Primary Care for Emergency Physicians

Bobby Desai
Alpa Desai
Editors

Springer

Primary Care for Emergency Physicians

Bobby Desai • Alpa Desai
Editors

Primary Care for Emergency Physicians

Springer

Editors
Bobby Desai
Department of Emergency Medicine
University of Florida College of Medicine
Gainesville, FL, USA

Alpa Desai
Department of Community Health &
Family Medicine
University of Florida College of Medicine
Gainesville, FL, USA

ISBN 978-3-319-44358-4 ISBN 978-3-319-44360-7 (eBook)
DOI 10.1007/978-3-319-44360-7

Library of Congress Control Number: 2016960755

Printed on acid-free paper

This Springer imprint is published by Springer Nature
The registered company is Springer International Publishing AG Switzerland
The registered company address is Gewerbestrasse 11, 6330 Cham, Switzerland

Preface

Primary care and emergency care share a common genesis; namely the overall care of the patient. In the estimation of some, they are hardest fields in medicine to master given the complexity involved. While the circumstances in which their patients are cared for is disparate, they nevertheless have in common aspects of treatment in those ailments that present for both. These ailments also share some common pitfalls that must be understood by both specialties. Emergency physicians primarily concentrate on the true emergencies that present to the emergency department; however, many patients present to the emergency department due to lack of access and require urgent, not emergent treatment. It is for these types of the patients this book is written. The basic outpatient management of those patients is something every emergency physician should understand. Of course, while follow-up with a primary care physician is often mandatory, the emergency physician can do the patient a great service by starting appropriate medications and performing appropriate testing if needed.

Contents

Contributors

Cheri N. Adgerson, MD Dermatology, UF Health Shands Hospital, Gainesville, FL, USA

Brandon R. Allen, MD Emergency Medicine, UF Health, Gainesville, FL, USA

J. Benjamin Barton, MD Emergency Medicine, UF Health Shands, Gainesville, FL, USA

Bobby Desai, MD, Med Emergency Medicine, University of Florida, Gainesville, FL, USA

Alpa Desai, DO Community Health and Family Medicine, University of Florida, Newberry, FL, USA

Tara Dyson, MD Division of Gastroenterology, Department of Internal Medicine, Gainesville, FL, USA

Julie Estrada Emergency Medicine, University of Florida, Gainesville, FL, USA

Desmond Fitzpatrick, MD Department of Emergency Medicine, University of Florida-Gainesville, Gainesville, FL, USA

Run Gan, MD Family Medicine, UF Health, Gainesville, FL, USA

Joshua Gordon, MD Emergency Medicine, UF Health Shands Hospital, Gainesville, FL, USA

Grant Harrell, MD Community Health and Family Medicine, UF Health Shands, Gainesville, FL, USA

Karl Huesgen, MD Department of Emergency Medicine, University of Florida College of Medicine, Gainesville, FL, USA

Charles Hwang, MD Department of Emergency Medicine, UF Health, Gainesville, FL, USA

Kyle M. Iketani, MD Emergency Medicine, University of Florida – Shands, Gainesville, FL, USA

Jason Jones, MD Emergency Medicine, Shands Hospital at the University of Florida, Gainesville, FL, USA

Gail A. Knight, MD Emergency Medicine, UF Health Shands Hospital, Gainesville, FL, USA

Michael Marchick, MD Emergency Medicine, UF Health Shands Hospital, Gainesville, FL, USA

Elvira Mercado, MD Department of Community Health and Family Medicine, UF Health Shands, Gainesville, FL, USA

Mohammad Reza Mohebbi, MD Department of Emergency Medicine, University of Missouri-Columbia, Coulmbia, MO, USA

Hasan Rasheed, MD Department of Emergency Medicine, Shands at University of Florida Health, Gainesville, FL, USA

Michael Seth Smith Department of Orthopedics, Shands at University of Florida Health, Gainesville, FL, USA

Tom A. Starnes, MD Community Health and Family Medicine, University of Florida Student Health Care Center, Gainesville, FL, USA

Erich T. Wyckoff, MD Obstetrics & Gynecology, Shand's Hospital, Gainesville, FL, USA

Ideen Zeinali, MD Emergency Department, UF Health-Shands, Gainesville, FL, USA

Chapter 1
Headache

Karl Huesgen

1.1 Introduction

Headache is the fourth most common reason for patient presentation to an emergency department (ED), with approximately 14 % per year of adults experiencing migraine or other severe headache in the previous 3 months [1]. Given the wide differential and potential morbidity and mortality of this nonspecific complaint, clinicians may find the workup and treatment of headache intimidating. Fortunately, the vast majority of presentations do not portend a more serious etiology.

Lacking intrinsic pain receptors, the brain itself is typically not the source of pain of headache. Rather, headache is usually caused by inflammation or pressure changes at vasculature or the meninges surrounding the central nervous system (CNS), whether this is caused by benign or serious causes (e.g., dehydration or intracranial neoplasm, respectively). Headache may also be a manifestation of other nearby structures (e.g., sinusitis, tension at cervical or frontotemporalis muscles, or inflammation of peripheral nerves).

The workup and treatment of the headache are generally directed by the history and physical exam. These will in turn direct delineate whether outpatient treatment, imaging, or immediate transport to the emergency department for emergent imaging (e.g., CT or MRI) and/or invasive testing (e.g. lumbar puncture) is indicated.

K. Huesgen, MD
Department of Emergency Medicine, University of Florida College of Medicine, Gainesville, FL, USA
e-mail: karlhuesgen@ufl.edu

B. Desai, A. Desai (eds.), *Primary Care for Emergency Physicians*,
DOI 10.1007/978-3-319-44360-7_1

1.2 Risk Factors

Risk factors vary depending on the specific etiology

- Prior headache
- Family history of migraine or other headache
- Analgesic use, overuse, or dependence
- Increased life stress
- URI for sinusitis, ear infection for mastoiditis
- Immunosuppression
- Uncontrolled hypertension
- Neoplasm and metastasis
- Coagulopathy
- Benign intracranial HTN
- Vascular risk factors for CVA

1.3 Differential Diagnosis

1.3.1 Common Complaints and Red Flags of Headache

Headache is divided into primary and secondary causes [1]. Headaches are considered primary when the headache is itself the main pathologic condition, whereas secondary headaches are thought to be a symptom of another pathology. For specific diagnostic criteria, practitioners are encouraged to refer to current International Headache Society guidelines.

The most common types of primary headache are migraine (with or without aura), tension-type headache, cluster headache, and hemicrania continua.

- Migraine:
  - Headaches are typically unilateral, pulsating, of moderate to severe intensity, and worsened by physical activity.
  - Symptoms may include nausea, vomiting, and photophobia.
  - Auras may include visual disturbances, paresthesias, numbness, and dysphonia (among others).
- Tension-type headache:
  - Headaches are typically bilateral, non-pulsating, of mild to moderate intensity, and not worsened by physical activity.
- Cluster headache:
  - During headache clusters, patients experience brief, excruciating unilateral pain.
  - Pain is periorbital and associated with ocular disturbances such as conjunctival injection, tearing, ptosis, or meiosis.
  - Pain is very responsive to high-concentration inhaled oxygen.

- Hemicrania continua:
  - Continuous daily unilateral pain is associated with conjunctival injection, rhinorrhea, ptosis, or miosis.
  - Pain is completely relieved by indomethacin.

Multiple other less common primary causes of headache exist, such as primary cough headache, primary headache associated with sexual activity, hypnic (sleep) headache, and benign thunderclap headache. These headaches must fulfill specific criteria, are not due to another disorder, and are generally diagnoses of exclusion.

The differential list for secondary causes of headache is extensive.

- Vascular and hematological:
  - Ischemic cerebrovascular accident (CVA) or transient ischemic attack (TIA)
  - Hemorrhagic CVA, including hypertensive
  - Aneurysmal subarachnoid hemorrhage (SAH):
    - Low-grade SAH (Hunt and Hess Grade 1) may have only mild headache.
  - Non-ruptured aneurysm (non-ruptured)
  - Arteritis (including temporal arteritis)
  - Cerebral venous thrombosis
  - Subdural hemorrhage (SDH)
  - Cervical artery dissection
- Nonvascular intracranial:
  - Neoplasm, either CNS primary or as site of metastasis
  - Benign intracranial hypertension/pseudotumor cerebri:
    - Impaired cerebrospinal fluid (CSF) absorption leads to increased intracranial pressure.
  - Inflammatory disease, e.g., systemic lupus erythematosus (SLE), Behçet's syndrome
  - Chiari malformation (Type 1):
    - Cerebellar tonsillar intrusion into foramen magnum leads to impaired CSF circulation and absorption.
    - Patients typically present with cervical pain and suboccipital headache provoked by exertion or cough.
  - CSF leak – Headache typically worse with standing
  - Seizure
- Posttraumatic:
  - Acute or chronic, for example, after concussion or whiplash
  - Epidural, subdural, subarachnoid hemorrhage
- Infectious:
  - Intracranial, e.g., bacterial meningitis, herpes encephalitis

  - Mastoiditis, osteomyelitis, sinusitis
  - As a symptom of system infection, e.g., influenza, HIV/AIDS
- Medications:
  - Many medications, most commonly phosphodiesterase inhibitors, nitroglycerin, nifedipine, nimodipine, digitalis, disulfiram, hydralazine, imipramine
- Medication overuse:
  - Defined by the presence of headache for 2–4 weeks after analgesic usage 10–15 days per month for 3 months
  - May be provoked by overuse of nonsteroidal anti-inflammatory drugs (NSAIDs), acetaminophen, opioids, triptans, ergotamine, or combination analgesics
  - May be associated with nausea, vomiting, tachycardia, anxiety (Ref. Evers et al [2].)
- Substance or withdrawal:
  - Caffeine, nicotine, alcohol, cocaine, cannabis, carbon monoxide, or other toxic exposure
- Headache due to or worsened by psychiatric disorder:
  - For example, depression, generalized anxiety disorder, somatization disorder
- Disorder of homeostasis:
  - Hypertension (typically SBP >160 or DBP >120 mmHg; with or without encephalopathy)
  - Hypercapnia or hypoxia (including sleep apnea)
  - Post-dialysis
  - Eclampsia/preeclampsia
- Eye pain and headache:
  - Acute glaucoma or other ocular inflammatory disorders
- Facial pain and headache due to rhinosinusitis
- Cranial pain due to bony destruction:
  - Multiple myeloma, Paget's disease
- Temporomandibular disorder:
  - Associated with painful jaw clicking, locking, tenderness at muscles of mastication, or temporomandibular joint (HA primary care guidelines)
- Cervicogenic headache:
  - Occipital pain associated with cervical paraspinous musculature tenderness
  - Exacerbated by neck movement

1.4 History

The single most important historical feature to elicit is whether the patient has had similar headaches in the past. However, even with benign headache presentations, if asked "is the worst headache of your life?," many patients will say "yes" regardless of prior identical presentations or diagnoses. The label "worst headache of one's life" may pigeonhole the emergency physician into unnecessary testing and should be used sparingly. A useful way to initiate this discussion is to ask "when was the last time you had a headache like this?" Patients with benign headaches will generally affirm prior similar headaches, patients with new-onset headaches will say never, and occasionally the patient will provide the diagnosis (e.g., an HIV-positive patient stating "the last time I had cryptococcal meningitis").

Headache Red Flags [3]

- A new or different headache
- Sudden onset and maximum severity at onset ("thunderclap")
- Precipitated by a Valsalva maneuver, exertion, or sex
- Fever and neck stiffness
- Progressive worsening over weeks to months
- The presence of neurological signs or seizure
- Worsened by recumbent position
- Uncontrolled hypertension
- New-onset headache after age 50
- Unexplained weight loss
- Scalp tenderness

Important historical features to elicit include:

- The presence of similar prior headaches or significant change from prior headaches
- Timing:
  - Time of onset
  - Time to peak intensity:
    - Maximal intensity at onset suggests SAH.
  - Discrete time of onset vs. gradual onset
- If chronic:
  - Frequency of recurrence.
  - Progressive worsening may suggest neoplasm, SDH, or hydrocephalus
- Provoking or palliating factors:
  - Worsened with exertion:
    - Suggestive of migraine (if recurrent) or SAH (if new-onset thunderclap)

  - Association with menstrual cycle:
    - Migraine often worsened in late luteal phase [US guideline Beithon]
  - Association with chewing (jaw claudication) associated with temporal arteritis
  - Worse in the morning:
    - Increased intracranial pressure, e.g., pseudotumor cerebri or due to space-occupying malignancy
    - Sleep apnea causing hypercapnia or hypoxia
  - HA from space-occupying lesion and cerebral venous thrombosis, worse with lying down
  - Sudden headache associated with decrease in environmental light (e.g., entrance into a darkened room) associated with acute angle closure glaucoma
  - Worse with cough or Valsalva may suggest Chiari 1 malformation
- Location – e.g., facial, bitemporal, unilateral, occipital, and radiation from the neck
- Quality:
  - Sharp vs. dull
  - "Throbbing" associated with migraine
  - "Band-like" associated with tension
- Severity:
  - Mild: able to continue daily activities with minimal alteration
  - Moderate: inhibits daily activities but is not incapacitating
  - Severe: incapacitating:
    - Low-grade SAH (Hunt and Hess Grade 1) may have mild or no headache, so this is not a predictive factor for differentiating this from other etiologies.
- Infectious symptoms:
  - Association with fever and meningismus should prompt immediate evaluation for meningitis:
    - Differentiate meningismus (neck stiffness) vs. diffuse myalgias
- Neurologic symptoms:
  - Visual disturbances or nausea may indicate migraine.
  - Altered level of consciousness with papilledema suggests space-occupying lesion, e.g., malignancy.
  - Loss of consciousness may suggest SAH.
- Ocular and nasal symptoms:
  - Rhinorrhea, tearing, eyelid ptosis, and edema may suggest cluster headache.

- Head or neck trauma:
  - Relatively minor head trauma may result in SDH, especially in older patients, alcoholics, or patients taking anticoagulants.
  - Neck trauma or manipulation (e.g., whiplash, yoga, chiropractic manipulation) may cause vascular injury (e.g., vertebral artery dissection).
- Medications:
  - Medications tried for relief
  - Medications used daily which may predispose to MOH
  - Anticoagulants or antiplatelet agents which may predispose to or worsen ICH
  - Exogenous estrogens may contribute to coagulopathy (e.g., dural venous thrombosis)
- Travel history
  - Possible exposure endemic viral or zoonotic diseases
- Sleep adequacy
- Significant life stressors
- Other medical conditions which may be contributory:
  - Hypertension.
  - Hypoxia due to COPD or obstructive sleep apnea (OSA).
  - Malignancy.
  - Dialysis.
  - Diabetes.
  - Immunosuppression, e.g., HIV/AIDS and transplant.
  - Recent upper respiratory tract infection or allergen exposure may predispose to sinus headache.

1.5 Physical Exam

The physical examination should focus on potential emergency conditions related to the headache. Numerous other etiologies may be identified during workup.

- Vital sign assessment:
  - The presence of fever, hypothermia, tachypnea, or unexplained tachycardia (indicative of an occult fever) should alert the physician to a possible infectious cause.
  - Hypertension with systolic blood pressure > 160 mmHg or diastolic blood pressure >120 mmHg may indicate hypertension as cause of headache:
    - Severely elevated blood pressure (e.g., SBP >200 mmHg or DBP >110) should prompt consideration of intracranial hemorrhage.
  - Tachycardia may indicate stimulants or dehydration.

- Neurologic examination:
  - Assess general alertness and orientation.
  - Evaluate cranial nerve function.
  - Assess for signs of cerebellar dysfunction.
  - Romberg test.
  - Assess reflexes.
  - Evaluate gait.
- Head, eyes, ears, nose, and throat examination:
  - Evaluate for signs of trauma which may have caused intracranial hemorrhage or concussion:
    - Battle sign
    - Raccoon eyes
  - Evaluate for signs of infection:
    - Mastoid tenderness suggestive of mastoiditis (temporal bone infection)
    - Pain reproduced or worsened with manual pressure to maxillary or frontal sinuses
    - Evaluate ears for evidence of:
      - Otitis media
      - Otitis externa
    - Evaluate sinuses for evidence of sinusitis.
    - Evaluate pharynx for evidence of:
      - Pharyngitis
      - Abscesses:
        - Peritonsillar
        - Retropharyngeal:
          - Typically will have odynophagia and voice changes
    - Evaluate dentition for evidence of abscesses.
  - Palpate for tenderness at the area of the temporal artery (tenderness may indicate temporal arteritis).
  - Evaluate pupillary size, symmetry, and reactivity:
    - Fundoscopic exam if possible to evaluate for papilledema as sign of increased intracranial pressure
    - Mid-range and minimally or nonreactive pupil concerning for acute angle closure glaucoma
- Neck examination:
  - Assess flexion, extension, and rotation.

  - Meningismus (neck stiffness suggesting meningitis or SAH) should be differentiated from diffuse myalgias (e.g.. due to influenza).
  - Tenderness of cervical paraspinous muscles may suggest cervicogenic headache.
- Skin:
  - Bruising, pallor, or other changes which might indicate coagulopathy
- Psychiatric evaluation:
  - Brief screen for depression and increased life stress

1.6 Additional Testing

Headache is generally diagnosed by history and physical exam. Imaging and/or laboratory tests are typically not needed [3].

Indications for imaging in the workup of headache include [4]:

- Headache that is new onset, worse than prior, or abrupt onset
- Progressive headache associated with neurological signs
- Headache in association with trauma, cough, exertion, or sexual activity
- Persistent and positional headache or headache associated with papilledema
- Headache associated with pregnancy, malignancy, or hypercoagulable disorders
- Headache with temporal location in older individuals
- Headache radiating to the neck
- New headache associated with HIV/AIDS

Head CT is generally indicated in investigation of possible hemorrhage, trauma, or gross anatomical disturbance such as normal pressure hydrocephalus or Chiari malformation. It may also provide evidence of sinusitis, mastoiditis, or other infection. CT angiography may demonstrate aneurysm. CT venography is an indication in the workup of possible cerebral venous thrombosis. If lumbar puncture is to be performed, CT is often performed first to evaluate for space-occupying lesions. Magnetic resonance imaging (MRI) is more sensitive for evaluation for soft tissues (e.g., malignancy) and, lacking ionizing radiation, is safe in pregnancy.

Similar to imaging, blood labs are generally not indicated in the workup of headache unless investigating specific diagnoses. For example, in an older individual, a normal ESR excludes the diagnosis of temporal arteritis (if abnormal this should be followed by referral to the appropriate specialist for temporal artery biopsy). Lumbar puncture and cerebrospinal fluid analysis should be performed if subarachnoid hemorrhage (sudden-onset, severe headache with a negative noncontrast head CT) or meningitis/encephalitis is suspected. Analyses should include cell count, gram stain, protein and glucose measurement, and fluid culture. In combination with negative head CT, absence of RBCs and xanthochromia on LP rules out SAH. If pseudotumor cerebri or cryptococcal meningitis is suspected, opening pressure should be measured.

1.7 Introduction to Treatment

If a specific underlying cause of the headache (e.g., caffeine withdrawal, dehydration) can be identified, treatment can be directed accordingly. In the emergency setting, however, the precise etiology of the headache may not be immediately identifiable. Fortunately, most headache sufferers get relief from a "headache cocktail" that simultaneously addresses multiple pharmacologic and physiologic targets, regardless of the specific headache etiology. It is important to reiterate that response to analgesia does not predict etiology and should not be used in diagnostic decision-making.

First-line headache cocktail:

- Metoclopramide (Reglan; 10–20 mg IV; 20 mg PO; dopamine antagonism and acetylcholine sensitization) or prochlorperazine (Compazine (brand name discontinued in the United States); 10 mg IV or 10 mg PO; dopamine and CNS adrenergic antagonism)
  - These medications have multiple side effects and contraindications, notably extrapyramidal side effects (EPS) including but not limited to dystonias, akathisia, neuroleptic malignant syndrome, etc.
- Diphenhydramine (Benadryl; nonselective antihistamine; 25 mg IV or 25–50 mg PO) to prevent EPS
- Ketorolac (Toradol; NSAID; 30 mg IV) or ibuprofen (Motrin; NSAID; 800 mg PO)
- Fluid bolus (e.g., normal saline 1–2 L IV)

In the ED setting, most headaches will respond to aforementioned "headache cocktail" with or without additional acetaminophen. For migraine headaches, second-line therapy consists of triptans (e.g., sumatriptan or zolmitriptan), but only if the patient has not used ergotamines (e.g., dihydroergotamine (DHE)) within prior 24 h as their additive mechanisms of action may cause vasospasm. Some patients may also benefit from additional IV or PO steroids, though the clinical literature has shown mixed results. Similarly, clinicians can also consider adding combination drugs that include butalbital (an intermediate-acting barbiturate), such as butalbital, acetaminophen, and caffeine combination therapy. Barbiturate-containing formulations should be used sparingly because they can lead to dependence and overuse. Likewise, opioids (e.g., hydrocodone, oxycodone, or hydromorphone) may be efficacious in treating headache, though most guidelines and consensus statement discourage their use. If barbiturates and opioids are the only substances from which the patient obtains relief, clinicians should consider investigation of dependence or medication overuse as the headache etiology. Long-term therapy for migraine prevention may include amitriptyline (a tricyclic antidepressant), beta-blockers, calcium channel blockers, antidepressants (e.g., venlafaxine, a serotonin-norepinephrine reuptake inhibitor), antiepileptic drugs (e.g., topiramate and divalproex), as well as multiple alternative and non-pharmacologic therapies (HA primary care guidelines). In practice these long-term therapies are rarely initiated in

the emergency setting. Treatment of tension-type headache is similar to that of migraine, though triptans and ergotamines are rarely used. Benzodiazepines, barbiturates, and opioids are also discouraged for tension headache treatment. A unique feature of treatment for cluster headache is response to high-flow oxygen. If the patient has a history suggestive of cluster headaches, a trial of high-concentration inhaled oxygen is indicated (100 % O_2 12 lpm for 15 via non-rebreather mask). Triptans can also be considered for cluster. If medication-overuse headache is suspected, guidelines recommend abrupt withdrawal for analgesic-, ergotamine-, or triptan-related headache. If overuse of opioids, benzodiazepines, or barbiturates is suspected, a more gradual taper is recommended (Evers et al.). If specific secondary headache can be diagnosed, varying therapies directed at the cause may be helpful (e.g., lumbar puncture for pseudotumor cerebri or CPAP for OSA).

1.8 Pharmacologic Treatment

- NSAIDS, acetaminophen, aspirin:
  - Block the inflammatory arachidonic acid cascade at COX-1 and COX-2 to inhibit prostaglandin synthesis.
  - Ibuprofen 200–400 mg PO.
  - Naproxen 275–550 mg PO.
  - Acetaminophen 500–1,000 mg PO or IV.
  - Aspirin 500–1,000 mg PO.
  - Ketorolac 30 mg IV or 60 mg IM or IV.
  - Indomethacin 25–75 mg PO or IV.
  - Pitfalls. These agents may cause many adverse reactions, notably gastric irritation, GI bleeding, and renal dysfunction. Aspirin causes permanent dysfunction of currently circulating platelets. Acetaminophen should be avoided in conjunction with ethanol and/or liver disease and should be limited to no more than 4 g daily. Ibuprofen has a black box association with cardiovascular disease.
- Neuroleptics, antiemetics, and antihistamines:
  - Multiple pharmacologic targets, including dopaminergic blockade and acetylcholine sensitization.
  - Metoclopramide 10 mg IV.
  - Prochlorperazine 10 mg IV.
  - Droperidol 2.5 mg IV or haloperidol 5 mg IV may have efficacy in headaches resistant to other drug classes.
  - Pitfalls: These agents also have many possible adverse reactions, notably dystonic reactions, sedation, and QTc prolongations. Clinicians are advised to familiarize oneself with preferred agents. Side effects may be decreased or eliminated by pretreatment with diphenhydramine which also provides ancillary benefit of drowsiness

- Triptans and DHE:
  - Causes vasoconstriction via selective serotonergic agonism (5HT-1 for triptans, 5HT-1D for DHE).
  - Triptan dose can be repeated once.
  - Sumatriptan 100 mg PO, 6 mg SC, 20 mg IN.
  - Zolmitriptan 5 mg IN.
  - Dihydroergotamine (DHE) 1 mg SC, IM, or IV.
  - Pitfalls: These classes may contribute to serotonin syndrome, chest pain, and possible coronary vasoconstriction. Avoid if there is history of Prinzmetal's angina, cardiovascular disease, uncontrolled hypertension or concurrent SSRI, MAOI, or other vasoconstrictive medications. Do not give triptans within 24 h of dihydroergotamine. DHE carries a black box warning for peripheral ischemia and cerebral vasospasm when given with 3A4 inhibitors (including macrolide antibiotics and protease inhibitors) and should not be used in hemiplegic or basilar migraine.
- Steroids:
  - Multiple anti-inflammatory, glucocorticoid, and mineralocorticoid effects.
  - Prednisone 60 mg PO.
  - Methylprednisolone 125 mg IV.
  - Daily steroid therapy should be initiated immediately if temporal arteritis is suspected.
  - Pitfalls: Usually well tolerated as short-term therapy, but efficacy data are mixed. Some research has shown benefit of steroids as monotherapy or in addition to other therapies in treatment of migraine, especially in those for whom migraines are recurrent, frequent, and refractory to other treatments [5].
- Opioids:
  - Analgesia through opioid receptor antagonism
  - Numerous preparations, routes, and combination therapies available
  - Hydrocodone 5 mg or oxycodone 5 mg
  - Hydromorphone 0.5–1 mg IV
  - Administered in 35 % of ED visits despite usage discouragement by guidelines and consensus statements [6]
  - Pitfalls: Opiates are pro-inflammatory and increase vasodilation, which is counterproductive to current understanding of migraine pathophysiology. If opioids are the only medications by which the patient obtains relief, the physician should consider MOH or dependence as cause.

References

1. Silberstein SD, Olesen J, Bousser MG, Diener HC, Dodick D, First M, Goadsby PJ, Göbel H, Lainez MJ, Lance JW, Lipton RB, Nappi G, Sakai F, Schoenen J, Steiner TJ; International Headache Society. The international classification of headache disorders, (ICHD-II)—revision of criteria for 8.2 medication-overuse headache. Cephalalgia. 2005:25(6), 460–5.
2. Becker WJ, Findlay T, Moga C, Scott NA, Harstall C, Taenzer P. Guideline for primary care management of headache in adults. Can Fam Physician. 2015;61(8):670–9.
3. Beithon J, Gallenberg M, Johnson K, Kildahl P, Krenik J, Liebow M, Linbo L, Myers C, Peterson S, Schmidt J, Swanson J. Diagnosis and treatment of headache. Bloomington: Institute for Clinical Systems Improvement (ICSI); 2013.
4. Edlow JA, Panagos PD, Godwin SA, Thomas TL, Decker WW. Clinical policy: critical issues in the evaluation and management of adult patients presenting to the emergency department with acute headache. J Emerg Nurs. 2009;35(3):e43–71.
5. Woldeamanuel YW, Rapoport AM, Cowan RP. What is the evidence for the use of corticosteroids in migraine? Curr Pain Headache Rep. 2014;18(12):1–5.
6. Burch RC, Loder S, Loder E, Smitherman TA. The prevalence and burden of migraine and severe headache in the United States: updated statistics from government health surveillance studies. Headache: J Head Face Pain. 2015;55(1):21–34.

Chapter 2
Eye Issues

Michael Marchick

2.1 Introduction

Ophthalmologic symptoms are frequent chief complaints in both the primary care office and emergency department settings [1]. While the majority of patients presenting with these symptoms will have a self-limiting etiology, rapidly sight-threatening diagnoses are on the differential, and the clinician must maintain a high degree of vigilance to properly rule out these conditions.

2.2 Differential Diagnosis

- Eyelid/periorbital conditions
  - Blepharitis
  - Hordeolum
  - Chalazion
  - Dacryoadenitis
  - Dacryocystitis
  - Periorbital (preseptal cellulitis)
  - Orbital cellulitis

M. Marchick, MD
Emergency Medicine, UF Health Shands Hospital, Gainesville, FL, USA
e-mail: marchick@ufl.edu

B. Desai, A. Desai (eds.), *Primary Care for Emergency Physicians*,
DOI 10.1007/978-3-319-44360-7_2

- Red/painful eye
  - Traumatic
    - Globe rupture
    - Hyphema
    - Subconjunctival hemorrhage
    - Corneal abrasions
    - Corneal foreign bodies
    - Chemical burns
  - Atraumatic
    - Acute angle-closure glaucoma
    - Conjunctivitis
    - Dry eye (keratoconjunctivitis sicca)
    - Episcleritis
    - Corneal ulcer
    - Scleritis
    - Keratitis
    - Corneal ulcer
    - Iritis
    - Pterygium

2.3 History

A full ophthalmologic history should be obtained in all patients with ocular/periorbital complaints, with particular attention to use of contact lenses and surgical history. Key components of the general medical history include the use of any anticoagulants and the presence of systemic disease with potential ocular manifestations, such as sarcoidosis, HIV, TB, and rheumatoid arthritis.

