tissue function. The latter application of imaging fluorescent agents is often called fluorescence tomography, and described in another section on Fluorescence Imaging. Imaging with optical tomography is commonly applied to imaging brain tissue for functional physiology studies, or neonatal cerebral imaging for tracking disease, or female breast cancer imaging. There are few commercially available devices which do optical tomography, but a larger number of experimental and investigational devices have been developed in research labs (1).

Tomography of tissue requires measurement of multiple cross-sectional measurements of light transmission from several different source and detector locations. When overlapping paths are measured, as with X-ray computed tomography, it is possible to mathematically reconstruct the interaction coefficient maps of the tissue interior. In the case of optical tomography, this inverse problem is not as analytically tractable as in the filtered backprojection methods used in X-ray tomography (2). The path of the light is highly scattered, and so the transport mechanism is dominated by elastic scatter. Optical tomography became more intensively studied when it was established the optical path length through a scattering medium could be measured by time-resolved or frequency-domain signals, by directly measuring the signal propagation time. Through modeling the transmission process as a diffusive transport problem, it is possible to mathematically quantify independent absorption and transport scattering coefficients (1). Existing systems typically use this approach to them compute concentration maps of hemoglobin, or relative oxygen saturation of the hemoglobin, or water and lipid fractions in the tissue, through computation of the exact absorption coefficient spectrum.

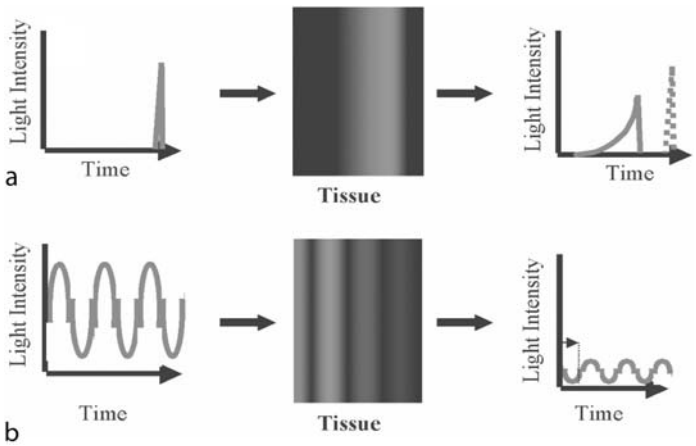
Characteristics

Most systems are based upon multiple fiber optic placements upon the tissue to be imaged, and illuminating the source light into the tissue while detecting the remitted light at all other fiber points. When this is completed, the light is cycled to each of the source fibers in turn to provide multiple overlapping measurements through the tissue volume, as indicated in Fig. 1. Recovery of the remitted light can also be done with noncontact application as well, delivering the light through a scanned beam and picking up the remitted intensity with optics to fibers or charge coupled devices (CCD) (3).

The type of light signals used in these systems are divided into time-resolved signals or frequency-domain signals or continuous wave signal illumination, as illustrated in Fig. 2. Each of these provide slightly different information about the tissue, but all have been shown to allow separation of absorption and scattering effects with different levels of success. It is largely agreed that time-resolved signals provide the optimal separation of the scattering and absorption coefficients, whereas frequency domain provides nearly equal ability. However, continuous wave signals are thought to be insufficient for separation of the effects of absorption versus scatter, without the use of multiple wavelengths or other



Optical Tomography. Figure 1 Illustrating two common geometries for imaging with optical tomography, namely an effectively circular or thick slabs of tissue volume with a single source and multiple detectors. The source location is cycled around to make multiple overlapping projection measurements, and the banana-shaped projections are the diffuse photon paths, which represent the dominant measurement path. *Inward arrows* represent the sources here and *outward arrows* represent the detectors.



Optical Tomography. Figure 2 An illustration of a time-resolved measurement (a) where a short pulse of light is transmitted through a hypothetical block of scattering tissue, spreading the pulse out in time, and a frequency-domain measurement scheme (b) using a sinusoidally modulated light intensity transmitted through the tissue.

constraining information. Measurement detectors are either photomultiplier tubes for the weakest light signals, or photodiodes, diode arrays or CCDs. Multichannel detector devices have been produced in many different configurations.

Diffusion theory based reconstruction requires the iterative solution to a perturbation type equation or a Newton method approach (2). Both of these require some initial guess of the tissue properties, often assumed to be the best homogeneous estimate, and then the image reconstruction process uses the Jacobian or sensitivity matrix to iteratively improve the estimate of the coefficients at each pixel within the image. The goal of the reconstruction is to minimize an objective function, which is often calculated as the normalized square difference between the calibrated data set and the calculated diffusion theory prediction of the signal. This minimization process requires inverting the Jacobian matrix, which is an ill-posed problem, and therefore requires careful regularization (addition of a constant term to the diagonal of the matrix) to make it invertible. This process inherently smooths the overall resulting image, and the value of the regularization parameter is often estimated for each particular problem, often in a Levenberg–Marquardt methodology.

Most current systems use multiple spectra measurements, thereby allowing recovery of the absorber concentration maps from the absorption coefficient spectra, and

with *a priori* knowledge of the chromophore extinction coefficients. It has been shown that direct reconstruction of the chromophores can also be achieved by including the spectral fitting process within the inverse image reconstruction problem, and this process reduces the ill-posed nature of the problem, and improves the accuracy.

Applications in Preclinical Studies

Small animal imaging with near-infrared tomography has been a highly used avenue, for studies in brain function and tumor imaging. Imaging of tumor characteristics *in vivo* has been possible both without and with a scattering coupling medium. Imaging of brain tissue function, in terms of hemoglobin and oxygen saturation has been done extensively and used for functional brain activation studies.

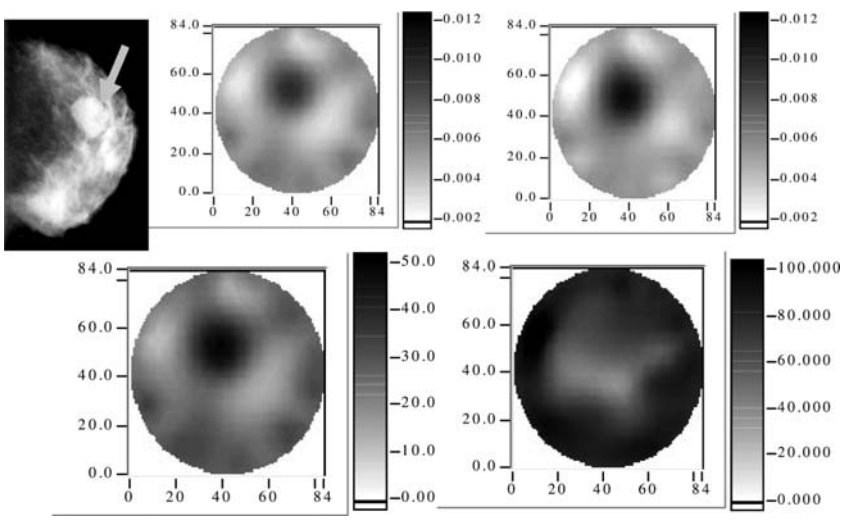
Applications in Brain Diagnostics

Significant interest in optical tomography was initiated with the possibility of monitoring or measuring neonatal cerebral hemodynamics *in vivo*. Commercial systems are available for spectroscopy monitoring of the brain in neonates with single point transmission measurements, and the desire to extend this to a full tomographic system was the driving force for much of the development of optical tomography throughout the past 20 years. Prototype systems have been developed in several labs and are still undergoing feasibility assessment for their potential use. Point spectroscopy of hemodynamics in the brain during childbirth and in utero have both also been shown and used in studies. Measurement of cytochrome oxidase changes in oxygenation within the brain have also been studied, with limited success in this area.

Brain topography systems have been studied extensively and commercialized by Hitachi as a tool to study cognitive function in humans. The difference between topography and tomography lies in the depth penetration of the light, with topography being surface measurements across large areas of the brain, without any reconstruction of the lower lying areas. This approach is simpler and more successful than tomography, and provides important information about the outer cortical layers where blood flow and oxygen consumption issues can readily be measured in response to energy demand in the brain cells. Study of cognitive function with optical topography and optical tomography is a highly active area of research and development (4).

Applications in Breast Cancer

Breast cancer imaging with optical tomography has been shown in many laboratories, with large clinical trials ongoing and being sponsored by the US National Cancer Institute. Commercial prototype systems have been created by a number of companies, with each having very different technological approaches and different clinical goals. Initial feasibility as a screening tool for widespread use has largely proven unsuccessful, however niche applications in screening high-risk groups, younger women or complicated tissue



Optical Tomography. Figure 3 (a) Radiographic image of a patient's breast, showing a well-localized fibroadenoma in the upper central region of the breast (marked with an *arrow*). The resulting NIR images of absorption coefficient at (b) 750 nm and (c) 800 nm wavelength are shown, along with the computed images of (d) total hemoglobin and (e) oxygen saturation. The gray-scale bar units for absorption coefficient are mm^{-1}. The units for total hemoglobin are in micromolar, and the units for oxygen saturation are in percent relative to 100% oxygenated hemoglobin.

cases remains a possible avenue for use. Integration of optical tomography into MRI scanners has been demonstrated and continues under development. A set of example images from a subject with a focal fibroadenoma tumor in the breast is shown in Fig. 3 (5).

The ability to image tumor properties of hemoglobin, oxygen saturation, water fraction, and scattering has undergone rapid development in the past 5 years. Direct recovery of these concentration images has been shown to improve the accuracy of the values, through multispectral fitting and reconstruction in a single step.

Optical tomography has also been coupled into standard clinical imaging systems, including MRI, ultrasound, X-ray tomography, X-ray tomosynthesis, MEG, EEG and is thought to provide complementary information, predominantly related to hemoglobin, oxygen saturation and uptake and retention of injected contrast agents.

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Optical Tracers

►Molecular Probes, Optical Probes

Oral Cavity, Inflammatory Diseases

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Synonyms

Inflammation; Inflammatory lesion

Definition

An inflammation is a non-specific immune response in reaction to any type of injury: pathogenic organisms, injuries, foreign bodies or ionising radiation. It may remain localized, sub-clinically and temporarily if the body's defensive mechanisms are effective. An inflammation can be acute, primary-chronic or an acute inflammation may persist and spread by extension to become a subacute or chronic state. Inflammations of the oral mucosa are called depending on their localization as follows:

- Glossitis—inflammation of the tongue
- Stomatitis—inflammation of larger parts of the oral mucosa
- Gingivitis—inflammation of the gum
- Cheilitis—inflammation of the lips

If deeper structures are involved it might be a ►phlegmon or ►abscess.

Pathology

Regarding the severity, macroscopic appearance, pathogenic agent and extent of the inflammatory process, various kinds of inflammation in the oral cavity do occur.

Aphthae are intra-epithelial cavities with a size up to 5 mm filled with serous fluid. They are caused by virus, toxic substances or medicaments. Habitual aphthae are characterized by chronical recurrent aphthae of unknown aetiology. A ►vasculitis is the underlying lesion of Behcet's disease—a seldom, systemic, HLA-B51 associated illness—in which recurrent aphthae of the oral cavity and genital region appear together with other organ manifestations as uvea, skin, joints and central nervous system.

In herpes simplex labialis, small vesicles at the border between the dermis and mucosa of the lips appear due to herpes simplex ►infection. If the inflammation extends on the entire oral cavity it is called herpetic stomatitis (synonym: stomatitis aphthosa). In a later stage of the disease, vesicles proceed to mucosal erosions and deeper ulcerations with red border.

Ulcerative stomatitis (synonym: necrotizing ulcerative gingivitis) is an extended, severe, necrotizing, oedematogenic inflammation caused by a complex of different organisms (streptococci, spirillum, fusiform bacteria and fungi) in immune-compromised patients or bad oral hygiene.

Oral herpes zoster infection is caused by the reactivation of varicella-zoster virus in the area of innervations of the maxillary, mandibular or seldom glossopharyngeal nerve. It is characterized by unilateral crops of clustered vesicles.

Oral candidiasis (synonym: thrush), an opportunistic infection especially in immune-compromised patients, is mainly caused by *Candida albicans*. It is usually limited to the skin and mucous membranes. White pseudomembranes consisting of mycetes and necrotic epithelium are a characteristic finding.

Acquired immunodeficiency syndrome (AIDS) favours the development of opportunistic bacterial, viral or mycotic infections including oral candidiasis and herpes simplex viral infection. Hairy leukoplakia—a column-shaped hyperkeratosis of the acanthoticaly broadened epithelium—can proceed years before the outbreak of AIDS.

In a phlegmon or ▶cellulitis, there is a diffuse spread of granulocytic infiltrates within the tissue. An ▶abscess consists of a necrosis—resulting from purulent colliquation of tissue—and a membrane, which borders on vital tissue. Both are kinds of bacterial infection (streptococci, staphylococci). Most often, they occur in the floor of the mouth due to secondary involvement from other spaces: masticator space (dental infection) or submandibular space (sialolithiasis).

Ludwig's angina is an extensive bacterial infection of the floor of the mouth that always involves both the sublingual and submandibular space. It is frequently bilateral and produces gangrene or serosanguinous phlegmon, but little or no frank pus; involves connective tissue, fascia and muscle, but not glandular structures (1).

In necrotizing fasciitis, there is severe or extensive phlegmon that extends into the superficial and deep fascia, producing thrombosis of the subcutaneous vessels and gangrene of the underlying tissues. The oral cavity may be involved.

The oral cavity can be involved in syphilis (synonyms: lues, treponemiasis), a *Treponema pallidum* infection. Primary affection shows a coarse infiltrate or ulceration of deep red colour (ulcus durum); secondary affection flat the infiltrates, ulcers with red halo and whitish blur on mucosa; tertiary affection causes bleeding, necrotizing and rubbery infiltrations, the so-called gumma.

Actinomycosis, caused by *Actinomyces israelii*, is characterized by hard, bluish-violet infiltrates—actinomyces drusen with leucocytes and foam cells—and fistulas to the skin as well as to bone (2, 3).

Clinical Presentation

Characteristic signs of nearly all oral cavity inflammations are pain, burning of the tongue and/or mucosa and dysphagia. Primary and tertiary affection in syphilis and infiltrates in actinomycosis are painless. In oral herpes zoster, the pain is of severe neuralgic character. Disturbances of tasting are typical for glossitis and oral candidiasis.

A reduced general condition and fever occur in ulcerative stomatitis, herpetic stomatitis, oral herpes zoster, purulent infections, Ludwig's angina, secondary syphilis and Behcet's disease. The fever is very high in purulent infections and subfebrile in Behcet's disease.

Swelling of lymph nodes accompanies ulcerative stomatitis, herpetic stomatitis, purulent infections, Behcet's disease and primary syphilis. Except for syphilis, the enlarged lymph nodes are usually painful. The clinical pictures of necrotizing fasciitis which occurs seldom in the head and neck region is characterized by a fulminating onset of the disease, painful edema, erythema, warmth, tenderness and septicemia.

Sialism, redness, necrotic odour, haemorrhage and swelling of the gingiva as well as the oral mucosal are seen in ulcerative stomatitis. Sialism also occurs in herpetic stomatitis and oral herpes zoster; necrotic odour in herpetic stomatitis and oral candidiasis. Some pseudomembranes in oral candidiasis are easily wiped off from the affected oral tissues and leave an erythematous, eroded or ulcerated surface, which may be tender.

Phlegmon and abscess of the floor of the mouth show a reddish, oedematous mucosa; they are hard in palpation; trismus may be present (2).

Imaging

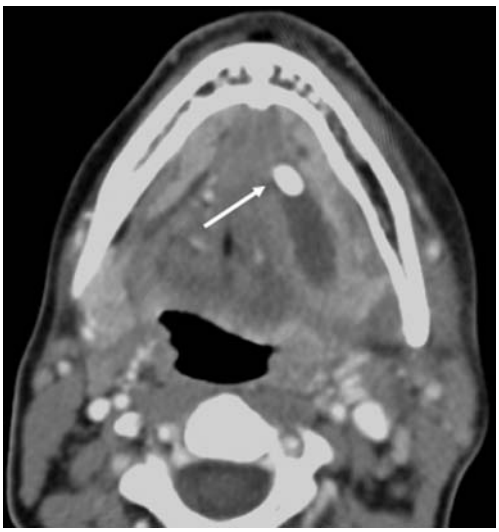
Most oral cavity inflammations are diagnosed by inspection due to their typically macroscopic appearance. An indication for imaging is given in patients in whom a deeper spread, mainly an abscess, is suspected or for exclusion of a tumour in chronic inflammation as actinomycosis or ulcerative stomatitis.

Ultrasound is the first performed imaging modality in acute inflammatory diseases of the oral cavity. If there are equivocal findings, CT or MRI can provide similar information. CT is clearly faster performed, less susceptible to motion artefacts, the better available technique and therefore the preferred method in adults. Considering the lack of radiation exposure, MRI should be given the preference in children.

Ultrasound

The floor of the mouth can be well investigated by ultrasound. The appearance of an abscess depends generally on its stage. In early stage, it is poorly defined and its demarcation against surrounding soft tissue is worse. With maturation it becomes more and more hypoechoic and has finally a dorsal sound-amplification. Fine echoes within the mass are suspicious of gas. If the

infection starts from the submandibular gland, the gland is painful enlarged and hypoechoic. Calculi can be identified up to a size of 3 mm. Ultrasound has limitations in the detection of mandibular involvement, the demonstration of spread into the retropharyngeal space and the visualization of phlegmonous processes.



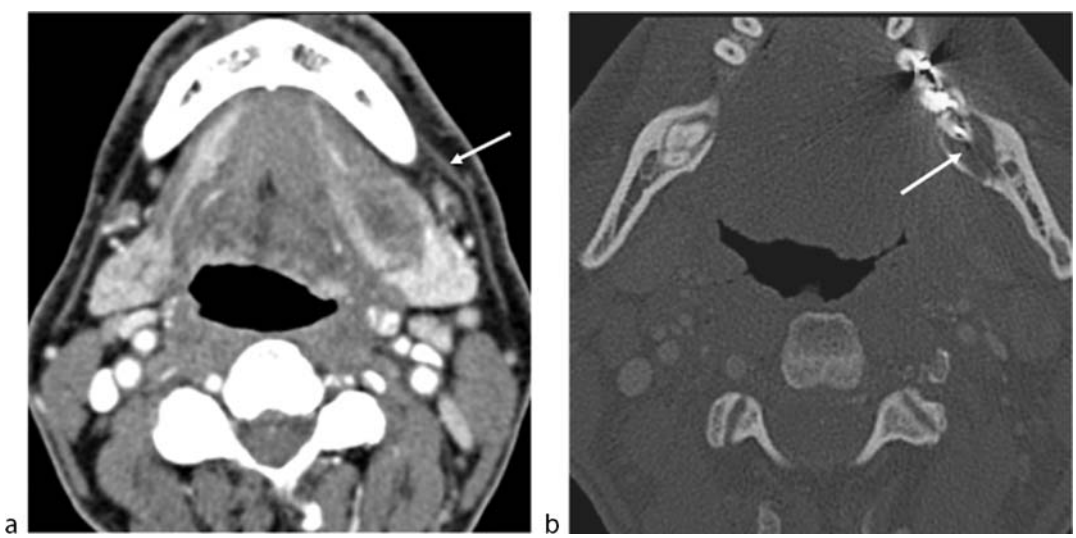
Oral Cavity, Inflammatory Diseases. Figure 1 Axial contrast-enhanced CT of a patient with clinically suspected abscess in the floor of the mouth. CT shows an area of low density as a sign for and abscess surrounded by swollen tissue of increased contrast enhancement and prominent lymphoid tissue on the left. Calculus as a cause could be detected (arrow). The patient was treated by incision and antibiotic therapy.

Computed Tomography

On CT, an abscess appears as an area of low density, with or without gas collection. In mature abscesses a rim enhancement is seen. Surrounding structures are usually involved: muscles are thickened, fat is oedematous with increased density, overlying fascia and skin are thickened showing an enhancement and draining lymph nodes are enlarged. Calculi in the salivary glands or major ducts, erosions of the mandibular cortex and a dental origin can be demonstrated (Figs 1 and 2). A phlegmon and an abscess cannot always be differentiated by clinical methods. Changes—as described in the neighbourhood of an abscess—without abscess formation are found on CT. In necrotizing fasciitis, CT supports the clinical diagnosis; identifies gas, the spread of the disease along vascular sheaths and into the mediastinum (4).

Magnetic Resonance Imaging

On MRI, an abscess has low T1-weighted and high T2-weighted signal intensities. As in CT only mature abscesses show a rim enhancement. Oedematous changes within the fat are not quite well visible as on CT. MR-sialography



Oral Cavity, Inflammatory Diseases. Figure 2 CT of a patient with dysphagia and swelling of the mouth floor for 2 days. After ultrasound a phlegmon was suspected. (a) Contrast-enhanced scan demonstrates a diffuse swelling of soft tissue in the floor of the mouth without clear abscess formation on the left. The neighbouring fat has an increased density and the fascia is thickened (arrow). (b) High-resolution scan in bone window gives a hint of the underlying dental infection (arrow). The molar was extracted and the infection treated by antibiotics.

has to be performed for the visualization of calculi. MRI is superior to CT in the demonstration of the medullary component, if there is an accompanying ▶osteomyelitis in the mandible.

Nuclear Medicine

In diagnostics of oral cavity inflammations, nuclear medicine plays a limited role. In suspected osteomyelitis of the mandible, bone scintigraphy can demonstrate an increased tracer uptake, but MRI has the same sensitivity and gives additionally exact anatomic information.

Diagnosis

The diagnosis of the oral cavity inflammation is based on anamnesis, inspection, mirror investigation and laboratory tests. In bacterial and mycotic diseases, smears are taken for identification of germs. In lues, spirochetes can be serologically proved 4 to 5 weeks after infection. Varicella-zoster-virus-titer is increased in herpes zoster. In Behcet's disease and oral candidiasis the differential diagnostics should include a HIV-test; in the first one also a lues-serologic-test. Actinomycosis and syphilitic gummas need a histological proof.

Imaging—ultrasound or CT, seldom MRI—is performed in suspected deeper inflammation to demonstrate the extent, to document gas-forming infection, to show the relationship to neighbouring bony structures and to give a support in the decision on a conservative or surgical therapy. CT is able to reveal underlying dental infection and sialolithiasis. The latter one can also be found by ultrasound.

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Organized Screening

Screening program organized at the locoregional or national level. With an explicit policy, it is a team responsible for organization and for delivery of the screening services, and a structure for quality assurance.
▶Screening, Breast Cancer

Oropharyngeal Foreign Bodies

▶Foreign Bodies, Gastrointestinal

Orthotopic Liver Transplantation

OLT represents the most common procedure for liver transplantation and consists in the replacement of the diseased liver by a liver coming from a cadaveric donor.
▶Transplantation, Liver

Os Carpale

The postarthritic ankylosis of two or more (incomplete os carpale) or all carpal bones as a typical feature of late-stage rheumatoid arthritis.
▶Rheumatoid Arthritis

Osgood–Schlatter Pattern

Overgrowth and fragmentation of the secondary growth center of the anterior tibial tubercle with swelling of the inferior patellar tendon; may be an overuse syndrome or related to repeated minor trauma.
▶Osteonecrosis in Childhood

Osmotic Myelinolysis

Osmotic myelinolysis, also referred to as central pontine myelinolysis when it involves the pons, is a disorder associated with chronic alcoholism and malnutrition. It is due to chronic electrolyte imbalances, most commonly hyponatremia. Osmotic myelinolysis is frequently precipitated by rapid iatrogenic sodium correction, resulting in breakdown of the blood–brain barrier and a

noninflammatory demyelination with relative preservation of neurons and their axons. The central pons is the most commonly affected site. Clinically, patients present with seizures, dysphagia, pseudobulbar palsy, dysarthria and movement disorders.

▶Toxic Disorders, Brain

Ossification of Posterior Longitudinal Ligament

OPLL results from growth of lamellar bone posterior to the vertebral bodies involving (by calcification) the posterior longitudinal ligament. It is usually diagnosed in elder patients from its characteristic radiographic appearance and may lead to severe neurologic deficit due to spinal canal stenosis.

▶Dish

Ossification or Calcification of Ligamenta Flava

Enthesopathy, ossification (mostly thoracolumbar spine), and calcification (often cervical spine) in (frequently) thickened ligamanta flava may contribute to the entity of OLF. There is coexistence of OLF with both DISH and OPLL.

▶Dish

Osteitis Condensans ilii

A benign condition characterized by sclerosis in the iliac bones adjacent to the sacroiliac joints. The sclerosis is generally bilateral and usually asymptomatic. The adjacent sacroiliac joint is unaffected. This entity is thought to represent a stress reaction and is most commonly found in postpartum patients.

▶Fractures, Stress

Osteitis Deformans

▶Paget Disease

Osteitis Pubis

A noninfectious inflammatory condition involving the pubic bone and pubic symphysis that is thought to occur secondary to periosteal trauma. It occurs most commonly in postsurgical patients or in athletes. Radiographic abnormalities include sclerosis and osteolytic changes. Its imaging characteristics resemble osteomyelitis, However, it is a separate clinical entity and responds to rest and anti-inflammatories.

▶Fractures, Stress

Osteo Sarcoma

▶Neoplasms, Bone, Malignant

Osteoarthritis

'Osteoarthritis' and 'osteoarthrosis' are both used in medical terminology, however, osteoarthrosis may be the more appropriate term since inflammatory changes are not pronounced in most of the joints. The best phrase to describe degenerative changes of articulations is 'degenerative joint disease'.

▶Degenerative Joint Disease, Peripheral Joints
▶Gout

Osteoblastoma

Osteoblastoma is a benign osteoblastic tumor that differs from osteoid osteoma in having a "nidus" larger than 1.5 cm in diameter, by showing more variable histologic features, and by possessing a potential for local bone destruction and aggressiveness.

▶Neoplasms, Bone, Benign

Osteochondrodysplasia

▶Osteodysplasia
▶Dysplasia, Skeletal

Osteochondroma

Osteochondroma (osteocartilaginous exostosis) represents a benign cartilage-forming lesion that consists of a bony outgrowth covered by a cartilaginous cap.
▶Neoplasms, Bone, Benign

Osteochondrosis Dissecans

Sometimes called osteochondritis dissecans, it is a traumatic osteochondral injury, usually along a convex articulating surface of a bone.
▶Osteonecrosis in Childhood

Osteoclast-Like Giant Cell Tumor, Pancreatic

Osteoclast-like giant cell tumor is a rare pancreatic tumor that closely resembles giant cell tumor of bone. It is composed of undifferentiated spindle-shaped epithelial or mesenchymal cells mixed with non-neoplastic osteoclast-like giant cells. Some tumors also contain areas of ductal adenocarcinoma. At imaging, the tumor may present as a solid inhomogeneous mass or as a cystic lesion. Invasion of adjacent structures is common, but metastatic spread is found in only 50% of patients. Prognosis is more favorable than for ductal adenocarcinoma.
▶Carcinoma, Pancreatic

Osteodysplasia

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Synonym

Osteochondrodysplasia

Definition

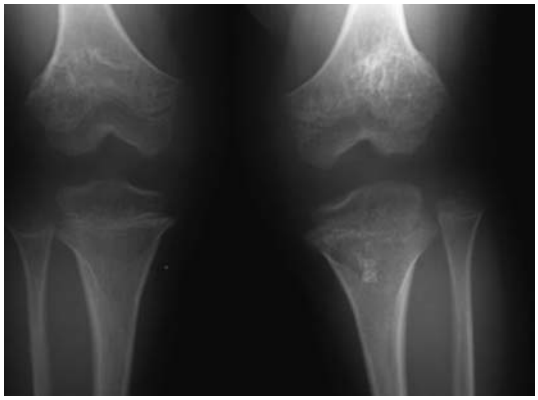
Osteodysplasias are well-defined abnormalities of bone growth, typically genetic in origin. They may be systematic, affecting one aspect of skeletal development, typically in proportion to growth potential of each site, or else "aleatoric," affecting sites of an aspect of bone development in a chance distribution, with a higher likelihood of occurrence at sites of greater normal growth. [I have borrowed the term aleatoric from modern music composition in which chance plays a role (1)] The aspect of growth may be slowed, disordered, or accelerated. The aspect involved is most often exclusively enchondral in nature or membranous in nature, with only secondary changes in the other. Most, but not all, dysplasias result in short stature (dwarfism). Kindred disorders include dysostoses, in which individual aspects of growth are affected, and sequences, in which an abnormal aspect of development causes a chain of consequent effects on bone (and other tissue) development. As more genetic markers for dysplasias are determined, more conditions are being grouped into "families" of dysplasias of like genetic origin.

Pathology/Histopathology

Various histologic manifestations of various dysplasias are known, some of which help explain radiographic findings. In multiple exostoses, the cartilage cap has a growth plate histologically close to the pattern of a normal physis, but somewhat more disorganized. In ▶achondroplasia, not only are the physeal and acrophyseal enchondral columns shorter and slower to progress from resting cartilage to provisional calcification, but also the columnar cells are grouped into separated clusters. The clustering and subsequent lack of ossification of the zones between the clusters, within the primary spongiosa zone of the metaphysis, may account for the enchondromatous areas in achondroplasia metaphyses and diametaphyses (Fig. 1). In Kniest disease, growth cartilage is characteristically irregular in pattern, described as Swiss cheese-like. Osteogenesis imperfecta shows osteoporosis. Cultured fibroblasts from persons with this dysplasia help characterize the condition. Osteopetrosis has denser than normal bone, similar to the hibernating bat, perhaps due to a temporary failure of osteocytic osteolysis, although most investigators favor a failure of osteoclast function as the direct cause.

Clinical Presentation

Disproportionate short stature is found in many systemic dysplasias; limb length discrepancy, with limp, is frequent in aleatoric dysplasias. Fractures (and blue sclerae) are a



Osteodysplasia. Figure 1 Example of prominent enchondral rests in the metaphyses of distal femur of a 9-year-old boy with achondroplasia. The medial and lateral margins of the femur metaphyses and diametaphyses are more concave than normal, partly as a manifestation of achondroplasia enchondral growth slowing and partly because the knees are somewhat flexed.

hallmark of osteogenesis imperfecta; teeth tend to show dentinogenesis imperfecta crowding and irregularity as well. Tall stature is seen in the ►dolichostenomelias, notably Marfan syndrome and homocystinuria. With regard to characterization of short limbs in dysplasias, the term rhizomelic refers to shortening of limbs most pronounced proximally, such as achondroplasia; ►mesomelic dysplasias have most of the relative shortening in the intermediate segments (radius/ulna and tibia/fibula); and acromelic refers to the greatest shortening distally. Other rather specific findings include hitchhiker thumb in diastrophic dysplasia; thumb extension beyond the fist in Marfan syndrome; early-in-life shortening, especially of the limbs and later shortening of the trunk in metatropic dysplasia; gargoyle-like facies in the mucopolysaccharidoses; and occasionally the ability to draw the shoulders together in front of the chest in cleidocranial dysplasia/dysostosis.

Imaging

Skeletal surveys for osteochondrodysplasia or dysostosis need to be customized for the suspected diagnoses; once certain findings appear, other views may be required. For example, whenever platyspondyly (flatter than normal vertebral bodies) is observed, one needs to check the dens and C1 region in detail, with careful flexion and extension views as necessary, to evaluate for associated subluxability between C1 and C2. The small tubular bones of the hand and feet should be included in any survey because they may show diagnostic features key to the diagnosis. Tall patients with dolichostenomelia (such as Marfan) need good lateral chest or thorax images to evaluate for pectus excavatum or carinatum. In aleatoric disorders such as multiple exostosis or enchondromatosis, two orthogonal views of each part are needed for complete evaluation of the lesions, even if not necessary for diagnosis.

In utero imaging for dysplasia or dysostosis begins with high-detail ultrasound. Questions that arise may then be further investigated with magnetic resonance imaging (MRI). If the answer is still not given, but is important, selected radiographs of the mother's abdomen may be considered, under close supervision by a specialized pediatric radiologist. If possible, for radiation protection considerations, radiographs of one twin should be avoided if the other is considered unaffected. Abnormal bone length, shape, or a positive family history of dysplasia or dysostosis should initiate the careful prenatal evaluation for specific entities.

MRI can give insight into the nature and quantity of cartilage in several dysplasias, including Kniest disease (2). MRI of the spinal cord is prudent before planned spinal surgery in children with any severely abnormal spinal curvature. Three-dimensional reconstruction of abnormal joints assists in surgical planning for specific procedures.

Accurate measurement of long bone lengths requires perpendicular X-ray beam images at each end of the bone with a radiopaque ruler alongside that is not shifted between the exposures. Alternately, measurements can be made from a computed tomography (CT) scout view.

Long bones for dysplasia evaluation need to be parallel to the film or screen, lest foreshortening in space be mistaken for dysplastic growth foreshortening through time.