A history of sudden vision loss or decrease should prompt emergent specialist consultation. A description of a curtain or veil being pulled across the visual field, as well as new onset or worsening floaters, suggests a retinal detachment. Sudden visual loss, particularly in a patient with atrial fibrillation or other cerebrovascular accident risk factors, suggests a central retinal artery occlusion. Vitreous hemorrhage and central retinal vein occlusion may also result in abrupt, painless vision loss.

- Other important historical features to elicit include:
  - Diplopia
  - Discharge
    - Amount
    - Consistency
    - Worsening in AM

- Discomfort
  - Aggravation of symptoms with blinking
  - Foreign body sensation
  - Itching
- Exposures
  - Chemical
    - *Red flag: Alkali and other caustic exposures
  - UV light (including skiing/high altitudes, welding)
- Genitourinary symptoms may suggest chlamydial or gonorrheal disease
- Trauma
- Upper respiratory infection symptoms
- Vomiting
  - *Red flag: Highly suspicious for angle-closure glaucoma if associated with visual disturbance and/or red eye

2.4 Physical Exam

Visual acuity is the vital sign of the eye and should be assessed in any patient presenting with an ocular complaint. If the patient's prescribed glasses are not at bedside, visual acuity measured through a pinhole will suffice [1]. An orderly progression through the physical examination of the periorbital region and the eye can then ensue as outlined below.

Pitfall: Following a traumatic injury, the integrity of the globe must immediately be assessed. The presence of pupillary irregularity (particularly a teardrop-shaped pupil), a corneal or scleral laceration, a positive Seidel's test, or a flat anterior chamber should prompt cessation of any further manipulation of the globe and immediate ophthalmology consultation.

Periorbital Exam

- Discharge
  - *Red flag: Copious purulent discharge in patients with risk factors for sexually transmitted diseases (gonorrheal conjunctivitis)
- Edema
- Erythema
- Papular lesions (i.e., hordeolum, chalazion)
- Proptosis
  - *Red flag: Proptosis is associated with retrobulbar hemorrhage (particularly in the setting of trauma), orbital cellulitis, cavernous sinus thrombosis, and malignancy.

- Vesicular rash (HSV, VZV)
  - *Red flag: Hutchinson's sign, a vesicular lesion on the tip of the nose, is highly associated with herpes zoster ophthalmicus.

Extraocular Movements

- *Red flag: Restriction of movement is associated with orbital cellulitis, traumatic entrapment, or cranial nerve palsies.

Conjunctival and Scleral Evaluation

- Conjunctival injection
- Chemosis
- Foreign bodies
- Laceration (*red flag for globe rupture)
- Scleral injection
- Subconjunctival hemorrhage

Pupil Exam

- Photophobia
- Reactivity
  - Assess for an afferent pupillary defect (Marcus Gunn pupil). A defect is present if the affected eye dilates in response to direct light and constricts in response to consensual light. Conditions affecting the retina, optic nerve, optic chiasm, and optic tract may result in this finding [2].
  - Shape
    - *Red flag: An irregularly shaped pupil, particularly a teardrop shape, strongly suggests globe rupture.
- Size
  - Subtle (<1 mm discrepancy) anisocoria is physiologic in approximately 20 % of patients, but may also result from third nerve palsy due to uncal herniation, Horner's syndrome, medications, and direct trauma [1].

Corneal Examination

- Fluorescein uptake
  - Abrasions
  - Dendritic lesions
    - *Red flag for HSV ophthalmicus
  - Seidel's sign (dye streaming from injury site)
    - *Red flag for globe rupture
- Foreign bodies
- Ulcer

Intraocular Pressure

- Normal 10–20 mmHg
  - *Red flag: Increased intraocular pressure suggests acute angle-closure glaucoma in the appropriate clinical scenario.
- Pitfall: Do not attempt to measure intraocular pressure if there is any concern for globe rupture.
- Pitfall: Falsely elevated pressure may result if any pressure is placed on globe by examiner during measurement.

Slit-Lamp Examination

- Cell and flare (the presence of white blood cells and protein in the anterior chamber) suggest iritis.
- Depth of the anterior chamber.

Funduscopic Exam Red Flags

- Papilledema
- Retinal detachment
- Retinal hemorrhage
- Retinal pallor
  - *Red flag for central retinal artery occlusion (particularly if associated with "cherry red spot")
- Pitfall: In general, a dilated exam should only be performed by an ophthalmologist. Do not dilate if any concern for acute angle-closure glaucoma.

2.5 Specific Conditions and Their Management

2.5.1 Eyelid/Periorbital Conditions

Blepharitis

- Acute or chronic inflammation of the hair follicles at the lid margin, commonly associated with staphylococcal infection, is termed blepharitis.
- Characterized by edema, erythema, pruritus, discharge, and crusting of the lid margin.
- Conjunctival injection may be present.
- Symptoms are typically most severe in the morning.
- Application of warm compresses and gentle cleaning with shampoo are typically effective.
  - In refractory cases, the use of topical antibiotics can be considered [3–5].

Hordeolum

- A hordeolum is an eyelid mass resulting from acute bacterial infection.
- An external hordeolum, commonly referred to as a stye, is a lesion originating from the glands of Zeiss or Moll.
- *Staphylococcus aureus* is the most commonly identified pathogen.
- Warm compresses applied several times daily are first line therapy.
- Topical bacitracin can also be considered.
- If persistent despite these measures, patients may be referred to an ophthalmologist for incision and drainage [6].

Chalazion

- Obstruction of a meibomian gland can result in formation of a chronic inflammatory lesion at the lid border termed a chalazion.
- The skin itself is typically normal; however, lid edema and/or a discrete nodular lesion may be noted.
- The presence of pain should suggest an alternative diagnosis.
- Warm compresses can be applied, but the lesions are typically self-limited.
  - Antibiotics are not indicated.
- If large and persistent, incision and drainage by an ophthalmologist can be considered [4, 6].

Dacryoadenitis and Dacryocystitis

- Infection of the lacrimal glands is termed dacryoadenitis, while infection of the lacrimal sac is termed dacryocystitis.
- Patients with dacryoadenitis typically present with abrupt onset eyelid edema, particularly near the upper lid margin.
- Dacryocystitis is typically preceded by an upper respiratory infection with development of erythema and edema just inferior to the medial canthus occurring several days later.
- Fever is typically present.
- With either condition, patients can appear quite ill.
- A variety of pathogens, both viral and bacterial, can be responsible for each.
  - *S. aureus* is the most common bacterial etiology in dacryoadenitis.
  - In neonates, *Streptococcus pneumoniae* is the predominant etiology.
  - *S. aureus* and *Staphylococcus epidermis* are more common in older children and adults.
- Therapy of dacryoadenitis includes parental antibiotics with anti-staphylococcal coverage.
  - MRSA coverage should be added as appropriate based on local susceptibility patterns.
  - Patients with dacryocystitis typically require parenteral antibiotics.
  - Infants in particular require admission to the hospital [6, 7].

Periorbital (Preseptal) and Orbital Cellulitis

- Cellulitis affecting the area surrounding the eye is divided into periorbital (preseptal) or orbital cellulitis depending on the presence of infection posterior to the orbital septum.
- Orbital cellulitis is acutely life- and sight threatening; therefore, differentiating the two entities is crucial.
- Periorbital cellulitis is typically more indolent in development and often preceded by minor skin trauma or a cutaneous facial infection such as a hordeolum.
- Orbital cellulitis is much more commonly abrupt in onset and the result of extension of sinusitis.
- Erythema, edema, and warmth surrounding the affected eye, as well as fever, may be present in both entities.
- The presence of visual changes, proptosis, and restriction of extraocular movement, as well as pain associated with such movement, suggest orbital cellulitis and/or abscess formation.
- If orbital cellulitis is suspected, patients should be emergently evaluated with CT or MRI to establish the presence or absence of inflammatory changes posterior to the orbital septum.
- In a well-appearing patient with periorbital cellulitis, treatment with an oral antibiotic regimen active against both MRSA and *S. pneumoniae* and *Haemophilus influenzae* is appropriate [4].
  - Clindamycin alone or, alternatively, amoxicillin/clavulanate along with sulfamethoxazole/trimethoprim are appropriate choices.
- Patients who do not experience improvement in symptoms within 24 h should be reassessed for evidence of orbital cellulitis.
- Patients with orbital cellulitis require hospital admission, broad-spectrum parenteral antibiotics, and ophthalmology consultation.

2.5.2 Intrinsic Orbit Conditions

2.5.2.1 Traumatic Injuries

Globe Rupture

- A globe rupture should be suspected after any high-impact blunt or penetrating injury to the orbit or periorbital region.
- Other traumatic injuries may coexist depending on the mechanism of injury.
- Eye pain is typically present and visual acuity is typically compromised.
- Globe rupture may be occult, and a low threshold for further evaluation and consultation should be maintained if the diagnosis is suspected despite lack of the cardinal findings noted above.

  - CT of the orbits is the typical next study in such cases.
  - MRI is contraindicated if there is any concern for a metallic foreign body.

- Management includes emergent ophthalmology consultation, broad-spectrum IV antibiotics (e.g., vancomycin and a fourth-generation cephalosporin), and placement of an eye *shield*.
  - No further manipulation of the globe should occur unless undertaken by an ophthalmologist.
  - Provide adequate analgesia and update tetanus immunization status as appropriate.
  - Patients should remain NPO.
  - Elevate the head of the patient's bed and treat any nausea or vomiting to protect against elevation of intraocular pressure.

Hyphema

- Blood in the anterior chamber following a traumatic injury is an indication for urgent ophthalmology evaluation.
- In the interim, patients should be evaluated for evidence of any other traumatic injuries.
- If no contraindication, patients should be advised to sit upright to minimize the opportunity for staining of the cornea.
- Pain control should be administered (avoid topical agents if any suspicion of a globe rupture).
- Due to rebleeding risk, NSAIDs and aspirin are contraindicated.
- Treat nausea and limit potential pupillary accommodation/constriction, which can result in further bleeding, by dimming/reducing ambient light.
- An eye shield can be utilized.

Subconjunctival Hemorrhage

- While the characteristic appearance of extravasated blood associated with a subconjunctival hemorrhage is often alarming to patients, the condition is typically benign.
- Patients may notice this finding after a direct traumatic injury, coughing, or Valsalva. Alternatively, the onset can be spontaneous.
- The presence of pain should prompt a search for an alternative/coexisting diagnosis, particularly in the setting of trauma.
- Evaluate for a corneal abrasion in these patients, and consider the possibility of an open globe injury.
- If the patient is anticoagulated, consider assessment of coagulation parameters.
- Reassurance of patients is indicated, as is counseling that up to 2–3 weeks may be needed for full resorption of the blood [3].

Corneal Abrasions

- Intense pain and photophobia are the most common presenting complaints among patients presenting with corneal abrasions.

- A history of eye trauma immediately preceding the onset of symptoms is often apparent.
- It is crucial to evaluate for more sinister injuries, particularly an open globe or hyphema, in the setting of a significant traumatic mechanism.
- Unfortunately, the physical exam, including measurement of visual acuity, is often limited by the patient's presenting symptoms.
- If there is no evidence of an open globe, a dose of topical anesthetic (i.e., proparacaine 0.5 %) should be administered and will provide near immediate relief.
  - Despite their efficacy, continued use of these agents is contraindicated due to concern for epithelial toxicity [8].
- Subsequent fluorescein instillation will allow for enhancement of the abrasion under a Wood's lamp or the cobalt blue filter of an ophthalmoscope or slit lamp.
- The presence of absence of a Seidel sign should be assessed concurrently.
- Eversion of the upper eyelid to evaluate for a retained foreign body is warranted.
- Patients with hypopyon (pus in the anterior chamber), hyphema, or corneal ulceration should be evaluated by an ophthalmologist emergently.
- Urgent referral is warranted for:
  - Patients without healing after 3–4 days
  - Patients with a significant decrease in visual acuity
  - Large abrasions (>1 quarter of the diameter of the cornea)
  - Those with purulent discharge
- Adequate analgesia is important for all patients.
- Most small abrasions will heal completely within 24 h at which time patients should be pain-free.
- In the interim, oral NSAIDs and/or opiate analgesia can be prescribed.
- Topical NSAIDs are also appropriate, although their cost can be significant.
- There is no evidence that patching of abrasions, in particular small lesions, leads to improvement in rate of healing or pain control [9].
- Contact lens wearers should never be patched.
- Larger abrasions require a longer course of oral analgesia.
- An ophthalmologist may consider the use of a cycloplegic agent in such cases.
- Appropriate antibiotic coverage should be administered. In abrasions associated with contact lens use, patients should receive coverage for *Pseudomonas aeruginosa* (e.g., ofloxacin or ciprofloxacin, one to two drops four times daily for 3–5 days).
- Appropriate coverage for others includes polymyxin-trimethoprim or erythromycin ointment (one drop or 0.5 in., respectively, four times daily for 3–5 days).

Corneal Foreign Body

- Corneal foreign bodies characteristically present with severe eye irritation worsened with blinking.
- Conjunctival injection, photophobia, and increased lacrimation are typically also present.

- Evaluate for a ruptured globe prior to any attempt at foreign body removal.
- Following application of topical anesthetic, attempts at foreign body removal can be made with irrigation or use of a moistened cotton swab.
- If unsuccessful, a clinician with experience in corneal foreign body removal with a 25-gauge needle or eye spud under magnification can make an additional attempt.
- Metallic foreign bodies present for more than several hours can result in formation of a rust ring.
  - If such a ring is noted following foreign body removal, the patient should be referred to an ophthalmologist within the next several days.
  - There is no need for emergent rust ring removal provided prompt ophthalmology follow-up is available.
- Urgent ophthalmology consultation is indicated for patients with foreign bodies which cannot be easily removed.
- All patients should be referred to ophthalmology on an outpatient basis to evaluate for delayed presentation of infection.
- Provide oral analgesia and topical antibiotics as would be given for patient with a corneal abrasion [5, 10].

Chemical Burns

- Any exposure of the globe to caustic material is a true ophthalmic emergency.
- Alkali exposures are particularly dangerous as they result in liquefactive necrosis, which results in a deeper progression of damage compared with the coagulation necrosis caused by acidic substances.
- The initial treatment for all chemical burns is copious irrigation with normal saline, preferably via a Morgan lens.
  - Do not delay irrigation to obtain visual acuity or other testing.
  - Following 30 min of irrigation, ocular pH should be checked.
  - Continue irrigation until the eye has maintained a pH of 7.0 for at least 30 min.
  - It is prudent to evert the eyelid to evaluate for the possibility of retained or crystallized particulate matter, which may result in persistent difficulties in achieving a neutral pH.
- Immediate ophthalmology consultation is indicated in all cases of caustic exposure [5, 11].

2.5.3 Atraumatic Conditions

2.5.3.1 Acute Angle-Closure Glaucoma

- An abrupt increase in intraocular pressure can result from impaired drainage of aqueous humor in anatomically predisposed individuals (typically patients with hyperopia).
  - Typical precipitants include dim lighting and medications with the side effect of pupillary dilatation (e.g., anticholinergics).

- This pressure increase can be acutely sight threatening and demands emergent reversal.
- Symptoms include blurred vision, visualization of colored halos near lights, nausea, vomiting, frontal headache, and severe eye pain.
- On physical exam, conjunctival injection, a fixed mid-dilated pupil, and a hazy cornea may be appreciated.
- If available, tonometry should be conducted by the clinician; if unavailable, emergent referral to an ophthalmologist is indicated.
  - Intraocular pressure is typically >30 mmHg.
- Pending ophthalmological evaluation, systemic acetazolamide can be given as well as consideration of topical timolol, apraclonidine, and/or pilocarpine.
- Laser peripheral iridotomy is the typical definitive treatment [11].

2.5.3.2 Conjunctivitis

Viral

- Viral conjunctivitis is the most common cause of a red eye [12], although bacterial infection and allergic mechanisms are also commonly responsible.
- Discerning the etiology responsible for a specific case of conjunctivitis can be difficult. Inflammation of the conjunctiva almost universally results in redness and discharge of the affected eye(s), as well as closure of the eyes upon awakening, regardless of etiology.
- Itching of the eyes in the setting of other conjunctivitis symptoms is a distinguishing factor implicating an allergic etiology.

Bacterial

- Bacterial infection is more common in children than adults.
- Concomitant or preceding upper respiratory infection symptoms suggest adenoviral conjunctivitis.
- Clinicians should be vigilant in performing an orderly physical exam in patients with conjunctivitis, with special attention to fluorescein staining to rule out more serious pathology which patients and/or parents will typically ascribe to "pink eye."
- Viral conjunctivitis is most commonly caused by one of many strains of adenovirus, although HSV can cause particularly severe disease as discussed below.
  - Adenoviral conjunctivitis is highly contagious, potentially transmissible up to 2 weeks following onset of symptoms.
  - Strict hand hygiene is therefore indicated.
  - Topical antibiotics are not necessary, as bacterial superinfection is rare and patients can be expected to have spontaneous resolution of symptoms generally within several days, with occasional persistence up to 2 weeks.

- The most common bacterial etiologies are *S. aureus* (particularly in older children and adults), *H. influenzae*, *Streptococcus pneumoniae*, and *Moraxella catarrhalis* predominate in young children [13].
  - Preferred agents for nonusers of contact lenses are erythromycin ointment (0.5 in. applied four times daily) or polymyxin-trimethoprim (one to two drops applied four times daily).
  - A 5–7-day course is typically prescribed.
    - Topical aminoglycosides are generally avoided due to their inherent toxicity to the corneal epithelium and tendency to cause further irritation with prolonged use.
  - The presence of a concomitant acute otitis media, particularly in children, implies *H. influenzae* infection [7].
    - Affected patients should also be treated with systemic antibiotics with activity against this pathogen (i.e., amoxicillin/clavulanate).
- *Pseudomonas* is a common pathogen in contact lens wearers.
- Such patients are also at high risk of corneal ulceration and keratitis, and a thorough evaluation for these conditions is indicated, as their presence necessitates prompt ophthalmology consultation.
  - In the absence of these findings, management in contact lens users typically employs topical fluoroquinolones.
- Patients should be advised to discard the lenses they were using just prior to symptom development and to avoid use of any lens until cleared to do so by an ophthalmologist.
- If there is no improvement in symptoms after 12–24 h of treatment, urgent ophthalmology referral is indicated.
- Chlamydial conjunctivitis should be suspected in at-risk patients refractory to standard bacterial conjunctivitis treatment.
  - Infection occurs through contact either directly from the genitalia or via the hands.
  - Symptoms of coexisting genital tract disease are variably present.
  - Systemic therapy (azithromycin 1 g PO × 1 or doxycycline 100 mg PO bid × 14 days) to clear the genital infection as well as topical therapy (e.g., erythromycin) should be administered.
  - Sexual contacts should also be tested and treated [3, 12].
- Hyperacute bacterial conjunctivitis, typically associated with *Neisseria gonorrhoeae*, is often heralded by a very abrupt onset of copious discharge from the affected eye(s) after introduction of the pathogen (≤12 h).
  - Spread is similar to that noted with chlamydial disease.
  - Urethritis symptoms are often present.
  - Cultures should be obtained and topical antibiotics (e.g., erythromycin) as well as systemic ceftriaxone (1 g IM) administered.
  - Due to the high risk of complications including corneal perforation and blindness, immediate ophthalmology consultation is warranted [12].

- Children born in US hospitals typically receive erythromycin ointment immediately after birth to reduce the likelihood of gonococcal ophthalmia neonatorum.
  - This condition most commonly presents during the first week of life with the onset of copious purulent eye discharge and conjunctival erythema.
  - Patients may present overtly septic due to disseminated infection.
  - Systemic ceftriaxone should be administered and patients admitted to the hospital with emergent ophthalmology consultation.
  - Chlamydia conjunctivitis typically presents somewhat later than gonorrhea (days 5–14).
  - Pneumonia is commonly associated.
  - Oral and topical erythromycin should be administered and prompt ophthalmology consultation obtained.
  - If untreated, corneal scarring can result [14].

Allergic

- Allergic conjunctivitis can be caused by environmental allergens such as pollen as well as through contact with irritating topical medications.
- In addition to the general supportive care recommended for all patients with conjunctivitis, topical and/or systemic antihistamines and a topical vasoconstrictor can be utilized.
- However, identification and avoidance of possible triggers are absolutely indicated.

Corneal Ulcer

- A deep infection of the cornea secondary to epithelial disruption due to direct bacterial invasion, trauma, or sloughing of cells due to dessication (such as with incomplete lid closure due to Bell's palsy) is termed a corneal ulcer.
- Soft contact lens wearers are at particularly high risk.
- Pain, photophobia, and foreign body sensation are typical symptoms.
- An area of white discoloration of the cornea is often appreciated; the lesion will enhance with fluorescein under a Wood's lamp.
- *P. aeruginosa* is of particular concern in contact lens wearers, while *S. pneumoniae* and *S. aureus* are typical culprits in other patients.
- Urgent ophthalmology consultation is indicated.
- A topical fluoroquinolone, with very frequent application, is typically prescribed following culture of the lesion [5, 14].

Episcleritis and Scleritis

- Differentiating episcleritis from scleritis is key, as the former is a generally benign condition, while the latter (which can also affect deeper structures of the globe) is potentially sight threatening.
- Features of episcleritis include onset of excessive lacrimation, irritation, and eye redness which typically only affect a segment of the globe.
- This stands in contrast to the more diffuse redness associated with conjunctivitis.
- Scleritis is typically associated with severe pain, an important distinguishing feature from the "grittiness" or mild irritation patients with episcleritis may report.

- Scleritis is often associated with systemic disease, specifically, rheumatoid arthritis and Wegener's granulomatosis.
- If scleritis is suspected, urgent referral to ophthalmology is warranted.
- Treatment typically consists of systemic NSAIDs, glucocorticoids, or other immunosuppressants [15].
- Although episcleritis is typically self-limited, topical lubricants, with or without topical NSAIDs, are often employed for symptomatic relief.

2.5.3.3 Keratitis

Herpetic

- Pitfalls: Only ophthalmologists should prescribe topical ophthalmic steroids. In the case of herpes simplex virus infection, topical steroid use can result in deeper involvement of eye structures and permanent visual impairment.
- Inflammation of the cornea is termed keratitis.
- Patients commonly present with severe pain, photophobia, and eye redness.
- Herpes simplex virus (typically reactivation rather than primary infection) is the most common cause; however, other viruses, bacteria, UV radiation, and incomplete closure of the eyelid, such as with Bell's palsy, are also responsible.
- Contact lens wearers are at increased risk due to *P. aeruginosa*. Herpes simplex infection involving the cornea is associated with ulceration and a characteristic dendritic pattern noted on fluorescein staining.
- Typical herpetic lesions may be present on the conjunctiva and eyelids.
- Treatment includes topical antivirals (e.g., trifluridine 1 % nine times daily). Patients with bacterial or viral keratitis should be seen by an ophthalmologist on a same day basis [5, 14].

UV Keratitis

- Associated with welding, skiing, and high-altitude exposure.
- Symptom onset is typically within 6–12 h of the insult.
- Avoidance of triggers and use of eye protection should be advised.
- Oral analgesia should be prescribed and the patient referred for outpatient ophthalmology follow-up.
- Pitfalls: Beware of the possibility of topical anesthetic abuse among patients with frequent exposure to insults which result in UV keratitis. Never prescribe topical anesthetic drops given the potential for corneal epithelial toxicity.

Iritis

- Iritis is commonly associated with infectious and systemic diseases such as herpes viruses, tuberculosis, syphilis, spondylarthritides, and sarcoidosis.
- Traumatic injuries may also be causative.

- The pupil is typically constricted and sluggishly reactive.
- Symptoms include pain, blurred vision, and conjunctival injection.
- Eye discharge is typically scant if present.
- A key exam finding is consensual photophobia (pain when light is exposed to the unaffected eye due to constriction of the iris).
- On slit-lamp examination, a "cell and flare" pattern due to the presence of white blood cells and proteinaceous material in the anterior chamber may be noted.
- In more severe infection, white blood cells may settle in the anterior chamber resulting in accumulation of a hypopyon.
- Sequelae of the diagnosis include glaucoma and cataract formation.
- Urgent ophthalmology consultation is warranted.

Herpes Zoster Ophthalmicus

- Herpes zoster ophthalmicus refers to reactivation of latent varicella zoster virus in the ophthalmic division of cranial nerve V.
- Patients often experience a prodrome of fatigue as well as pain affecting the dermatome in the days prior to onset of rash.
- Hutchinson's sign, the presence of vesicles on the tip of the nose, is associated with a higher likelihood of ocular involvement.
- Ocular manifestations can include blepharitis, conjunctivitis, keratitis, scleritis, iritis, retinitis, and optic neuritis.
- Management includes urgent ophthalmology consultation if there is any evidence of ocular involvement and administration of systemic antivirals (e.g., acyclovir 800 mg PO five times daily for 7–10 days).
- Antivirals are of most use when started within 72 h of onset of rash.
- Adequate analgesia should also be administered.
- Hospitalization and IV antivirals should be considered for the immunocompromised and those with severe disease (i.e., retinitis) [16].

Pterygium

- A wedge-shaped proliferation of conjunctival tissue which extends onto the cornea is termed a pterygium.
- The pathogenesis is unclear; however, excessive exposure to UV radiation is a known risk factor.
- The symptoms are typically slowly progressive and the lesion itself painless.
- However, patients may present due to cosmetic concerns, eye irritation, and potentially visual loss due to covering of the visual axis or induced astigmatism is possible.
- Patients with eye irritation should be counseled to use eye lubrication.
- In the event of visual disturbance, non-emergent ophthalmological referral is appropriate for consideration of removal [12].

References

1. Robinett DA, Kahn JH. The physical examination of the eye. Emerg Med Clin N Am. 2008;26:1–16.
2. Duong DK, Leo MM, Mitchell EL. Neuro-ophthalmology. Emerg Med Clin N Am. 2008;26:137–80.
3. Cronau H, Kankanala RR, Mauger T. Diagnosis and management of red eye in primary care. Am Fam Physician. 2010;81:137–44.
4. Carlisle RT, DiGiovanni J. Differential diagnosis of the swollen red eyelid. Am Fam Physician. 2015;92:106–12.
5. Wirbelauer C. Management of the red eye for the primary care physician. Am J Med. 2006;119:302–6.
6. Wald ER. Periorbital and orbital infections. Infect Dis Clin N Am. 2007;21:393–408.
7. Sethuraman U, Kamat D. The red eye: evaluation and management. Clin Peds. 2009;48: 588–600.
8. Yagci A, Bozkurt B, Egrilmez S, Palamar M, Ozturk BT, Pekel H. Topical anesthetic abuse keratopathy: a commonly overlooked health care problem. Cornea. 2011;30:571–5.
9. Turner A, Rabiu M. Patching for corneal abrasion. Cochrane Database Syst Rev. 2006;7:CD004764.
10. Wipperman JL, Dorsch JN. Evaluation and management of corneal abrasions. Am Fam Physician. 2013;87:114–20.
11. Pokhrel PK, Loftus SA. Ocular emergencies. Am Fam Physician. 2007;76:829–36.
12. Leibowitz HM. The red eye. N Engl J Med. 2000;343:345–51.
13. Hovding G. Acute bacterial conjunctivitis. Acta Ophthalmol. 2008;86:5–17.
14. Deibel JP, Cowling K. Ocular inflammation and infection. Emerg Med Clin N Am. 2013;31:387–97.
15. Okhravi N, Odufuwa B, McCluskey P, Lightman S. Scleritis. Surv Ophthalmol. 2005;50: 351–63.
16. Shaikh SA, Ta CN. Evaluation and management of herpes zoster ophthalmicus. Am Fam Physician. 2002;66:1723–30.

Chapter 3
Ear Pain and Cerumen Impaction

Michael Marchick

3.1 Introduction

Ear-related complaints are common presenting symptoms in the primary care setting and in emergency departments. Intrinsic ear disease, which is most commonly infectious in etiology, is the most common cause of otalgia in the pediatric population. Secondary, or referred, etiologies predominate in adults [1]. The ear and periauricular tissues receive sensory innervation from cranial nerves V, VII, IX, and X, as well as innervation from cervical roots C2 and C3, leading to a broad differential of conditions which result in referred pain, as listed below [2].

3.2 Differential Diagnosis

3.2.1 Auricular/Periauricular

- Infectious:
  - Acute otitis media
  - Bullous myringitis
  - Cellulitis
  - Chondritis/perichondritis (may also be post-traumatic in etiology)
  - Mastoiditis
  - Otitis externa/malignant otitis externa

M. Marchick, MD
Emergency Medicine, UF Health Shands Hospital, Gainesville, FL, USA
e-mail: marchick@ufl.edu

B. Desai, A. Desai (eds.), *Primary Care for Emergency Physicians*,
DOI 10.1007/978-3-319-44360-7_3

- Cerumen impaction
- Cholesteatoma
- Foreign body
- Keratosis obturans
- Neoplastic
- Relapsing polychondritis
- Traumatic:
  - Acoustic
  - Barotrauma
  - Blunt
  - Penetrating

*3.2.2 Referred (*Most Common Referred Etiologies)*

- Bruxism
- Cervical lymphadenitis
- Cervical spine arthritis
- Dental abscess/caries*
- Eustachian tube dysfunction
- Myofascial pain
- Oropharyngeal/retropharyngeal:
  - Abscess
  - Foreign body
  - Neoplasm
  - Pharyngitis/tonsillitis
- Parotitis
- Posttonsillectomy pain
- Sinusitis
- Temporomandibular joint syndrome*
- Thyroiditis
- Rare but serious presentations – angina, giant cell arteritis, thoracic aneurysms

3.2.3 Facial Neuralgias

- Bell's palsy
- Great auricular neuralgia
- Ramsay Hunt syndrome
- Trigeminal neuralgia

3.3 History

As with any patient presenting with pain, standard questions regarding the context, duration, quality, severity, and progression of discomfort should be elicited, as well as any associated symptoms. Unfortunately, the history is obviously limited in the large proportion of pediatric patients who consist of those presenting with otalgia.