Nuclear Medicine

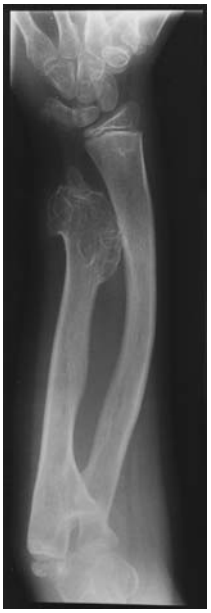
Multiple cartilaginous exostoses each have a cartilage cap with a growth plate (I call it "paraphysis") that will have uptake on bone scan similar to physes. Fractures in such conditions as osteogenesis imperfecta, Ollier enchondromatosis, and osteopetrosis will be hot on bone scan. The periosteal reaction causing the thick cortices of Engelmann disease will be hot on bone scan in the active stage, as it will be in other membranous bone overactivity dysplasias or dysostoses, such as van Buchem disease. Osteopetrosis usually gives a superscan of high activity of all involved bone segments. Uptake is also increased in active fibrous dysplasia.

Diagnosis

Only a few dysplasias and dysostoses will be described here.

Systematic dysplasias are symmetric side-to-side and generally change one rule or rate of growth, with the sites of greatest normal growth being affected the most. The prototype is achondroplasia with rhizomelic shortening; lumbar pedicles abnormally close side-to-side with the distance decreasing downward, greater than normal concavity of medial and lateral margins of metaphysis (Fig. 1), frontal bossing of the skull, petrous ridges closer to each other than normally, frequent cartilage rests in metaphyses, smooth but delayed growth centers with epiphyseal shape following the shape of abnormally concave physes, proximal ends of the femurs in infancy showing the pattern of an ice cream scoop on end, horizontal sacrum, shorter than normal metaphyseal collar, and trident (Vulcan salute) hands, among the many imaging findings. Hypochondroplasia, an allelic (due to the same gene locus) disorder, is less severe; thanatophoric dysplasia, also allelic, is more severe (demonstrating femurs resembling European telephone receivers and quite pronounced platyspondyly). Achondroplasia and its family results from slowed enchondral growth, whereas Marfan syndrome and homocystinuria have dolichostenomelic (predominant proximal segment) lengthening due to accelerated enchondral growth. The medial and lateral margins of long bone metaphyses are less concave than normal. Children with Marfan syndrome show pectus carinatum or excavatum, tortuous aortic arch, and wide lumbar canal; homocystinuria shows truncal osteoporosis.

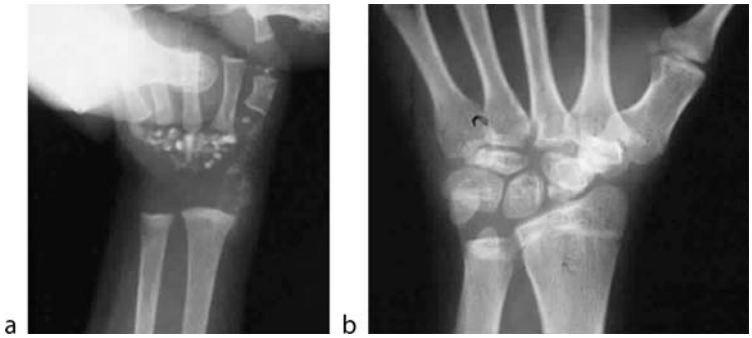
Among the aleatoric dysplasias, the findings of exostosis or enchondromas occur in a chance distribution, more likely occurring in areas of greater growth potential. The more and the larger the exostosis, the greater the impairment of longitudinal growth of an affected long bone. If one paired bone is more affected than its mate, bowing and, often, dislocations occur (Fig. 2). Exostoses of epiphyses and their equivalents are termed Trevor disease (also known as dysplasia epiphysealis hemimelica); exostoses also can occur at terminal tufts of phalanges and the nonepiphyseal ends of other short tubular bones. Osteogenesis imperfecta is systemic with regard to osteoporosis but aleatoric with regard to fractures. The multiple wormian bones also show side-to-side symmetry in distribution. The weakened bones may lead to tam-o'-shanter skull (protrusio occipiti; basilar invagination) from the calvarium sinking on the cervical spine (if this deformity is severe, the clivus may run upward). Teeth are numerous and irregularly placed. The lateral clavicles may show increased upward convexity; the ribs resemble coat hangers. Prenatal sites of fracture may yield bones that look wide at birth from healing fractures. Treatment of osteogenesis imperfecta with bisphosphonates results in tell-tale nearly parallel thin lines similar in shape to the growth plates in the metaphyses (and equivalent areas in secondary growth centers) that record the jolt to the skeletal system from each dose.



Osteodysplasia. Figure 2 Growth disparity from multiple cartilaginous exostosis/osteochondromatosis in a 10-year-old boy. The ulna has far more exostoses than the radius distally, resulting in a shorter ulna and a secondary lateral bowing of the radius. The more the exostoses, the greater the impairment of longitudinal growth. (From Oestreich AE, Crawford AH (1985) Atlas of Pediatric Orthopedic Radiology. Thieme Verlag, Stuttgart, p 27)

Chondrodysplasia punctata (multiple stippled epiphyses) in infancy consists of dense dots of calcification within unossified growth cartilage; the involved bones are short, irregularly shaped, or even abnormally unossified (Fig. 3). As the child gets older, the stipples resolve, and the pattern becomes one of misshapen epiphyses and their equivalents, a pattern then called multiple epiphyseal dysplasia (Fig. 3).

In metatropic dysplasia, the metaphyseal collar bone bark seems to lack its usual ability to restrict too rapid transverse growth of physis and metaphysis, so that bones resemble dumbbells with unusually broad metaphyses. The megaepiphyseal dysplasia Kniest disease shows coronal cleft vertebral bodies and delayed ossification of the (large) centers for the femoral head. Severe cervical kyphosis is seen (when lateral images are obtained) in diastrophic dysplasia and camptomelic dysplasia and is one of the cervical vertebral anomalies seen in Larsen



Osteodysplasia. Figure 3 From multiple stippled epiphyses to multiple epiphyseal dysplasia. (a) In infancy, one sees the multiple epiphyseal stipples of the distal carpal row as well as the (longitudinally short) first metacarpal. (b) In a 16-year-old, one sees highly dysplastic and narrow distal carpals (i.e., multiple epiphyseal dysplasia) with relatively normal proximal carpals, as well as the longitudinally short first metacarpal. [After F. Silverman. From Oestreich AE (2004) Epiphyseal dysplasias and dysostoses. In: Ferrucci JT (ed) Taveras and Ferrucci's Radiology on CD-ROM Diagnosis Imaging Intervention, Vol 5, chapter 5]

syndrome. A small mandible with an abnormally concave undersurface is the key finding of Pierre-Robin sequence and is seen in camptomelic dysplasia, cerebrocostomandibular syndrome, and Seckel syndrome. Progressive pseudorheumatoid dysplasia has joint region changes closely resembling rheumatoid arthritis, but also has both early and unusually large os trigonum centers behind the talus, and bullet-shaped or Scheuermann-like (irregular endplates) thoracolumbar vertebral bodies unlike rheumatoid arthritis. Every normal child, incidentally, has at least one small os trigonum center that appears near the end of the first decade of life and fuses with the talus in about a year, appearing somewhat earlier in girls than in boys. Then, in the second decade, another os trigonum center appears in some 10–20% of children, which may or may not also fuse to the talus to remain as a posteriorly protruding process.

Postaxial polydactyly and mesomelic short limbs are characteristic in Ellis–van Creveld syndrome. An interesting accompaniment to hand polydactyly is the ham-shaped hamate (a wider than normal hamate ossification in the form of a cured ham).

Various characteristic vertebral body shapes on lateral images occur in the several spondyloepiphyseal and spondylometaphyseal dysplasias, as well as mucopolysaccharidosis dysostosis multiplex conditions. Twisted ribs and long bones are the prime feature of Melnick–Needles osteodysplasty.

On *in utero* imaging, achondrogenesis, the second most frequent lethal skeletal dysplasia, is characterized by a lack of ossified vertebral bodies. Very short ribs, such as in short rib polydactyly syndromes, also predict neonatal demise, as do features of thanatophoric dysplasia (the best-known lethal dysplasia). The shorter the ribs in Jeune syndrome, the less likely the neonatal survival.

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Osteofibrous Dysplasia

Osteofibrous dysplasia (ossifying fibroma, Kempson–Campanacci lesion) is a benign fibroosseous lesion which mostly occurs in the tibia of young children. Histologically, the entity differs from fibrous dysplasia by containing scattered bony trabeculae that are rimmed by active osteoblasts.

▶Neoplasm-Like Lesions, Bone

Osteoid

Uncalcified organic bone phase consisting of collagen fibers (approximately 94%), proteoglycans, and glycoproteins.

▶Osteomalacia

Osteoid Osteoma

Osteoid osteoma is a benign bone-forming lesion of limited size that usually induces reactive new bone

formation. The lesion itself has classically been described as the "nidus" and should, by definition, not override a maximum diameter of 1.5 cm. Larger lesions should be termed osteoblastoma.
▶Neoplasms, Bone, Benign

Osteoidosis

▶Osteomalacia

Osteoma

The most frequent benign osseous tumor of the face and sinusal cavities, usually affecting men over 50 years of age. These tumors are commonly asymptomatic, fortuitously discovered on a radiologic examination, in the frontoethmoidal cavity or in the external auditory canal.
▶Fibro-Osseous Lesions, Facial Skeleton

Osteomalacia

Osteomalacia (OM) is a pathological condition in adult bone metabolism characterized by the impaired and delayed mineralization of osteoid, causing the accumulation of osteoid and altering the mechanical properties of bone.
▶Osteomalacia

Osteomalacia

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Synonym

▶Osteoidosis

Definition

In adult bone metabolism – after closure of the epiphyseal growth plates – ▶osteomalacia (OM) is defined to be a condition of impaired and delayed mineralization of ▶osteoid (organic bone phase) in cortical and trabecular bone. In children, this condition is known as ▶rickets and includes the defective mineralization of the cartilaginous part of the epiphyseal growth plates.

Pathology/Histopathology

Physiological bone remodeling is a dynamic process of constant bone formation and resorption, respectively, carried out on the cellular basis through osteoblasts and osteoclasts. Chronologically, bone formation can be divided into two phases. In phase I the osteoblasts produce osteoid in the organic bone matrix consisting of collagen fibers, proteoglycans, and glycoproteins. In phase II the osteoblasts produce calcium, phosphate, hydroxyl, and carbonate creating a plate-like crystal known as hydroxyapatite, which mineralizes on the osteoid-forming calcified bone. Pathophysiological disturbances in phase II based on alterations of the calcium phosphate homoeostasis – in which the serum calcium X phosphorus product is low – lead to the condition of OM in adults and to rickets in children. The calcium phosphate homoeostasis is maintained by a multitude of factors, and thus the etiology of OM and rickets is very diverse. A large number of disorders are associated with OM and rickets. Nonetheless, hypovitaminosis D is considered to be the most frequent cause of OM. The primary role of vitamin D is its physiological function in opposing a decline of serum calcium. Examples of disturbances in vitamin D metabolism that may cause OM are:

- Low dietary intake
- Insufficient UV absorption of the skin (reduced natural sunlight exposure, skin covering garments, dark skin complexion which leads to the absorption of only certain spectra of the UV light)
- Intestinal malabsorption, e.g., after gastrectomy, bowel resection, or nontropical sprue
- Accelerated vitamin D excretion by virtue of anticonvulsive drugs inducing liver enzymes
- Chronic renal failure leading to a deficient hydroxylation of the liver 25(OH)-vitamin D_3 into the active $1,25(OH)_2$-vitamin D_3 form

As described above, a low calcium X phosphorus product is the pathophysiological starting point for the genesis of OM and rickets, and therefore conditions leading to low phosphorus levels must also be considered:

- Acquired phosphate depletion due to malnutrition, alcoholism, and use of aluminum-containing antacids which bind phosphorus intestinally
- Increased renal excretion of phosphorus caused by an impaired tubular resorption (e.g., adult-onset vitamin D-resistant hypophosphatemic osteomalacia, Fanconi syndrome)

Attention should also be paid to oncogenic conditions causing OM (tumor-induced or oncogenic OM), although they occur seldom. This paraneoplastic syndrome is associated with several different neoplasms, of which the majority are of benign and mesenchymal origin, namely, bone and soft tissue tumors. These tumors release circulating factors that increase the phosphorus renal clearance consecutively causing low serum phosphorus levels, while the calcium serum concentration is normal.

Clinical Presentation

The clinical presentation of OM is unspecific. Frequent complaints of patients suffering from OM are bone pain and muscle weakness. Bone pain is of diffuse and dull character. In general the symptoms begin as lower back pain and spread symmetrically into the pelvic region and the hips, or upward to the vertebral column into the rib cage and the shoulder girdle. Compression, vibration, or mere muscular activity of symptomatic regions may provoke and increase pain, in some cases leading to the adjusted pain-avoidance behavior of the patient, such as cautious walking, in extreme cases even immobilization or the fear of coughing.

OM-related muscle weakness is generally localized proximal to the body trunk, e.g., around the hips involving the gluteal muscle group leading to a waddling gait in late stages of the disease. Whether muscle weakness is secondary to bone pain or of primary genesis is difficult to distinguish. In advanced stages of OM, morphological deformities of the skeleton – due to bone softening and hence reduced mechanical strength – can be observed resulting in hyperkyphosis of the thoracic vertebral column or coxa vara and a high fracture susceptibility.

In children suffering from rickets, morphological deformities of the skeleton vary in their dimension according to the time of onset in relation to the phase of epiphyseal bone growth. In periods of rapid growth, rickets tends to achieve high clinical severity. Craniotabes (softening of the calvaria), late fontanelle closure, rachitic rosary (palpable bloating of the costochondral junctions), Harrison's groove (indentation of the lower ribs at the diaphragm), bowing of the long bones (tibia, femur, radius, ulna), and thickening of the wrist, knees, and ankles (on grounds of metaphyseal widening) are the most visible clinical complications. Muscle weakness to severe muscle hypotonia is another major complication of rickets.

Imaging

With conventional radiographs, computed tomography, and magnetic resonance imaging, a wide range of modalities for diagnosing OM are at hand.

In OM, conventional radiographs are usually obtained first. The overall radiographic appearance of bone can be described as homogenous and fuzzy. The reason for this is the decline of contrast differences between bone marrow and calcified bone due to the increased density of unmineralized osteoid. In contrast to this rather non-specific "ground-glass" appearance, so-called ▶Looser's zones – found in only 5–10% of patients suffering from OM – are considered to be typical. Looser's zones – also known as ▶milkman's zones or ▶pseudofractures – are radiolucent bands of unmineralized osteoid usually oriented perpendicular to the surface of the bones ranging in size from a few millimeters to several centimeters. Looser's zones are frequently found bilaterally, symmetric, at sites where larger arteries are adjacent to the bones (inner aspects of the femur, pubic rami, the lateral edge of the scapula, and the metatarsals); the hypothesis being that the pulsation of these vessels and the reduced mechanical strength of the bones cause pseudofractures of the latter which heal only insufficiently with osteoid.

On computed tomography images the fuzzy "ground-glass" appearance of bones affected by osteomalacia is also observed, and Looser's zone may be identified more accurately in contrast to pathological fractures of other genesis.

The relative increase of unmineralized osteoid in relation to mineralized bone or the relative decrease of mineralized to unmineralized bone can be quantified measuring the overall bone mineral density using either dual x-ray absorptiometry or quantitative computed tomography.

Pseudofractures may be identified as hypointense lines or fissures on T1- and T2-weighted magnetic resonance (MR) images as well as on images acquired using short T1 inversion recovery (STIR) sequences. High signal intensity around the fracture area on T2-weighted and STIR MR images reveals an acute process, whereas isolated hypointense regions are considered to be chronic. Therefore, the clinical activity of OM may be determined using MR imaging.

Nuclear Medicine

For isotope bone scanning, technetium 99m-labeled methylene diphosphonate – which preferably adsorbs onto bone surfaces undergoing new bone formation – is being used. The diagnostic strength of isotope bone scanning for metabolic bone diseases is based on its high sensitivity in detecting and localizing functional alterations before structural changes have taken place. In OM the bone scan often shows a nonspecific increased uptake of technetium 99m involving the entire skeleton. However, Looser's zones may be identified on bone scans

before they are visible on conventional radiographs. For the diagnosis of oncogenic OM and the identification of the primary tumor, it has been reported that tracers selectively binding onto somatostatin receptors (octreotide) may improve the detection rate.