Any history of traumatic injury to the auricle and periauricular areas should also be elicited, including exposure to loud noises such as fireworks. Infectious etiologies are much more likely to cause acute, continuous, but gradually worsening pain than other causes, as well as be associated with a history of fever. A history of brief, lancinating pain may suggest trigeminal neuralgia. Association with chewing may suggest temporomandibular joint disorder, dental abscess, or caries. In older patients, a history of pain worsened with range of motion of the neck could indicate cervical spine arthritis. Associated symptoms suggesting an intrinsic otologic cause include tinnitus, vertigo, hearing loss, aural fullness, and otorrhea. With regard to secondary causes, inquire regarding associated rash, dental pain, and sore throat. Finally, in adults, risk factors for malignancy including smoking or chewing tobacco use and heavy alcohol consumption should be assessed [2].

3.4 Physical Examination

The physical exam should consist of examination not only of the auricle and the post auricular area but the oropharynx, nose, and neck.

First, inspect the auricle for erythema, tenderness, or edema. Increased pain with traction on the pinna or pressure on the tragus suggests otitis externa or other pathology involving the external auditory canal.

Next, evaluate for edema, warmth, and erythema over the mastoid, obliteration of the postauricular fold, as well as displacement of the pinna anteriorly and inferiorly, findings which are consistent with mastoiditis. Particularly in the setting of trauma, examination for Battle's sign (swelling, ecchymosis, and tenderness over the mastoid) should be undertaken, which could suggest an underlying basilar skull fracture.

Cerumen may have to be removed in order to perform a thorough exam, as described below in the section on cerumen impaction. Assess for discharge and narrowing of the external auditory canal associated with otitis externa and malignant otitis externa, and inspect for masses consistent with malignancy or cholesteatoma. Inspect the tympanic membrane for perforation, erythema, bulging, and reduced mobility with insufflation, and evaluate the presence of any effusion behind the tympanic membrane. Opacity of the tympanic membrane, the presence of an air fluid level, compromised tympanic membrane mobility, and otorrhea without evidence of otitis externa all indicate the presence of a middle ear effusion. Note that a previously normal tympanic membrane may take on an erythematous appearance immediately after any attempt at cerumen removal.

If the examination of the ear and periauricular areas is unrevealing, particularly careful evaluation for secondary causes of otalgia should be undertaken. Evaluate the oropharynx for any evidence of swelling or fluctuance consistent with an odontogenic abscess and percuss the teeth. In particular for patients with risk factors for malignancy, carefully evaluate for the presence of any masses. The neck should also be palpated to examine for masses suggestive of malignancy or lymphadenitis. Anterior neck tenderness is suggestive of thyroiditis. Range of motion assessment and palpation of the cervical spine can elicit findings suggesting arthritis or radiculopathy. Palpation of the temporomandibular joint while the patient opens and closes the jaw may reveal tenderness and crepitus associated with temporomandibular joint syndrome. Tenderness in the temporal region suggesting giant cell arteritis in older patients or parotid tenderness and swelling indicative of parotitis should also be assessed.

3.5 Important Intrinsic Etiologies and Their Treatment

3.5.1 Acute Otitis Media

Acute otitis media (AOM) accounts for a significant number of visits to primary care offices and emergency departments and is the most frequent indication for antibiotic prescriptions in children [3]. The peak incidence occurs between the ages of 6–18 months. Daycare attendance, family history of acute otitis media, air pollution (including exposure to tobacco smoke), and lack of breastfeeding are all associated with increased risk for AOM.

AOM is typically preceded by a viral URI. Inflammation and increased respiratory secretions result in obstruction of the eustachian tube. This results in pooling of secretions in the middle ear, providing a milieu amenable to bacterial and/or viral growth. *Streptococcus pneumoniae* (~45 % of bacterial isolates), non-typeable *Haemophilus influenzae* (~45 %), and *Moraxella catarrhalis* (~10 %) are the predominantly implicated bacterial pathogens. *S. pneumoniae* once accounted for a much larger proportion of infections, but the use of polyvalent pneumococcal conjugate vaccine has resulted in a marked decline in its prevalence in middle ear isolates [3]. Respiratory syncytial virus, influenza, and adenoviruses are also associated with the diagnosis.

Otalgia is the most common symptom in patients mature enough to communicate. Fever and malaise may be the only initial clues to the diagnosis in younger patients. A fever > 40 °C implies bacteremia or an alternative source of infection. Diagnosis of acute otitis media requires both evidence of middle ear inflammation and the presence of tympanic membrane bulging or otorrhea not due to otitis externa. Inflammation is commonly manifested as erythema of the tympanic membrane, otalgia, or fever [4]. A bulging, cloudy, or erythematous tympanic membrane, with lack of tympanic membrane mobility on pneumatic otoscopy, is very strongly suggestive of the diagnosis [5].

Adequate analgesia should always be administered. Options include topical agents or oral NSAIDs or acetaminophen. Topical agents can be considered if the tympanic membrane is intact. While immediate initiation of antibiotic therapy was once routine,

recent guidelines have advocated a 24–48 h watchful waiting approach prior to initiation of antibiotics in non-severe cases, as the condition frequently resolves spontaneously in 24–72 h, and there is significant concern for development of antibiotic resistance. Patients generally considered to require immediate treatment include those under 2 years of age, patients with otorrhea, and patients with the presence of bilateral acute otitis media [4, 6]. First-line treatment is amoxicillin 80–90 mg/kg divided bid. In penicillin-allergic individuals who did not experience an urticarial or anaphylactic reaction, a second- or third-generation cephalosporin, such as cefdinir, is appropriate. In individuals with a severe type I hypersensitivity, clindamycin 30–40 mg/kg/day divided tid or azithromycin 10 mg/kg on day 1, followed by 5 mg/kg daily on days 2–5, can be used. With the exception of azithromycin, a treatment duration of 10 days is typically employed for those under age 2, those with recurrent AOM, and those with associated tympanic membrane perforation [4].

Lack of improvement within 48–72 h of initiation of antibiotics signifies treatment failure, which is often the result of infection with a beta-lactamase-producing pathogen. Patients who fail amoxicillin are typically treated with amoxicillin-clavulanate. Alternatively, third-generation cephalosporins, including ceftriaxone with or without the use of clindamycin, are options. Persistent treatment failure should prompt referral to an otolaryngologist [4].

Recognized sequelae of acute otitis media include conductive hearing loss, balance difficulties, tympanic membrane perforation, cholesteatoma formation, and progressive extension of infection to contiguous structure (potentially resulting in mastoiditis, meningitis/intracerebral abscess, and septic lateral or cavernous sinus thrombosis) [5].

3.5.2 Otitis Media with Effusion

Middle ear effusion is a persistent finding in many patients with a recent diagnosis of AOM, regardless of whether antibiotics were initially prescribed, with a prevalence of 70% at 2 weeks and 10% at 3 months [7]. Eustachian tube dysfunction, tobacco abuse, and chronic sinus disease can also result in a chronic effusion. Symptoms can include diminished hearing and a sense of aural fullness. Nonurgent ENT referral is indicated if an effusion has been present for > 3 months, as there is concern for development of permanent hearing impairment in children. Antibiotics, antihistamines, and decongestants are *not* indicated [5].

3.5.3 Otitis Externa

Infection of the external ear canal is commonly referred to as swimmer's ear. Disruption of the protective barrier provided by slightly acidic cerumen and the skin of the external auditory canal allow bacteria to invade. Common scenarios leading to bacterial introduction include prolonged exposure of the canal to moisture (swimmers), earplug/earbud use, and instrumentation of the ear such as with cotton swabs [1]. The most common bacterial pathogens identified are *Pseudomonas aeruginosa*

(38 % of cases), *Staphylococcus epidermis* (9 %), and *Staphylococcus aureus* (8 %). Polymicrobial infection is common; fungal infection is rare [8].

Symptoms include pruritis, otorrhea, hearing loss, and pain and tenderness of the external ear. On physical exam, typical findings include tenderness with traction on the auricle or tragus. The external auditory canal is typically edematous and erythematous, with the presence of debris in the canal.

Treatment of otitis externa includes gentle cleaning of debris from the canal, pain control, and topical therapy to treat inflammation and infection. A variety of topical treatment options exist. Simple acidification of the ear, i.e., with acetic acid, is commonly used in the developing world, as replication of *P. aeruginosa* and *S. aureus* is inhibited at pH < 6. However, there is a concern for ototoxicity if the tympanic membrane is perforated, and this method may be irritating to the inflamed ear canal. Typically in the developed world, a topical antibiotic with activity against *P. aeruginosa* and *S. aureus* is utilized. Steroids have been shown effective in reducing pain and inflammation, and several antibiotic/glucocorticoid preparations are commercially available [5].

Neomycin is an aminoglycoside often prescribed in conjunction with a steroid as first line therapy for acute otitis externa. Advantages include low cost and efficacy similar to other antibiotic/steroid combinations. Due to concern for aminoglycoside ototoxicity if introduced to the middle ear, preparations containing this agent should be avoided if tympanic membrane rupture may be present or in the presence of tympanostomy tubes. Neomycin can also directly cause skin irritation, complicating recovery for affected patients. Fluoroquinolone drops (ofloxacin or ciprofloxacin) are generally preferred due to less concern for ototoxicity and inflammation, although their cost is higher. Ciprofloxacin is also commercially available in combination with dexamethasone and hydrocortisone.

In patients with a severely occluded ear canal, an ear wick moistened with antibiotic drops may need to be placed in order to facilitate antibiotic delivery. Patients should be advised to follow up in several days with an otolaryngologist if there is no improvement in symptoms, as this could indicate acute otitis media with tympanic membrane rupture (in which case oral antibiotics may be appropriate), progression to malignant otitis externa, or need for cleaning of the external ear canal in order to allow appropriate antibiotic delivery. Patients with ear wicks, severe inflammation, and high-risk groups for development of malignant otitis externa should also have close otolaryngology follow-up [1].

Pitfall Failure of acute otitis externa to resolve within 2–3 weeks despite adherence to appropriate treatment strongly suggests the diagnosis of malignant otitis externa.

3.5.4 Malignant Otitis Externa

Extension of otitis externa to the surrounding cartilage, bone, and soft tissues is termed malignant otitis externa (also known as necrotizing otitis externa). Patients at risk include diabetics, the immunocompromised, and the elderly [1]. Historically, > 90 % of all cases have been due to *Pseudomonas*. Recently, however, MRSA has been increasingly associated with the condition [9]. In patients with AIDS and a

CD4 count < 50/mm^3, fungal etiologies become much more common. Symptoms may include foul smelling and/or purulent otorrhea, severe pain, and later in the course, facial nerve paralysis and other cranial nerve palsies. The affected ear will typically be edematous and erythematous, often with granulation tissue in the floor of the external auditory canal. Exposed bone may also be present. The tympanic membrane, when visualized, often appears normal. Pain out of proportion to exam is likely to be present. MRI and CT are the most readily available modalities to establish the diagnosis and its extent, although a technetium bone scan can also be utilized. Empiric treatment should include prompt initiation of an IV antipseudomonal agent with additional coverage for MRSA. The exudate should ideally be cultured prior to initiation of antibiotics. Prompt otolaryngology consultation for consideration of debridement, as well as admission, is indicated [5].

3.5.5 Mastoiditis

Mastoiditis, infection of the air cells in the mastoid process, was once a common sequela of AOM; however the incidence is much lower in the age of antibiotics. Recognized pathogens include *S. aureus*, *S. pneumoniae*, *Streptococcus pyogenes*, *H. influenzae*, *P. aeruginosa*, and enteric gram-negative rods [10]. AOM very frequently coexists with mastoiditis; hence the condition is most commonly observed in children, particularly those under age 2. Presenting symptoms include fever, posterior ear pain, edema, warmth, and erythema posterior to the ear. On exam, anterior/inferior displacement of the pinna and obliteration of the postauricular fold may be appreciated, as well as findings consistent with associated AOM.

CT imaging is typically utilized to confirm the diagnosis. Of note, mastoid effusion is a common finding on CT in patients with acute otitis media and is typically not of clinical significance. Diagnostic findings of mastoiditis include resorption of the bony septae dividing the mastoid air cells, destruction of the mastoid cortex, periosteal thickening and/or disruption, and subperiosteal abscess [5].

Patients with mastoiditis are typically admitted to the hospital with ENT consultation for administration of IV antibiotics. A typical regimen is vancomycin with the addition of a fourth-generation cephalosporin, especially if *P. aeruginosa* is a concern (i.e., recent antibiotic therapy and patients with recurrent AOM). Myringotomy is often performed to assist in tailoring of antibiotic selection. Surgical debridement may be required in the absence of appropriate response to antibiotics [11].

Pitfalls If inadequately treated, mastoiditis can progress to osteomyelitis of the petrous bone (petrositis), resulting in sixth and seventh cranial nerve palsies, permanent hearing impairment, meningitis, brain abscess, and venous sinus thrombosis.

The possibility of masked (subacute) mastoiditis in patients with AOM unresponsive to antibiotics should be considered. In such patients, complications of mastoiditis such as those listed above may arise without classic signs and symptoms of mastoiditis [12].

3.5.6 Foreign Bodies

A variety of foreign bodies can be inserted or make their way into the external ear canal. Beads, insects (particularly cockroaches), pebbles, and beans are among the most commonly cited in the literature [13]. Button batteries [14], live insects, and foreign bodies with potential to penetrate the tympanic membrane should be removed promptly. Insects may be removed by instilling 2 % lidocaine, which will paralyze the insect, as well as provide topical analgesia for the patient. Suctioning of the canal and direct retrieval of the insect are then enabled. For some small objects, irrigation can be considered if the tympanic membrane is intact. Otherwise, removal is dependent on adequate direct visualization and appropriate equipment retrieval, typically alligator forceps or a small suction catheter. The presence of a penetrating foreign body with concern for middle ear damage is an indication for urgent ENT consultation. Referral to an otolaryngologist is indicated if removal attempts are unsuccessful, tympanic membrane perforation occurs (iatrogenic or otherwise), there is preexisting damage to ear structures, or a sharp foreign body is present.

Pitfall Disk batteries should never undergo attempted removal via irrigation due to the risk of burns from a short circuit.

3.5.7 Traumatic Tympanic Membrane Perforation

Traumatic perforation of the tympanic membrane may arise from a blow to the ear (most common), barotrauma, blast injury, or direct trauma. Presenting symptoms include fullness, tinnitus, and hearing loss. Once diagnosed, patients should not undergo cleansing or instrumentation of the external ear canal for at least 2 weeks unless there is visible contamination, in which case careful suctioning may be employed. Patients should be advised that the ear should be kept dry to reduce the likelihood of infection. There is no clear indication for use of systemic or topical antibiotics. Patients should be reassured that within 1 month, the vast majority of ruptures not caused by a blast injury heal spontaneously. The length of time required for healing varies directly with the size of the perforation. Very large perforations (involving 80 % or more of the area of the tympanic membrane) tend not to heal spontaneously. Appropriate supportive care results in improved healing compared with early surgical repair. Thus, urgent referral is generally *not* warranted, as otolaryngologists typically wait 3–6 months for healing before considering repair.

Pitfall Severe vertigo following a ruptured tympanic membrane suggests the presence of a perilymphatic fistula or rupture of the round window. In this scenario, urgent ENT consultation is necessary [15].

3.5.8 Cerumen Impaction

Cerumen impaction is a common issue facing clinicians, with a prevalence of approximately 10% in children (in which case limited evaluation of the tympanic membrane is possible), and much higher rates ever noted in elderly and developmentally delayed patients [16]. The condition is defined as accumulation of cerumen resulting in symptoms (including hearing loss, vertigo, or pain) and/or inability to adequately examine the ear [17]. A variety of potential means of removal exist, including mechanical methods, irrigation, and the use of cerumenolytic/lubricating agents. Patient-specific factors, including the integrity of the tympanic membrane, history of immunocompromising conditions including diabetes mellitus, the use of anticoagulants, ear canal stenosis, and prior surgical history should influence the choice of the method utilized. Removal methods may be combined, such as the use of cerumenolytics prior to irrigation.

Mechanical removal can be achieved with a variety of instruments including curettes, loops, spoons, alligator forceps, and suction tips. Adequate visualization is of the utmost importance to minimize complications and may be obtained using an otoscope with speculum, headlight, or binocular microscope, although the latter is rarely available in an ED or primary care office. Manual removal is often preferred for patients with immunocompromising conditions, a perforated tympanic membrane, or a history of ear surgeries.

Irrigation using tap water or saline may be performed using a dedicated ear syringe, an oral jet irrigator, an 18 gauge IV catheter, or one of several commercial devices designed for this purpose. The irrigation device should not be inserted beyond the outer third of the external auditory canal, and the irrigant should be directed toward the superior aspect of the canal to minimize direct pressure on the tympanic membrane. The use of body-temperature liquid can help reduce the incidence of vertigo due to caloric stimulation. Patients with a history of previous ear surgeries, which can result in atrophic areas of the tympanic membrane, are at increased risk of perforation due to the pressure inherent to irrigation attempts [17].

Cerumenolytics are divided into oil based, water based, and non-oil or water based, as listed below. While cerumenolytics are more effective than no treatment, there is no evidence to suggest superiority of one preparation versus another [18]. Most preparations are applied for a period of 15–30 min, after which attempts at removal may be made.

3.5.9 Cerumenolytic Agents

Oil Based

- Almond oil
- Arachis oil
- Mineral oil/liquid petroleum

Water Based

- Acetic acid
- Docusate sodium (Colace)
- Hydrogen peroxide
- Sodium bicarbonate
- Sterile saline
- Triethanolamine polypeptide oleate (Cerumenex)

Non-oil or Water Based

- Choline salicylate, glycerin (Audax)
- Carbamide peroxide (Debrox, Murine)

Pitfalls Any cerumen removal method may lead to pain, tympanic membrane perforation, vertigo, and otitis externa.

Irrigation of the canal of a patient with a non-intact tympanic membrane may lead to inner ear infection, caloric vertigo, and ototoxic hearing loss.

Tap water canal irrigation of diabetics and the immunocompromised – particularly if an abrasion is present – may result in malignant otitis externa. Consider the use of acidifying eardrops post-procedure if this method is utilized, and provide explicit return precautions for the development of symptoms of otitis externa.

The use of cotton-tipped applicators to attempt removal of cerumen often leads to worsened impaction and can result in infection and rupture of the tympanic membrane. Patients should be counseled to avoid this technique.

3.5.10 Ramsay Hunt Syndrome

Also known as herpes zoster oticus, Ramsay Hunt syndrome refers to the reactivation of *Varicella zoster virus* resulting in unilateral facial paralysis, ear pain, and vesicles affecting the ear canal and auricle. Cranial nerve VIII can also be affected, resulting in varying degrees of hearing loss and vertigo. While systemic antivirals such as valacyclovir in conjunction with systemic glucocorticoids are typically prescribed, there exists a paucity of data regarding their efficacy [19, 20].

References

1. Neilan RE, Roland PS. Otalgia. Med Clin N Am. 2010;94:961–71.
2. Ely JW, Hansen MR, Clark EC. Diagnosis of ear pain. Am Fam Physician. 2008;77(5):621–8.
3. Coker TR, Chan LS, Newberry SJ, Limbos MA, Suttorp MJ, Shekelle PG, Takata GS. Diagnosis, microbial epidemiology, and antibiotic treatment of acute otitis media in children: a systematic review. JAMA. 2010;304(19):2161–9.

4. Lieberthal AS, Carroll AE, Chonmaitree T, Ganiats TG, Hoberman A, et al. The diagnosis and management of acute otitis media. Pediatrics. 2013;131(3):e964–99.
5. Conover K. Earache. Emerg Med Clin N Am. 2013;31:413–42.
6. Venekamp RP, Sanders SL, Glasziou PP, Del Mar CB, Rovers MM. Antibiotics for acute otitis media in children. Cochrane Database Syst Rev. 2015;6:CD000219.
7. Teele DW, Klein JO, Rosner BA. Epidemiology of otitis media in children. Ann Otol Rhinol Laryngol. 1980;89(3 Pt 2):5–6.
8. Roland PS, Stroman DW. Microbiology of acute otitis externa. Laryngoscope. 2002;112(7 Pt 1):1166–77.
9. Hobson CE, Moy JD, Byers KE, Raz Y, Hirsch BE, McCall AA. Malignant otitis externa: evolving pathogens and implications for diagnosis and treatment. Otolaryngol Head Neck Surg. 2014;151(1):112–6.
10. Luntz M, Brodsky A, Nusem S, Kronenberg J, Keren G, et al. Acute mastoiditis – the antibiotic era: a multicenter study. Int J Pediatr Otorhinolaryngol. 2001;57:1–9.
11. Geva A, Oestreicher-Kedem Y, Fishman G, Landsberg R, DeRowe A. Conservative management of acute mastoiditis in children. Int J Pediatr Otorhinolaryngol. 2008;72:629–34.
12. Holt GR, Gates GA. Masked mastoiditis. Laryngoscope. 1983;93:1034–7.
13. Schulze SL, Kerschner J, Beste D. Pediatric external auditory canal foreign bodies: a review of 698 cases. Otolaryngol Head Neck Surg. 2002;127(1):73–8.
14. Kavanagh KT, Litovitz T. Miniature battery foreign bodies in auditory and nasal cavities. JAMA. 1986;255(11):1470–2.
15. Maitland CG. Perilymphatic fistula. Curr Neurol Neurosci. 2001;1:486–91.
16. Roeser RJ, Ballachanda BB. Physiology, pathophysiology, and anthropology/epidemiology of human ear canal secretions. J Am Acad Audiol. 1997;8:391–400.
17. Roland PS, Smith TL, Schwartz SR, Rosenfeld RM, Ballachanda B, et al. Clinical practice guideline: cerumen impaction. Otolaryngol Head Neck Surg. 2008;139(3 S2):S1–21.
18. Burton MJ, Dorée CJ. Ear drops for the removal of ear wax. Cochrane Database Syst Rev. 2003;3:CD004400.
19. Uscategui T, Dorée C, Chamberlain IJ, Burton MJ. Antiviral therapy for Ramsay Hunt syndrome (herpes zoster oticus with facial palsy) in adults. Cochrane Database Syst Rev. 2008;(4):CD006851.
20. de Ru JA, van Benthem PP. Combination therapy is preferable for patients with Ramsay Hunt syndrome. Otol Neurotol. 2011;32:852–5.

Chapter 4
Sore Throat, Dental Pain, and Other Oral Issues

Michael Marchick

4.1 Introduction

Chief complaints such as "sore throat" and "dental pain" are some of the most common encountered by primary care and emergency physicians. In the vast majority of cases, the diagnosis is quite benign, which can lull the clinician into a false sense of security when evaluating patients with these complaints. However, the differential ranges from routine self-limited etiologies to immediately life-threatening conditions. Furthermore, the availability of emergent dental consultation is often quite limited during much of the week, while dental emergencies occur continuously. Therefore, the clinician must possess a solid knowledge base with respect to the evaluation and management of these conditions.

4.2 History

The history obtained in patients presenting with oropharyngeal complaints should rapidly assess for any red flags for impending respiratory failure. A change in voice (particularly a "hot potato" voice), dyspnea, submandibular swelling, and rapidly worsening symptoms are red flags for a potential obstructive process such as a deep space infection that may mandate immediate intervention.

Once immediate evidence of an obstructive process has been excluded, further historical detail can be obtained to narrow the differential. In addition to the standard elements of the patient history that should be elicited for a complaint of pain in any region of the body, assess for associated symptoms including fever, malaise, congestion,

M. Marchick, MD
Emergency Medicine, UF Health Shands Hospital, Gainesville, FL, USA
e-mail: marchick@ufl.edu

B. Desai, A. Desai (eds.), *Primary Care for Emergency Physicians*,
DOI 10.1007/978-3-319-44360-7_4

rhinorrhea, and cough. The presence of any urogenital symptoms is relevant if there exists concern for gonorrheal pharyngitis or oral herpes simplex infection.

Past medical history should focus on immunocompromising conditions (including splenectomy), which may predispose to rapid propagation of infection to a potentially life-threatening process. If oral abscess drainage is being considered, ask about any history of cardiac conditions such as a valve replacement or congenital defect that requires endocarditis prophylaxis. Immunization history is of importance when epiglottitis and diphtheria are in the differential. Finally, assess for risks factors for the development of oropharyngeal cancer, including past or present use of chewing tobacco and/or smoking, alcohol abuse, family history of malignancies, and occupational exposures.

4.3 Physical

The initial step in the physical examination of any patient with symptoms affecting the oropharynx should be evaluation for any evidence of respiratory distress (tripod posture, cyanosis, inability to handle secretions), which will require immediate securing of the airway and ventilator support due to the potential for a progressive obstructive process. Following this, a systematic approach to the physical exam should be employed:

- External exam:
  - "Hot potato" voice (*red flag): deep space infections
  - Submandibular swelling (*red flag): Ludwig's angina
  - Trismus (*red flag): deep space infections
  - Cervical lymphadenopathy: numerous infectious/inflammatory processes, malignancy
  - Facial asymmetry: odontogenic infection/abscess, sialoadenitis
  - Lip swelling: if atraumatic (*red flag) – angioedema; traumatic – hematoma
- Internal exam:
  - Posterior oropharynx:
    - Tonsillar asymmetry (*red flag): peritonsillar abscess
    - Uvular edema (*red flag): uvulitis
    - Pseudomembrane (*red flag): diphtheria
    - Erythema
    - Exudates
  - Tongue:
    - Protrusion/displacement (*red flag): Ludwig's angina, angioedema
    - Swelling (*red flag): angioedema
    - Discoloration

  - Teeth/gums:
    - Caries
    - Dental fractures
    - Tenderness: tenderness of teeth to percussion suggests pulpitis/abscess
    - Tenderness of gums
    - Fluctuance
  - Masses (*red flag): malignancy
- Ear exam:
  - Evaluate for acute otitis media, otitis externa, and other potential sources of referred pain

4.4 Specific Etiologies

4.4.1 Sore Throat

Viral Pharyngitis

- Viral pharyngitis is the most common cause of sore throat.
- Symptoms are typically associated with a progression of other upper respiratory infection symptoms including congestion, rhinorrhea, and cough.
- Physical exam findings are of limited utility in distinguishing viral pharyngitis from a bacterial etiology.
- A variety of viral pathogens have been implicated, including influenza, Epstein-Barr (see below), cytomegalovirus, rhinovirus, and adenoviruses. In the vast majority of cases, identification of a specific etiology is unnecessary, as there is no specific treatment and the illness is self-limited.
- If influenza is suspected and the patient has been symptomatic for less than 48 h, consider treatment with oseltamivir.
- Testing may also be considered for mononucleosis if suspected clinically (see below).
- In patients at high risk for human immunodeficiency virus infection, consider the possibility of the acute retroviral syndrome.
- Otherwise, in the vast majority of patients, treatment is purely supportive.
- Nonsteroidal anti-inflammatory drugs and acetaminophen are mainstays of therapy.
- Adult patients can alternatively use aspirin-containing products.
- Formal studies regarding the efficacy of topical treatments are lacking.
- Numerous over-the-counter preparations are available, including lozenges containing benzocaine or menthol and sprays containing phenol.
- Patients should be counseled to avoid grossly excessive use of products containing benzocaine due to the theoretical possibility of inducing methemoglobinemia.
- All patients should be encouraged to maintain adequate hydration [1].

Streptococcal Pharyngitis

- Streptococcal pharyngitis is a common, easily treatable etiology of sore throat.
- The incidence is highest in children ages 5–15, although adults are also commonly infected.
- In addition to sore throat and dysphagia, patients with strep pharyngitis also commonly complain of fever, headache, and abdominal pain.
- The presence of cough, rhinorrhea, congestion, and conjunctivitis argue against the diagnosis.
- On exam, palatal petechiae, pharyngeal erythema, tonsillar exudates, tender anterior cervical lymphadenopathy, and scarlet fever (an erythematous, finely papular rash which spares the face) may be noted [2].