Diagnosis

The difficulty in diagnosing OM arises from its very nonspecific clinical presentation as described above. In the population of elderly patients, diffuse bone pain and muscle weakness can be misinterpreted as rheumatological conditions, thus OM remains undiagnosed, although in most cases it is a curable diseases. Higher awareness and suspicion of osteomalacia in daily clinical routine is therefore essential.

Laboratory test results are given in the form of a table.

Imaging modalities for the diagnosis of OM have been described above; nonetheless, a brief summary in the context of the overall diagnostic procedure is as follows: patients presenting with unspecific symptoms and laboratory findings suggesting that OM might be the underlying cause may first undergo isotope bone scanning in order to identify typical Looser's zones. Conventional radiographs of suspicious regions can validate the diagnosis in some cases, but one should keep in mind that radiographically visible morphological changes may occur rather late in the course of OM as compared to pathological uptake behavior on bone scans. On the other hand, if patients can localize peak pain spots, conventional radiographs may be sufficient for identifying Looser's zone without the use of isotope bone scan. Bone mineral density measurements are rather unspecific for the diagnosis of OM because a multitude of disorders, particularly osteoporosis, are associated with a decreased bone mineral density. Besides having a higher spatial accuracy, computed tomography can be used if it is difficult to ascertain with conventional radiographs whether fractures are caused by OM or other pathological conditions. MR imaging, in addition, can help describe the acuteness of fractures. If all imaging modalities and laboratory tests fail, the best – gold standard – diagnostic modality for identifying OM is the histological analysis of bone biopsies in which the accumulation of osteoid is visible as an increase in the average osteoid volume (>10%) and thickness (>15 μm).

Serum concentration	Vitamin D deficiency	Chronic renal failure	Renal tubular disorders
Ionized calcium	Normal/↓	↓	Normal
Inorganic phosphorus	↓	↑	↓
25(OH)-vitamin D_3	↓	Normal	Varies

(*continued*)

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Osteomyelitis

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Synonym

Bone infection

Definition

The term *osteomyelitis* implies an infection of bone and bone marrow, whereas *osteitis* indicates contamination of the cortical bone only. *Chronic osteomyelitis* results most commonly after trauma or surgery and results from unsuccessful treatment of acute bone infection. A *sequestrum* represents a segment of necrotic bone that is separated from living bone by granulation tissue. An *involucrum* denotes a layer of living bone that has formed about the dead bone. An opening in the involucrum is termed a *cloaca*. A Brodies abscess is an intraosseous abscess, frequently seen in children caused by *Staphylococcus aureus* and represents a subacute or chronic infection. *Chronic recurrent multifocal osteomyelitis* is a rare sterile (noninfectious) disorder primarily involving

children and adolescents and characterized by a prolonged, fluctuating course most often involving the tubular bones, the clavicle, and less often the spine.

Pathology/Histology

Pathology: Osseous structures can be contaminated by three principal routes (1):

1. Hematogenous spread of infection through the blood stream as in osteomyelitis of the child or spondylodiscitis.
2. Direct spread from a contiguous source of infection as seen in diabetic foot infections or decubital ulcers in paralyzed patients.
3. Direct implantation of infectious material into the bone as seen after penetrating injuries, punctures, or other surgical procedures.

Hematogenous osteomyelitis of the *child* is most frequently located in the metaphysis of long bones. In the infant up to 1 year of age, bone infection may be complicated by epiphyseal involvement. Subperiosteal abscesses and septic arthritis may complicate osteomyelitis in older children.

In *adults*, osteomyelitis most frequently results from direct spread from a contiguous source of infection and most of these patients have pedal osteomyelitis due to longstanding diabetes mellitus. Prevalence of *posttraumatic* osteomyelitis directly depends on the degree of traumatization of the involved limb (extensive soft tissue damage, necrotic bone, and presence of foreign bodies). Infection results from direct implantation of microorganism in most cases and *S. aureus* is the most common pathogen. *Postoperative* infection occurs via direct implantation, spread from a contiguous septic focus or hematogenous contamination.

Histology: Bone biopsy is performed if clinical and radiologic evaluation is not conclusive, if a neoplasm is suspected or if microbial diagnosis is attempted. Definite diagnosis of osteomyelitis relies on positive culture results of causative organisms from a biopsy sample. Characteristic histologic findings of bone infection include aggregates of inflammatory cells (including neutrophils, lymphocytes, histiocytes, and plasma cells), erosions of trabecular bone and marrow changes that range from loss of normal marrow fat in acute osteomyelitis to fibrosis and reactive bone formation in chronic disease. Limitations of percutaneous and surgical bone biopsy include sampling error, false negative cultures in patients receiving antibiotics, difficulties in distinguishing other osteopathy from osteomyelitis histopathologically, and the risk to damage the bone as a result of trauma or iatrogenic infection. Culture results of percutaneous bone biopsy specimens in pedal infection may be unreliable due to contamination from underlying infected soft tissue. Bone biopsy cultures in osteomyelitis may be false negative in up to 50%, whereas accuracy of histopathologic diagnosis of osteomyelitis is high.

Clinical Presentation

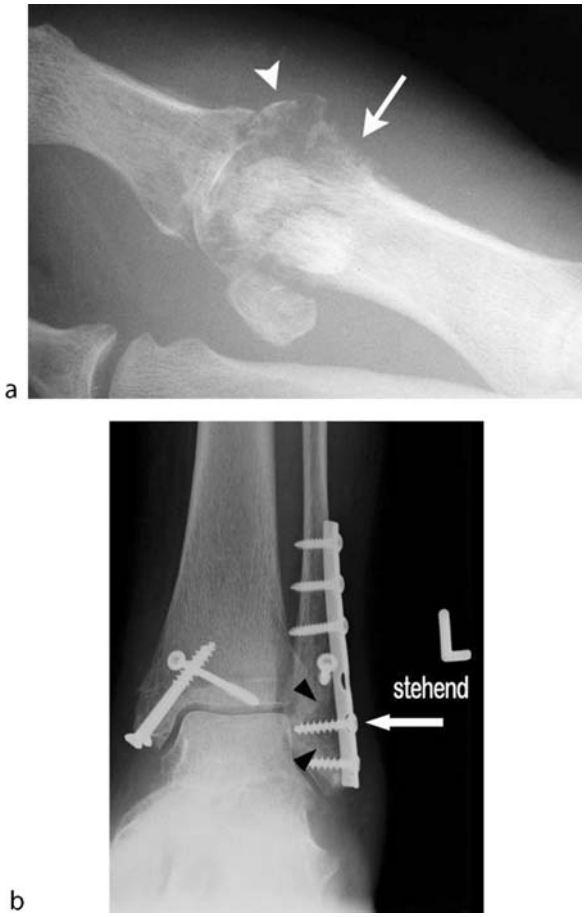
The clinical manifestations of the different forms of bone infections vary considerably depending on the activity of infection. Childhood osteomyelitis is often associated with a sudden onset of high fever, a toxic state, and local signs of inflammation. Diabetic foot infection most commonly has a chronic, relatively indolent course but may quickly lead to septicemia and toxic shock. Tuberculous osteomyelitis differs from pyogenic osteomyelitis by the absence of fever and pain. Posttraumatic and postoperative infections may, in the acute and early stages, lead to exquisite focal symptoms with significant inflammation, fever, and leukocytosis. If the infection can not be cured in the acute stage, chronic recurrent osteomyelitis develops with chronic recurrent bouts of infection, abscesses, bony sequestra, and fistula.

Imaging

Radiographs

Radiographs are usually the first radiological examination performed if bone infection is suspected. In children, first signs of hematogenous osteomyelitis may be perceptible a few days after onset of symptoms. First radiographic signs of hematogenous osteomyelitis are a subtle swelling of the juxtacortical soft tissues, which may only be evident by direct comparison with the other extremity. Destructive metaphyseal osteolysis and periostitis become visible after 8 to 10 days. Brodies abscesses are typically seen radiographically as well-defined metaphyseal radiolucencies with sclerotic borders, "dripping" to the physeal plate.

In adults, radiographic findings of osteomyelitis do not appear in adults for 10 to 14 days after infection and until 35 to 50% of the bone has been destroyed. Radiographic changes of osteomyelitis are not only delayed, but sensitivity is poor. Typical radiographical signs of acute osteomyelitis are permeative bone destruction with periosteal reaction and surrounding soft tissue swelling (Fig. 1a). Beginning cortical bone resorption can be identified as endosteal scalloping, intracortical tunneling, and poorly defined subperiosteal bony defects. Radiographs are also the basic imaging modality in posttraumatic and postoperative osteomyelitis displaying postoperative bony remodeling, sequestra, foreign bodies and osteosynthetic material, and deformities. Acute



Osteomyelitis. Figure 1 Illustration of typical radiographic signs of acute osteomyelitis. (a) Advanced osteomyelitis of the first metatarsal head in a diabetic patient with an ulcer at the plantar aspect of the first metatarsophalangeal joint. The radiograph reveals extensive permeative bone destruction of the first metatarsal head (arrow) with fragmentation (arrowhead). Also note the narrowing of the joint space due to concomitant septic arthritis. (b) Postoperative acute osteomyelitis of the distal fibula with permeative bone destruction (arrowheads) around the second most distal screw (arrow), which is loosened at the plate with a fine radiolucent rim around the screw head.

postoperative and posttraumatic osteomyelitis leads to ill-defined bone destruction, which usually develops in bone directly adjacent to the metal (Fig. 1b). Chronic osteomyelitis can lead to reactive bone formation, periosteal reaction, bony fistula, intraosseous abscesses, and bony sequestra. Presence of surgical implants and posttraumatic bone remodeling may considerably complicate evaluation of chronic osteomyelitis. To evaluate activity of chronic osteomyelitis, it is very helpful to compare current and old films together as changes such as a new linear periosteal reaction, osteolysis, and sequestration are suspicious of active infection. Delayed union or nonunion of fracture may also be caused by chronic infection.

Computed Tomography

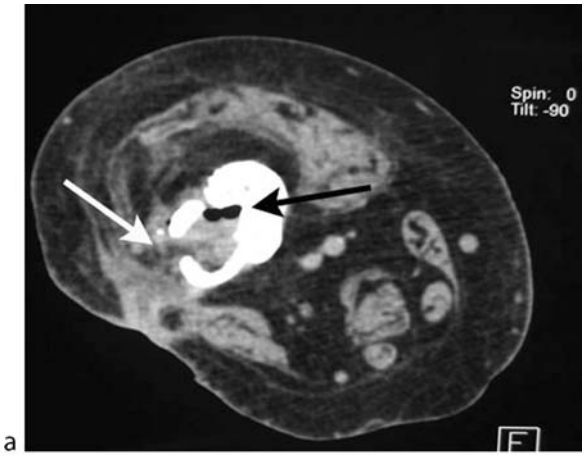
Computed tomography has been largely replaced by magnetic resonance (MR) imaging to evaluate osteomyelitis. CT may however be useful to evaluate the presence of sequestra and involucra in chronic osteomyelitis. Since CT offers exceptional detail of the bony architecture in a cross-sectional display, it can also be used to evaluate cortical destruction and fistula, periosteal new bone formation, and the presence of intraosseous gas (Fig. 2), all of which are less conspicuous on MR images.

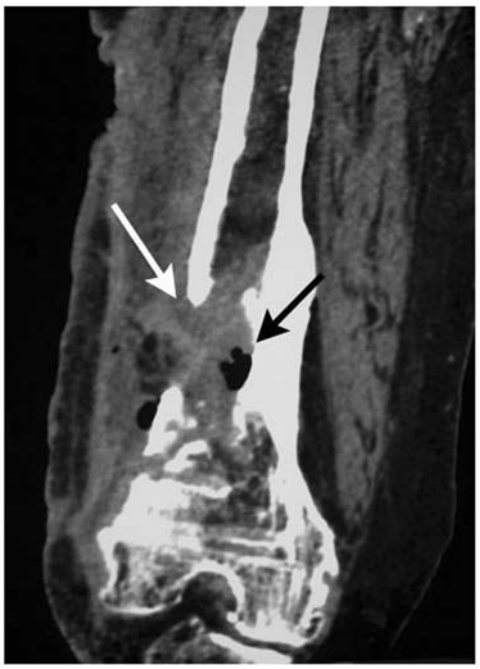
Magnetic Resonance Imaging

MR imaging allows early detection of osteomyelitis in contrast to radiographs and CT. Diagnosis of osteomyelitis on MR images is based on the identification of altered bone marrow signal (Fig. 3). Infection of the marrow compartment results in loss of the normal fatty marrow signal on T1-weighted images, with edema on T2-weighted or STIR images, and enhancement on post-gadolinium T1-weighted images. MR protocols should include fat-saturated T2-weighted images and contrast-enhanced, T1-weighted fat-suppressed images (2). Identification of such marrow signal alterations away from the subchondral bone results in high sensitivity for osteomyelitis; however, other entities can alter the bone marrow signal in similar fashion, including fracture, tumor, severe inflammatory arthritis or neuropathic disease, or recent postoperative changes. The lack of bone marrow edema on STIR images excludes osteomyelitis with a specificity of 98% (2).

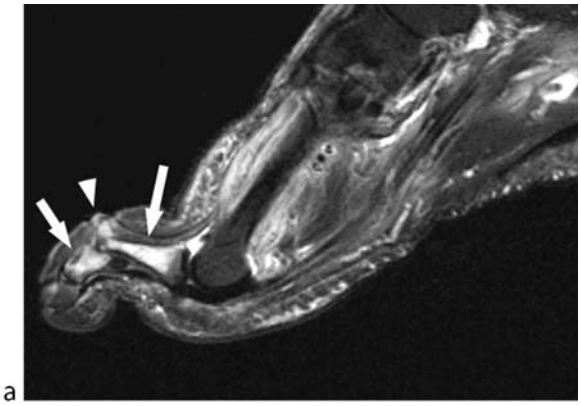
Pedal osteomyelitis, which is by far the most frequent form of osteomyelitis in adult diabetic patients, results in over 95% of patients from contiguous spread of a soft tissue infection; the majority of these patients have some combination of adjacent skin ulceration, cellulitis, soft tissue abscess, and sinus tract. These signs are also called "secondary signs" of osteomyelitis, and can improve specificity. Ischemic tissue in pedal infection is best visualized on fat-suppressed, contrast-enhanced images (3). Osteomyelitis and abscesses may not enhance in necrotic tissue (3).

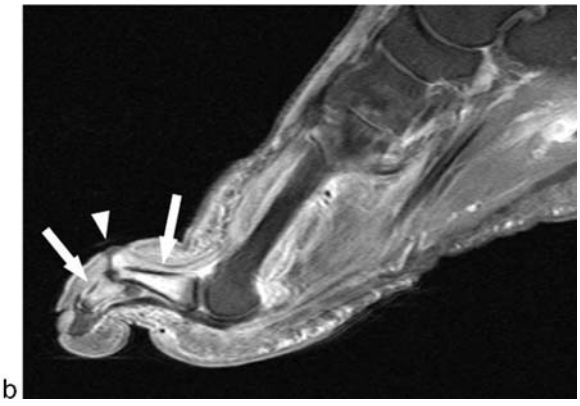
In complicated posttraumatic cases, postoperative bone marrow signal alterations can persist as long as 1 year (4). MR protocols in postoperative osteomyelitis should include fast spin-echo (FSE) sequences with high bandwidth to decrease susceptibility artifacts from the





Osteomyelitis. Figure 2 Illustration of CT findings in chronic active posttraumatic osteomyelitis. (a) Axial scan reveals a cortical defect (white arrow) in the severely remodeled cortex of the distal femur and gas inclusions (black arrow) in the marrow space. (b) Coronal reformat reveals extensive bony remodeling and a large cortical defect (white arrow) filled with enhancing tissue extending to the bone marrow. Intramedullary gas inclusions (black arrow) in the marrow are also indicative of active infection.





Osteomyelitis. Figure 3 Typical MR signal alterations of osteomyelitis in a diabetic patient with an ulcer dorsal to the proximal interphalangeal joint of the second toe. (a) Fat-suppressed T2-weighted image reveals hyperintense signal in the bone marrow of the proximal and middle phalanx (arrows) and interruption of the dorsal skin surface with hyperintense sinus tract (arrowhead). Note the destruction of the proximal interphalangeal joint with subluxation indicating septic arthritis. (b) Contrast-enhanced, T1-weighted fat-suppressed image with diffuse hyperintense signal in the bone marrow of the proximal and middle phalanges confirming osteomyelitis. Note the delineation of the small dorsal ulcer (arrowhead) and the linear hypointense sinus tract extending to the destroyed joint.

metal implants. Gradient echo images and frequency selective fat suppression lead to extensive metal artifacts. T1-weighted subtraction images may be used instead after gadolinium administration.

An *intraosseous abscess* leads to typical findings of a "target" appearance with a center of low signal intensity on T1-weighted images and high signal on T2-weighted images with rim enhancement. Chronic osteomyelitis and sclerosing osteomyelitis are indolent processes with areas of bone necrosis and sclerosis; as a result, MR imaging may show areas of low signal on T1- and T2-weighted images. Differentiation between reactive, noninfective tissue edema with inflammation from true bacterial infection may be difficult using MR imaging in the following situations (1): (a) differentiation of secondary osteomyelitis from reactive bone marrow edema in septic arthritis, (b) differentiation of postoperative and

posttraumatic reparative signal alterations from infection (4), and (c) differentiation of pedal osteomyelitis and diabetic neuroarthropathy (see also Neuropathic Joint Disease).

Ultrasound

Ultrasound imaging can be very useful in pediatric patients due to the lack of radiation. Sonographic signs of osteomyelitis include asymmetric juxtacortical soft tissue swelling, thickening of the periosteum, and demonstration of an adjacent hypoechoic collection. In advanced cases, frank cortical destruction can be seen.

Nuclear Medicine

Technetium-99m MDP three-phase bone scan has a high sensitivity for osteomyelitis and is excellent for excluding bone infection in case of a normal radiograph, but specificity is low. Increased accumulation of tracer can be seen in other conditions such as bone tumors, neuropathic osteoarthropathy, fractures, and following trauma and surgery. One advantage of Tc99m MDP studies over radiography is that it can detect osteomyelitis within 24 to 48 h of onset.

Labeled white blood cell scans or Tc99m labeled monoclonal antigranulocyte antibodies raise the specificity to detect osteomyelitis, and may be helpful in excluding infection in a Charcot joint.

Diagnosis

Timely diagnosis of acute osteomyelitis is best achieved by either MR imaging or scintigraphy if radiographs are normal. Sonography may be very useful in acute pediatric osteomyelitis or septic arthritis. Activity of chronic posttraumatic or postoperative osteomyelitis is often best evaluated by comparison of recent and older radiographs. In complex cases with chronic posttraumatic osteomyelitis, a multimodality approach involving specialists from different disciplines (infectious disease, radiology, orthopedic surgery) is usually required.

▶Oral Cavity, Inflammatory Diseases
▶Neoplasms, Odontogenic

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Osteomyelitis, Neonates, Childhood

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Synonyms

Bone infections; Bone marrow infections; Joint infections; Soft tissue infections

Definition

Infections of individual bony parts, joints, and other supporting musculoskeletal tissue, including combinations of the three.

The responsible agent may be bacterial, fungal, parasitic, viral, or other organisms. A condition resembling osteomyelitis, but without a known agent, is chronic recurrent multifocal osteomyelitis (▶CRMO), with various skeletal manifestations behaving symptomatically and radiographically similar to infection. An open fracture is associated with a break in the skin or at the nail bed. A closed fracture has no such direct communication with the outside environment.

Pathology/Histopathology

According to the causative organism, the histopathology of bone (and joint and soft tissue) infection varies. Additionally, the histopathology of subacute and chronic infection is different from the acute type. Moreover, failures in body defense mechanisms vary between these underlying conditions – for example, in ▶chronic granulomatous disease of childhood, white cells can ingest bacteria and other organisms, but not digest them when the organisms do not contain hydrogen peroxide (for this reason, Ed Neuhauser called the condition "dyspeptic granulomatosis"). The physis is a relative barrier to

passage of infection in bones, except in infancy or when affected by trauma. The bone bark of the metaphyseal collar is a relative barrier to pus in bone, so that breaking through cortex to form periosteal reaction generally stops at the step-off between periosteum and bone bark (in luetic bone disease, this feature is known as the Wimberger sign). In those joints that surround metaphyseal bone, infection travels readily between bone and joint. Neonates of mothers infected with syphilis who show "leukemic lines" at birth do not, at the time of neonatal x-ray imaging, have skeletal infection yet, but rather show the generalized effect of intrauterine stress (of the same nature of such lucent bands when the mother is given magnesium sulfate during late pregnancy (2)). Later in childhood syphilitic bone disease may or may not include local spirochetes. Biopsy of lesions in chronic recurrent multifocal osteomyelitis shows many plasma cells, but no microorganisms.

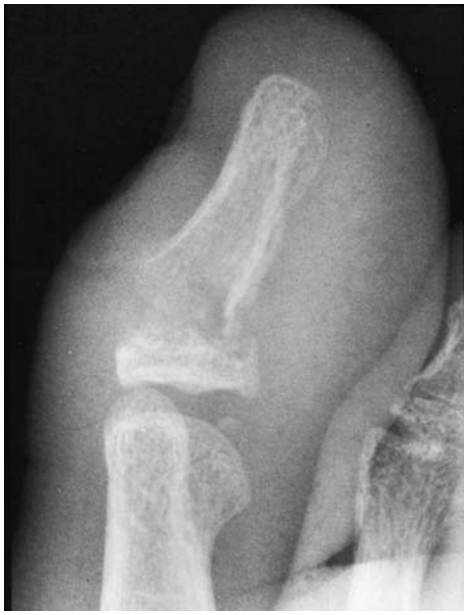
Clinical Presentation

Cardinal signs and symptoms of osteomyelitis include fever, malaise, pain, tenderness, swelling, warmth, redness, and loss of motion. The same signs occur with septic arthritis, and most are found with pyomyositis and other soft tissue infections, so that clinically the differentiation of the sites of infection may be difficult indeed. In patients with immune deficiency and other disorders of defense mechanisms, symptoms may be more silent.

Any ▶stubbed toe or stubbed finger fracture (dorsal Salter II fracture of a distal phalanx) must be suspected of being infected (3) (Fig. 1). Bones adjacent to tissue sites of tuberculosis or actinomycosis are vulnerable to becoming infected, as those infections tend to cross tissue planes. In osteopetrosis, one should be alert for jaw osteomyelitis.

Imaging

Since bone local demineralization and periosteal reaction do not appear on plain radiographs for 10 days after an infection begins, it is essential that the radiologist not dismiss a "normal" study before 10 days as ruling out infection (Fig. 2). It is also wrong to give the advice of solely a return for repeat imaging before the 10 days have elapsed or even after that time – the time to diagnose and treat osteomyelitis is right away (not only after plain film abnormality is evident). Nuclear imaging, ultrasound, and magnetic resonance imaging (MRI) are particularly good for early detection of infection. With ultrasound, elevation of the periosteum may be detected within a day, far earlier than the 10 days required for plain imaging.

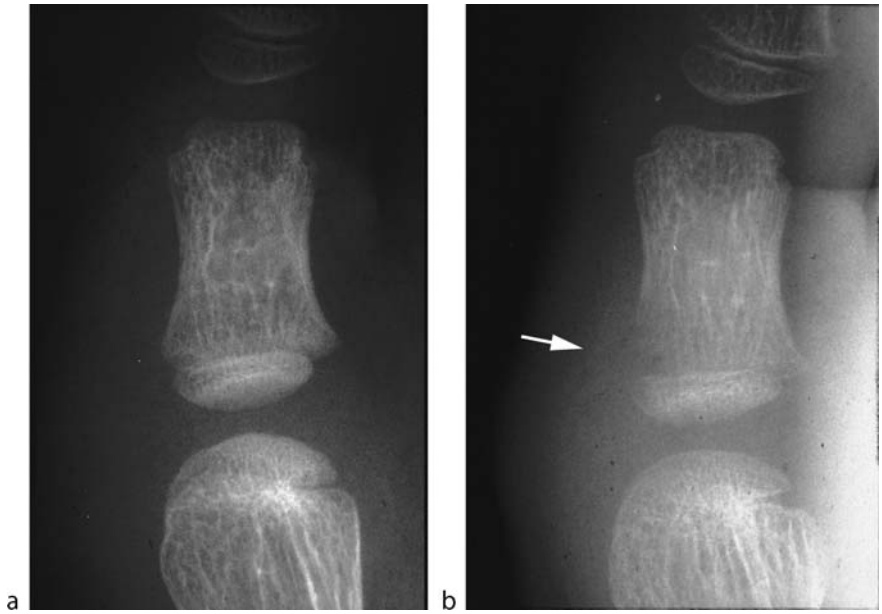


Osteomyelitis, Neonates, Childhood. Figure 1
Osteomyelitis of the distal phalanx of the great toe in a boy several weeks after a stubbed toe Salter II fracture. Bone destruction and periosteal reaction have appeared. From Oestreich AE, Crawford AH (1985) Atlas of Pediatric Orthopedic Radiology, Thieme Verlag, Stuttgart p. 70.

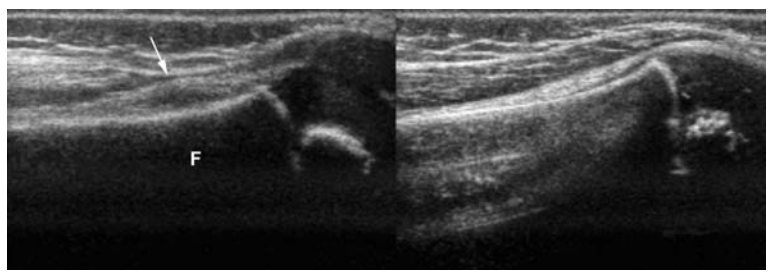
The echogenic periosteum is separated by a hypoechoic zone from the echogenic cortex (Fig. 3). MRI can reveal edema in marrow and surrounding soft tissue, as well as show the organizing fluid elevating periosteum. A needle stick under fluoroscopic, ultrasound, or computed tomography (CT) guidance can also give a diagnosis quickly. CT can distinguish soft tissue infection from its normal surroundings, as can ultrasound and MRI. Dental films give high detail of the periapical region if infection beyond teeth is suspected. Fluid surrounding a tendon is well depicted by ultrasound in tendonitis.

Nuclear Medicine

Acute osteomyelitis that has caused necrosis or avascularity will be cold (less activity than normal bone) on bone scan, unlike the majority of bone infection – acute, subacute, or chronic – which will be hot. A bone scan alone may not distinguish infarction from new sterile infarction in sickle cell anemia, but gallium scanning gives relatively much stronger uptake in infection than infarction. Infection limited to joint or soft tissue should not increase activity in adjoining bone on precise nuclear images. Since it takes 10 days for bone demineralization or periosteal reaction to become visible on radiographs,



Osteomyelitis, Neonates, Childhood. Figure 2 (a) Less than 10 days from onset of symptoms, a plain image of the great toe shows some soft tissue swelling, but the bone is normal in appearance. (b) Ten days later, destruction of the proximal medial metaphysis and metadiaphysis is evident (arrow). It is a medical error to wait until the radiograph is abnormal; other imaging or some treatment should have been initiated at the time of presentation. From Oestreich AE, Crawford AH (1985) Atlas of Pediatric Orthopedic Radiology, Thieme Verlag, Stuttgart p. 247.

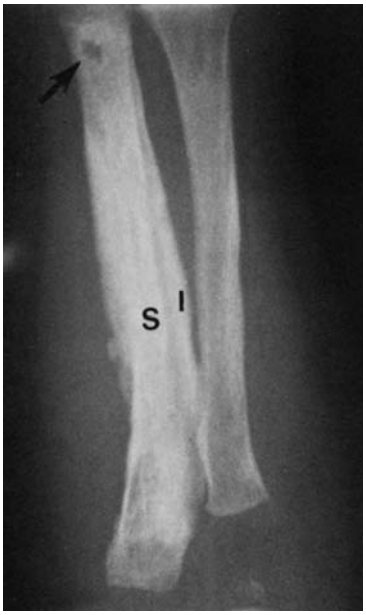


Osteomyelitis, Neonates, Childhood. Figure 3 A young child with acute osteomyelitis of the proximal femur (F), with no plain image bone abnormality yet. The arrow points to periosteum elevated by the sonolucent pocket of pus external to the femoral shaft (F) on longitudinal ultrasound. The right-sided image is the normal contralateral side.

the radiologist should strongly consider bone scanning for diagnosis if symptoms have lasted less than that time. Gallium 67 citrate is generally positive in osteomyelitis by 24–48 h. Labeled white cells with indium 111 or Tc99m HMPAO are other means for seeking infection by nuclear scanning. Active chronic recurrent multifocal osteomyelitis is expected to be positive on bone scan during active phases. In Langerhans cell histiocytosis (one theory considers the disease formerly known as histiocytosis X to be of infectious origin), increased bone scan activity with a photopenic center may be seen. For spondylodiskitis in a child, gallium scan shows high activity; MRI is an alternative useful modality.

Diagnosis

Perhaps the most important point to restate about radiographs of osteomyelitis is that bone demineralization and periosteal reaction are not visible until 10 days after the infection begins; so the diagnosis should not wait for that. Something else should be done (nuclear imaging, needle biopsy, ultrasound, CT, MRI, or heuristic treatment possibly guided by blood culture). Tarsal and carpal bones and the patella and other growth centers have no periosteum, so they will not have periosteal reaction. Infection around the roots of teeth leads to loss locally of the lamina dura and bone demineralization. Infection of



Osteomyelitis, Neonates, Childhood. Figure 4 Four weeks after only soft tissue swelling and a positive elbow fat pad, this 6-month-old infant has full-blown osteomyelitis seldom seen today, with an involucrum (I) surrounding sequestrum (S) of the ulna and a distal cloaca evident (arrow). From Oestreich AE, Crawford AH (1985) Atlas of Pediatric Orthopedic Radiology, Thieme Verlag, Stuttgart p. 66.

the long bone shaft usually does not disturb the 1–3 mm metaphyseal collar, but bone demineralization and periosteal reaction begin beyond it. Untreated or unsuccessfully treated tubular bone osteomyelitis may eventually lead to involucrum (from periosteum) surrounding sequestrum (the damaged avascular bone) and discharge of pus through a radiographically evident cloaca (Fig. 4). Subacute osteomyelitis (Brodie abscess) tends to be seen as ovoid bone demineralization, longitudinally directed, near a physis or perhaps crossing it.

Infection, and other fluid, in the hip or humerus is generally not perceived on plain images (until eventual local bone infection or regional demineralization occurs). Infection in the knee and ankle is easily seen (but not specific) from interfaces, the suprapatellar bursa and the ankle tear drop just adjacent to the talus, respectively.

Soft tissue infections on plain images are seen as soft tissue swelling and disturbance of the local interfaces between tissues, including subcutaneous fat. Exceptionally, gas density is seen from gas-forming organisms.

Interventional Radiological Treatment

Interventional pediatric radiologists may be asked to drain deep abscesses, generally done under ultrasound, but sometimes CT, guidance. If fluid is obtained, it should be investigated for organisms, including tuberculosis, regardless of the gross appearance.

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Osteonecrosis, Adults

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Synonyms

Aseptic bone necrosis; Avascular necrosis

Definition

Negative bacteriologic studies from well-documented cases of bone necrosis led to the use of the term "aseptic necrosis." Subsequent observations indicated that such cases were not only aseptic but also avascular. Hence, the terms "ischemic necrosis," "►avascular necrosis," and "►bone infarction" were suggested (1). By convention, the term "bone infarction" is reserved for bone necrosis in the metaphyseal and diaphyseal regions, whereas "avascular necrosis" (ischemic necrosis) applies to the epiphyseal and subarticular regions.

Pathology/Histopathology

Knowledge of the anatomy and histology of the articular and subarticular region are important for understanding the pathophysiology of osteonecrosis.

In the hyaline cartilage, four different zones can be differentiated microscopically because of different orientation of the fibers. Below the three superficial zones lies the very thin zone of calcified cartilage, divided from the uncalcified zone by the so-called tide mark that represents

the zone of new cartilage formation. The chondrocytes produce the fibers and the ground substance, which is composed of collagens (5–10%), proteoglycans (10–30%; important for osmotic pressure), and water (65–80%).

At its base, adult articular hyaline cartilage is bordered by the subchondral plate (cortical endplate) with a high number of arterial vessels, capillaries, sinusoids, and venous vessels, with a declining number by 20% from adolescence until the seventh decade. While the upper (outer) zones of cartilage get their nourishment from the synovial fluid, there is some evidence that the very important zone of calcified cartilage and, definitely, the subchondral regions are fed by the subchondral vessels. The medullary bone has a dual blood supply, from the medullary arteries and periosteal arteries (2). Bony structures with a relatively high amount of cartilage surface (head of femur, talus) are very prone to problems in vascularization.