4.4.2 Centor Criteria

- The Centor criteria are a widely used scoring system to estimate the probability of a diagnosis of streptococcal pharyngitis.
- One point is assigned for the presence of each of the following factors:
  - Fever >38 °C
  - Tender anterior cervical lymph nodes
  - Presence of tonsillar exudates
  - Absence of cough
- A score of 0 is associated with a likelihood of streptococcal pharyngitis of approximately 2.5 %.
- A score of 4 has been associated with a >50 % probability of infection.
- Guidelines issued by several professional societies concur that in a patient with a Centor score of 0, no further testing or treatment is necessary.
- Many authorities agree that in those with a score of 1 or 2, a rapid strep test with subsequent culture if negative is reasonable, while the use of additional testing in those with scores of 3–4 is controversial [1]:
  - The sensitivity of rapid streptococcal testing is quoted between 70 and 90 %, with excellent specificity [2].
  - Patients with negative rapid strep testing should have a throat culture obtained.
- Without treatment, streptococcal pharyngitis is self-limiting, with improvement in symptoms expected within 1 week.
- However, as streptococcal pharyngitis can result in rheumatic fever and post-streptococcal glomerulonephritis, all US guidelines call for treatment in patients with a positive rapid strep test or culture:

  - Acceptable treatment regimens include either oral penicillin VK for 10 days or one-time IM benzathine penicillin G
  - Cephalosporins, azithromycin, erythromycin, and clindamycin are acceptable alternatives in penicillin-allergic patients, depending on the severity of the reaction
- Supportive care for sore throat, as detailed above for viral pharyngitis, is also appropriate.

Mononucleosis

- Infectious mononucleosis is caused by the Epstein-Barr virus, which is transmitted through oral secretions.
- Children as well as adults can be affected, with younger patients typically having a less severe course [3].
- The condition typically presents with flu-like symptoms, including prominent sore throat, fatigue, headache, and fever.
- A prolonged duration of symptoms is a particular clue to the diagnosis, which can be difficult to differentiate from other causes of sore throat.
- Physical exam findings include marked anterior and posterior cervical lymphadenopathy, hepatomegaly, and splenomegaly.
- A maculopapular rash is sometimes observed, typically following the use of an antibiotic due to presumed earlier diagnosis of bacterial pharyngitis.
- The monospot test is commonly used to confirm the diagnosis.
- However, as this test relies on the presence of antibodies to Epstein-Barr virus, false-negative testing can be observed early in the disease course.
- Treatment is supportive, with the use of NSAIDs or acetaminophen for fever and pain control, the mainstay of therapy
- To reduce the likelihood of splenic rupture, all patients should be counseled to abstain from sports until their symptoms have fully resolved.

Epiglottitis

- Epiglottitis was once predominantly a disease of children caused primarily by *Haemophilus influenzae* type b.
- Due to immunizations, a shift in the responsible pathogens has occurred, with *Staphylococcus aureus*, *Streptococcus pneumoniae*, and *Streptococcus pyogenes*, as well as anaerobes now commonly implicated.
- Along with this, the burden of disease has shifted primarily to adults.
- Classically, pediatric patients presented with a triad of drooling, stridor, and tripod positioning, a presentation that has become much less common with the epidemiologic shift.
- Recent case series have demonstrated that painful or difficult swallowing and voice changes are the most common presenting symptoms [4].
- Fortunately, as a consequence of adult patients' larger airways, the need for airway protection in those with epiglottitis has decreased.

- Patients with respiratory distress or stridor associated with a presumed or confirmed diagnosis should be intubated, preferably in an operative room setting or by awake fiberoptic intubation.
- Clinicians should remain vigilant regarding the need for potential airway intervention in others, particularly those with:
  - Reported dyspnea
  - Rapid onset of symptoms
  - Tachycardia
  - Tachypnea [4]
- Diagnosis can be confirmed by lateral soft tissue X-rays showing an enlarged epiglottis ("thumbprint sign") or CT.
- If such imaging is pursued, it must be done in a closely supervised manner.
- Early otolaryngological consultation and empiric antibiotics should be initiated.
- Typical regimens include ampicillin/sulbactam or a third-generation cephalosporin (such as ceftriaxone) with vancomycin.

Peritonsillar Abscess

- Tonsillitis and other oropharyngeal infections can occasionally progress to abscess formation posterior to the tonsil.
- Patients present with sore throat and fever and may also have voice changes, trismus, and drooling.
- On exam, inferomedial displacement of the affected tonsil results in uvular deviation away from the abscess.
- Group A streptococci are the most frequent cause; however, many infections are polymicrobial, with anaerobes frequently identified in cultured aspirates.
- If the diagnosis is unclear after physical exam, CT and ultrasound (in the hands of an experienced operator) have been shown to have excellent sensitivity and specificity for the diagnosis.
- Ultrasound can also be useful in assisting drainage at the bedside.
- Treatment consists of drainage (typically by needle aspiration), antibiotics, and pain control.
- Typically antibiotic regimens include amoxicillin/clavulanate 875 mg bid or clindamycin 300 mg four times daily:
  - The role of steroids is controversial.
- Admission is appropriate for patients that:
  - Cannot tolerate PO
  - Have intractable pain
  - Have immunocompromising conditions
  - Have concern for ability to maintain their airway [1, 5, 6]

Retropharyngeal Abscess

- Retropharyngeal abscesses are most common in children under 5 years old.
- Adult patients typically have a history of an immunocompromising conditions or have recently undergone surgery or experienced trauma to the oropharynx.
- Symptoms range from fever and neck pain to neck stiffness, a "hot potato voice," and respiratory difficulty.
- The symptoms are generally quite nonspecific – and unfortunately can be rapidly progressive – therefore a high index of suspicion is warranted.
- CT is the diagnostic test of choice.
- Infection is typically polymicrobial, including anaerobic bacteria.
- Treatment includes airway protection as needed, IV antibiotics (ampicillin/sulbactam or clindamycin, with vancomycin added if concern for MRSA), and early surgical consultation for consideration of drainage.
- Delay in diagnosis can lead to extension to contiguous structures, resulting in airway obstruction, sepsis, mediastinitis, and neurologic sequelae.

Diphtheria

- As a result of vaccination, diphtheria is exceedingly rare in the developed world.
- However, it is important to consider this disease in unvaccinated patients from underdeveloped nations.
- Symptoms are similar to many more benign viral illnesses, including gradual onset of sore throat, low-grade fever, malaise, and lymphadenopathy.
- The presence of an adherent gray pseudomembrane in the throat is pathognomonic, but not universally present.
- Treatment includes airway protection, diphtheria antitoxin, and penicillin.

4.4.3 Salivary Gland Disease

- Three paired sets of glands (parotid, submandibular, and sublingual) are largely responsible for saliva production.
- The parotid gland is drained by Stensen's duct, while Wharton's duct drains the submandibular gland, as well as some of the secretions of the sublingual gland.
- Salivary duct stones (sialolithiasis) most frequently occur in Wharton's duct.
- Patients present with abrupt onset of pain, typically exacerbated by eating.
- Swelling of the associated gland is also commonly noted.
- The presence of purulent drainage signifies a concomitant bacterial infection. A palpable or visible stone may be present, making the diagnosis obvious. In other cases, imaging with CT or ultrasound is necessary to clarify the diagnosis. Management includes massage/milking of the affected gland and duct, analgesia, and use of sialogogues such as hard candy and acidic juice. Prompt otolaryngology follow-up is also indicated [7].
- Inflammation of a salivary duct is termed sialoadenitis.

4.4.4 Parotitis

- The parotid gland is most commonly affected, and the resulting process referred to as parotitis.
- Bacterial parotitis is most commonly due to *S. aureus*, although polymicrobial infection also involving *Streptococcus viridans* and anaerobes is not unusual.
- Dehydration is a very common predisposing factor.
- Patients typically present with painful unilateral swelling of the cheek.
- Fever is common, and patients may appear quite ill. Bilateral involvement can occur in up to 25 % of patients.
- Purulent drainage from Wharton's duct clinches the diagnosis.
- In other patients, ultrasonography or CT can be considered (These modalities can also assess for the presence of any abscess or stone).
- Treatment includes antibiotics with coverage of *S. aureus* and anaerobes.
- Patients with toxic appearance, significant comorbidities, and poor access to otolaryngology follow-up should be admitted.
- Others can be discharged with close follow-up and an oral agent such as clindamycin.
- Amoxicillin/clavulanate may be employed if methicillin-resistant *S. aureus* is not a particular concern.
- All patients should receive hydration, analgesia, sialogogues, and parotid massage.
- Any medications that could be contributing to salivary stasis (e.g., anticholinergics) should be discontinued if possible.
- Viral parotitis is commonly associated with mumps, but influenza, parainfluenza, and other viral pathogens are also responsible.
- In contrast to bacterial parotitis, patients typically present with bilateral parotid pain and swelling which develop following a several day period of influenza-like symptoms.
- The presence of facial erythema or warmth or purulent drainage from Stensen's duct is consistent with a bacterial, rather than viral, etiology.
- Treatment of viral parotitis is largely supportive and consists of analgesics and hydration.
- If there is any uncertainty as to whether the patient may have a bacterial etiology, antibiotics should be considered [5–7].

4.4.5 Dental Issues

Minor Dental Trauma

- The Ellis classification is used to describe the depth of a dental fracture.
- Ellis I fractures involve only the enamel and will not produce pain:
  - Patients can be referred to a dentist for further care if appearance is a concern.

- Ellis II fractures involve the enamel and the yellow dentin:
  - Pain may be present.
  - Treatment consists of analgesia and application of calcium hydroxide paste over the affected area.
  - Patients should be referred to a dentist promptly and advised to avoid any trauma to the area (including chewing), until evaluated by a dentist
- Ellis III fractures involve the enamel, dentin, and pulp and are tooth threatening:
  - The presence of a red area in the affected tooth is the hallmark of diagnosis.
  - Patients typically report severe pain.
  - Calcium hydroxide should be applied and the patient referred to a dentist within 48 h.
- Avulsed permanent teeth should be reimplanted as soon as possible as the likelihood of healing and functionality long term is inversely proportional to time out of socket.
  - To perform this, gently rinse the tooth – *do not wipe the root* – and place into the socket taking care only to handle the tooth by the crown.
  - Stabilization of the affected tooth must then be achieved, either through a temporary splint performed in the ED or by a dentist or oral maxillofacial surgeon as soon as possible following reimplantation.
  - A soft diet is an imperative for such patients until they are cleared to advance by a dentist or oral maxillofacial surgeon [5, 6].
  - Evaluate for the possibility of an aspirated tooth with a chest X-ray in patients with a newly missing tooth that cannot be located, as a post-obstructive pneumonia can result.
  - Bronchoscopic retrieval is necessary if an aspirated tooth is identified.

Atraumatic Tooth Disease: Dental Caries and Pulpitis

- Erosion of the enamel results in development of dental caries.
- If left untreated, the erosion may extend to the dentin and the pulp, causing pain, which is typically worsened by heat or cold.
- This erosion also enables invasion of bacteria from the oropharynx.
- Inflammation of the pulp resulting from this process is termed pulpitis and can be divided into reversible (acute) and irreversible (chronic) forms.
- Reversible pulpitis is typically associated with brief episodes of pain, while the pain of irreversible pulpitis is typically much longer in duration (although it tends to be less severe).
- All patients should be provided with adequate analgesia, including the offering of local anesthesia with a long-acting agent such as bupivacaine.
- NSAIDs and acetaminophen are first-line therapy for dental pain.
- If refractory, brief, sparing courses of opiates may be considered.
- Systemic antibiotic are often prescribed and should be prescribed if clear evidence of infection is present (e.g., an abscess as described below) [5–7]:
  - Otherwise, the utility of antibiotics is unclear [8, 9].

- If utilized, antibiotics should cover *Streptococcus mutans* and other oral anaerobes:
  - Penicillin VK 500 mg four times daily and amoxicillin 500 mg four times daily are appropriate first-line choices (clindamycin or erythromycin may be used in penicillin-allergic patients).
- Prompt dental referral is appropriate for all patients with pulpitis. Definitive treatment of irreversible pulpitis is either extraction or root canal therapy.

Periapical Abscess
- If pulpitis is left untreated, bacteria may invade the alveolar bone and contiguous tissues, forming a periapical abscess.
- Patients may present with pain, swelling, or complaint of drainage of pus (with its associated foul taste).
- Treatment includes immediate incision and drainage, irrigation, as well as antibiotics as described above.
- Patients should be referred to a dentist for root canal therapy or extraction.

Ludwig's Angina
- Ludwig's angina, cellulitis of the submandibular space, may result if the above processes occur in the region of the second and third molar, allowing bacterial invasion of the submandibular space (extension of deep space infections of the oropharynx can also cause Ludwig's angina).
- Patients typically present with pain, edema, and a hoarse voice.
- Submandibular swelling, which can precipitously progress to tongue elevation resulting in airway occlusion, is noted on exam.
- Initial management includes immediate airway assessment and securing of a definitive airway if indicated.
- Close reevaluation is imperative in patients without an established definitive airway.
- Prompt oral maxillofacial surgery consultation is necessary, as is administration of parenteral antibiotics to cover the typically polymicrobial infection:
  - Ampicillin/sulbactam and clindamycin are appropriate choices [5–7].

4.4.5.1 Periodontal Disease

Gingivitis
- Gingivitis, inflammation of the gingiva, is a reversible inflammatory response to irritants including plaque.
- Edema, erythema, and occasional bleeding or discharge may be present.
- Patients should be referred to a dentist for debridement.
- In the interim, patients should be counseled on the role of improved oral hygiene.

Periodontitis

- If untreated, gingivitis can progress to periodontitis, an irreversible inflammatory condition that can result in destruction of the periodontal ligament and alveolar bone.
- As a result, patients may present with loosened/lost teeth.
- Prompt referral to a dentist is indicated.

Periodontal Abscesses

- With progression of periodontitis, an abscess may form in the areas of eroded gum and alveolar bone tissue; such abscesses are termed periodontal abscesses and differ from periapical abscesses in that the adjacent tooth may be otherwise healthy
- Incision and drainage as well as irrigation of such abscesses should be performed at the time of diagnosis.
- Patients are typically prescribed a short course of antibiotics active against typical oral flora, as described above.
- All patients should be referred promptly for dental follow-up for consideration of further debridement of tissue, as well as provided with adequate analgesia.

Necrotizing Ulcerative Gingivitis

- Necrotizing ulcerative gingivitis (Vincent's disease, trench mouth) refers to a direct bacterial invasion of gingival tissue, rather than the simple inflammation characteristic of gingivitis.
- Risk factors include:
  - Malnutrition
  - Smoking
  - Emotional stress
  - Immunocompromise (particularly HIV)
  - Poor hygiene
- Patients commonly present with rapid onset of pain, as well as foul-smelling breath and a metallic taste, and may also have associated fever and lymphadenopathy.
- On exam, edematous interdental gingival papillae with ulceration and bleeding are noted.
- A friable pseudomembrane may also be present.
- Treatment consists of improved oral hygiene, antiseptic mouthwash (e.g., 3% hydrogen peroxide, chlorhexidine), systemic analgesics, and topical anesthetics (e.g., viscous lidocaine).
- If systemic symptoms are present, oral antibiotics should also be prescribed.
- Patients should be referred to a dentist for further care [5–7].

Pericoronitis

- Inflammation of the gingival tissue overlying an impacted tooth crown (typically the third molar) is termed pericoronitis.
- Due to proximity to vital structures, left untreated, the condition has the potential to become life-threatening.
- Pain is often severe and is worsened by chewing and mouth opening.
- Trismus may be present. Warm saline irrigation, rinses with antiseptic mouthwash and analgesics, should be provided.
- If symptoms are severe, or there are signs of systemic illness, antibiotics should be provided.
- If an abscess is present, careful incision and drainage should be performed.
- Patients should be referred to an oral maxillofacial surgeon for extraction of the offending molar.

4.4.6 Post-extraction Pain

- Pain in the immediate 1–2 days following dental extraction is common and ideally treated with systemic analgesics such as acetaminophen, NSAIDs, or opiates, as well as application of ice packs. An opioid may be considered for breakthrough pain.
- Alveolar osteitis, commonly referred to as dry socket, typically develops 3–4 days post-extraction if the blood clot at the site of the extraction is displaced.
- A localized infection may occur at the site:
  - Pain is typically abrupt in onset and quite severe.
- Following local anesthesia, management typically consists of gentle irrigation followed by packing of the site with gauze soaked in eugenol or one of several commercially available dressings designed for this indication.
- Patients should be discharged with oral analgesics and follow-up with their dentist within 24 h.
- Antibiotics active against oral flora are also typically provided.

References

1. Cirilli AR. Emergency evaluation and management of the sore throat. Emerg Med Clin N Am. 2013;31:501–15.
2. Wessels MR. Clinical practice: streptococcal pharyngitis. N Engl J Med. 2011;364:648–55.
3. Son KH, Shin MY. Clinical features of Epstein-Barr virus-associated infectious mononucleosis in hospitalized Korean children. Korean J Pediatr. 2011;54:409–13.

4. Guardiani E, Bliss M, Harley E. Supraglottitis in the era following widespread immunization against Haemophilus influenzae type B: evolving principles in diagnosis and management. Laryngoscope. 2010;2010(120):2183–8.
5. Marx JA, Hockberger RS, Walls RM. Rosen's emergency medicine – concepts and clinical practice. 8th ed. Philadelphia: Elsevier Saunders; 2013.
6. Tintinalli JE, Stapczynski J, Ma O, Yealy DM, Meckler GD, Cline DM, editors. Tintinalli's emergency medicine: a comprehensive study guide. 8th ed. New York: McGraw-Hill; 2016.
7. Armstrong MA. Salivary gland emergencies. Emerg Med Clin N Am. 2013;31:481–99.
8. Hodgdon A. Dental and related infections. Emerg Med Clin N Am. 2013;31:465–80.
9. Agnihotry A, Fedorowicz Z, van Zuuren EJ, Farman AG, Al-Langawi JH. Antibiotic use for irreversible pulpitis. Cochrane Database Syst Rev. 2016;(2):CD004969.

Chapter 5
Cough, Cold, and Congestion

Desmond Fitzpatrick and Hasan Rasheed

5.1 Introduction

The common cold is a benign, self-limited, acute viral infection with associated symptoms of sneezing, rhinorrhea, nasal congestion, cough, and malaise. It is the most frequent acute illness in the industrialized world [1]. Adults typically have two to three episodes of illness yearly while children can have up to five [2]. The common cold is typically caused by viruses (including rhinovirus, RSV, coronavirus, and others), and often, no infecting organism is detected [2].

The differential diagnosis for cough, cold, and congestion is broad: ranging from non-emergent causes such as rhinitis to life-threatening illnesses such as pulmonary embolism. The primary objective when evaluating patients who have acute cough complaints is often to exclude pneumonia [3]. However, the goal of the emergency physician is to differentiate non-emergent causes from emergencies such as pulmonary embolism, CHF exacerbation, COPD exacerbation, etc. A thorough history and physical examination are important to guide diagnosis, and vital sign abnormalities can often distinguish these illnesses. The American College of Chest Physicians has defined three categories of cough based on duration. Acute cough lasts less than 3 weeks, while a subacute cough lasts 3–8 weeks, and a chronic cough lasts more than 8 weeks [4].

Again, careful assessment is necessary as misidentification can lead to inappropriate discharge of potentially lethal conditions.

D. Fitzpatrick, MD (✉)
Department of Emergency Medicine, University of Florida-Gainesville, Gainesville, FL, USA
e-mail: defitzpatrick@ufl.edu

H. Rasheed, MD
Department of Emergency Medicine, Shands at University of Florida Health, Gainesville, FL, USA
e-mail: rasheedh@ufl.edu

B. Desai, A. Desai (eds.), *Primary Care for Emergency Physicians*,
DOI 10.1007/978-3-319-44360-7_5

5.2 Risk Factors for Cough, Cold, and Congestion

- Transmission of common cold occurs most through hand-to-hand contact.
- Malnutrition.
- Cigarette smoking.
- Immunodeficiency.
- Extremes of age.

5.3 Differential Diagnosis [3]

- *Acute*
  - Bacterial sinusitis
  - Bronchitis:
    - 1–5 days of fevers, malaise, and myalgias followed by persistent cough, phlegm production, and possible wheezing lasting 1–3 weeks
  - Influenza:
    - Cough + sudden onset of high fever (>101 F), headache, myalgias, and fatigue
  - Rhinitis
  - Pharyngitis (viral)
  - Asthma exacerbation
- *Don't miss*
  - Pneumonia
  - Pulmonary embolism
  - COPD exacerbation
  - CHF
  - Scarlet fever
  - Acute epiglottitis or supraglottic inflammation
  - Peritonsillar abscess
  - Gonococcal pharyngitis
  - Kawasaki disease
  - Diphtheria
- *Chronic*
  - *Gastrointestinal:*
    - GERD
  - *Pulmonary:*
    - COPD
    - Smoking

    - Cough variant asthma
    - UACS – upper airway cough syndrome
    - Post-viral cough
  - *Drug induced:*
    - ACE inhibitor
    - Cocaine
    - Methamphetamines
    - Beta-blockers
    - Chlorpromazine
    - NSAIDS, aspirin, and OCPs

5.4 Common Complaints and Red Flags* of Chronic CCC [(*) = Common]

- Cough
- Congestion and/or rhinorrhea
- Conjunctivitis
- Fever – uncommon in adults but common in children
- Hemoptysis*
- Malaise
- Myalgias
- Nausea/vomiting
- Pleuritic chest pain
- Recurrent infections*
- Severe chest pain*
- Sore throat
- Syncope*
- Weight loss*

5.5 History

When evaluating a patient with this complaint, the emergency physician can use details from patient history to differentiate common cold from conditions that require antibiotics as well as emergent conditions.

Important historical features to elicit include:

- Duration of symptoms:
  - Acute vs. subacute vs. chronic
- The presence of potential secondary factors (correlate with past medical history) [* = red flags]:

  - Acute medical complaints (or acute exacerbations of chronic medical conditions:
    - Headache
    - Shortness of breath (*)
    - Chest pain (*)
    - Signs and symptoms of diabetes mellitus (*):
      - Polyuria/polydipsia
    - Abdominal pain or discomfort (*)
- Use of medications or drugs (including tobacco and alcohol)
  - Pitfall: not inquiring about over-the-counter medications which may contain stimulants

5.6 Duration of Symptoms [2]

- *Acute cough* (*<3 weeks*):
  - Most commonly seen with common cold but also seen in emergencies such as pneumonia, CHF exacerbation, COPD exacerbation, and pulmonary embolism.
  - The most important step in evaluation of acute cough is for the emergency physician to differentiate between benign and serious conditions.
- *Subacute cough* (*3–8 weeks*):
  - Most often follow an upper respiratory infection.
  - If not, postinfectious, should be treated as chronic cough.
  - Usually postinfectious cough that is caused by postnasal drip, upper airway irritation, and mucus accumulation of bronchial hyperresponsiveness due to asthma.
  - Consider allergen/irritant exposure, pneumonia, or chronic bronchitis exacerbation.
  - Antitussives can be used when necessary.
- *Chronic cough (>8 weeks):*
  - Can have multiple causes including upper airway cough syndrome (UACS), asthma, non-asthmatic eosinophilic bronchitis, and GERD.
  - Important to optimize therapy for each diagnosis and check compliance with treatment and maintenance of all effective therapies.
  - Further outpatient investigations will likely be needed for management.

5.7 Physical Exam

The physical examination should focus on potential emergency conditions for the reported cough, cold, and congestion.

- Vital sign assessment:
  - The presence of fever or hypothermia should alert the physician to a possible infectious etiology.
  - Tachycardia may have multiple etiologies including:
    - An appropriate response to fever
    - The potential use of medications or drugs, including over-the-counter medications and illicit substances
  - Pulse oximetry:
    - May alert the clinician to the presence of an underlying pulmonary pathology
- Neurologic assessment should focus on:
  - Level of alertness
- Head, eyes, ears, nose, and throat examination:
  - Note evidence of trauma:
    - For example, battle and raccoon signs
  - Pupillary size and reactivity:
    - May be important in evaluation for withdrawal or acute ingestions
- Cardiovascular and pulmonary examination:
  - Specifically, for acute conditions including angina, flash pulmonary edema, or exacerbations of asthma or COPD
- Gastrointestinal examination:
  - The presence or absence of abdominal pain, vomiting, or diarrhea
- The presence or absence of purulent sputum is not an accurate predictor of bacterial infection [3].

5.8 Introduction to Treatment

After evaluation and stabilization of emergency conditions, the emergency physician has several medication options to treat patients. These include:

1. Medication management introduction:
   (a) Considerations for medication management include:
      (i) Using the lowest effective dose
      (ii) Use of the medication on an intermittent basis
      (iii) Prescription of enough medication for a short-term basis only
      (iv) Avoiding antibiotics in uncomplicated cases of URI
      (v) Consideration of chronic medical conditions that may increase the side effects of the prescribed medication – specifically the sedative side effects

5.9 Non-pharmacologic Treatment

Nasal saline irrigation may reduce the need for pain meds and improve overall comfort; however, there have been mixed results with regard to benefit as some trials have shown that this causes more irritation. A systematic review from 2015 has concluded symptomatic relief, but studies were small with high risk of bias [5]. In a systemic review and meta-analysis of six trials, it was found that inhalation of humidified air or steam has been found to reduce symptoms but did not change viral shedding or create other objective clinical improvement [6]. Warm tea and chicken soup may further help to provide symptomatic relief and comfort for patients.

5.10 Pharmacologic Treatment

There are several over-the-counter medications that can be used for the symptomatic treatment of cough, cold, and congestion. While it is easy to inform patients of over-the-counter medications, it is also imperative to be aware of possible pitfalls and side effects of these medications. Over-the-counter medications often have serious side effects in children, with no proven benefit over placebo [7].

5.11 Analgesics

Analgesics
NSAIDs
Aspirin
Acetaminophen

Several studies have looked at NSAIDs and acetaminophen in symptomatic relief (headache, otalgia, myalgias, etc.), and it has been found that NSAIDs and acetaminophen were more effective than placebo at relieving symptoms [8]. Aspirin and acetaminophen were equally effective [8]. Short courses are usually considered to be safe.

Pitfalls

1. Watch for signs of toxicity including GI, CNS, and renal systems.
2. Patients may present with chronic overdose as well.

5.12 Antihistamines

Antihistamines
Diphenhydramine
Cetirizine
Loratadine

First-generation antihistamines can help alleviate rhinorrhea and sneezing; however, they are sedating. Second-generation antihistamines are often less sedating. A Cochrane Review found that antihistamines were more effective than placebo in the first 2 days of treatment, but had little to no improvement of symptoms after 6–10 days [9]. Combination of antihistamines with decongestants may be more beneficial [10].

Pitfalls

1. Have anticholinergic effects, beware of overdose.
2. May cause CNS depression.
3. Sedating, second-generation antihistamines are less sedating.

5.13 Decongestants

Decongestants
Oxymetazoline
Phenylephrine
Pseudoephedrine

Topical and oral decongestants can help relieve nasal congestion secondary to the common cold [10].

Pitfalls

1. Overdose can cause sympathomimetic toxidrome including hypertension, seizures, tachycardia (or reflex bradycardia), mydriasis, diaphoresis, agitation, and altered mental status.
2. Can lead to cardiac arrhythmia in overdose.
3. Increase risk for rhabdomyolysis in overdose.
4. Mixed preparations can lead to overdose of other medications (IE acetaminophen or salicylates).
5. Limit topical decongestant use to 2–3 h to prevent rebound rhinitis, including complications such as epistaxis, agitation, insomnia, and worsened hypertension [10].

5.14 Antitussives

Antitussives
Dextromethorphan
Codeine
Benzonatate

Antitussives are not recommended by the American College of Chest Physicians for cough secondary to URIs. This therapy is rarely necessary in the initial stages with variable outcomes in the later course of infection. Dextromethorphan was found to modestly decrease cough severity and frequency in a systematic review [11]. Codeine is the traditional opiate for cough suppression and was more effective than placebo in reducing severity and frequency of cough [11]. Benzonatate anesthetizes the stretch receptors of the lungs and pleura and is more effective when combined with guaifenesin [4].

Pitfalls

1. Do not take dextromethorphan concurrently or within 2 weeks of discontinuing MAO inhibitors.
2. Dextromethorphan can cause CNS symptoms including serotonin syndrome, confusion, excitement, irritability, and nervousness.
3. Codeine can cause CNS depression, constipation, hypotension, and respiratory depression. Be careful prescribing to pediatrics, debilitated patients, and elderly.
4. Benzonatate can lead to hallucinations as well as hypersensitivity reactions such as bronchospasm, cardiovascular collapse, and laryngospasm.
5. Benzonatate has also led to overdose in children younger than 10 years of age. Signs of overdose in children include restlessness, tremors, convulsion, coma, and cardiac arrest. It is not approved for use in children younger than 10 years of age.

5.15 Expectorants

Mucolytics
Guaifenesin

Expectorants help to thin secretions and may promote clearance of drainage. A systematic review from 2014 showed no good evidence for or against effectiveness of OTC medications, such as guaifenesin, for acute cough [12].

Pitfalls

1. Increases sedative effectives of alcohol, sleeping pills, muscle relaxers, and anesthetics
2. Can cause nausea, vomiting, and diarrhea
3. Increases the risk of kidney stone formation

5.16 Antibiotics and Antivirals

Antibiotics and antivirals
Amoxicillin-clavulanate
Doxycycline
Tamiflu

Antibiotic therapy for uncomplicated URI may cause more harm than benefit [13]. When compared to placebo in a systemic review of randomized trials, patients with URI symptoms of less than 7 days did not have a change in symptom persistence in antibiotic and placebo groups, with antibiotic groups having great risk of adverse effects [14]. Per the Infectious Disease Society of America, 5–7-day courses of empiric antibiotics are recommended for treatments of signs and symptoms that are:

(a) Persistent and not improving (>10 days)
(b) Severe (≥3–4 days)
(c) Worsening or "double-sickening" (≥3–4 days) [15]

Amoxicillin-clavulanate 500–125 mg TID or 875–125 mg BID is recommended rather than amoxicillin alone as empiric therapy [15]. Doxycycline can be used for patients allergic to penicillin [15]. Macrolides or second-/third-generation cephalosporins are not recommended for empiric therapy because of high resistance to *S. pneumoniae* [15]. Trimethoprim-sulfamethoxazole is not recommended for empiric therapy because of high resistance among both *S. pneumoniae* and *H. influenzae* [15].