Ischemic necrosis of bone and bone infarction occur in areas of predominantly fatty marrow, which has a much lower blood flow. Infarcts (ischemic necrosis) occurring within the epiphysis or in small round bones (such as the talus) are covered by compact subchondral bone and cartilage.

Mineralized bone does not appear to be directly materially altered by ischemic necrosis. Bone density is unchanged within the dead bone but is usually lowered in the surrounding bone due to reactive changes (osteopenia).

Clinical Presentation

The clinical symptoms depend on the anatomic location of the osteonecrosis. A small proportion of patients may even present without symptoms, but usually in ►Association Recherche Circulation Osseous (ARCO) stage 2 avascular necrosis is associated with considerable pain. If the femoral head is affected, patients suffer from slight pain in the hip joint, groin, or buttock or may experience limping and even be unable to walk at all. The symptoms are unspecific, so many other conditions, including arthrosis, insufficiency fractures, and sciatica, have to be considered in the differential diagnosis. In patients with Ahlback's disease the clinical onset is relatively abrupt, with pain that worsens during the night and accompanying effusion and tenderness. The clinical presentation may mimic meniscal disease or arthrosis and can only be diagnosed with ►magnetic resonance imaging (MRI). Avascular necrosis of the lunate bone leads to localized pain and swelling of the wrist. Again, without proper imaging modalities, the diagnosis can be delayed. Early diagnosis is of utmost importance for proper treatment of the disease because failure to promptly initiate appropriate therapy can lead to bone and joint destruction with subsequent need for total joint replacement.

Imaging

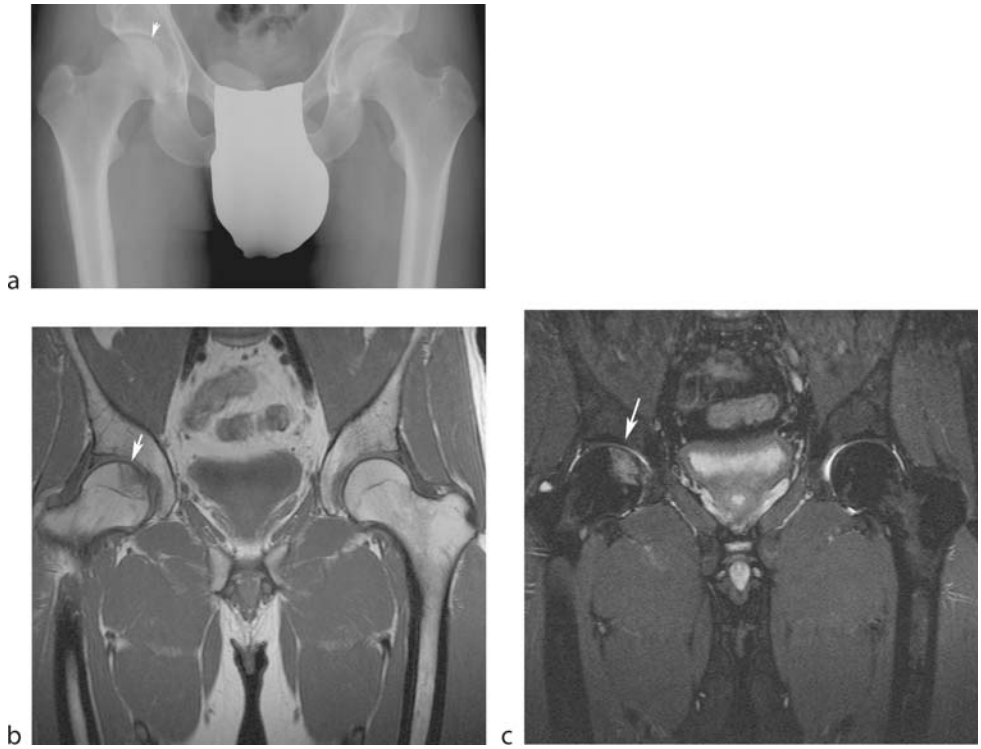
Standard radiographs, computed tomography (CT), and MRI are used in the diagnosis of osteonecrosis/bone infarction.

The early diagnosis must be based on the visualization of changes in the soft tissue/bone marrow, which are altered on MRI. The role and relevance of high-resolution CT in detecting subchondral (micro)insufficiency fractures in early diagnosis of epiphyseal osteonecrosis is still under discussion. The only existing imaging modality to visualize necrotic marrow tissue is MRI. The combination of the inner linear, high-intensity rim with the parallel-running outer, low-intensity rim on T2-weighted or contrast-enhanced MR images is typically called the "double-line" sign and is characteristic for epiphyseal ischemic osteonecrosis with repair (3) (Fig. 1a–c). In this stage (ARCO 2), an arc-shaped subchondral lucent lesion with a thin sclerotic rim can be seen on standard radiographs. These findings are even better demonstrated with CT. CT also shows the abnormal configuration of the trabeculae resulting in irregular localized tiny defects with neighboring sclerosing areas. This ARCO stage 2 may last for weeks and months, but subchondral bony fractures ("crescent sign") representing ARCO stage 3 may finally develop. They are best seen on standard radiographs and/or CT (4). Finally, a complete impression fracture will occur. In ARCO stage 4, all typical signs of degenerative osteoarthritis are summarized, which includes joint deformity with flattening of the most involved part, subchondral "cystic" and sclerotic changes, formation of osteophytes, and joint space narrowing. Application of intravenous MR contrast medium is advocated by some authors, which could be helpful in unclear cases to exactly differentiate the necrotic part with no contrast enhancement from the surrounding viable tissue (5). As the most sensitive MR sequence, STIR images should be used first. If any abnormality is seen, T1-weighted fat-suppressed and T2-weighted gradient-echo (or fast spin-echo) should be added.

In (medullary) bone infarction the early phase with central necrosis and surrounding reactive and usually irregularly shaped tissue, which shows significant contrast enhancement, is followed by increasing calcification. Finally, either the medullary calcification can be found as the only remaining sign, or there will be complete healing.

Nuclear Medicine

Cessation of blood flow can be visualized by a three-phase bone scan, in which the first, vascular phase shows a low uptake of the radioactive marker, which is surrounded by a circular area of elevated uptake in later phases ("cold in hot



Osteonecrosis, Adults. Figure 1 (a) Anteroposterior view of both hips in a 30-year-old man. Ill-defined translucency represents ARCO stage 2 of right hip necrosis. (b) Coronal magnetic resonance imaging (STIR) shows marked edema of the right femoral head. (c) Coronal magnetic resonance image (T1-SE) shows hypointense demarcation in the right femoral head; ARCO stage 2.

spot"). Nuclear medicine studies, however, have been largely replaced by MRI because of better specificity and availability and the lack of ionizing radiation with MRI (6).

Diagnosis

The most preferred classification system for the staging of epiphyseal osteonecrosis was published by ARCO, in which emphasis was placed on a four-part staging system comparing radiographs, CT, bone scintigraphy, and MRI with histology and including prognostic factors (Table 1).

Osteonecrosis can be differentiated in necrosis of the epiphysis (usually called osteonecrosis) and in necrosis of the metadiaphysis (usually called infarction). Spontaneous medullary bone infarcts are often incidental findings in the metaphyses of long tubular bones. They appear as peripheral rims or shells of calcifications in the humerus, femur, tibia, or fibula and must be distinguished from enchondromas.

In the majority of cases, the exact etiology of osteonecrosis is unknown and hence is called idiopathic osteonecrosis. Most commonly, the femoral head or femoral condyles are involved in idiopathic epiphyseal necrosis (Ahlback's disease, or SONK). The differential diagnosis of idiopathic (epiphyseal) osteonecrosis includes bone marrow edema syndrome (BMES), osteomyelitis, bone tumor, and malignancies such as leukemia. BMES is a self-limiting disease with a relatively large zone of bone marrow edema and accompanying joint effusion and demineralization ("transient osteoporosis") but without double-line sign.

Secondary osteonecrosis may be due to trauma, corticosteroid therapy or hypercortisolism (such as after transplantation), hemoglobinopathies, Caisson disease (dysbaric conditions), small vessel disease (such as collagen disease), alcoholism and pancreatitis, gout and hyperuricemia with reactive vessel obstructions, Gaucher disease, irradiation/chemotherapy with direct vessel damage, and high intra-articular pressure (such as in infection and hemophilia).

The prognosis of osteonecrosis depends on the size and location of the ischemia. Osteonecrosis that occurs in a weight-bearing position and involves more than 30% of the articular head (such as the femoral head) has a much worse prognosis than lesions in a nonweight-bearing position and involving less than 15% of the articular head.

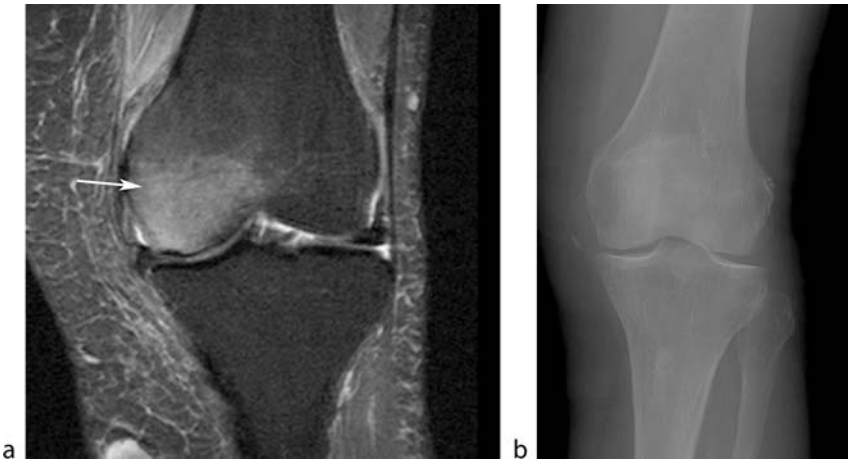
Osteonecrosis, Adults. Table 1 ARCO Classification (1992)

ARCO stage	Histology	Radiographs	Computed tomography	Bone scintigraphy	Magnetic resonance imaging
0	Microosteonecrosis	0	0	0	0
1	• Osteopenia, fractures • Microosteonecrosis • Marrow edema • Hyperemia	0	• Osteopenia • Insufficiency-micro-fractures	• Centrally in lesion low uptake	• Localized edema
2	• Osteonecrosis • Reactive changes: hyperemia, osteopenia, new bone formation	• Osteopenia • Reactive • sclerosis • Cysts	• Osteopenia • Sclerosis • Cysts • Asterisk sign	• High uptake	• Double-line sign (reactive margin of infarct with hyperemia and new bone formation) • Single-line sign in insufficiency fractures
3	• Subchondral impression fracture in weight-bearing area	• Crescent sign	• Crescent sign	• High uptake	• Crescent sign • Cartilage fracture (loss)
4	• Reactive degenerative changes	• Flattened articular surface • Joint space narrowing • Subchondral cyst formation • Reactive osteophytes • Subchondral sclerosis • Deformity, malpositioning, atrophy		• High uptake, deformity	• Cartilage defects • Deformity • Effusion, • Subchondral sclerosis • Osteophytes

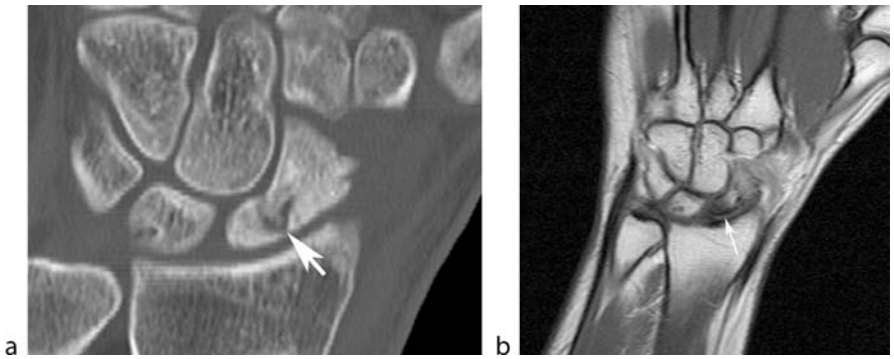
The most common sites for posttraumatic osteonecrosis are the femoral head, the body of the talus, the humeral head, and the carpal scaphoid (Preiser's disease). Rarely, the tarsal navicular, capitate, carpal hamate, or lunate bone is involved. The development of the osteonecrosis may take 6–36 months. The likelihood of development of osteonecrosis gets higher with the severity of the trauma. The principal blood supply of the affected bones is responsible for the risk and location of osteonecrosis. Early diagnosis is made by MRI, and sequential follow-up is with CT and standard radiographs. MRI shows persistent edema with localized zones of necrosis. The osseous flattening, collapse, and fragmentation are usually delayed for a period of 9 months and also depend on the weight-bearing load of the epiphysis. Finally, the involved bony parts may become very dense, smaller, and irregularly shaped (Fig. 3).

Adult idiopathic osteonecrosis may be due to chronic overloading of the hip joint (head of femur) and the femoral condyle (Ahlback's disease). Early diagnosis is possible with MRI and three-phase bone scan. Primary necrosis of the femoral head probably affects men more frequently than women and is usually seen between the fourth and seventh decades of life. The reported prevalence of bilateral disease varies between 35 and 72%. The typical radiographic signs are round, oval, or triangular low densities that are neighbored by a high-density rim, followed by the crescent sign and impression fracture (ARCO stages 1–3). Early diagnosis depends on proof of bone marrow edema (with MRI) or diminished blood supply (with three-phase bone scintigraphy).

Femoral condyle osteonecrosis (Ahlback's disease, spontaneous osteonecrosis) is a typical condylar idiopathic osteonecrosis localized in the medial condyle of the femur and occurs in patients older than 50 years and in women more than in men, but it may be also found in the lateral condyles and the tibial plateau. On standard radiographs, the first sign is a subtle flattening of the neighboring joint surface, followed by a narrow zone of increased density adjacent to the depressed osseous surface. A radiolucent area in the condyle can be detected over the ensuing weeks, becoming more sharply demarcated by time. If untreated, further depression of the bony margins and progressive sclerosis and intra-articular osseous bodies will follow. Over a period of months or years, all signs of secondary degenerative osteoarthritis will develop. Bone collapse, varus deformity, and displacement can also be noted (Fig. 2).



Osteonecrosis, Adults. Figure 2 (a) Coronal magnetic resonance image (STIR) shows marked edema of the medial condyle in a 90-year-old woman (Ahlback's disease). (b) Anteroposterior view shows no evidence of osteonecrosis in this digital radiograph.



Osteonecrosis, Adults. Figure 3 (a) Coronal MPR of multidetector computed tomography reveals nonunion of the scaphoid in a 35-year-old woman after trauma. (b) Coronal magnetic resonance image (T1-SE after intravenous gadolinium) demonstrates no contrast enhancement in the proximal fragment (Preiser's disease) of the scaphoid bone.

Kienbock's disease occurs most commonly between the ages of 20 and 40 years and has a predilection for the right lunate bone. The male-to-female ratio is 2:1, with the following stages:

In stage I the radiograph is normal or shows a subtle fracture. MRI visualizes a bone marrow edema, and bone scintigraphy a hot spot. In stage II, abnormalities in radiodensity with changes in size and shape are seen. MRI will reveal necrotic hypointense areas in all sequences. In stage III, the lunate bone collapses and shows a high density with irregular borders. In stage IV, signs of degenerative osteoarthritis are demonstrated. The etiology seems to be chronic trauma. In patients with short ulnae (ulnar minus variant), the loading forces on the lunate are much higher than normally.

In dysbaric osteonecrosis (Caisson disease), in cases of too rapid decompression the released nitrogen may produce bubbles that act as gas-emboli. Those may occlude vessels partially or completely. A delay of at least 6 months or years between the exposure and the onset of radiologically evident juxtaarticular or diaphyseal and metaphyseal radiodense foci/radiodense lesions is typical. Early diagnosis is possible with three-phase bone scintigraphy and whole-body MRI.

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Osteonecrosis, Childhood

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Synonyms

Avascular necrosis (Perthes disease = Legg–Calvé–Perthes disease = idiopathic avascular necrosis of the hip in childhood); Bone infarction; Chondrolysis (of cartilage)

Definitions

Arterial or venous ischemia leading to cell death and/or disturbance of growth in bone. Historically, several (eponymous) variations in skeletal growth, now known not to be due to necrosis, were once so considered and are still often discussed in the context of true necrosis states. Death or disturbance of the zone of resting cartilage leads to delay, deformity, or cessation of enchondral growth; other conditions such as frostbite and ►Kashin–Beck disease may locally impair enchondral growth as well. The conditions in infancy known as multiple stippled epiphyses may well be secondary to necrosis of growth cartilage not yet in a growth plate.