If a patient presents with <48 h since onset of symptoms for influenza, consider Tamiflu 75 mg BID ×5 days (2 mg/kg BID ×5 days in children).

People at high risk, which should have treatment for influenza, include the following [16]:

- Residents of nursing homes or chronic care facilities
- Adults ≥65 years of age
- Native Americans and Alaska Natives
- Morbidly obese patients
- Pregnant women and women up to 2 weeks postpartum
- Chronic medical conditions including:
  - Cardiovascular disease
  - Active malignancy
  - Chronic renal insufficiency
  - Chronic liver disease
  - Diabetes
  - Hemoglobinopathies
  - Pulmonary disease (including asthma)

References

1. Kirkpatrick GL. The common cold. Prim Care. 1996;23:657.
2. Coughlin L. Cough: diagnosis and management. Am Fam Physician. 2007;75(4):567–75.
3. Buttaravoli PM, Leffler SM. Minor emergencies. Philadelphia: Elsevier/Saunders; 2012. p. 221–6.
4. Silvestri RC, Weinberger SE. Treatment of subacute and chronic cough in adults. UpToDate, 05 May 2015. Web. 03 Feb 2016.
5. King D, Mitchell B, Williams CP, Spurling GK. Saline nasal irrigation for acute upper respiratory tract infections. Cochrane Database Syst Rev 2015: CD006821.
6. Singh M, Singh M. Heated, humidified air for the common cold. Cochrane Database Syst Rev. 2013; (6):CD001728.
7. Pappas, D. The common cold in children: Management and prevention. UpToDate, 01 June 2016. Web. 03 June 2016.
8. Bachert C, Chuchalin AG, Eisebitt R, et al. Aspirin compared with acetaminophen in the treatment of fever and other symptoms of upper respiratory tract infection in adults: a multicenter, randomized, double-blind, double-dummy, placebo-controlled, parallel-group, single-dose, 6-hour dose-ranging study. Clin Ther. 2005;27:993.
9. De Sutter AI, Saraswat A, van Driel ML. Antihistamines for the common cold. Cochrane Database Syst Rev. 2015;11:CD009345.
10. Sexton DJ, McClain MT. The common cold in adults: Treatment and prevention. UpToDate, 05 Apr 2016. Web. 27 May 2016.
11. Yancy Jr WS, McCrory DC, Coeytaux RR, et al. Efficacy and tolerability of treatments for chronic cough: a systematic review and meta-analysis. Chest. 2013;144:1827.
12. Smith SM, Schroeder K, Fahey T. Over-the-counter (OTC) medications for acute cough in children and adults in community settings. Cochrane Database Syst Rev 2014: CD001831.
13. Harris AM, Hicks LA, Qaseem A. High value care task force of the American College of Physicians and for the Centers for Disease Control and Prevention. Appropriate antibiotic use

for acute respiratory tract infection in adults: advice for high-value care from the American College of Physicians and the Centers for Disease Control and Prevention. Ann Intern Med. 2016;164:425.
14. Kenealy T, Arroll B. Antibiotics for the common cold and acute purulent rhinitis. Cochrane Database Syst Rev. 2013;(6):CD000247.
15. Chow AW, Benninger MS, Brook I, Brozek JL, Goldstein EJ, Hicks LA, Pankey GA, Seleznick M, Volturo G, Wald ER, File Jr TM. IDSA clinical practice guideline for acute bacterial rhinosinusitis in children and adults. Clin Infect Dis. 2012;54(8):e72–112.
16. Zachary, KC. Treatment of seasonal influenza in adults. UpToDate, 10 May 2016. Web. 27 May 2016.

Chapter 6
Sinonasal Diseases

Charles Hwang, Bobby Desai, and Alpa Desai

6.1 Allergic Rhinitis

Allergic rhinitis (AR) is one of the most common diseases affecting both adults and children, impacting approximately one in six Americans [1, 2]. It is the most common chronic disease in children and is the fifth most common chronic disease overall in the United States [3]. Because of its prevalence, it results in a significant 6–11 billion dollars in healthcare costs annually, as well as a 2–4 billion dollar loss in productivity annually [1, 2, 4].

AR is an immunoglobulin E (IgE)-mediated disease characterized by inflammation of the lining of the nasal passages. An inciting allergen causes numerous inflammatory cells (mast cells, T cells, B cells, macrophages, and eosinophils) to infiltrate the nasal mucosa. T cells stimulate IgE production by plasma cells; this triggers the release of histamine and leukotrienes that cause the classic symptoms of rhinitis, including increased vascular permeability, mucous secretion, pruritus, rhinorrhea, nasal congestion, sneezing, postnasal drip, conjunctivitis, cough, and/or nasal itching [4–6].

AR may be classified by (1) the temporal pattern of the symptoms, such as seasonal, perennial, or episodic; (2) the frequency of the symptoms, such as intermittent (<4 days per week or <4 weeks per year) or persistent (>4 days per week and >4 weeks per year); and (3) the severity of the symptoms (mild or moderate severe) [5, 7]. The severity is classified based on the impact on quality of life; mild severity indicates

C. Hwang, MD (✉)
Department of Emergency Medicine, UF Health,
1329 SW 16th Street, PO Box 100186, Gainesville, FL 32610, USA
e-mail: c7places@ufl.edu

B. Desai, MD
Emergency Medicine, University of Florida, Gainesville, FL 32610, USA
e-mail: bdesai@ufl.edu

A. Desai, DO
Community Health and Family Medicine, University of Florida, Newberry, FL, USA

B. Desai, A. Desai (eds.), *Primary Care for Emergency Physicians*,
DOI 10.1007/978-3-319-44360-7_6

symptoms that do not interfere with quality of life, whereas moderate severe symptoms do interfere [8]. Although these categories separate AR into rigid compartments, AR is an inflammatory disease that exists on a continuum. For example, patients living in more temperate climates may experience significant overlap between seasonal and perennial symptoms. Moreover, patients with perennial AR may have seasonal exacerbations based on local temperature and climate changes. Furthermore, many experts consider AR on a spectrum of diseases that includes the entire respiratory tract, including asthma and reactive airway disease [5, 7]. However, the classification of AR helps guide the clinician toward the appropriate treatment for each patient [4].

The diagnosis of AR is based on history and physical examination. Frequently, AR goes undetected because patients do not realize the impact AR has on quality of life nor do providers regularly screen patients for its signs and symptoms [5]. Symptoms suggestive of AR include nasal itching, nasal congestion, sniffling, rhinorrhea, sneezing, and conjunctivitis triggered by an allergic cause. These triggers may include dust mites, pollen, animal dander, upholstery, cockroach residues, mold, and humidity. Perennial symptoms are more suggestive of indoor allergens such as dust mites and animal dander, while seasonal symptoms are more suggestive of outdoor allergens, although outdoor allergens may cause perennial symptoms in more temperate climates. Atypical symptoms, such as unilateral nasal symptoms, severe headache, epistaxis, or anosmia, may suggest a more insidious diagnosis (CSF rhinorrhea, chronic rhinosinusitis, sinonasal tumor, granulomatous disease) [4, 9]. Other sinonasal pathologies, such as sinusitis, granulomatous disease, and viral upper respiratory infections, can cause symptoms similar to AR and should be further delineated [4].

The patient may have a past medical history or family history of asthma, sleep apnea, sinusitis, otitis media, nasal polyps, atopic dermatitis, or conjunctivitis [4, 5, 10, 11]. Medications may cause nasal symptoms and should be elicited as well; these include antihypertensives, acetylsalicylic acid, nonsteroidal anti-inflammatory drugs, psychotropic medications, topical decongestants, and recreational misadventures [4, 5, 12].

The physical examination may include clear rhinorrhea, nasal congestion, pale discoloration of the nasal mucosa, postnasal drip, and lacrimation. Conjunctival edema, as well as the classic “allergic shiner” or periorbital darkness and puffiness, may be present. Patients may be observed to have sequelae or behavioral adaptations due to longstanding AR, including mouth breathing, frequent sniffling, throat clearing, or the development of an “allergic crease” from frequent rubbing of the nose, also known as the “allergic salute.” The physical examination should evaluate for other insidious causes of nasal obstruction and rhinorrhea, including CSF leak, nasal polyps, or foreign bodies [4, 5].

Although the definitive diagnosis of AR is by IgE testing and identification of a specific allergen that causes symptoms, it is reasonable to make a clinical diagnosis based on the history and physical exam and to initiate empiric therapy, especially if the symptoms are adversely impacting quality of life. Providers should make the provisional diagnosis of AR when patients present with symptoms of AR and the history and physical examination are suggestive of an allergic cause [4]. Moreover, symptomatic improvement from allergen avoidance and/or empiric therapy further supports the diagnosis of AR and may preclude further allergy testing altogether [4].

Routine imaging does not play a role in the diagnosis of uncomplicated AR; it exposes patients to excessive cost and the risk of ionizing radiation without any benefits in diagnosis. However, if there is clinical concern for other pathology, including neoplasm, abscess, or rhinosinusitis, imaging may be indicated [13].

Empiric therapy for allergic rhinitis includes avoidance of triggers, environmental modification, and medical management. Clinicians may advise patients to avoid known allergen triggers and recommend low-cost, potentially beneficial lifestyle modifications that reduce the allergen burden within the environment, including removal of pets, installation of air purification systems, and use of bed covers. Numerous studies have evaluated pet ownership, dander elimination by frequent pet washing, the use of impermeable bed covers, air filtration using high-efficiency particulate air (HEPA) filters, and acaricides (chemical agents to kill dust mites). Although these interventions reduce allergen levels, no single intervention has been proven to be effective for AR, and a multiple intervention strategy may prove useful; further studies need to be performed [4, 14–18].

Intranasal steroids are considered the most effective medication in treating AR and are more effective than oral antihistamines [19–25]. Intranasal steroids include mometasone, beclomethasone, budesonide, fluticasone, and triamcinolone. Patients with intermittent symptoms may be treated with second-generation, nonsedating antihistamines and/or decongestants. The second-generation antihistamines include cetirizine, fexofenadine, loratadine, levocetirizine, and desloratadine and are efficacious in decreasing symptoms of AR but not nasal congestion [25, 26]. Intranasal antihistamines, such as azelastine, are also effective at managing AR symptoms while minimizing systemic side effects [21, 27]. Patients with refractory AR should be referred to a specialist for possible immunotherapy.

Clinicians should refer patients for IgE allergy testing when the diagnosis is uncertain, or when patients do not respond to empiric therapy, or when identification of the specific allergen will help guide therapy [4]. IgE allergy testing is performed either by skin or blood testing. Skin testing is a highly sensitive and specific test that allows direct observation of the body's response to a suspected allergen after introducing the allergen intradermally (either by skin pricking or by intradermal injection) [28, 29]. Blood testing is performed by an immunoassay; allergen-specific IgE is incubated with the suspected allergen, which has been attached to a solid phase. The bound IgE complex is then measured by introducing anti-IgE antibody (specific for the allergen) that has been radioactively or enzymatically labeled [4]. There are other tests that are used to evaluate suspected AR, including acoustic rhinometry, olfactory testing, microarray testing, nasal allergen challenges, and nasal eosinophilia smears although there is currently insufficient evidence on their utility [4].

6.2 Sinusitis

Sinusitis affects approximately one in eight American adults, or 35 million American adults, per year [30, 31]. More than 20 % of prescribed antibiotics are for sinusitis, making sinusitis the fifth most common diagnosis treated with antibiotics [32, 33].

The healthcare-related costs associated with managing sinusitis exceed $11 billion per year, in addition to losses in productivity and quality of life [31, 34]. Despite the prevalence of sinusitis, significant variations exist in its management [35]. Moreover, sinusitis is not a benign disease; besides the prevalence and financial impact of sinusitis, uncommon but serious complications can include meningitis, intracerebral abscess, orbital cellulitis, and orbital abscess [36].

Acute sinusitis is commonly caused by viral infections. Direct inoculation by viruses (commonly rhinovirus, influenza virus, and parainfluenza virus) results in direct contact with the nasal mucosa. These viruses can spread to the paranasal sinuses by direct spread, or by nose blowing which elevates intranasal pressures and propels viruses into the paranasal sinuses. The subsequent inflammation, vascular permeability, hypersecretion, and impaired ciliary clearance result in sinus obstruction and the classic syndrome of sinusitis [37]. Because this inflammation typically also involves the contiguous nasal passages as well, many experts prefer the more correct term *rhinosinusitis*. Although viruses are the most common cause of acute rhinosinusitis, any inflammatory cause that obstructs the sinonasal passages can cause acute rhinosinusitis, including allergic rhinitis, nasal obstruction or polyps, dental infection, and impaired mucociliary clearance [38]. Acute rhinosinusitis can become secondarily infected by bacteria and occurs in 0.5–2 % of patients with acute rhinosinusitis [35, 37, 39]. Common bacteria associated with bacterial sinusitis are *Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Moraxella catarrhalis*.

Rhinosinusitis is categorized by the duration of symptoms and by the etiology. Acute rhinosinusitis refers to symptoms lasting shorter than 4 weeks, while chronic rhinosinusitis refers to symptoms that persist more than 12 weeks [35]. Acute rhinosinusitis can be further categorized by the etiology, bacterial versus viral rhinosinusitis. This distinction is important, as antibiotics are not indicated for viral causes. Four or more discrete episodes of sinusitis in a year are termed recurrent rhinosinusitis. Expert opinion varies on the terminology for symptoms that last between 4 and 12 weeks, with some experts labeling this acute rhinosinusitis and others labeling this subacute. Therefore, the management of symptoms lasting from 4 to 12 weeks must be tailored to the individual patient [35].

Symptoms suggestive of rhinosinusitis include nasal congestion, rhinorrhea, postnasal drip, purulent (not clear) nasal discharge, teeth discomfort, facial pain localized over the frontal or maxillary sinuses, facial pressure or fullness, fever, fatigue, cough, malaise, anosmia, headache, and ear pressure/fullness. Symptoms may worsen with straining or bending over [35, 40–43].

Clinical findings of rhinosinusitis include nasal discharge, rhinorrhea, purulent drainage from the nose or oropharynx, mucosal edema, or turbinate hyperemia or hypertrophy. Patients may exhibit alteration in speech tone secondary to nasal obstruction. The affected sinus may have overlying erythema, edema, or tenderness to palpation and percussion. The maxillary teeth may exhibit tenderness to percussion if the maxillary sinus is involved. Transillumination of the sinuses may show opacification of the frontal and/or maxillary sinuses although this is neither sensitive nor specific [35, 43, 44]. Of these physical findings, purulence within the nasal

cavity is the only finding shown to have diagnostic value [35]. The provider should also evaluate for complications of sinusitis, including nuchal rigidity, abnormal extraocular movements, altered mental status, visual changes, facial or periorbital or orbital cellulitis, and proptosis [35].

The diagnosis of acute rhinosinusitis is a clinical diagnosis; the diagnosis is made when a patient has up to 4 weeks of purulent nasal discharge associated with nasal obstruction and/or facial pain or pressure. Distinguishing between viral and bacterial rhinosinusitis is trickier, as the symptoms of acute viral (nonbacterial) rhinosinusitis have significant overlap with that of acute bacterial rhinosinusitis [35].

The clinical course of acute viral rhinosinusitis is similar to other viral upper respiratory tract infections (URIs); patients typically are afebrile but may have a fever early on that precedes the onset of self-limited respiratory symptoms, including coughing, rhinorrhea, sore throat, nasal congestion, and sneezing. Once the fever abates early in the disease course, respiratory symptoms usually peak in severity between day 3 and 6 of illness and should continue to improve by day 10 of illness [43].

Symptoms of acute bacterial rhinosinusitis, on the contrary, typically last longer than 10 days, and symptoms are generally more severe. The American Academy of Otolaryngology Clinical Practice Guideline suggests that a diagnosis of bacterial rhinosinusitis be made "when (a) symptoms or signs of acute rhinosinusitis (purulent nasal discharge accompanied by nasal obstruction, facial pain-pressure-fullness, or both) *persist without evidence of improvement for at least 10 days* beyond the onset of upper respiratory symptoms, or (b) symptoms or signs of acute rhinosinusitis *worsen within 10 days* after an initial improvement (double worsening)" [35]. It is important to note that the presence or absence of purulent drainage does not distinguish viral from bacterial infections [45]. Moreover, the Clinical Practice Guideline does not include fever as a distinguishing factor between viral and bacterial rhinosinusitis, as fever may be present in patients with viral rhinosinusitis. Fever has a sensitivity and specificity of approximately 50 % for bacterial rhinosinusitis [46, 47]. Certain experts, however, encourage careful consideration of acute bacterial rhinosinusitis in the subgroup of patients with "high fever" and purulent nasal discharge for 3–4 days. The Infectious Disease Society of America defines "high fever" as greater than 39 °C [42, 43, 48].

It is impossible for radiologic imaging to distinguish bacterial from viral rhinosinusitis. For routine acute rhinosinusitis, radiographic imaging is not indicated unless a complication or alternative diagnosis is suspected [35, 43]. Complications of sinusitis include orbital or intracranial extension, such as preseptal cellulitis, orbital cellulitis, subperiosteal abscess, meningitis, intracranial or epidural abscess, osteomyelitis, or cavernous sinus thrombosis [49]. Clinicians should also have a heightened level of suspicion in patients with an immunocompromised state or diabetes which predisposes patients to complications. Alternative diagnoses such as malignancy or granulomatous disease may warrant imaging as well.

In viral rhinosinusitis, viruses cause a marked inflammatory response mediated by cytokines, resulting in mucosal edema, mucus production, and impaired ciliary function. Antibiotics are not effective for viral illnesses; rather, the

management should be directed at symptomatic relief. Palliative medications directed toward symptom control, such as antipyretics, analgesics, nonsteroidal anti-inflammatory drugs, nasal saline irrigation, decongestants, antihistamines, antitussives, and topical corticosteroids, may drastically improve symptomatic relief [50, 51].

For bacterial rhinosinusitis, symptomatic management (as discussed above) may prove useful because pain relief is often a principal reason patients seek healthcare. When comparing placebo versus antibiotic therapy, the evidence remains equivocal with respect to cure rate, duration of illness, and adverse events [35]. Therefore, clinicians may offer watchful waiting or antibiotic therapy, both of which are appropriate, evidence-based practices. Watchful waiting refers to symptomatic management while deferring antibiotics for 7 days. Antibiotics are started if symptoms do not improve after 7 days or worsen at any time. Watchful waiting, along with anticipatory guidance and return precautions, should only be offered if follow-up is guaranteed to ensure that antibiotics may be started in a timely manner [35, 43]. Patients concerned about healthcare access may also be offered a safety net antibiotic prescription, which can be filled after a period of watchful waiting. Joint decision-making between the clinician and patient, including the risks, benefits, and alternatives of antibiotic therapy versus watchful waiting and symptomatic relief, is important [35].

The first-line antibiotic of choice for acute bacterial rhinosinusitis is amoxicillin or amoxicillin-clavulanate for 5–10 days. The addition of clavulanate should be considered in patients where bacterial resistance is more likely or in the setting of comorbidities, for example, recent antibiotic use, failure of prior antibiotic therapy, close contact with children or healthcare environment, smokers, high prevalence of resistant bacteria within the community, elderly, immunocompromised, or diabetics [35, 43, 52, 53]. For penicillin-allergic patients, doxycycline and fluoroquinolones are alternatives [48]. For patients that do not improve after 72 h of antibiotic therapy, the clinician should reassess the patient and alternate diagnoses should be entertained, including complications of rhinosinusitis, such as preseptal cellulitis, orbital cellulitis, subperiosteal abscess, meningitis, intracranial or epidural abscess, osteomyelitis, or cavernous sinus thrombosis. Other disease entities that cause symptoms similar to rhinosinusitis include migraine headache, tension headache, cluster headache, temporal arteritis, dental caries and abscesses, temporomandibular joint dysfunction, and allergic rhinitis. Computed tomography imaging may be helpful if alternative insidious conditions are entertained [35, 43].

Chronic rhinosinusitis is defined as rhinosinusitis that persists unabated for greater than 12 weeks. It is imperative to distinguish recurrent acute rhinosinusitis from chronic rhinosinusitis. The diagnosis of chronic rhinosinusitis should be performed by a specialist using anterior rhinoscopy, nasal endoscopy, or computed tomography and is outside the scope of this chapter. Patients suspected of chronic rhinosinusitis should be referred to an otolaryngologist for further management [35].

References

1. Blaiss MS. Allergic rhinitis: direct and indirect costs. Allergy Asthma Proc. 2010;31(5): 375–80.
2. Meltzer EO, Bukstein DA. The economic impact of allergic rhinitis and current guidelines for treatment. Ann Allergy Asthma Immunol. 2011;106(2 Suppl):S12–16.
3. Tran NP, Vickery J, Blaiss MS. Management of rhinitis: allergic and non-allergic. Allergy Asthma Immunol Res. 2011;3(3):148–56.
4. Seidman MD, Gurgel RK, Lin SY, et al. Clinical practice guideline: allergic rhinitis. Otolaryngol Head Neck Surg. 2015;152(1 Suppl):S1–43.
5. Small P, Kim H. Allergic rhinitis. Allergy Asthma Clin Immunol. 2011;7 Suppl 1:S3.
6. Borish L. Allergic rhinitis: systemic inflammation and implications for management. J Allergy Clin Immunol. 2003;112(6):1021–31.
7. Bousquet J, Khaltaev N, Cruz AA, et al. Allergic Rhinitis and its Impact on Asthma (ARIA) 2008 update (in collaboration with the World Health Organization, GA(2)LEN and AllerGen). Allergy. 2008;63 Suppl 86:8–160.
8. Wallace DV, Dykewicz MS, Bernstein DI, et al. The diagnosis and management of rhinitis: an updated practice parameter. J Allergy Clin Immunol. 2008;122(2 Suppl):S1–84.
9. McCrory DC, Williams JW, Dolor RJ, et al. Management of allergic rhinitis in the working-age population. Evid Rep Technol Assess (Summ). 2003;(67):1–4.
10. Kiyohara C, Tanaka K, Miyake Y. Genetic susceptibility to atopic dermatitis. Allergol Int: Off J Japanese Soc Allergol. 2008;57(1):39–56.
11. Nayak AS. The asthma and allergic rhinitis link. Allergy Asthma Proc. 2003;24(6):395–402.
12. Varghese M, Glaum MC, Lockey RF. Drug-induced rhinitis. Clin Exp Allergy. 2010;40(3): 381–4.
13. Cornelius RS, Martin J, Wippold 2nd FJ, et al. ACR appropriateness criteria sinonasal disease. J Am Coll Radiol: JACR. 2013;10(4):241–6.
14. Custovic A, Simpson BM, Simpson A, et al. Effect of environmental manipulation in pregnancy and early life on respiratory symptoms and atopy during first year of life: a randomised trial. Lancet. 2001;358(9277):188–93.
15. Fretzayas A, Kotzia D, Moustaki M. Controversial role of pets in the development of atopy in children. World J Pediatr: WJP. 2013;9(2):112–19.
16. Sheikh A, Hurwitz B, Shehata Y. House dust mite avoidance measures for perennial allergic rhinitis. Cochrane Database Syst Rev. 2007;(1):CD001563.
17. Nurmatov U, van Schayck CP, Hurwitz B, Sheikh A. House dust mite avoidance measures for perennial allergic rhinitis: an updated Cochrane systematic review. Allergy. 2012;67(2): 158–65.
18. Terreehorst I, Oosting AJ, Tempels-Pavlica Z, et al. Prevalence and severity of allergic rhinitis in house dust mite-allergic patients with bronchial asthma or atopic dermatitis. Clin Exp Allergy. 2002;32(8):1160–5.
19. van Bavel J, Findlay SR, Hampel Jr FC, Martin BG, Ratner P, Field E. Intranasal fluticasone propionate is more effective than terfenadine tablets for seasonal allergic rhinitis. Arch Intern Med. 1994;154(23):2699–704.
20. Welsh PW, Stricker WE, Chu CP, et al. Efficacy of beclomethasone nasal solution, flunisolide, and cromolyn in relieving symptoms of ragweed allergy. Mayo Clin Proc. 1987;62(2): 125–34.
21. Kaszuba SM, Baroody FM, de Tineo M, Haney L, Blair C, Naclerio RM. Superiority of an intranasal corticosteroid compared with an oral antihistamine in the as-needed treatment of seasonal allergic rhinitis. Archiv Intern Med. 2001;161(21):2581–7.
22. Herman H. Once-daily administration of intranasal corticosteroids for allergic rhinitis: a comparative review of efficacy, safety, patient preference, and cost. Am J Rhinol. 2007;21(1): 70–9.

23. Rodrigo GJ, Neffen H. Efficacy of fluticasone furoate nasal spray vs. placebo for the treatment of ocular and nasal symptoms of allergic rhinitis: a systematic review. Clin Exp Allergy. 2011;41(2):160–70.
24. Weiner JM, Abramson MJ, Puy RM. Intranasal corticosteroids versus oral H1 receptor antagonists in allergic rhinitis: systematic review of randomised controlled trials. BMJ. 1998;317(7173):1624–9.
25. Benninger M, Farrar JR, Blaiss M, et al. Evaluating approved medications to treat allergic rhinitis in the United States: an evidence-based review of efficacy for nasal symptoms by class. Ann Allergy Asthma Immunol. 2010;104(1):13–29.
26. Meltzer EO. Performance effects of antihistamines. J Allergy Clin Immunol. 1990;86(4 Pt 2): 613–19.
27. Horak F, Zieglmayer UP, Zieglmayer R, et al. Azelastine nasal spray and desloratadine tablets in pollen-induced seasonal allergic rhinitis: a pharmacodynamic study of onset of action and efficacy. Curr Med Res Opin. 2006;22(1):151–7.
28. Bousquet J, Heinzerling L, Bachert C, et al. Practical guide to skin prick tests in allergy to aeroallergens. Allergy. 2012;67(1):18–24.
29. Bernstein IL, Li JT, Bernstein DI, et al. Allergy diagnostic testing: an updated practice parameter. Ann Allergy Asthma Immunol. 2008;100(3 Suppl 3):S1–148.
30. Lucas JW, Schiller JS, Benson V. Summary health statistics for U.S. adults: National Health Interview Survey, 2001. Vital and health statistics. Series 10, Data from the National Health Survey. 2004;(218):1–134.
31. Blackwell DL, Lucas JW, Clarke TC. Summary health statistics for U.S. adults: national health interview survey, 2012. Vital and health statistics. Series 10, Data from the National Health Survey. 2014;(260):1–161.
32. Anon JB, Jacobs MR, Poole MD, et al. Antimicrobial treatment guidelines for acute bacterial rhinosinusitis. Otolaryngol Head Neck Surg. 2004;130(1 Suppl):1–45.
33. Bishai WR. Issues in the management of bacterial sinusitis. Otolaryngol Head Neck Surg. 2002;127(6 Suppl):S3–9.
34. Rudmik L, Smith TL, Schlosser RJ, Hwang PH, Mace JC, Soler ZM. Productivity costs in patients with refractory chronic rhinosinusitis. Laryngoscope. 2014;124(9):2007–12.
35. Rosenfeld RM, Piccirillo JF, Chandrasekhar SS, et al. Clinical practice guideline (update): adult sinusitis executive summary. Otolaryngol Head Neck Surg. 2015;152(4):598–609.
36. Hytonen M, Atula T, Pitkaranta A. Complications of acute sinusitis in children. Acta Otolaryngol Suppl. 2000;543:154–7.
37. Gwaltney Jr JM. Acute community-acquired sinusitis. Clin Infect Dis: Off Publi Infect Dis Soc Am. 1996;23(6):1209–23; quiz 1224–5.
38. Scheid DC, Hamm RM. Acute bacterial rhinosinusitis in adults: part I. Evaluation. Am Fam Physician. 2004;70(9):1685–92.
39. Fokkens WJ, Lund VJ, Mullol J, et al. EPOS 2012: European position paper on rhinosinusitis and nasal polyps 2012. A summary for otorhinolaryngologists. Rhinology. 2012;50(1):1–12.
40. Lanza DC, Kennedy DW. Adult rhinosinusitis defined. Otolaryngol Head Neck Surg. 1997;117(3 Pt 2):S1–7.
41. Piccirillo JF. Clinical practice. Acute bacterial sinusitis. N Engl J Med. 2004;351(9):902–10.
42. Meltzer EO, Hamilos DL. Rhinosinusitis diagnosis and management for the clinician: a synopsis of recent consensus guidelines. Mayo Clin Proc. 2011;86(5):427–43.
43. Wald ER, Applegate KE, Bordley C, et al. Clinical practice guideline for the diagnosis and management of acute bacterial sinusitis in children aged 1 to 18 years. Pediatrics. 2013;132(1):e262–80.
44. Low DE, Desrosiers M, McSherry J, et al. A practical guide for the diagnosis and treatment of acute sinusitis. CMAJ: Can Med Assoc J = J de l'Assoc Med Can. 1997;156 Suppl 6:S1–14.
45. Lacroix JS, Ricchetti A, Lew D, et al. Symptoms and clinical and radiological signs predicting the presence of pathogenic bacteria in acute rhinosinusitis. Acta Otolaryngol. 2002;122(2): 192–6.