Pathology/Histopathology

Clinical bone necrosis is usually more dynamic than mere death of osteocytes. The body has ways of repairing damage, so that the histopathologic pattern may be composed of both destruction and rebuilding. In Perthes disease, for example, the first step is impairment of vascular supply. The first irreversible structural change is a crumbling fracture of bone just below the lateral zone of provisional calcification, seen on frog leg radiographs. Then waves of bone necrosis appear with lack of viable osteocytes, followed by waves of overlying new bone (creeping substitution). Years later, a viable but deformed head is usually formed, more or less congruent with its acetabulum. However, because the shape is less spherical than normal and does not fully fit in its acetabulum, secondary arthrosis tends to appear relatively early in adulthood. Because of avascularity in the femoral neck and primary spongiosa in Perthes' disease, cartilage from the physis persists in the otherwise ossified neck, giving gouges of lucency on X-ray images. Both the head and neck of the femur widen transversely compared to normal as the disease progresses—perhaps related to increased periosteal formation in the neck from the processes of healing. An adverse event in the progress of Perthes is tethering across the physis in some patients. The greater trochanter growth is generally not impaired in Perthes, so it is relatively overgrown, leading to varus deformity of the hip, which is further exaggerated if tethering of the main physis occurs.

Osteochondrosis (some still refer to it as osteochondritis) dissecans is an injury to bone and overlying cartilage, which is painful and more severe, indeed unstable, whenever joint fluid enters the space between the lesion and normal bone.

Schmorl nodes are protrusions of normal vertebral disk substance through weak areas in the vertebral end plates (zones of provisional calcification). Schmorl nodes are often a component of Scheuermann disease of the spine.

In frostbite and Kashin–Beck disease (1), portions or the entire involved physis or acrophysis becomes nonviable, leading to local lack of growth (and perhaps early physeal fusion). A cone epiphysis is a result of a similar loss of viability of the central (more senior) portion of a physis, whether due to injury, infection, or genetic causes.

Clinical Presentation

Pain (in the groin, thigh, or referred to a knee), limping, and favoring a limb may reflect Perthes disease; however, Perthes may be clinically silent for months and discovered serendipitously on radiographs acquired for other reasons. Symptoms for avascular necrosis from other causes and at other sites are similar. In Gaucher disease, the spleen is often quite large; in sickle cell anemia, the spleen is usually small or absent. Traumatic avascular necrosis occurs after hip dislocation. For steroid-induced hip necrosis, other symptoms of steroid use could be sought. The incidence of osteochondrosis dissecans rose when teenagers were dancing The Twist, which indicates that certain challenging repetitive motions might be a predisposing factor. For frostbite, the history of severe cold exposure if available is helpful for the diagnosis (although the pattern of involvement on X-ray images is virtually diagnostic—especially if the thumb is spared by being

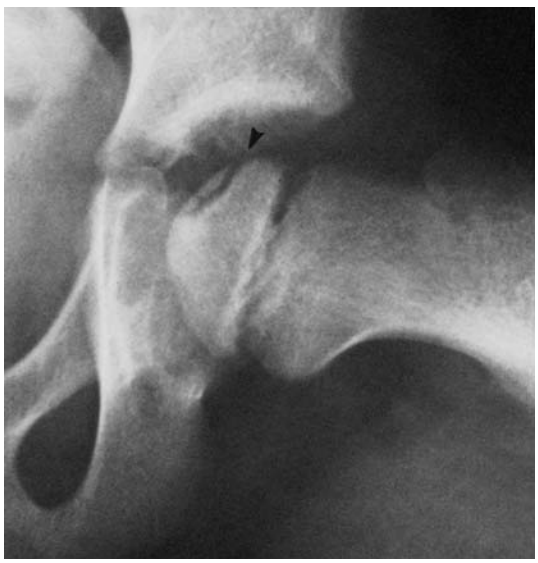
held in the fist during the exposure). Kashin–Beck disease is geographically limited to China, Mongolia, and Tibet, where thousands of cases have occurred.

Imaging

In Perthes disease, magnetic resonance imaging (MRI) and nuclear images show avascularity earlier than the first plain image finding, which, in turn, appears first on the frog-leg view (Fig. 1). However, unilateral retarded maturation of one femoral head might be appreciated on the frontal radiograph as well. Ultrasound shows associated hip effusion easily; plain images do not. As Perthes disease progresses, computer tomography (CT) images show the current integrity of bone, extent of involvement, and nonosseous components; however, MRI is preferred because it does not use radiation and also displays the femoral head and neck and acetabulum throughout the long course of Perthes (2).

►Osteochondrosis dissecans of the distal femur is usually situated posteriorly in the involved condyle, and thus is better seen on a notch (angled) view tangential to it. Evaluation for instability or fluid between the osteochondrosis and adjacent healthy bone could be made with MRI or a CT arthrogram.

When there is doubt about whether a normal variant of ossification or an osteonecrotic bone is present, a display of the osseous and cartilaginous elements on MRI may help one decide, with overlying normal cartilage favoring the normal variant. For example, Meyer dysplasia of the hip (3) can be distinguished from Perthes disease by the finding of normal cartilage and normally vascularized bone. Ultrasound can show the findings in Osgood–Schlatter condition of the patellar tendon and anterior tibial apophysis.



Osteonecrosis, Childhood. Figure 1 Perthes disease lucent crescent (*arrowhead*) of the outer femoral head on frog-leg view in an 8-year-old boy. From Oestreich AE, Crawford AH (1985) Atlas of Pediatric Orthopedic Radiology. Thieme Verlag, Stuttgart p 190.

Nuclear Medicine

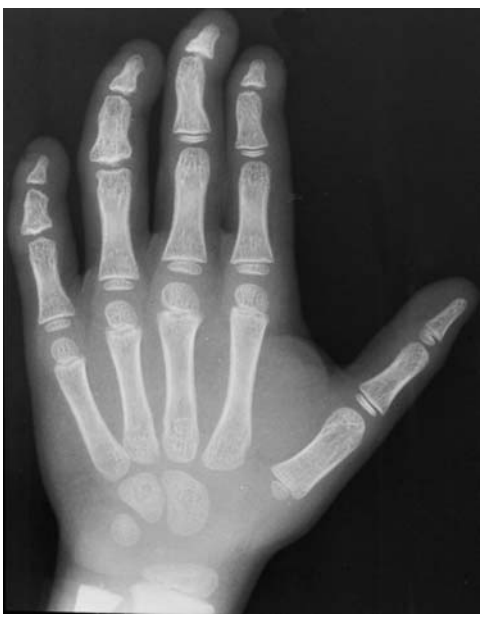
Nuclear imaging, like MRI, shows findings earlier in Perthes disease than the upper outer lucent crescent found on radiographic frog-leg images. On bone scans, the earliest finding is a cold defect in the femoral head, a larger zone indicating more extensive disease. This phase, however, is reversible without progression to irreversible definitive Perthes disease. The perthetic femoral head eventually regains scan activity in the reparative phase later in the disease evolution. One week after the onset of sterile infarction of bone, whether from sickle cell disease, Gaucher disease, pancreatitis, or other noninfectious causes, bone scans begin to show high activity (as healing begins) rather than the earlier lower than normal (cold) activity. Lack of bone scan activity is also a sign of avascularity after certain fractures, for example, in the proximal scaphoid following a transverse fracture of the scaphoid waist. The differential diagnosis between infection and infarction of bone in sickle disease can be assisted by dual scanning: in infection, gallium 67 should be much more avidly taken up than technetium in conventional bone scan.

Diagnosis

Bones with osteonecrosis are generally denser than the nearby bones, whether because of the loss of volume putting more bone substance into a smaller space from collapse or because living bones demineralize from local irritation whereas dead bones do not. A classic example is the proximal scaphoid being denser than the distal one when its blood supply is lost from a fracture across the bone's waist. Infarction of bone shafts can also incite callus, which is denser than the nearby bone.

The early plain image changes of Perthes disease are a smaller epiphysis than contralaterally because of decreased vascularity and, on the frog-leg view alone, a slit of lucency below the outer femoral head zone of provisional calcification, representing crumbling fracture (Fig. 1). The extent of the slit reflects the severity of involvement. As the disease progresses (with or without surgery to improve position or protect the hip), the head becomes irregularly denser and longitudinally shorter,

while the head and neck become transversely wider (and thus incompletely covered by the bony acetabular roof). Gouges of lucency may extend down the metaphysis of the neck from the physis, reflecting avascular zones in the primary spongiosa. Without ultrasound or cross-sectional imaging, one cannot determine whether effusion accompanies Perthes disease. The perthetic hip through the progress of disease becomes varus as the greater trochanter continues to grow nearly normally, while the femoral physis is slowed, or occasionally tethered, by premature fusion across part of the physis.

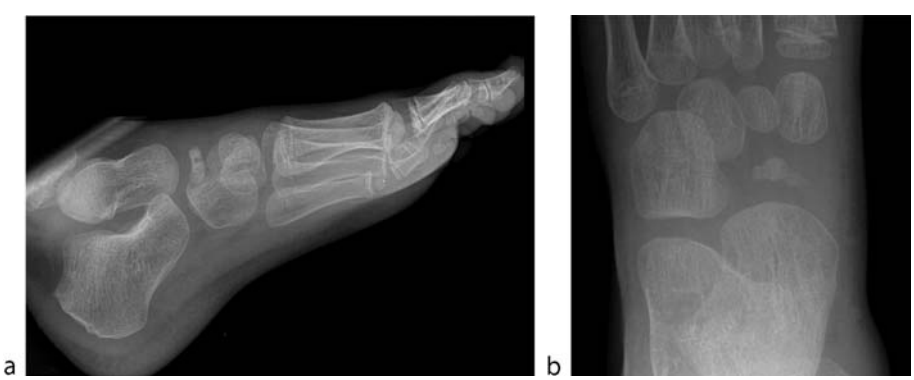


Osteonecrosis, Childhood. Figure 2 Frostbite sequelae: distal phalanges 2 through 5 are short from physeal closure, as are middle phalanges 4 and 5. The distal (acrophyseal) ends of the middle phalanges are also irregular from enchondral damage. As is often the case, the thumb was presumably spared by being protected in the fist during the cold exposure. From Oestreich AE, Crawford AH (1985) Atlas of Pediatric Orthopedic Radiology. Thieme Verlag, Stuttgart p 169.

Sickle cell infarction of small tubular bones, generally about 1 year of age, is painful accompanied by periosteal reaction and called "hand–foot" syndrome. This may be the presenting symptom of previously unknown sickle cell disease. The typical Lincoln log vertebral bodies in sickle cell disease result from infarction or impairment of the more central portions of the vertebral end plates. Distinguishing infarction from infection in long bones in sickle cell disease by imaging can be quite difficult; dual nuclear scanning with bone scan and gallium scan (or labeled white blood cells) may help by showing especially high activity on the latter.

In frostbite, Kashin–Beck disease, and some sequelae of rat bite (1), enchondral bone growth is locally destroyed, so that portions or the entire bones do not grow (Fig. 2). Cone-shaped epiphyses, fused epiphyses, pumice-shaped carpal bones, and irregular nonepiphyseal ends of small tubular bones are manifestations. In frostbite the distribution is acral, from the fingertips proximally, because of the nature of the cold injury, often sparing the thumb if it had been protected in the fist. Distribution of enchondral damage in Kashin–Beck disease is more scattered—involvement asymmetrically of the lower extremity bones leads to length discrepancy and hence limp.

The apophysis of the posterior calcaneus normally appears denser than the rest of the bone. It is no longer considered "Sever disease" unless localized symptoms occur and the nuclear scan is abnormal. Many diagnoses of "Köhler disease" of the tarsal navicular are actually an overlap of multiple ossification centers rather than true osteonecrosis (Fig. 3). Close perusal of oblique and lateral images will usually solve the question. Similarly, the ossification across the closing inferior ischiopubic synchondrosis is often both vigorous and asymmetric from



Osteonecrosis, Childhood. Figure 3 Mimic of Köhler disease in a 6-year-old patient. Lateral image suggests sclerosis and irregularity of the navicular; but the frontal view shows it results merely from overlap, the bone developing from three normal-density growth centers. The pain, incidentally, was lateral, not medial.

side-to-side. Unless abnormally increased bone scan activity can be shown, van Neck osteonecrosis should not be considered. Kienböck disease or lunatomalacia is a real entity, however, often associated with ulna minus.

Meyer dysplasia of the hip in the early years of life is an irregular appearing ossification of the femoral head. However, it has normal vascularity on nuclear scan or MRI and uncommonly progresses to true Perthes disease.

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Osteopenia

In childhood, a reduction of bone substance either from osteoporosis or hyperparathyroidism or, after physes fuse, also from osteomalacia.

▶Demineralization, Bone, Childhood

▶Osteoporosis

Osteophytes

Bony spurs or outgrowths in the proximity of a joint, osteophytes, are found in a variety of musculoskeletal disorders, including degenerative joint disease and DISH. Presence of multiple osteophytes is referred to as osteophytosis.

▶Dish

Osteoporosis

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Synonyms

Osteoporosis and osteopenia are precisely defined according to the World Health Organization (WHO) Criteria (see later)

Definitions

Osteoporosis is defined as a disease associated with a loss of bone mass and a deterioration of bone structure, both resulting in increased bone fragility and susceptibility to fracture (1). In 2000, the definition given by the National Institutes of Health consensus development conference in 1993 was modified (2). Osteoporosis was now defined as a skeletal disorder characterized by compromised bone strength predisposing to an increased risk of fracture. Because this definition appears fairly abstract, the following statements were added.

Bone strength reflects the integration of two main features: bone density and bone quality. Bone density is expressed as grams of mineral per area or volume and in any given individual is determined by peak bone mass and amount of bone loss. Bone quality refers to architecture, turnover, damage accumulation (e.g., microfractures), and mineralization.

Because bone density is the parameter that can be determined best *in vivo*, has a high precision, and correlates well with the biomechanically determined bone strength [it explains approximately 70% of bone strength (2)], the WHO defined osteoporosis on the basis of bone mineral density (BMD) (3). A BMD that is more than 2.5 standard deviations below that of a white, young, healthy female adult reference population (T-score) is defined as osteoporosis. A BMD that is 1–2.5 standard deviations below that of the young and healthy reference population is defined as ▶osteopenia. This definition, however, was originally only established for BMD of the proximal femur determined using ▶dual-energy X-ray absorptiometry (DXA), but it has been applied to define diagnostic thresholds at other skeletal sites, such as the spine (anterior–posterior) and the distal radius, and for other technologies.

Pathology/Histopathology

Osteoporosis is characterized by reduced activity of osteoblasts and increased activity of osteoclasts. Because trabecular bone has a turnover up to seven times higher than the cortical shell, bone loss occurs here first. The trabecular network transforms from a platelike to a rodlike structure with thinner trabeculae and wider intertrabecular spaces. Connections between trabeculae

are lost as osteoclasts form resorption lacunae that may have the size of thinner trabeculae. To quantify trabecular structure changes, histomorphologic parameters can be calculated from biopsies of the iliac crest, or noninvasively using high-resolution magnetic resonance imaging (MRI) or peripheral MicroCT at the distal radius, the tibia, or the calcaneus.

Clinical Presentation

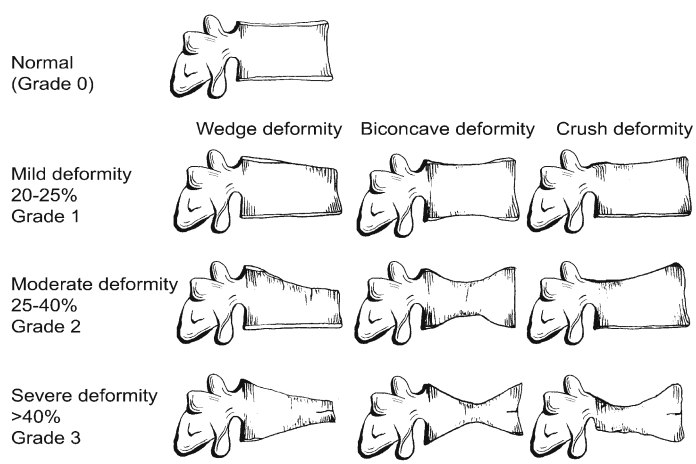
Osteoporosis may be undiagnosed for a long time and frequently manifests itself with insufficiency fractures. Early symptoms such as back pain and height loss are nonspecific, and a hunchback represents already advanced osteoporosis that is frequently associated with several vertebral fractures. The most severe insufficiency fractures affect the proximal femur with high associated disability and mortality. Osteoporosis-related vertebral fractures also have important health consequences for older women and men, including disability and increased mortality. The presence of one vertebral fracture increases the risk of any subsequent vertebral fracture fivefold, and 20% of women who have had a recent diagnosis of fracture will sustain a new fracture within the next 12 months. Since a number vertebral fractures do not come to clinical attention, the radiographic diagnosis is particularly important. Because these fractures can be prevented with appropriate medications, recognition and treatment of high-risk patients is warranted.

Imaging

Conventional radiographs of the spine are not suited to determine bone mass in the early stage of osteoporosis because it takes a bone loss of more than 20–40% before a loss of bone mass is visualized on radiography. Morphological signs described on spine radiographs, such as a coarse trabecular structure and a framelike appearance of the vertebrae, are also not very reliable. Conventional radiographs, however, are important in diagnosing fractures and in the differential diagnosis of osteoporosis because a number of other diseases may present with bone loss and fractures.

According to Genant et al (4), a vertebral deformity in T4-L4 of more than 20% of loss in height with a reduction in area of more than 10–20% is defined as a fracture. Using this fracture threshold, a semiquantitative score to grade the severity of vertebral fractures as visually determined from radiographs has been described (Fig. 1). In rare cases, osteoporosis may present with a coarse trabecular structure with thick vertical trabeculae suggestive of vertebral hemangioma. This so-called hypertrophic atrophy, however, is generalized, and the trabecular bone structure appears more coarse than in hemangioma. Important differential diagnoses in osteoporosis are osteomalacia, hyperparathyroidism, renal osteopathia, and malignant bone marrow disorders such as multiple myeloma and diffuse metastatic disease. Endplate fractures are found in Scheuermann's disease and malignant lesions. The differential diagnosis of osteoporotic and malignant pathologic fractures may be difficult. Fractures located above the Th 7 level present with a soft tissue mass, osseous destruction, and fractures of the posterior part of the vertebrae in conventional radiographs are more likely to be malignant.

Conventional radiographs of the proximal femur and the distal radius are usually obtained after a low-impact trauma with persistent symptoms in postmenopausal elderly individuals. It should be noted that osteoporotic



Osteoporosis. Figure 1 Spinal fracture index as defined by Genant et al (4) used to classify osteoporotic vertebral fractures.

fractures may be difficult to detect on conventional radiographs due to demineralization of the bone and are not infrequently occult.

Computed tomography (CT) and MRI may be helpful in detecting occult fractures (Fig. 2), differentiating osteoporotic and malignant fractures, depicting multiple lesions, and soft tissue masses or destructive changes. Bogost et al showed that 37% of proximal femur fractures were not detected in conventional radiographs, which were demonstrated in MR scans of these patients (5). Nonenhanced T1 and STIR (respectively T2-weighted fat-saturated) sequences are recommended in patients with a high clinical suspicion of fracture but negative radiographs. Diffusion-weighted MR sequences and iron oxide contrast media in MRI have been successfully used to differentiate malignant and benign bone marrow pathology. CT is less sensitive in depicting bone marrow pathology; however, it is better suited to assess the stability of an osteolytic lesion or fracture because it directly visualizes the bony structures and demonstrates fracture lines in a detailed fashion.

Nuclear Medicine

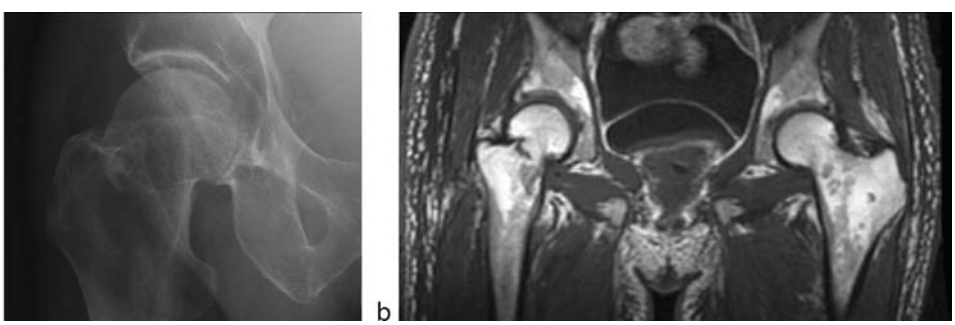
Bone scintigraphy has a limited role in the assessment of osteoporosis. The technique may be useful to detect occult fractures (though it is less sensitive than MRI), to depict multiple lesions, and to differentiate between old and new vertebral fractures.

Diagnosis

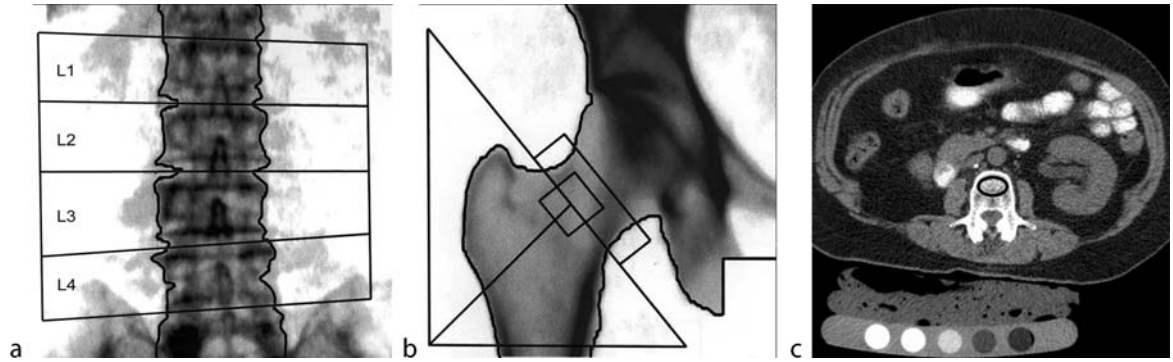
Diagnosis of osteoporosis is usually made on the basis of bone density or the presence of osteoporotic fractures. Currently the most important techniques in osteodensitometry are DXA and ►quantitative computed tomography (QCT).

DXA: The principle of DXA is a dual-energy measurement that is based on the fact that radiation of distinct energies is attenuated by tissues to different extents. In both soft tissue and bone, a low-energy beam is attenuated to a greater degree than a high-energy beam is. Contrast in attenuation between bone and soft tissue is greater for the low-energy beam than for the high-energy beam, such that the attenuation profile of bone may be determined by subtracting both the low- and high-energy attenuation profiles. DXA scanners provide either pencil or fan beam techniques. Fan beam techniques are faster. The precision of DXA is high, and radiation exposure is low. BMD is most frequently determined at the spine (anteroposterior or lateral) (Fig. 3a) and at the proximal femur (Fig. 3b). Whole-body measurements as well as measurements at the distal radius and the calcaneus may also be obtained. The anteroposterior examination of the lumbar spine is a standard procedure with a precision *in vivo* of 1%, a radiation exposure of 1(-50) μSv (the higher dose is required for digital high-resolution images), and a fairly high accuracy (4–10%). For monitoring BMD, the precision alone, however, is not the only parameter required to assess the diagnostic performance of a technique. We also need to know the annual rate of BMD loss in normal patients using this technique as well as the least significant change between two measurements, which are 1–2% and 3–4%, respectively, for the anteroposterior spine.

Using automated software, areal BMD (g/cm^2) is determined, usually at L1–L4 (Fig. 3a). These projection images, however, have a number of limitations: (i) vertebrae with a larger size have a higher BMD, (ii) aortic calcification and all other soft tissue calcifications in the regions of interest (ROIs) increase BMD, and (iii) degenerative changes of the spine including osteophytes, facet sclerosis, and degenerative disc disease may also falsely increase BMD. In elderly patients with substantial degenerative changes of the lumbar spine, anteroposterior DXA of the lumbar spine may therefore not be a suitable technique. Lateral



Osteoporosis. Figure 2 Radiographically occult insufficiency fracture of the right proximal femur in a postmenopausal woman. On the radiograph of the right femur (a), no fracture is shown, whereas the coronal T1-weighted magnetic resonance image (b) clearly shows a fracture of the right femur neck.



Osteoporosis. Figure 3 Standard techniques to measure bone mineral density: dual-energy X-ray absorptiometry images of the anteroposterior spine (a) and the proximal femur with standard regions of interest (b) as well as quantitative computed tomography of the lumbar spine (c) with an oval region of interest in the vertebral body.

DXA is influenced less by these changes because it assesses only the vertebral bodies and thus focuses more on trabecular bone. However, drawbacks of this technique are a lower precision, a higher radiation exposure, and superimposition of the pelvis and the ribs, which may limit analysis of the lumbar spine to L3. So far, anteroposterior DXA is still the standard DXA procedure to assess the lumbar spine. When analyzing DXA scans, a number of pitfalls have to be considered that may be operator-dependent, such as mislabeled vertebrae, misplaced disk space markers, wrongly sized ROIs, and artifacts in the analysis region. These analysis errors are of greater magnitude than the machine's intrinsic precision errors.

DXA of the proximal femur is a particularly important examination because it is currently one of the best techniques to assess fracture risk of the hip (Fig. 3b). But the examination of the hip is more demanding than that of the spine; the proximal femur has to be positioned in a standardized fashion, and a number of ROIs have to be placed correctly. The correct location of these ROIs varies according to the manufacturer. Standard ROIs are the neck region, the trochanteric region, and the intertrochanteric region. The ROI used most frequently is the total femur. The total femur ROI consists of the neck region, the trochanteric region, and the intertrochanteric region. Ward's triangle has an inferior precision compared with the other ROIs and is currently not used as a standard ROI. The precision for hip BMD and the annual rate of loss are lower compared with anteroposterior spine, and the least significant change is higher.

As in DXA of the lumbar spine, a number of operator-dependent errors may occur in the proximal femur and should be detected by the radiologist. Most of these errors are due to improper positioning of the patient and the ROIs. Correct positioning of the patient includes internal rotation of the hip with a straight femoral shaft (the lesser trochanter should not or just barely be visualized). Correct positioning and size of the ROIs, in particular the neck box, may vary according to the manufacturer; for example, Lunar/GE systems have a standardized size of the neck box, which is placed automatically in the region of the neck with the smallest diameter. Osteoarthritis, Paget's disease, fracture, vascular calcifications, calcific tendinitis, enostosis, and avascular necrosis of the hip are also potential sources of error. Conventional radiographs may be required if an atypical density profile is shown. If these lesions are too large or if developmental dysplasia of the hip is found, BMD has to be determined at a different site.

QCT: In contrast to DXA, QCT allows a true densitometric measurement (in mg/mL) of trabecular bone, whereas DXA gives an areal BMD (in mg/cm^2) that includes trabecular and cortical bone. Since the trabecular bone has a substantially higher metabolic turnover, it is more sensitive to changes in BMD (annual rate of bone loss in QCT 2–4% vs. 1% in anteroposterior DXA of the spine). On the other hand, the precision of two-dimensional (2D) QCT (but not of volumetric QCT) is lower than that of DXA (1.5–4% vs. 1%). A big advantage of QCT is that it is not as susceptible to degenerative changes of the spine as DXA is. Osteophytes and facet joint degeneration as well as soft tissue calcifications (in particular of aortic calcifications) usually do not falsely elevate the BMD in QCT. As in DXA, however, fractured or deformed vertebrae must not be used for BMD assessment because these vertebrae usually have an increased BMD.

QCT may be performed with any CT system; however, a calibration phantom is required to transform the attenuation measured in Hounsfield units (HU) into BMD (mg/mL). Dedicated software improves the precision of the examination. The patient is examined lying supine on the phantom, usually with a water- or gel-filled cushion in between to avoid artifacts due to air gaps. Standard 2D QCT is performed of the lumbar spine; usually, the first to third lumbar vertebrae are analyzed

using a single midvertebral section that is aligned along the endplates. Volumetric QCT can be performed of the spine and the proximal femur using axial, contiguous 3-mm sections of each site.

BMD data obtained by QCT are compared to an age-, sex-, and race-matched database. T-scores used for the assessment of osteoporosis according to the WHO definition have been established for DXA but not for QCT, though they may be given by the manufacturers of the software. If these T-scores are used to diagnose osteoporosis, a substantially higher number of individuals compared to DXA will be diagnosed as osteoporotic, since BMD measured with QCT shows a faster decrease with age than DXA. Researchers have therefore advocated using BMD measurements analogous to the WHO definition but with thresholds corresponding to lower T-scores. Thus, BMD values from 80–120 mg/mL have been classified as osteopenic, and BMD values below 80 mg/mL as osteoporotic (6), which corresponds to a T-score of approximately -3.0.

Peripheral QCT and DXA are less frequently used and clinically of limited significance. New techniques to assess microarchitecture and macroarchitecture of bone have not been introduced into the clinical arena but may give additional information on fracture risk and have future potential.

▶Acromegaly
▶Demineralization, Bone

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Osteoporotic Vertebral Fractures

According to the spinal fracture index, deformities of the vertebrae of more than 20% are defined as fractures. These are not infrequently asymptomatic but are excellent indicators for future fractures. Even if bone mineral density is not below a T-score of -2.5, the presence of fractures is indicative of osteoporosis.

▶Osteoporosis

Osteosarcoma

Most frequent malignant primary bone tumour found predominantly in young adults. Typically aggressive morphology in radiological images with complex periosteal reaction and osteoblastic matrix pattern.

▶Neoplasms, Bone, Malignant

Osteosclerosis

The hardening or the abnormally high density of bone.

▶Neoplasms, Odontogenic

Outlet Obstruction Syndrome

Outlet obstruction syndrome, also called obstructed defecation is defined as incomplete evacuation of fecal contents from the rectum.

▶Pelvic Floor Dysfunction, Anorectal Manifestations

Outlet Obstruction Syndrome – Obstructed Defecation

▶Pelvic Floor Dysfunction, Anorectal Manifestations

Ovarian Cancer

Ovarian cancer is in the majority (85%) of cases epithelial in origin. Familial evidence of ovarian cancer is the strongest risk factor for ovarian cancer. At diagnosis in more than 75% of patients with epithelial ovarian cancer, peritoneal tumor spread outside the pelvis or lymphatic metastases are detected.

▶Carcinoma, Ovarium

Ovarian Cancer Screening

Screening with CA-125 and sonography are currently only suggested in high-risk patients. These are patients with a positive family history of ovarian cancer or BRCA1 or BRCA2 gene mutations.

▶Carcinoma, Ovarium

Ovarian Cancer Staging

The most commonly used staging system of ovarian cancer is the FIGO (International Federation of Gynecologists and Obstetricians) classification system. Alternatively, ovarian cancer is staged on the basis of the TNM classification. Staging is based on the findings detected during explorative laparotomy including cytologic assessment of the peritoneum.

▶Carcinoma, Ovarium

Ovarian Cancer

▶Carcinoma, Ovarium

Ovarian Metastases

Ovarian metastases comprise approximately 5–15% of malignant ovarian tumors, and derive most commonly from colon, stomach, breast, and melanomas as primary cancers. Krukenberg tumors display characteristic imaging features, which include bilateral, solid ovarian tumors, often with central necrosis. Other ovarian metastases present with similar imaging findings as ovarian cancer.

▶Masses, ovarian

Ovarian Neoplasm

▶Masses, Ovarian

Ovarian Teratomas

Ovarian teratomas consist of a series of tumors which derive from primordial germ cells. They comprise mature teratomas, immature teratomas, and monodermal teratomas. Mature teratomas constitute the vast majority of these tumors (99%).

▶Teratoma, Ovaries, Mature, Ovalar

Ovarian Torsion

Ovarian torsion is the most important complication of dermoids. It is reported in 3.2–16% of cases and is a gynecological emergency. Increasing tumor size correlates with increased risk of torsion.

▶Teratoma, Ovaries, Mature, Ovalar

Ovarian Vein Obstruction

▶Thrombosis, Vein, Ovarian

Ovarian Vein Occlusion

▶Thrombosis, Vein, Ovarian

Oxygen–Ozone Therapy

A mixture of oxygen–ozone gas is injected into the centre of the intervertebral disc at non-toxic concentrations. Ozone has a direct lytic effect on the proteoglycan molecules that form the nucleus pulposus. The resulting proteoglycan fragments have limited water-binding abilities, which leave intradiscal water molecules free to diffuse into the surrounding tissues. The resulting loss of water causes a decrease of intradiscal pressure. This causes the herniated portion of the nucleus pulposus to recede towards the centre of the disc, relieving pressure on the nerve root.

▶Percutaneous Interventions for Lumbar Radicular Syndrome