46. Williams Jr JW, Simel DL, Roberts L, Samsa GP. Clinical evaluation for sinusitis. Making the diagnosis by history and physical examination. Ann Intern Med. 1992;117(9):705–10.
47. Hauer AJ, Luiten EL, van Erp NF, et al. No evidence for distinguishing bacterial from viral acute rhinosinusitis using fever and facial/dental pain: a systematic review of the evidence base. Otolaryngol Head Neck Surg. 2014;150(1):28–33.
48. Chow AW, Benninger MS, Brook I, et al. IDSA clinical practice guideline for acute bacterial rhinosinusitis in children and adults. Clin Infect Dis: Off Public Infect Dis Soc Am. 2012;54(8):e72–112.
49. Setzen G, Ferguson BJ, Han JK, et al. Clinical consensus statement: appropriate use of computed tomography for paranasal sinus disease. Otolaryngol Head Neck Surg. 2012;147(5): 808–16.
50. Arroll B, Kenealy T. Are antibiotics effective for acute purulent rhinitis? Systematic review and meta-analysis of placebo controlled randomised trials. BMJ. 2006;333(7562):279.
51. Arroll B, Kenealy T. Antibiotics for the common cold and acute purulent rhinitis. Cochrane Database Syst Rev. 2005;(3):CD000247.
52. Brook I, Gober AE. Resistance to antimicrobials used for therapy of otitis media and sinusitis: effect of previous antimicrobial therapy and smoking. Ann Otol Rhinol Laryngol. 1999;108(7 Pt 1):645–7.
53. Jenkins SG, Farrell DJ, Patel M, Lavin BS. Trends in anti-bacterial resistance among Streptococcus pneumoniae isolated in the USA, 2000–2003: PROTEKT US years 1–3. J Infect. 2005;51(5):355–63.

Chapter 7
Neck Pain

Michael Seth Smith and Tom A. Starnes

7.1 Introduction

Neck pain is a common complaint among patients, with up to two-thirds of people experiencing this malady at some point in their lives [1]. Neck pain led to 16.3 million patient visits in the United States in 2010, 5 % of which were emergency department visits [2]. The majority of patients presenting with neck pain do not require hospitalization, indicating that most people presenting with neck pain may be safely managed in the outpatient setting. It is well-known that many patients, especially those of lower socioeconomic status, will seek non-emergency care in the emergency department [3]. Even though these patients will not likely require admission when they present with neck pain, the differential diagnosis of neck pain does contain serious and even life-threatening etiologies of which the emergency physician must be cognizant.

Neck pain can be a manifestation of a wide variety of medical conditions besides musculoskeletal or soft tissue injury. Careful history taking and physical examination, when coupled with a high index of suspicion in the appropriate cases, will allow the emergency physician to triage patients with neck pain into low- and high-risk categories. Low-risk patients can be managed in the outpatient setting; high-risk patients may warrant admission and possibly subspecialty consultation.

M.S. Smith (✉)
Department of Orthopedics, Shands at University of Florida Health, Gainesville, FL, USA
e-mail: michael.smith@ufl.edu; Smithms@ortho.ufl.edu

T.A. Starnes, MD
Community Health and Family Medicine, University of Florida Student Health Care Center, Gainesville, FL, USA
e-mail: Thomas.Starnes@medicine.ufl.edu; tstarnes@ufl.edu

B. Desai, A. Desai (eds.), *Primary Care for Emergency Physicians*,
DOI 10.1007/978-3-319-44360-7_7

7.2 Risk Factors for Neck Pain

- Participation in contact sports or other activities predisposing to acute injury [4]
- Presence of underlying medical conditions which can manifest as neck pain
- Poor posture and/or postural awareness and poor ergonomics
- Psychosocial factors, including occupational history and presence of underlying psychologic disorders or poor coping skills
- Advancing age

7.3 Differential Diagnosis

The differential diagnosis of neck pain is broad, involving the musculoskeletal structures of the neck as well as other organs which can refer pain to the neck. The following list is not comprehensive but rather representative of the multiple etiologies of neck pain:

- Traumatic
  - Cervical paraspinal muscular strain or cervical ligamentous sprain
    Classic "whiplash" injury from forced cervical extension resulting in tension-compression forces in the cervical spine [5]
    Can be immediate-onset or delayed in presentation from when injury actually occurred
  - Noncontact fractures (clay-shoveler's fracture) [4, 5]
  - Fractures secondary to impact or contact [6]
- Non-traumatic
  - Musculoskeletal
    - Cervical spine osteoarthritis
      Poor correlation between radiographic findings and patient complaints [7]
    - Longus colli tendinitis/retropharyngeal calcific tendinitis
      Most common symptoms are acute onset of neck pain, odynophagia, and reduced ROM. Benign but difficult to distinguish from retropharyngeal abscess [8, 9]
    - Diffuse idiopathic skeletal hyperostosis (DISH)
      C-spine involvement found in up to 76% of patients with DISH. Can be complicated by dysphagia, hoarseness, and stridor depending on which local structures are affected [10, 11]
    - Cervical intervertebral disk herniation
    - Thoracic outlet syndrome
    - Myofascial trigger points
    - Wry neck [12]

  - Zygapophyseal: associated with sudden movements and acute pain, more common in younger population
  - Diskogenic: associated with more chronic course, more common in older population
- Referred shoulder pain
  - Rotator cuff tendinitis/impingement syndrome
  - Adhesive capsulitis

– Infectious

- Infectious thyroiditis
- Pharyngitis
  Need to distinguish bacterial from viral infections and treat bacterial infections to prevent complications [13].
- Deep space infections (retropharyngeal or precervical infections)
  Must look for an underlying cause; most retropharyngeal abscesses occur in the setting of immunodeficiency or an injury by a foreign body [14].
- Diskitis/osteomyelitis
  More common in patients older than 50 years. Risk factors include intravenous drug abuse, chronic hemodialysis, and other immunocompromising states [15].
- Lymphadenitis
  Should prompt evaluation into infectious etiologies affecting the head and neck
- Herpes zoster
  Pain present several days prior to onset of rash

– Cardiovascular

- Carotid artery dissection
  May not always be associated with typical cardiovascular risk factors but can be a sequela of neck injury, including chiropractic manipulation [16]. Can be associated with focal neurologic signs, including amaurosis fugax, partial Horner's syndrome, hypogeusia, and visual field disturbances
- Spontaneous hematoma
  Patients may be prescribed multiple blood-thinning agents for cardiovascular indications [17].
- Myocardial infarction (referred pain)
  Neck pain present in approximately 25 % of patients with an acute myocardial infarction in one study. No significant difference seen between male and female patients [18]

– Rheumatologic

- Rheumatoid arthritis
  Neck pain prevalent in 43–86 % of patients with RA, with radiographic changes noted in greater than 80 % [19]

    - Ankylosing spondylitis
      Neck pain may be a presenting symptom of AS in between 25 and 33 % of patients, with a slight prominence in women [20].
    - Autoimmune thyroiditis
  - Metabolic
    - Gout/pseudogout
      Cervical spine is a more unusual site of tophi formation but can be seen in advanced cases of untreated hyperuricemia and gout. MRI may be more helpful in establishing the diagnosis as the clinical picture can mimic rheumatoid arthritis [21].
    - Paget's disease
  - Neurologic
    - Chiari I malformation
    - Cervicogenic headache
  - Visceral
    - Esophageal dysmotility/obstruction
    - Referred gallbladder pain

7.4 Red Flags of Neck Pain

Red flags of neck pain include the 5 D's [12]:

1. Dizziness
2. Diplopia
3. Dysphagia
4. Dysarthria
5. Drop attacks

Presence of these symptoms should alert the emergency physician to the possibility of cardiovascular etiologies such as vertebrobasilar insufficiency or carotid artery dissection.

7.5 History

The wide differential diagnosis of neck pain can be narrowed based on the history obtained from the patient. Details will not only help with diagnosis but also with treatment plans.

- Symptom onset
  - Acute versus insidious/chronic

- Location of pain
  - Localized versus diffuse
- Presence of additional symptoms
  - Headache
  - Radiating neuropathic pain
  - Red flags (5 D's as listed above)
- Aggravating and alleviating factors, including medications
- Occupational or recreational factors

7.6 Physical Examination

As with physical examination of any other area of the body, history obtained from the patient or witness will guide the physical examination. An abrupt onset of symptoms following a traumatic event will obviously lead the examiner to look for acute musculoskeletal injuries, whereas an insidious onset would cause one to look for alternative reasons for the patient's complaints. If the cause of pain is thought to be structural, initial evaluation should focus on determining whether the pain generator in question is articular, muscular, or neural in nature [12].

- Vital signs
  - The presence of fever should prompt a search for an infectious etiology of symptoms.
- Examination of musculoskeletal structures
  - Examination of contour
    Loss of normal cervical lordosis can be caused by ankylosing spondylitis (back to wall test).
  - Assess range of motion in six directions
    - Flexion/extension
    - Rotation
    - Side bending
  - Palpation of vertebral bodies, paraspinal musculature, upper trapezius, sternocleidomastoids
  - Special testing
    - Spurling maneuver (neck compression test)
      Used to identify lateral rupture of a cervical intravertebral disk causing nerve root compression
    - Roos test and Adson's test
      Used to evaluate for thoracic outlet syndrome in patients presenting with neck pain in conjunction with numbness and tingling of an upper limb
  - Neurologic exam if radiation of symptoms below the shoulder

- Examination of other structures in the neck as indicated by history
  - Visual examination of oropharyngeal structures
  - Palpate thyroid. May be able to auscultate a bruit in hyperthyroidism
  - Palpate posterior and anterior cervical lymph nodes. Preauricular, postauricular, and occipital nodes are usually visible if significantly involved in underlying process [22].

7.7 Introduction to Treatment

Much as the physical examination will be guided by the history of current injury, so will treatment options be directed by the nature of the underlying process. Neck pain caused by non-musculoskeletal pathology should be treated as indicated to address the underlying process (i.e., treating an infection, myocardial infarction, etc.). The following are some broad principles for treatment of both acute and chronic musculoskeletal neck pain:

- Acute/traumatic musculoskeletal injury
  - In trauma patients, one must decide which patients require imaging of the C-spine. The NEXUS Low-Risk Criteria is a validated model that can assist the emergency physician in decision-making [23].

NEXUS Low-Risk Criteria
No tenderness at posterior midline of C-spine
No focal neurologic deficit
Normal level of alertness
No evidence of intoxication
No distracting injury

  - Fractures involving multiple levels, involving C3–C5, or demonstrating widened disk space or facet dislocation on plain films should be evaluated for adjacent level ligamentous injury (ALLI) by MRI [24].
  - Less serious acute musculoskeletal injuries (e.g., whiplash) can often be managed symptomatically with short courses of acetaminophen, nonsteroidal anti-inflammatories, and muscle relaxants.
- Musculoskeletal injury of a chronic nature
  - Acute injury such as whiplash can turn into a chronic pain syndrome.
  - Most chronic neck pain treatment plans will require a multimodality/multidisciplinary approach:
    - Patient education, including expected outcomes
    - Physical therapy with postural rehabilitation, muscle strength, and control techniques

- Medications for short durations
- Referral to psychology/psychiatry as needed
- Invasive procedures (corticosteroid injections, surgical intervention) only when an adequate amount of conservative management has failed

7.8 Pharmacologic Treatment

Pharmacologic treatment options listed below assume that the patient's condition does not warrant admission to the hospital and can be managed conservatively:

- Acute musculoskeletal injury
  - Acetaminophen
    - Acts as an analgesic and antipyretic on cannabinoid receptors in the central nervous system
    - Dosed 650–1,000 mg every 8 h as needed, maximum daily dose 3 g (FDA reduced this recommended maximum dose from 4 g in 2014)
    - No benefit seen from taking doses higher than 1 g at a time [12]
    - Pitfalls
      1. Prescribing acetaminophen when a patient is already taking acetaminophen-containing medications such as oxycodone-acetaminophen. Patients must be counseled that 3 g of acetaminophen from all sources counts toward the maximum daily limit.
      2. Prescribing to patients with liver injury.
      3. Unlikely to alleviate severe pain.
  - Nonsteroidal anti-inflammatory drugs (NSAIDs)

Commonly prescribed NSAIDs	Typical dosing
Ibuprofen	400–800 mg PO every 4–6 h
Meloxicam	7.5–15 mg PO every 24 h
Diclofenac	25–50 mg PO every 8–12 h
Naproxen	250–1,000 mg PO every 12–24 h

    - Inhibit cyclooxygenase (COX) enzyme which has a role in the inflammatory cascade after acute injury. Antithrombotic effects are seen in addition to analgesic and antipyretic effects.
    - There is evidence that NSAIDs are more efficacious than placebo for the treatment of neck pain [25].
    - Pitfalls:
      1. Prescribing a prolonged course (>5 days) [12]
      2. Prescribing to patients at risk for peptic ulcer disease without co-prescribing a PPI or H2 blocker

3. Prescribing to patients with history of renal insufficiency
4. Prescribing to patients with elevated risk of cardiovascular disease
5. Prescribing to patients with NSAID allergy

– Skeletal muscle relaxants

Commonly prescribed muscle relaxants	Typical dosing
Cyclobenzaprine	5–10 mg PO every 8 h as needed
Methocarbamol	1,500 mg PO every 6 h for 2–3 days with maximum dose of 8 g/day and then reduce to 4 g/day
Carisoprodol	250–350 mg PO every 8 h and at hour of sleep

- Related to cyclic antidepressants and have multiple effects on neurotransmitter pathways resulting in sedation.
- One study showed that there was no benefit in pain control in prescribing NSAIDs plus muscle relaxants versus NSAIDs alone, but there was a higher rate of nervous system complications because of the central action of muscle relaxants [26].
- Pitfalls:

1. Skeletal muscle relaxants cause sedation as a class effect; patients must be cautioned about sedation as a probable side effect.
2. Cyclobenzaprine should not be prescribed to patients:
   – Who are taking or have taken a MAO inhibitor within the past 14 days
   – Who have hyperthyroidism
   – Who are recovering from a myocardial infarction, have cardiac conduction abnormalities, or have congestive heart failure
3. The dose of methocarbamol should be reduced in patients with hepatic impairment.

- Chronic musculoskeletal pain
  Acetaminophen, NSAIDs, and muscle relaxants should be prescribed at the lowest possible effective dose and for the shortest duration possible. They should not play a role in chronic musculoskeletal neck pain due to the adverse effects of long-term administration.

References

1. Binder AI. Cervical spondylosis and neck pain. BMJ. 2007;334(7592):527–31.
2. United States Bone and Joint Initiative. Summary of resource allocation of total health care occurrences for back pain and related conditions by sex, United States 2010. http://www.boneandjointburden.org/docs/T2.4.1.pdf. Accessed 17 Nov 2015.

3. Wexler R, Hefner JL, Sieck C, et al. Connecting emergency department patients to primary care. J Am Board Fam Med. 2015;28(6):722–32.
4. Paulus S, Kennedy DJ. Return to play considerations for cervical spine injuries in athletes. Phys Med Rehabil Clin N Am. 2014;25(4):723–33.
5. Akhaddar A, El-asri A, Boucetta M. Multiple isolated thoracic spinous process fractures (Clay-Shoveler's fracture). Spine J. 2011;11(5):458–9.
6. Erbulut DU. Biomechanics of neck injuries resulting from rear-end vehicle collisions. Turk Neurosurg. 2014;24(4):466–70.
7. Rudy IS, Poulos A, Owen L, et al. The correlation of radiographic findings and patient symptomatology in cervical degenerative joint disease: a cross-sectional study. Chiropr Man Ther. 2015;23:9.
8. Gabra N, Belair M, Ayad T. Retropharyngeal calcific tendinitis mimicking a retropharyngeal phlegmon. Case Rep Otolaryngol. 2013;2013:912628.
9. Mannoji C, Koda M, Furuya T, Aramomi M. Calcific tendinitis of the Longus Colli. Intern Med. 2015;54(12):1573.
10. Hermann KG, Bollow M. Rethinking diffuse idiopathic skeletal hyperostosis. Rheumatology (Oxford). 2014;53:1917–9.
11. Mader R. Clinical manifestations of diffuse idiopathic skeletal hyperostosis of the cervical spine. Semin Arthritis Rheum. 2002;32(2):130–5.
12. Brukner P, Khan K. Brukner & Khan's clinical sports medicine. 4th ed. Sydney: McGraw-Hill; 2012.
13. Noh HJ, de Freitas CA, de Paula ESFdSR, Simoes JC, Kosugi EM. Lemierre syndrome: a rare complication of pharyngotonsillitis. Braz J Otorhinolaryngol. 2015:568–570.
14. Schott CK, Counselman FL, Ashe AR. A pain in the neck: non-traumatic adult retropharyngeal abscess. J Emerg Med. 2013;44(2):329–31.
15. Berbari EF, Kanj SS, Kowalski TJ, et al. Executive summary: 2015 Infectious Diseases Society of America (IDSA) clinical practice guidelines for the diagnosis and treatment of native vertebral osteomyelitis in adults. Clin Infect Dis. 2015;61(6):859–63.
16. Thomas LC. Cervical arterial dissection: an overview and implications for manipulative therapy practice. Man Ther. 2015:2–9.
17. Shirakura Y, Fukuoka T. Man with sudden neck pain. J Emerg Med. 2015:e199–e200.
18. Berg J, Bjorck L, Dudas K, Lappas G, Rosengren A. Symptoms of a first acute myocardial infarction in women and men. Gend Med. 2009;6(3):454–62.
19. Gillick JL, Wainwright J, Das K. Rheumatoid arthritis and the cervical spine: a review on the role of surgery. Int J Rheumatol. 2015;2015:252456.
20. Roussou E, Sultana S. Spondyloarthritis in women: differences in disease onset, clinical presentation, and Bath Ankylosing Spondylitis Disease Activity and Functional indices (BASDAI and BASFI) between men and women with spondyloarthritides. Clin Rheumatol. 2011;30(1):121–7.
21. Nunes EA, Rosseti Jr AG, Ribeiro DS, Santiago M. Gout initially mimicking rheumatoid arthritis and later cervical spine involvement. Case Rep Rheumatol. 2014;2014:357826.
22. Orient JM, Sapira JD. Sapira's art & science of bedside diagnosis. 3rd ed. Philadelphia: Lippincott Williams & Wilkins; 2005.
23. Hoffman JR, Mower WR, Wolfson AB, Todd KH, Zucker MI. Validity of a set of clinical criteria to rule out injury to the cervical spine in patients with blunt trauma. National Emergency X-Radiography Utilization Study Group. N Engl J Med. 2000;343(2):94–9.
24. Awad BI, Carmody MA, Lubelski D, et al. Adjacent level ligamentous injury associated with traumatic cervical spine fractures: indications for imaging and implications for treatment. World Neurosurg. 2015;84(1):69–75.
25. Wong JJ, Cote P, Ameis A, et al. Are non-steroidal anti-inflammatory drugs effective for the management of neck pain and associated disorders, whiplash-associated disorders, or non-specific low back pain? A systematic review of systematic reviews by the Ontario Protocol for Traffic Injury Management (OPTIMa) Collaboration. Eur Spine J. 2015:34–61.
26. Turturro MA, Frater CR, D'Amico FJ. Cyclobenzaprine with ibuprofen versus ibuprofen alone in acute myofascial strain: a randomized, double-blind clinical trial. Ann Emerg Med. 2003;41(6):818–26.

Chapter 8
Dysphagia and Odynophagia

Charles Hwang, Bobby Desai, and Alpa Desai

8.1 Dysphagia

Dysphagia is a common problem encountered by primary care physicians. Dysphagia occurs in up to 30–40 % of patients in long-term care facilities and up to 50 % of elderly patients or patients with neurological conditions [1, 2]. It is not a benign process and may cause dehydration, weight loss, nutritional deficiencies, aspiration pneumonia, or airway obstruction [3, 4].

Dysphagia is objectively defined as the abnormal transit of solids and/or liquids from the mouth to the hypopharynx (oropharyngeal phase) or through the esophagus (esophageal phase) during swallowing [5, 6]. Subjectively, patients experience the sensation of a delay in transit. The subjective sensation and objective findings of dysphagia may be congruent or disproportionate; for example, patients with functional dysphagia may experience the sensation of transit delay despite objectively normal transit. Conversely, patients with decreased esophageal sensation may have true dysphagia but may not perceive the severity of their symptoms [5].

C. Hwang, MD (✉)
Department of Emergency Medicine, UF Health, Gainesville, FL, USA
e-mail: c7places@ufl.edu

B. Desai, MD
Emergency Medicine, University of Florida, Gainesville, FL, USA

A. Desai, DO
Community Health and Family Medicine, University of Florida, Newberry, FL, USA

B. Desai, A. Desai (eds.), *Primary Care for Emergency Physicians*,
DOI 10.1007/978-3-319-44360-7_8

Table 8.1 Esophageal dysphagia

Structural causes	Motor causes
Peptic stricture	Achalasia
Zenker's diverticulum	Nutcracker esophagus
Schatzki's ring	Diffuse esophageal spasm
Esophageal web	Scleroderma
Esophagitis	Functional
Neoplasm	
Aortic compression	
Lymphadenopathy	
Radiation-induced stricture	
Esophageal adenocarcinoma	
Squamous cell cancer	
Caustic medications or ingestions (bisphosphonates, tetracycline)	

8.1.1 Etiology

Dysphagia can be categorized in several ways. The location of dysphagia, oropharynx versus esophagus, is of significant importance because the potential pathology, and subsequent evaluation and management, varies by anatomical location.

The oropharyngeal phase of swallowing is mediated by a complex mechanism involving the cerebrum, brain stem, cranial nerves, and coordination between striated and smooth muscle, which are innervated by the somatic and autonomic (enteric) nervous systems, respectively. Therefore, *oropharyngeal dysphagia* typically involves broad multisystem musculoskeletal and nervous system pathology and affects three main populations – elderly patients, patients with neurological diseases, and patients with head and neck diseases. Examples of such pathologies include stroke, dementia, Parkinson's disease, myasthenia gravis, multiple sclerosis, botulism, progressive supranuclear palsy, polymyositis, amyotrophic lateral sclerosis, and muscular dystrophy, among other neuromuscular diseases. Local anatomic pathologies include abscess, neoplasm, thyromegaly, cervical hyperostosis, Zenker's diverticulum, post-head-and-neck surgical or radiation changes, and ingestion of caustic substances [1–3, 5–7].

Esophageal dysphagia refers to abnormal transit during the esophageal phase of swallowing, which can be further categorized into structural and motor abnormalities. In many conditions, however, dysphagia is mediated by both structural and motor pathologies. Structural abnormalities include disproportionate size and consistency of the food bolus or narrow esophageal lumen (i.e., Schatzki's rings, esophagitis, head-and-neck or esophageal neoplasms, peptic strictures, or extrinsic compression of the esophagus by lymphadenopathy or cancer). The luminal diameter of a normal esophagus is 2–3 cm; luminal diameters less than 1.3 cm can manifest in solid food esophageal dysphagia. Motor pathologies include abnormal peristaltic wave or inappropriate sphincter relaxation [2, 3, 5–7]. See Table 8.1 for causes of esophageal dysphagia.

The two most common causes of obstructive structural lesions are *Schatzki's rings* (located at the squamocolumnar junction) and *esophageal webs* (located in the upper esophagus). Both are thin membranous structures that obstruct the esophageal lumen. Solid food ingestion causes food impaction, resulting in dysphagia with associated symptoms of heartburn and chest pain [2]. *Esophageal strictures* and *esophagitis* develop from esophageal inflammation that ultimately results in fibrosis. Inflammatory causes may include peptic diseases, autoimmune diseases, infection, ingestion of caustic medications or substances, trauma, and radiation [2]. Infectious causes are typically those associated with immunosuppression, such as HIV/AIDS, *Candida albicans*, herpes simplex virus, cytomegalovirus, varicella zoster, and Epstein-Barr virus. *Zenker's diverticulum* is a herniation of the pharyngeal mucosa through the Killian triangle which can cause regurgitation of food, halitosis, and dysphagia of solid and liquid foods. Finally, *esophageal carcinoma* is characterized by the progressive dysphagia of solids to liquids over several weeks to months, accompanied by weight loss, anorexia, and anemia. Risk factors include smoking, alcohol abuse, and long-standing history of reflux [2].

Achalasia is characterized by elevated lower esophageal sphincter pressure and absence of distal esophageal peristalsis; this results in the stasis and regurgitation of ingested foods, the dilatation of the distal esophagus, and progressive liquid and solid food dysphagia that can result in coughing, aspiration, weight loss, and heartburn [2]. *Nutcracker esophagus* is the most common esophageal motility disorder, accounting for more than 40% of esophageal motility disorders. It is characterized by coordinated peristalsis of excessive amplitude, resulting in chest pain [2]. *Diffuse esophageal spasm* is characterized by uncoordinated contractions during peristalsis, preventing the normal forward transit of food. Symptoms are usually intermittent and can include globus sensation, heartburn, and regurgitation [2].

8.1.2 History

A careful history is of paramount importance in identifying the type, location, and etiology of dysphagia and helps to guide evaluation and treatment [3, 7]. Besides the important elements of onset, type of food, duration, severity, localization, progression, and associated symptoms, the category of dysphagia and specific causes/diseases should be evaluated. A broad differential is imperative as dysphagia is not limited to the gastrointestinal tract; for example, motor pathology may be caused by cranial nerve defects (pertinent questions include drooling, voice change, aspiration), and structural pathology may be caused by dilated aortic compression or spinal kyphosis [2, 3, 5].

Patients with oropharyngeal dysphagia have difficulty *initiating* swallowing, with associated symptoms of coughing, choking, drooling, regurgitation, and aspiration. Symptoms are localized to the oropharynx or hypopharynx. History of head-and-neck surgery or radiation, previous diagnosis of head-and-neck cancer, stroke, and neurological disorders are important elements of the past medical

history. As a result of their symptoms, patients may change their dietary habits, resulting in unexpected weight loss [3–5].

Patients with esophageal dysphagia experience food "sticking" in the throat, upper chest, suprasternal notch, or retrosternal area *after* swallowing. Constant dysphagia with solids and liquids is suggestive of motor pathology as the neuromuscular peristalsis affects both solids and liquids. Conversely, intermittent dysphagia with solids is suggestive of structural abnormalities. The principal symptoms of structural/anatomic causes of dysphagia occur primarily with ingestion of solids and not liquids; symptoms are aggravated with larger and denser solids [3, 5]. Progressive dysphagia over an extended period of weeks to months raises concern for neoplasms, whereas dysphagia that is episodic and unchanged over years suggests a benign disease process such as Schatzki's rings or esophagitis. Of note, the location and severity of solid food dysphagia is not necessarily proportional to the severity of luminal narrowing, as patients can modify and adapt their dietary habits to accommodate their dysphagia by avoiding certain foods, chewing foods fully, and mixing liquids with their solid meals [3, 5].

Chest pain or discomfort and heartburn frequently accompany dysphagia regardless of its etiology. Cardiac causes of chest pain (myocardial ischemia or infarction or acute coronary syndrome) should be ruled out before evaluating the patient for causes of dysphagia [2]. Associated odynophagia suggests inflammation in the upper gastrointestinal tract; common causes include infection, medication induced, or ulceration. Any immune system suppression can cause infectious esophagitis, and patients may have concurrent cardiac, respiratory, or abdominal symptoms of immunosuppression [2]. Various foods, toxins, and medications can cause direct mucosal injury or decrease lower esophageal sphincter tone. Table 8.2 contains a list of medications, foods, and toxins that can cause or worsen dysphagia [1–4].

Past medical history may include autoimmune diseases, such as CREST syndrome which can cause scleroderma. Patients with a history of gastritis, reflux, peptic ulcer disease, or caustic ingestion can have esophagitis or peptic strictures [2].

Other important considerations include compensatory and adaptation mechanisms that patients may develop to mitigate their symptoms. For example, patients may avoid certain foods, eat slower and be the last to finish a meal to decrease the severity of their dysphagia, or learn to drink fluids with solids to facilitate transit [2, 5, 6]. Ultimately, patients with food aversion or decreased oral intake may experience unexpected weight loss and dehydration [5].

8.1.3 Physical Exam

The examination may elucidate the cause of dysphagia. The primary goals of the physical examination are to (1) identify underlying systemic or metabolic disorders, (2) identify neurological disorders and their severity, and (3) to identify sequelae of dysphagia, including dehydration, metabolic derangements, or respiratory/pulmonary compromise. Therefore, the physical exam should include the head and neck, mouth, oropharynx, larynx, cardiopulmonary and nervous systems, and cranial nerves [4, 5].

Table 8.2 Medications and toxins that can affect swallowing

Sedation	Xerostomia
Benzodiazepines	Anticholinergics
Barbiturates	Antihypertensives
Neuroleptics	Antihistamines
Anticonvulsants	Antipsychotics
Myopathy	Anxiolytics
HMG-CoA reductase inhibitors (statins)	Antidepressants
Corticosteroids	Anticonvulsants
Impaired motility	Antiparkinsonian agents
Anticholinergics	Narcotics
Calcium channel blockers	Inflammation
Beta-blockers	Antibacterial agents such as tetracycline, doxycycline, clindamycin, trimethoprim-sulfamethoxazole, erythromycin
Nitrates	Iron
Botulinum toxin	Nonsteroidal anti-inflammatory drugs and aspirin
Phenothiazines	Corticosteroids
Amiodarone	Bisphosphonates
Procainamide	Zidovudine
Foods	Quinidine
Alcohol	Chemotherapeutic agents
Chocolate	
Peppermint	
Onion	

Vital signs may demonstrate fever, dehydration, or respiratory distress. The neurological exam should be centered around the suspected pathology, namely, to assess the mental status and identify cranial nerve deficits, proximal or asymmetric extremity weakness, fasciculations, reflexes, dysarthria, dysphonia, glossal atrophy, tremors, and cognitive dysfunction [2, 5]. The neurological assessment may identify impaired cognition, stroke, or other chronic illnesses.

The head and neck examination is necessary to evaluate dentition, oropharyngeal lesions, glossal fasciculations, signs of dehydration, palatal or uvular or glossal deviation, thyromegaly, and lymphadenopathy [2, 5]. Evaluate the abdomen for tenderness and masses. Positive fecal occult blood testing may indicate neoplastic disease or severe esophagitis. The skin may demonstrate changes of autoimmune or connective tissue diseases, such as scleroderma [2].

One of the easiest and most important parts of the physical exam is to watch the patient swallow. Oftentimes, patients may not recognize or may underestimate their difficulty in swallowing. Observation of swallowing may provide information with respect to the degree of dysphagia, especially if the patient takes multiple swallows for a single food bolus, if the patient takes sips of water after each food bolus, or if the patient regurgitates food after swallowing [5].

8.1.4 Further Studies

Although the history and physical can provide numerous clues to arrive at possible diagnoses, additional tests are frequently needed, and the provider can tailor the workup to further evaluate the clinical suspicion.

In patients with suspected oropharyngeal disorders, the videofluoroscopic swallowing study is the gold standard for evaluating the swallowing mechanism [3–5, 8]. In this study, patients eat and drink foods that are mixed with barium to make them radiopaque. It is similar to the barium swallow, but the barium swallow specifies small food boluses and does not include drinking from a cup [3]. Videofluoroscopic swallowing study evaluates the anatomic structures, the movement of these structures, how food transits during swallowing, and how effective compensatory maneuvers (neck flexion or breath holding) are during swallowing. In this way, it is possible to develop an individualized diet by determining which food consistency is best and minimizes aspiration [3].

For patients with suspected esophageal dysphagia, providers may consider performing a barium swallow, upper endoscopy, or esophageal manometry. It is important to understand these studies are not identical or redundant; rather, they may provide complementary information. The decision on which study to perform first should be based on which test is most likely to yield the diagnosis and/or treat the pathology [3–6, 8].

If the clinical picture is more consistent with motility dysfunction, a barium esophagram is the initial diagnostic test of choice. Barium esophagography allows for direct visualization of muscular function and coordination, evaluation of esophageal response to various food consistencies to replicate patients' symptoms, and evaluation of distal esophageal motility disorders including achalasia, scleroderma, or esophageal strictures, which are better evaluated by barium swallow as compared to endoscopy. Moreover, additional advantages of barium swallow are that it is noninvasive, inexpensive, can be done in patients that are poor candidates for upper endoscopy, does not require sedation, has few complications, and can help plan future endoscopy, along with any associated procedures including botulinum injection or pneumatic dilatation. Barium studies are contraindicated in patients with suspected complete or near complete obstruction, food impactions, and foreign body ingestions, for which endoscopy is diagnostic and therapeutic [2, 5, 6].

Upper endoscopy not only provides accurate information on esophageal anatomy, but it also allows for mucosal evaluation, the ability to biopsy, and the opportunity for therapeutic procedures if indicated [5]. By directly visualizing the gastrointestinal tract, endoscopy can diagnose intraluminal tumors, esophagitis, infection, food impaction, foreign body, and reflux disease, and it serves as a useful adjunct in the evaluation of motility disorders. Endoscopy is insensitive to diagnosing motility disorders, but contrary to barium esophagography, upper endoscopy has the opportunity for therapeutic potential either by pneumatic balloon dilatation, cautery, sclerotherapy, myotomy, or botulinum toxin injection [2, 6].

Table 8.3 Treatment of common causes of dysphagia

Achalasia – treated by reducing lower esophageal sphincter pressure by either pneumatic dilation or surgical myotomy. Pharmacologic therapy (botulinum toxin injection) may be used in patients with refractory symptoms or are unwilling to undergo dilation or myotomy
Diffuse esophageal spasm and nutcracker esophagus – acid suppression with PPI, sublingual nitroglycerin, and calcium channel blockers. Botulinum toxin injection may reduce symptoms
Scleroderma and other autoimmune diseases are best treated with systemic management of the disease. Acid suppression with PPI is recommended
Rings, webs, and strictures are best managed with lifestyle and behavioral modifications. Acid suppression with PPI is recommended. With large lesions and refractory cases, endoscopic dilation surgical rejection, or electrocautery may be considered
Reflux esophagitis and Barrett esophagus are best managed with lifestyle and behavioral modifications. Acid suppression with PPI or histamine-2 antagonists
Infectious esophagitis should be treated with the appropriate antiviral or antifungal agent. As infectious esophagitis is also commonly associated with immunosuppression, the disease should also be managed systemically
Esophageal carcinoma should be managed with chemotherapy, radiation, and/or surgery
Zenker's diverticulum can be managed via endoscopic cricopharyngeal myotomy

Esophageal manometry is currently the most sensitive technique and the gold standard in assessing and diagnosing esophageal motility disorders. Manometry, a diagnostic adjunct for patients with esophageal dysphagia, measures the peristaltic and sphincter function at intervals less than 1 cm apart along the entire esophagus [2–6, 8]. Recent evidence has demonstrated that the combination of manometry with impedance measurements is the most sensitive indicator of abnormal esophageal motility [5]. However, it is not a primary diagnostic study; rather, it is performed when a careful history, barium study, and endoscopy have not elucidated a diagnosis [2].

The greatest concern in patients presenting with dysphagia is the possibility of having cancer as the cause of their symptoms. The evaluation of a patient who is suspected or confirmed to have esophageal cancer includes the previously mentioned studies, including upper endoscopy and endoscopic biopsy and barium esophagram. However, additional imaging is used to stage the cancer, including computed tomography to evaluate the mass and surrounding structures. Endoscopic ultrasound can identify adjacent nodal metastases and can direct needle biopsies of suspected nodal disease [6].

8.1.5 Management

The management of dysphagia depends on the etiology and typically involves medications and/or surgery (Table 8.3) [2]. Low-cost, low-risk, efficacious interventions include dietary modification with softer and smaller foods, behavioral or postural changes, and referral to a speech and language pathologist to strengthen oropharyngeal muscle groups. Careful monitoring of hydration, electrolyte, and nutrition is

essential. Additional nutritional support may be warranted via parenteral or gastrostomy tube. Referral to specialist at an early stage may be indicated if the diagnosis remains unclear despite initial workup or if additional therapeutic expertise is required [2–5].

8.2 Odynophagia

It is important to distinguish dysphagia, which is the abnormal transit of solids and/or liquids, from odynophagia, which is pain during swallowing. As with dysphagia, odynophagia can be due to pain anywhere from the oropharynx to the esophagus. Odynophagia can also present alongside dysphagia, as the causes of dysphagia can also cause pain during swallowing.

8.2.1 Etiology

Pain during swallowing is due to inflammation of the gastrointestinal mucosa extending from the mouth to the distal esophagus. Inflammation can take the form of infection or local irritation. This topic will address the common causes of odynophagia. The treatment of each cause is beyond the scope of this chapter.

Common infectious causes include:

- Cytomegalovirus (CMV) esophagitis occurs in patients with immunocompromised states, such as posttransplantation, human immunodeficiency virus (HIV) infection or acquired immunodeficiency syndrome (AIDS), and corticosteroid therapy. Because of the systemic effects of immunosuppression, CMV may present with symptoms involving secondary organ systems unrelated to the esophagitis. Symptoms of CMV esophagitis may include nausea, vomiting, abdominal pain, odynophagia, dysphagia, weight loss, and retrosternal chest pain [9].
- Herpes simplex virus (HSV) esophagitis is commonly seen in immunocompromised patients, such as posttransplantation, corticosteroid therapy, HIV and AIDS, and post-chemotherapy. Symptoms include retrosternal chest pain, odynophagia, dysphagia, fever, weight loss, nausea, vomiting, and food impaction. Case reports have described gastrointestinal bleeding and the development of tracheoesophageal fistula as possible sequela of HSV esophagitis [10–13].
- Candidiasis (thrush) is also commonly seen in immunocompromised states. Patients with HIV who are not on highly active antiretroviral therapy (HAART) or who are on HAART but have not achieved immune reconstitution are at particular risk for candidiasis. The absence of oropharyngeal candidiasis does not rule out candidal esophagitis. However, the presence of oropharyngeal candidiasis carries a positive predictive value of 90 % for esophageal candidiasis [14].

- Pharyngitis is a common source of infection that causes sore throat and odynophagia and may be caused by viral (influenza, CMV, EBV, HSV, HIV) or bacterial (commonly group A streptococcus) causes. Other non-group A *Streptococcus* bacterial causes include other streptococci strains, *Corynebacterium diphtheriae*, and *Mycoplasma pneumoniae*, among others. The emergent conditions, however, include epiglottis, peritonsillar abscess, retropharyngeal abscess, and submandibular deep tissue infections. These conditions typically present with copious secretions, dysphonia, drooling, muffled "hot potato" voice, hoarseness, trismus, and neck or submandibular swelling. The clinician should have a high level of suspicion for these infections; if there is any suspicion, the patient should be emergently transferred to the emergency department for definitive care.
- Aphthous ulcers are recurrent, round, small, painful ulcers located in the oropharynx and usually last less than 2 weeks. The cause of these ulcers is poorly understood [15].

Causes of local irritation include:

- Esophageal foreign bodies may cause retrosternal or chest pain, dysphagia, globus sensation, drooling, stridor, wheezing, or choking. High-risk foreign bodies (i.e., sharp objects, batteries, magnets) or foreign bodies that have been present for extended periods of time can cause esophageal erosion, resulting in esophageal perforation, fever, pneumomediastinum, crepitus, or gastrointestinal bleeding.
- Local trauma to the esophagus can result from diagnostic investigations (i.e., endoscopy), drinking hot foods/drinks, ingesting caustic substances, reflux, or localized radiation in the treatment of cancer. Localized trauma can cause mucosal irritation and subsequent fibrosis, resulting in not only odynophagia but also dysphagia.

References

1. Clave P, Shaker R. Dysphagia: current reality and scope of the problem. Nat Rev Gastroenterol Hepatol. 2015;12(5):259–70.
2. Kruger D. Assessing esophageal dysphagia. JAAPA: Off J Am Acad Physician Assistants. 2014;27(5):23–30.
3. Palmer JB, Drennan JC, Baba M. Evaluation and treatment of swallowing impairments. Am Fam Physician. 2000;61(8):2453–62.
4. American Gastroenterological Association medical position statement on management of oropharyngeal dysphagia. Gastroenterology. 1999;116(2):452–54.
5. Abdel Jalil AA, Katzka DA, Castell DO. Approach to the patient with dysphagia. Am J Med. 2015;128(10):1138 e1117–1123.
6. Navaneethan U, Eubanks S. Approach to patients with esophageal dysphagia. Surg Clin North Am. 2015;95(3):483–9.
7. Hendrix TR. Art and science of history taking in the patient with difficulty swallowing. Dysphagia. 1993;8(2):69–73.
8. Lind CD. Dysphagia: evaluation and treatment. Gastroenterol Clin North Am. 2003;32(2):553–75.

9. Wilcox CM, Straub RF, Schwartz DA. Prospective evaluation of biopsy number for the diagnosis of viral esophagitis in patients with HIV infection and esophageal ulcer. Gastrointest Endosc. 1996;44(5):587–93.
10. Rattner HM, Cooper DJ, Zaman MB. Severe bleeding from herpes esophagitis. Am J Gastroenterol. 1985;80(7):523–5.
11. Cirillo NW, Lyon DT, Schuller AM. Tracheoesophageal fistula complicating herpes esophagitis in AIDS. Am J Gastroenterol. 1993;88(4):587–9.
12. Obrecht Jr WF, Richter JE, Olympio GA, Gelfand DW. Tracheoesophageal fistula: a serious complication of infectious esophagitis. Gastroenterology. 1984;87(5):1174–9.
13. Marshall JB, Smart 3rd JR, Elmer C, Lillich MA, Diaz-Arias AA. Herpes esophagitis causing an unsuspected esophageal food bolus impaction in an institutionalized patient. J Clin Gastroenterol. 1992;15(2):179–80.
14. Wilcox CM, Straub RF, Clark WS. Prospective evaluation of oropharyngeal findings in human immunodeficiency virus-infected patients with esophageal ulceration. Am J Gastroenterol. 1995;90(11):1938–41.
15. Ship JA. Recurrent aphthous stomatitis. An update. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 1996;81(2):141–7.

Chapter 9
Noncardiac Chest Pain (Including Chest Wall Pain)

Jason Jones

9.1 Introduction

Chest pain is of particular interest to emergency physicians, both for its high prevalence and its potential to reflect serious disease. Each year, seven million Americans seek emergency department care for chest pain, representing 5 % of all visits and the second most common presenting symptom overall [1].

Emergency physicians are perhaps most attuned to evaluating for cardiac causes of chest pain. Heart disease is the leading cause of death in the United States, and 1–4 % of patients presenting to the ED with chest pain are suffering an acute coronary syndrome or myocardial infarction [2, 3].

All chest pain investigations should at least consider the possibility of cardiac diseases as a cause. These include coronary artery disease, unstable angina, myocardial infarction, congestive heart failure, valvular disease, and aortic dissection. Several predictive tools are available to help risk stratify the likelihood of acute coronary conditions [4]. Potentially life-threatening noncardiac causes, including pneumothorax and pulmonary embolism, should also be considered.

Nevertheless, most chest pain is not life-threatening or cardiac in origin. In one meta-analysis, only 14–16 % of chest pain was cardiac [3]. In comparison, 25–50 % was musculoskeletal, 10–18 % pulmonary, 6–10 % gastrointestinal, and 10–18 % psychogenic. Recurrent noncardiac chest pain is especially common, affecting approximately one in four American adults [5]. Patients suffering from such pain are more likely to report a diminished quality of life and higher rates of depression and anxiety [6]. Thus, care must be given to investigate and treat noncardiac causes as well.

J. Jones, MD
Emergency Medicine, Shands Hospital at the University of Florida, Gainesville, FL, USA
e-mail: jasonjones@ufl.edu

B. Desai, A. Desai (eds.), *Primary Care for Emergency Physicians*,
DOI 10.1007/978-3-319-44360-7_9

9.2 Risk Factors for Chest Pain

Compared to patients with cardiac chest pain, patients with noncardiac chest pain are younger, smoke more tobacco, drink more alcohol, and have higher rates of anxiety [5].

- Hypertension
- Hyperlipidemia
- Tobacco use
- Alcohol use
- Diabetes
- Coronary artery disease
- Family history of coronary artery disease
- Increasing age
  - Likelihood of noncardiac chest pain decreases with age
  - Likelihood of cardiac chest pain increases with age
- Sedentary lifestyle
- Obesity
- Heartburn
- Acid reflux
- Dysphagia
- Emotional stress
- Depression
- Anxiety

9.3 Differential Diagnosis

- Cardiac chest pain
  - Ischemic
    - Stable angina
    - Unstable angina
    - Myocardial infarction
    - Congestive heart failure
  - Nonischemic
    - Pericarditis
    - Myocarditis
    - Valvular heart disease
    - Aortic dissection

- Noncardiac chest pain
  - Gastroesophageal
    - Gastroesophageal reflux disease (GERD)
    - Peptic ulcer disease
    - Gastritis and esophagitis
    - Esophageal spasm
    - Esophageal dysmotility
    - Pancreatitis
  - Pulmonary
    - Pneumonia
    - Bronchitis
    - Lung disease
      - COPD
      - Asthma
      - Restrictive lung disease
    - Lung cancer
      - Chest pain is a presenting symptom in 53 % [7].
    - Pneumothorax
    - Pulmonary embolism
    - Pleuritis
  - Musculoskeletal
    - Costochondritis
    - Tietze syndrome
    - Chest wall syndromes
    - Overuse injuries
    - Trauma
      - Rib fractures
      - Thoracic spinal fractures
      - Sternal fractures
    - Skin and soft tissue
      - Breast lesions
      - Herpes zoster
  - Psychiatric
    - Emotional stress
    - Hyperventilation

- Anxiety disorders
- Panic disorders
- Somatoform disorders

Red Flags of Chest Pain

- Cardiac chest pain red flags:
  - Signs of cardiac ischemia or infarction:
    - Exertional chest pain
    - Pain radiating to the arm, shoulder, neck, or jaw
    - Nausea
    - Diaphoresis
    - New EKG changes:
      - ST segment elevations
      - ST segment depressions
      - T wave inversions
      - Hyperacute T waves
      - Q waves
      - Conduction deficits
  - Murmur
  - Friction rub
  - Abnormal rhythm
  - Pulmonary edema
  - Sudden onset of acute ripping or tearing pain
    - May suggest aortic dissection
    - Incidence is 6 cases per 100,000 patient years [7]
- Noncardiac chest pain red flags:
  - Gastrointestinal
    - Repeated vomiting
    - Decreased appetite
    - Unintentional weight loss
    - Dysphagia or odynophagia
    - Hematemesis
    - Melena
  - Pulmonary
    - Acute, pleuritic chest pain with dyspnea, tachypnea, tachycardia, or hypoxia
      - Suggests pneumothorax
        - Acute pleuritic pain with dyspnea is present in 64–85 % of pneumothorax cases [7].

    - Dyspnea, tachycardia, clear lung sounds, cough, hemoptysis, or signs of deep vein thrombosis (unilateral limb swelling)
        - Suggests pulmonary embolism (PE).
        - Wells Score less than two and normal D-dimer assay excludes PE.
        - Patients with elevated Wells Score or D-dimer may require CT angiography or ventilation-perfusion scan.
    - Cough, sputum, fever, and tachypnea with signs of severe sepsis or septic shock
        - Suggests pneumonia with concerning features
    - Dyspnea, hoarseness, cough, hemoptysis, and unintentional weight loss, especially in a smoker
        - May suggest lung cancer
- Musculoskeletal
    - Severe mechanism
    - Poor air movement secondary to pain
- Psychiatric
    - Suicidal thoughts
    - Thoughts of self-harm

9.4 History

History is directed at detecting red flag symptoms and eliciting information that may clarify the timing, chronicity, severity, and source of the patient's chest pain.

Important historical features to elicit include:

- Characteristics of chest pain
    - Duration
        - Acute vs. chronic
    - Quality
    - Time course
    - Location
    - Possible inciting factors
    - Alleviating and aggravating factors
- Other acute medical complaints
    - Dyspnea
    - Palpitations

  - Diaphoresis
  - Cough or sputum
  - Wheezing
  - Fever
  - Heartburn
  - Nausea or vomiting
  - Weight loss
  - Anxiety

- Past medical history
- Family history (especially of early myocardial infarctions)
- Use of medications or drugs (including caffeine, alcohol, tobacco, and supplements)
- Psychiatric history

9.5 Physical Examination

The physical examination should focus on potential emergency conditions.

- Vital sign assessment
  - Fever or hypothermia may suggest an infectious etiology.
  - Hypoxia may indicate a pulmonary or vascular etiology.
  - Tachycardia.
  - Pulse oximetry can detect hypoxia.
- Cardiovascular assessment should focus on:
  - Heart sounds
  - Regular or irregular rhythm
  - Murmurs, friction rub, or gallop rhythms
  - Jugular venous distention (JVD)
  - Peripheral edema or swelling
  - Pulse differential in the upper extremities
- Pulmonary assessment should focus on:
  - Cyanosis
  - Lung sounds
    - Crackles
    - Wheezes
    - Rhonchi
    - Stridor
    - Egophony

  - Dullness to percussion
  - Unilateral absence or decrease of lung sounds
    - May suggest pneumothorax
  - Reproducible pain
- Musculoskeletal assessment should focus on:
  - Localized tenderness
  - Swelling
  - Skin lesions
  - Reproducible pain
- Gastrointestinal assessment should focus on:
  - Epigastric tenderness
  - Vomiting
  - Heme-positive stool (if pertinent)
  - Signs of anemia
- Psychiatric assessment should focus on:
  - Hyperventilation
  - Suicidal thoughts
  - Thoughts of self-harm
  - Anxiety or panic states

9.6 Introduction to Treatment

After evaluation and stabilization of emergency conditions, the emergency physician has several options to treat chest pain. These include:

1. Education and lifestyle modification
   (a) Tobacco and alcohol cessation
   (b) Avoidance of caffeine, stimulants, and fresh citrus juice
   (c) Minimization of opioids and benzodiazepines
   (d) Home medication compliance
   (e) Exercise and dietary modifications
   (f) Stress-reduction techniques
2. Medications
   (a) Analgesics for pain relief
      (i) Aspirin
      (ii) Nonsteroidal anti-inflammatory drugs (NSAIDs)
      (iii) Opioids

(b) Anti-inflammatory medications

(i) Nonsteroidal anti-inflammatory drugs (NSAIDs)
(ii) Aspirin

(c) Anti-reflux medications for gastrointestinal pain

(i) Proton pump inhibitors (PPIs)
(ii) H2 receptor antagonists

(d) Antibiotics for infections (e.g., pneumonia)
(e) Psychiatric medications for anxiety, depression, or pain modulation

(i) Includes benzodiazepines and antidepressants.
(ii) Many emergency physicians will defer initiation of psychiatric medications to a psychiatrist or primary care physician.

3. Referral and follow-up

(a) Primary care physician
(b) Cardiology for suspected coronary artery disease
(c) Gastroenterology

(i) Lifestyle modifications
(ii) Upper endoscopy
(iii) Esophageal manometry
(iv) Empiric PPI trial
(v) Muscle relaxant trial
(vi) Pain modulators
(vii) Botulinum toxin
(viii) Surgical or endoscopic therapy

(d) Psychiatry or psychology

9.7 Pharmacologic Treatment

9.7.1 Nonsteroidal Anti-inflammatory Drugs (NSAIDs)

Provide immediate pain relief and anti-inflammatory effect.

Side effects of these medications include gastrointestinal ulcers, bleeding, and acute kidney injury.

Examples of NSAIDs
Aspirin
Ibuprofen
Naproxen
Ketorolac
Meloxicam
Indomethacin
Ketoprofen

Pitfalls
1. Prescribing to patients at risk for bleeding
2. Prescribing to patients with kidney disease
3. Prescribing long-term doses
4. Failing to suggest to take with food

9.7.2 Opioid Analgesics

Provide potent and immediate pain relief.

Side effects of these medications include gastrointestinal ulcers, bleeding, and acute kidney injury.

Examples of opioids
Codeine
Fentanyl
Hydrocodone
Morphine
Oxycodone

Pitfalls
1. Sedation
2. Respiratory depression and apnea
3. No anti-inflammatory effect
4. Constipation
5. Dependence, addiction, and withdrawal
6. Risk of overdose

9.7.3 Proton Pump Inhibitors (PPIs)

Provide long-lasting reduction of gastric acid production.

GERD is present in up to 60% of patients with noncardiac chest pain [8]. Acid reflux causes symptoms of heartburn in some patients and chest pain in others. In patients with suspicion of GERD, gastroenterologists recommend that a proton pump inhibitor (PPI) be initiated at a high dose and tapered to the lowest dose that maintains symptomatic relief. Along with lifestyle modifications, this technique has a high success rate [5].

Examples of proton pump inhibitors (PPIs)
Esomeprazole
Lansoprazole
Omeprazole
Pantoprazole
Rabeprazole

Pitfalls
1. Failure to taper dose once relief is achieved.
2. Some studies show an association between PPIs and diarrheal infection or pneumonia.

9.7.4 H2 Receptor Antagonists (H2 Blockers)

Provide long-lasting reduction of gastric acid production.

H2 blockers are less effective than PPIs in reducing GERD-related noncardiac chest pain [5]. They successfully control the symptoms of GERD-related noncardiac chest pain in 42–52 % of patients [9].

Examples of H2 receptor antagonists
Cimetidine
Famotidine
Ranitidine

Pitfalls
1. Generally well tolerated.
2. Some studies show an association between PPIs and diarrheal infection or pneumonia.

9.7.5 Benzodiazepines

These are a class of psychoactive medications producing immediate short-term relief of anxiety or panic disorder.

A short course of this class of medication may be considered in consultation with the patient's primary care physician or psychiatrist.

Examples of benzodiazepines
Alprazolam
Chlordiazepoxide
Clonazepam
Diazepam
Lorazepam

Pitfalls
1. Respiratory depression or apnea
2. Sedation
3. Dependence, addiction, and withdrawal
4. Risk of overdose

9.7.6 Antidepressants

A short course of this class of medication may be considered in consultation with the patient's primary care physician or psychiatrist.

Examples of antidepressants
Amitriptyline
Bupropion
Citalopram
Duloxetine
Escitalopram
Fluoxetine
Paroxetine
Sertraline
Venlafaxine

Pitfalls

1. Use in pregnancy
2. Anticholinergic effects
3. Sedation
4. Weight changes
5. Sexual dysfunction
6. May paradoxically increase risk of suicide

References

1. National Hospital Ambulatory Medical Care Survey: 2011 Emergency Department Summary Tables. http://www.cdc.gov/nchs/data/ahcd/nhamcs_emergency/2011_ed_web_tables.pdf. Accessed 21 Feb 2016.
2. Ebell MH. Evaluation of chest pain in primary care patients. Am Fam Physician. 2011;83(5):603–5.
3. Haasenritter J, Biroga T, Keunecke C, et al. Causes of chest pain in primary care – a systematic review and meta-analysis. Croat Med J. 2010;56(5):422–30.
4. Ayerbe L, González E, Gallo V, et al. Clinical assessment of patients with chest pain; a systematic review of predictive tools. BMC Cardiovasc Disord. 2016;6(1):18.
5. Schey R, Villarreal A, Fass R. Noncardiac chest pain: current treatment. Gastroenterol Hepatol. 2007;3(4):255–62.
6. Eslick GD, Jones MP, Talley NJ. Non-cardiac chest pain: prevalence, risk factors, impact and consulting — a population-based study. Aliment Pharmacol Ther. 2003;17(9):1115–24.
7. Yelland M, Cayley Jr WE, Vach W. An algorithm for the diagnosis and management of chest pain in primary care. Med Clin N Am. 2010;94(2):349–74.
8. Liuzzo JP, Ambrose JA. Chest pain from gastroesophageal reflux disease in patients with coronary artery disease. Cardiol Rev. 2005;13:167–73.
9. Fang J, Bjorkman D. A critical approach to noncardiac chest pain: pathophysiology, diagnosis, and treatment. Am J Gastroenterol. 2001;96:958–68.

Chapter 10
Hypertension

Mohammad Reza Mohebbi and Sara Tehranchian

10.1 Introduction

Hypertension is a very common condition seen in primary care and may lead to myocardial infarction, stroke, renal failure, and death if not treated [1]. Abundant evidence has shown benefit of antihypertensive treatment in reducing adverse health outcomes in patients with hypertension [1]. Most asymptomatic patients with poorly controlled hypertension do not have acute target organ damage and, therefore, immediate workup or treatment is not necessary. When a patient presents with severely elevated blood pressure (BP), the emergency physician need to differentiate hypertensive emergency from asymptomatic elevated BP with potential risk factors for target organ damage. Patients who present to the emergency department (ED) with asymptomatic hypertension will need outpatient follow-up and medical treatment. However, some patients may have an underlying emergent pathology that may require investigation, and that is where the emergency physician should be able to determine if this is a true emergency or/and has the potential of an adverse outcome.

M.R. Mohebbi, MD (✉)
Department of Emergency Medicine, University of Missouri-Columbia, Columbia, MO, USA
e-mail: mrmohebbi@gmail.com

S. Tehranchian, PharmD
Napa State Hospital, Napa, CA, USA

B. Desai, A. Desai (eds.), *Primary Care for Emergency Physicians*,
DOI 10.1007/978-3-319-44360-7_10

10.2 Definition

10.2.1 Hypertension

Per the Seventh Report of the Joint National Committee on the Prevention, Detection, Evaluation, and Treatment of High BP (JNC7) [2]:

- Normal BP: SBP <120 mmHg or DBP <80 mmHg
- Prehypertension: SBP 120–139 or DBP 80–89
- Stage 1 hypertension: SBP 140–159 or DBP 90–99
- Stage 2 hypertension: SBP ≥160, or DBP ≥100

JNC8 however does not define hypertension and prehypertension but defines the thresholds for pharmacologic treatment (see treatment) [1].

There is no universal set limit for severe hypertension in adults. Severe hypertension in adults is defined as SBP ≥180 mmHg and/or DBP ≥110 or 120 mmHg in the literature [3, 4].

Other terms such as hypertensive urgency, hypertensive crisis, etc. have been used in the literature with some ambiguity. Rosen's emergency medicine textbook suggests a more practical classification for management of hypertension at the ED [5]:

- *Hypertensive emergency*:
  - Significantly elevated BP with signs or symptoms of acute, ongoing target organ damage.
  - Acute target organ damage in the setting of a hypertensive emergency may include the following:
    - Neurologic: hemorrhagic or ischemic cerebral vascular accident, hypertensive encephalopathy.
    - Cardiovascular: myocardial ischemia, acute pulmonary edema, aortic dissection, acute left ventricular dysfunction, and unstable angina pectoris.
    - Other conditions such as acute renal failure, retinopathy, eclampsia, microangiopathic hemolytic anemia [6].
- *Poorly controlled chronic hypertension*:
  - Elevated BP in a patient with established hypertension without attributable symptoms or evidence of acute target organ damage.
- *Elevated BP without prior history of hypertension*:
  - Although the majority of these patients will end up being diagnosed with hypertension, the diagnosis cannot be established with one ED visit.

10.3 Etiology and Risk Factors

Non-modifiable

- Genetics (twice as much if one or two parents hypertensive).
- Race (African Americans at higher risk).
- Age (advanced age associated with increased risk).
- Gender (male>female).
- Reduced adult nephron mass may predispose to hypertension, which may be genetic or acquired (hypoxia, drugs, nutritional deficiency), premature birth, or infection.

Modifiable

- Being overweight
- High sodium intake
- Excessive alcohol intake
- Sedentary lifestyle
- High stress
- Smoking
- Diabetes
- Hyperlipidemia
- Personality traits and depression

10.4 Differential Diagnosis and Underlying Causes [2]

Cardiovascular

- Primary hypertension
- Acute coronary syndrome
- Aortic dissection
- Coarctation of aorta
- Vasculitis
- Heart failure
- Collagen vascular disease

Renal

- Acute/chronic renal failure
- Glomerulonephritis
- Renal artery stenosis,

Endocrine/Electrolytes

- Pheochromocytoma
- Cushing syndrome
- Hyperthyroidism
- Primary aldosteronism
- Congenital adrenal hyperplasia
- Acromegaly
- Hypercalcemia

Respiratory

- Sleep apnea
- Respiratory distress

Psych/CNS

- Anxiety
- White coat syndrome
- Ischemic/hemorrhagic stroke
- Brain tumor
- Bulbar poliomyelitis
- Intracranial hypertension

Obstetrics

- Pregnancy-induced hypertension
- Preeclampsia and eclampsia

Drug/Medication Related

- Rebound hypertension (abrupt stopping of antihypertensive medications)
- Over-the-counter medications:
  - NSAIDs, decongestants containing ephedrine
- Immunosuppressants:
  - Cyclosporine, tacrolimus
- Recreational sympathomimetic drugs
  - Cocaine, amphetamines, phencyclidine, etc.
- Herbals:
  - St. John's Wort
- Industrial chemicals:
  - Lead, mercury, thallium, and lithium salts
- Alcohol withdrawal
- Antipsychotics, antidepressants, and anxiolytics (clozapine, venlafaxine, buspirone, etc.)
- Serotonin syndrome

- Cortisone and other steroids
- Estrogens
- Weight loss medications
- Erythropoietin
- Metoclopramide
- Ketamine

10.5 History

ED physicians should always look for potential acute, ongoing, target organ damage to rule out hypertensive emergency. A quick history should determine whether any of the following are present [7]:

- Recent head trauma
- Generalized neurologic symptoms
  - Mental status alteration
  - Agitation
  - Delirium
  - Seizure
- Nausea and vomiting
  - May be a sign of increased intracranial pressure
- Focal neurologic symptoms
  - May be a sign of ischemic or hemorrhagic stroke
- Chest discomfort
  - May be a sign of myocardial ischemia or aortic dissection
- Dyspnea
  - May be a sign of pulmonary edema
  - May be a sign of pulmonary embolism
- Acute, severe back pain
  - May be a sign of aortic dissection
- Pregnancy
  - Preeclampsia
  - Increased risk of aortic dissection in pregnancy
- Sympathomimetic drugs
  - Cocaine
  - Amphetamine(s)
  - Phencyclidine
  - Monoamine oxidase inhibitors

- Discontinuation of sympatholytic agents
- Non-adherence to current antihypertensives

10.6 Physical Exam

Physicians should look for symptoms or signs suggestive of secondary causes of hypertension or presence of end-organ damage [8].

Important aspects of the physical examination in the hypertensive [9]:

- Accurate measurement of BP
- Orthostatic vital signs in older patients and in patients with diabetes or suspected postural hypotension
- General appearance
  - Distribution of body fat
  - Skin lesions
  - Muscle strength
  - Alertness
- Fundoscopy
  - Hemorrhage
  - Papilledema
  - Cotton wool spots
- Neck
  - Palpation and auscultation of carotids
  - Thyroid
- Heart
  - Size
  - Rhythm
  - Sounds
- Lungs
  - Rhonchi
  - Rales
- Abdomen
  - Renal masses
  - Bruits over aorta or renal arteries
  - Femoral pulses
- Extremities
  - Peripheral pulses
  - Edema

- Neurologic assessment
  - Visual disturbance
  - Focal weakness
  - Confusion

10.7 Work Up

Reviewing the medical literature shows no consensus about the laboratory workup necessary for patients with severe asymptomatic hypertension. The JNC7 recommends work up only before starting the treatment in newly diagnosed patients [2, 8]. Per the American College of Emergency Physicians 2013 clinical policy, in ED patients with asymptomatic markedly elevated BP, routine screening such as serum creatinine, urinalysis and ECG is not required. In certain patient populations such as those with poor follow-up, screening for an elevated serum creatinine level may identify renal injury which may affects making a decision for disposition [10].

In case of suspicion for hypertensive emergency, clinical judgment must be used to order appropriate work up such as CMP, CBC, UA, ECG, cardiac enzymes (R/O acute coronary syndrome), drug screening, chest X-ray, head CT (R/O intracranial hemorrhage) or other imaging modalities are recommended if needed.

10.8 Treatment

10.8.1 Management of Hypertension in Primary Care Setting

10.8.1.1 Nonpharmacologic Treatment

All hypertensive patients should undergo appropriate nonpharmacologic (lifestyle) modification. Smoking cessation has shown to reduce overall cardiovascular risk (Table 10.1) [2].

10.8.1.2 Pharmacologic Treatment

Initiate pharmacologic treatment if [1]*:

- General population age ≥60 years if SBP ≥150 or DBP ≥90
  - Goal: SBP <150 and DBP <90
- General population age <60 years *or* age ≥18 years with chronic kidney disease (CKD) *or* age ≥18 years with diabetes if SBP ≥140 or DBP ≥90
  - Goal: SBP <140 and DBP <90

Table 10.1 Lifestyle modifications to prevent and manage hypertension

Modification	Recommendation	Approximate systolic BP reduction, range
Weight reduction	Maintain normal body weight (BMI, 18.5–24.9 kg/m^2)	5–20 mmHg per 10 kg weight loss
Adopt dietary approaches to stop hypertension	Consume a diet rich in fruits, vegetables, and low-fat dairy products with a reduced content of saturated and total fat	8–14 mmHg
Dietary sodium reduction	Reduce dietary sodium intake to no more than 100 meq/day (2.4 g sodium or 6 g sodium chloride)	2–8 mmHg
Physical activity	Engage in regular aerobic physical activity such as brisk walking (at least 30 min per day, most days of the week)	4–9 mmHg
Moderation of alcohol consumption	Limit consumption to no more than two drinks (e.g., 24 oz beer, 10 oz wine, or 3 oz 80-proof whiskey) per day in most men and to no more than one drink per day in women and lighter-weight persons	2–4 mmHg

Reproduced from JNC7 [2]

Initial antihypertensive treatment should include [1]:

- In general nonblack population:
  - A thiazide diuretic, calcium channel blocker (CCB), angiotensin-converting enzyme (ACE) inhibitor, *or* angiotensin II receptor blockers (ARB) alone or in combination
- In the general black population:
  - A thiazide diuretic or CCB alone or in combination
- In patients with chronic kidney disease older than 18 years old:
  - ACE inhibitor or ARBs alone or in combination with other drug classes
- In patients with heart failure:
  - Thiazide diuretics, ACE inhibitors, and CCBs are more effective, respectively.
- JNC8 does not recommend α-blockers and β-blockers for the initial treatment of hypertension.

If the target BP is not reached within 1 month after starting treatment, either the dosage should be increased, or a second medication should be added (thiazide, CCB, ACE inhibitor, or ARB).

ACE inhibitors and ARBs should not be used together in the same patient.

**A recent NIH sponsored trial (SPRINT trial) suggests a lower SBP goal of <120 mmHg for patients ≥50 at high risk for cardiovascular events but without diabetes* [11].

10.8.2 Antihypertensive Medications

Evidence-based dosing for antihypertensive drugs.*

10.8.3 ACE Inhibitors

Medication	Initial daily dose, mg	Target dose, mg	No. of doses per day
Captopril	50	150–200	2
Enalapril	5	20	1–2
Lisinopril	10	40	1

Pitfalls

- Contraindicated in pregnancy, caution in women of child-bearing age.
- May cause a chronic cough.
- May cause life-threatening angioedema of head and neck.
- Avoid in bilateral renal stenosis/renal impairment.
- May cause high normal potassium or hyperkalemia.
- May cause increase in serum creatinine.
- Captopril may cause neutropenia with myeloid hypoplasia and agranulocytosis especially in patient with renal impairment.

10.8.4 Angiotensin Receptor Blockers

Medication	Initial daily dose, mg	Target dose, mg	No. of doses per day
Eprosartan	400	600–800	1–2
Candesartan	4	12–32	1
Losartan	50	100	1–2
Valsartan	40–80	160–320	1
Irbesartan	75	300	1

Pitfalls

- Contraindicated in pregnancy, caution in women of child-bearing age
- Avoid in bilateral renal stenosis
- May cause angioedema
- May cause high normal potassium or hyperkalemia
- May cause increase in serum creatinine

10.8.5 β-Blockers

Medication	Initial daily dose, mg	Target dose, mg	No. of doses per day
Atenolol	25–50	100	1
Metoprolol	50	100–200	1–2

Pitfalls
- Abrupt stopping may cause tachyarrhythmia and potential cardiac ischemia.
- JNC8 does not recommend β-blockers for the initial treatment of hypertension.
- Bronchospasm in asthma or COPD (nonselective agents).

10.8.6 Calcium Channel Blockers

Medication	Initial daily dose, mg	Target dose, mg	No. of doses per day
Amlodipine	2.5	10	1
Diltiazem XR	120–180	360	1
Nitrendipine	10	20	1–2

Pitfalls
- May cause second- and third-degree heart block, left ventricular dysfunction
- May cause peripheral edema

10.8.7 Thiazide-Type Diuretics

Medication	Initial daily dose, mg	Target dose, mg	No. of doses per day
Chlorthalidone	12.5	12.5–25	1
Hydrochlorothiazide	12.5–25	25–50	1–2
Indapamide	1.25	1.25–2.5	1

Pitfalls
- May cause electrolyte imbalance (hypokalemia, hyponatremia, hypercalcemia).
- Check BUN/Cr and electrolytes at baseline and make sure patient follows up.
- Usually ineffective with CrCl <30 mL/min unless in combination with a loop diuretic with the exception of Chlorthalidone that is effective with CrCl ≥10 mL/min.

**The abovementioned list and dosing is adopted from JNC8 based on the randomized clinical trials reviewed by JNC8 and does not include other medications or drug classes that are not recommended as first-line therapy by JNC8* [1].

Many people will require treatment with more than one antihypertensive drug to achieve BP control. JNC8 suggests that any of the four classes (ACE inhibitors, ARB, CCB, and thiazide diuretics) would be good choices as add-on agents. Also the JNC8 recommendation is specific for thiazide-type diuretics and does not include loop or potassium-sparing diuretics [1].

10.8.8 Management of Hypertension at the ED

10.8.8.1 Asymptomatic Hypertension

Managing asymptomatic hypertension at the ED is different from the primary care setting. Rapidly lowering BP in the ED is usually unnecessary in asymptomatic patients and may be harmful. In most cases, no intervention is necessary and an outpatient follow-up should be scheduled with the primary care provider. Hypertensive emergencies, however, require admission and immediate treatment. Even in the emergent setting, BP should not be acutely lowered due to the risk of hypoperfusion. American College of Emergency Physicians 2013 clinical policy has the following recommendations for management of asymptomatic hypertension:

- In patients with asymptomatic markedly elevated BP, routine ED medical intervention is not required. In select patient populations (eg, poor follow-up), emergency physicians may treat markedly elevated BP in the ED and/or initiate therapy for long-term control. Patients with asymptomatic markedly elevated BP should be referred for outpatient follow-up [10].

10.8.8.2 Hypertensive Emergency

In hypertensive emergencies, the BP should be aggressively lowered within minutes to an hour by no more than 25 % and then lowered to 160/100–110 mmHg within the next 2–6 h [2, 6].

10.8.8.3 IV Agents for Management of Hypertensive Emergencies [5, 12]

Indication and treatment goal	Agents (*figures in parenthesis show infusion rates*)	Pitfalls
Acute coronary syndrome: *to diminish cardiac workload and improve coronary artery perfusion*	**Primary: nitroglycerin (5–200 µg/min)** Secondary: metoprolol 5 mg q5 min, labetalol 20–80 mg q 10 min (1–2 mg/min) *Alternative*: *esmolol*, *nicardipine*	Routine use of beta-blockers is controversial
Acute heart failure: *to reduce impedance to forward flow and diminish cardiac workload*	**Primary: nitroglycerin (5–200 µg/min), furosemide 40–240 mg q12 h (10–40 mg/h)** Secondary: enalaprilat 0.625–1.25 mg q15 min (1–2 mg/h) *Alternative*: *clevidipine*, *nicardipine*, *sodium nitroprusside*	Intubation or noninvasive ventilatory support decreases preload and may drop BP. Enalaprilat may cause sustained hypotension

Indication and treatment goal	Agents (*figures in parenthesis show infusion rates*)	Pitfalls
Aortic dissection: *to reduce shear force*	**Primary: esmolol 0.5–1 mg/kg ×1 (50–300 µg/kg/min)+ sodium nitroprusside (1–2 mg q5 min (0.25–10 µg/kg/min)** Secondary: labetalol 20–80 mg q 10 min (1–2 mg/min) *Alternative*: *esmolol*+(*clevidipine or nicardipine*), *diltiazem*, *verapamil*	Avoid beta-blockers if aortic regurgitation is present
Acute ischemic stroke: *to reduce hemorrhagic conversion and edema while maintaining perfusion*	**Primary: nicardipine (5–15 mg/h)** Secondary: : labetalol 20–80 mg q 10 min (1–2 mg/min) *Alternative*: *esmolol*	Permissive hypertension. Acute BP reduction only indicated if giving tPA or secondary target organ dysfunction or BP >220/110. Nitric oxide donors and hydralazine should be avoided
Acute intracerebral hemorrhage: *to reduce hematoma expansion and edema*	**Primary: nicardipine (5–15 mg/h)** Secondary: labetalol 20–80 mg q 10 min (1–2 mg/min) *Alternative*: *esmolol*	Nitric oxide donors and hydralazine should be avoided
Hypertensive encephalopathy: *to decrease edema*, *reduce intracranial pressure*, *and improve autoregulatory control*	**Primary: nicardipine (5–15 mg/h)** Secondary: labetalol 20–80 mg q 10 min (1–2 mg/min) labetalol 20–80 mg q 10 min (1–2 mg/min) *Alternative*: *esmolol*, *enalaprilat*	Nitric oxide donors and hydralazine should be avoided
Acute kidney injury: *to decrease renal parenchyma and glomerular pressure*	**Primary: fenoldopam (0.1–0.3 µg/kg/min)** Secondary: clevidipine (2–32 µg/h), nicardipine (5–15 mg/h) *Alternative*: *labetalol*, *sodium nitroprusside*	ACE inhibitors and diuretics should be avoided
Preeclampsia and eclampsia: *to decrease intracranial pressure while maintaining placental perfusion*	**Primary: hydralazine 5–20 mg q30 min (1.5–5 µg/kg/min)** Secondary: : labetalol 20–80 mg q 10 min (1–2 mg/min) *Alternative*: *Nicardipine*	IV magnesium (6 g initial dose) administered in all cases. Cesarean section is the definitive treatment
Sympathetic crisis: *to reduce alpha-1 adrenergic receptor-mediated vasoconstriction*	**Primary: phentolamine 5–15 mg q5 min (0.2–0.5 mg/min)** Secondary: nitroglycerin (5–200 µg/min) *Alternative*: *fenoldopam*, *clevidipine*, *nicardipine*, *sodium nitroprusside*	Benzodiazepines are first-line therapy when caused by cocaine or amphetamines Beta-blocker monotherapy contraindicated

Adapted from Rosen's Textbook of Emergency Medicine [5] and Koda-Kimble & Young's Applied Therapeutics [12]

10.9 Disposition

Lowering BP in the ED is usually unnecessary in asymptomatic patients. We recommend the American College of Emergency Physicians 2013 clinical policy as mentioned above. In most cases, no intervention is necessary and an outpatient follow-up should be scheduled with the primary care provider. Hypertensive emergencies, however, require admission, most often in an intensive care unit.

References

1. James PA, Oparil S, Carter BL, Cushman WC, Dennison-Himmelfarb C, Handler J, et al. 2014 evidence-based guideline for the management of high BP in adults: report from the panel members appointed to the Eighth Joint National Committee (JNC8). JAMA. 2014;311(5):507–20.
2. Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL Jr, et al. National Heart, Lung, and Blood Institute Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High BP; National High BP Education Program Coordinating Committee. The seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High BP: the JNC 7 report. JAMA. 2003;289(19):2560–72.
3. ESH/ESC Task Force for the Management of Arterial Hypertension. Practice guidelines for the management of arterial hypertension of the European Society of Hypertension (ESH) and the European Society of Cardiology (ESC): ESH/ESC Task Force for the Management of Arterial Hypertension. J Hypertens. 2013;31(10):1925–38.
4. Shayne PH, Pitts SR. Severely increased BP in the emergency department. Ann Emerg Med. 2003;41(4):513–29.
5. Marx J, Hockberger R, Walls R. Rosen's emergency medicine – concepts and clinical practice, 8th ed. Philadelphia: Elsevier/Saunders; 2014. p. 1113–1123.
6. Madhur MS, Riaz K, Dreisbach AW, Harrison DG. Hypertension. In Medscape. Editor: Maron DJ. At: http://emedicine.medscape.com/article/241381-overview. Accessed 19 July 2016.
7. Johnson W, Nguyen ML, Patel R. Hypertension crisis in the emergency department. Cardiol Clin. 2012;30(4):533–43.
8. Kessler CS, Joudeh Y. Evaluation and treatment of severe asymptomatic hypertension. Am Fam Physician. 2010;81(4):470–6.
9. Basile J, Bloch MJ. Overview of hypertension in adults. In: UpTODate, Bakris GL, Kaplan NM, editors. UpToDate, Waltham. At: http://www.uptodate.com/contents/overview-of-hypertension-in-adults. Accessed 10 Dec 2015.
10. Wolf SJ, Lo B, Shih RD, Smith MD, Fesmire FM, American College of Emergency Physicians Clinical Policies Committee. Clinical policy: critical issues in the evaluation and management of adult patients in the emergency department with asymptomatic elevated BP. Ann Emerg Med. 2013;62(1):59–68.
11. SPRINT Research Group, Wright Jr JT, Williamson JD, Whelton PK, Snyder JK, Sink KM, Rocco MV, et al. A randomized trial of intensive versus standard blood-pressure control. N Engl J Med. 2015;373(22):2103–16.
12. Alldredge BK, Corelli RL, Ernst ME, Guglielmo BJ, Jacobson PA Kradjan WA, Williams BR. Koda-Kimble and Young's applied therapeutics: the clinical use of drugs, 10th ed. Philadelphia: Lippincott Williams & Wilkins; 2013. p. 520–35.

Chapter 11
Gastroesophageal Reflux Disease (GERD)

Tara Dyson

11.1 Introduction

Gastroesophageal reflux disease (GERD) is the reflux of gastric content into the esophagus causing discomfort. It is the most commonly diagnosed GI disorder. The exact prevalence of the disease is unclear as the most common symptom, heartburn, is not ubiquitous which will underestimate the prevalence. Additionally, a large subset of patients will have no subjective symptoms at all but will have objective evidence of reflux disease on endoscopy or pH monitoring and thus are underrepresented on survey-based population studies. An Olmsted County survey found 42 % of people queried had an episode of heartburn in the preceding year while 45 % noted regurgitation. Of this cohort, approximately 5 % visited a physician for the symptoms during this same time period [5].

11.2 History

The diagnosis of GERD can be made by history alone in a patient with the typical symptoms of heartburn and regurgitation, and empiric therapy can be initiated. Given the myriad of over-the-counter (OTC) preparations available, patients have often tried various therapies unsuccessfully prior to seeking medical care. The ineffectiveness of OTC preparations, including proton pump inhibitors (PPIs), does not exclude the diagnosis of GERD, and further investigation is warranted [3].

T. Dyson, MD
Division of Gastroenterology, Department of Internal Medicine,
PO BOX 100277, Gainesville, FL 32610-0277, USA
e-mail: dysont@ufl.edu

B. Desai, A. Desai (eds.), *Primary Care for Emergency Physicians*,
DOI 10.1007/978-3-319-44360-7_11

When chest pain is a presenting symptom, a cardiac etiology should be excluded prior to empiric GERD treatment and/or gastrointestinal work-up. Thought of a cardiac work-up should also be considered for women, who often present with atypical ischemia symptoms, if GERD symptoms are atypical and/or the diagnosis is unclear.

11.3 Risk Factors for GERD

- Obesity, particularly central
- Hiatal hernia
- Pregnancy
- Smoking
- Acid hypersecretion, such as Zollinger-Ellison syndrome
- Lower esophageal dysfunction, such as post myotomy
- Dysfunctional esophageal clearance, as with scleroderma
- Delayed gastric emptying as seen in gastroparesis

11.4 Symptoms (Red Flags*)

- Heartburn
- Regurgitation
- Dysphagia*
- Burping
- Dyspepsia
- Nausea
- Epigastric pain
- Bloating
- Chest pain
- Bronchospasm
- Odynophagia*
- Sore throat
- Hoarseness
- Water brash
- Chronic cough
- Chronic otitis media/sinusitis
- Sleep disturbance
- Weight loss*

11.5 Physical Exam

As the esophagus is not directly accessible, the physical evaluation for GERD focuses on evaluating for other possible etiologies particularly for extraesophageal symptoms, when present:

- *Vital signs*
  - Pulse oximetry
    GERD is not likely the primary cause of hypoxia but can exacerbate hypoxia in an individual with underlying pulmonary disease.
- *Head*, *neck*, *and throat*
  - Laryngeal and pharyngeal inflammation
  - Dental enamel erosion
  - Sinus inflammation
  - Otitis media
    The above symptoms are related to chronic recurrent irritation of regurgitated acid resulting in inflammation and hyperplasia.
- *Pulmonary*
  - Wheezing and crackles
    Severe GERD can result in primary pulmonary disease but more often triggers an exacerbation of an underlying chronic pulmonary disease.
- *Abdomen*
  - Substernal chest pain or epigastric pain
    Possible if GERD is associated with mucosal-based disease like erosive esophagitis or ulceration
  - Abdominal pain with palpation
    Not a common finding. It may indicate alternative diagnosis.
  - Bowel sounds
    If high-pitched or tympanic sounds are present, it may be suggestive of a bowel obstruction, particularly if nausea or vomiting is present.

11.6 Differential Diagnosis

Diagnostic testing is reserved for alarm symptoms, recurrent symptoms, complications, or when the diagnosis remains unclear [7].

- Coronary artery disease
- Esophageal disease
  - Esophageal motility disorder
  - Esophagitis
  - Functional esophageal dysfunction
  - Zenker's diverticulum
  - Eosinophilic esophagitis

- Gastric disease
  - Gastroparesis
  - Gastritis
  - Peptic ulcer disease
  - Gastric outlet obstruction
- Biliary disease
  - Cholangitis
  - Choledocholithiasis

11.7 Treatment

Treatment of GERD is accomplished via multiple mechanisms: lifestyle modification, creation of a physical barrier, neutralization of acid, blockage of acid production, increase in gastric emptying, and structural enhancement of the gastroesophageal junction. Treatment can be administered on an as needed basis or chronically depending on their frequency or severity of symptoms. Additional work-up is indicated in individuals with alarm symptoms, refractory symptoms, or symptoms that require unusually large doses of medication for control.

- *Lifestyle modification*
  - Avoidance of dietary triggers*
    - Caffeine
    - Alcohol
    - Chocolate
    - Peppermint
    - Carbonated beverages
    - Spicy food
  - Weight loss
  - Avoidance of late-night eating, within 3hours of bedtime
  - Elevation of the head of the bed
    (*Studies are inconsistent regarding benefit and avoidance recommended based on personal symptom correlation.)
- Pharmacologic
  - *Antacids*
    - Calcium carbonate
    - Aluminum hydroxide
      Over-the-counter formulations used for rapid treatment of acute reflux symptoms by directly neutralizing gastric acid. Onset of action is quick as is the duration of action, 30–60 min.

- Pitfalls

  1. Diarrhea
  2. Renal impairment
  3. Electrolyte abnormalities

– *Mucosal-based agent*

- Aluminum sucrose sulfate
  Directly coats the esophageal mucosa creating a physical barrier to gastric content promoting healing. This agent is useful in the treatment of mild reflux disease as well as for use in pregnancy, Category B.
- Pitfalls

  1. Caution in renal insufficiency
  2. Aluminum toxicity
  3. Hypophosphatemia

- Bismuth subsalicylate
  Over-the-counter preparation used for treatment of mild to moderate reflux symptoms. It has poorly understood antisecretory properties as well as provides a direct mucosal barrier. It is useful for its rapid onset but has a short duration of action. Its antimicrobial effects are adjunctive in the treatment of *H. pylori*.
- Pitfalls

  1. Blackens stool, which can be confused with melena
  2. Bismuth toxicity

– *Histamine-2 receptor antagonists*

- Famotidine
- Cimetidine
- Ranitidine
- Nizatidine
  Over-the-counter preparations used for treatment of mild to moderate reflux, which work by blocking the histamine-2 receptor on parietal cells, thereby decreasing acid secretion. It is useful for on-demand treatment of reflux symptoms and can be taken once or twice daily for long-term therapy. Nocturnal dosing can be considered in patients with persistent symptoms and nighttime symptoms despite daily PPI usage. Ranitidine and cimetidine felt to be safe for use in pregnancy, Category B [4].
- Pitfalls

  1. Tachyphylaxis limits effectiveness to 6–8 weeks of usage.
  2. Require dose reduction for renal insufficiency.
  3. Myelosuppression.
  4. Gynecomastia.
  5. Impotence.
  6. Hepatitis.
  7. Multiple drug-drug interactions, primarily with cimetidine.

– *Proton pump inhibitors*

- Omeprazole*
- Omeprazole/sodium bicarbonate*
- Rabeprazole
- Esomeprazole
- Pantoprazole
- Lansoprazole*
- Dexlansoprazole

  The antacid effect of this group is obtained by direct irreversible inhibition of the hydrogen-potassium ATPase pump located on the parietal cell. As a group, they are the most effective pharmacologic antacid therapy available. There are seven formulations that are effectively equivalent in their efficacy on a milligram per milligram basis. Three formulations are available over the counter*, while the others require a prescription. Once daily AM dosing is recommended with an increase in frequency, change in administration time, or change in formulation based on an individual's response. They are administered 30–60 min prior to a meal and may take up to 5 days before maximal effect is demonstrated. Chronic therapy is indicated in patients with recurrent symptoms with discontinuation and/or complications related to reflux like erosive esophagitis, strictures, or Barrett's esophagus. The lowest effective dose should be used for long-term maintenance [3].

- Pitfalls

  1. Short-term use may increase the risk of community- and hospital-acquired pneumonia.
  2. There may be increase in the risk of *Clostridium difficile* infection in at-risk patients.
  3. Discontinuation in patients with osteoporosis *not* indicated, although therapy may increase the risk of hip fracture or additional bone loss in the setting of other risk factors for hip fracture, and change in therapy should be considered.
  4. No alteration of therapy indicated for concomitant clopidogrel usage.
  5. Potential calcium, iron, and vitamin B12 malabsorption.

11.8 Treatment Failures

Medially refractory symptoms require additional work-up and specialty evaluation. Risk factors that predict treatment failure include family history of GERD, dose escalation of antacid therapy, inability to discontinue antacid therapy, esophagitis on endoscopy, and failure of esophagitis to heal despite adequate therapy [8].

- Diagnostic evaluation
  - *Esophagram*
    - Allows evaluation for mass, strictures, and stenosis; often deferred in place of endoscopy which allows for diagnosis and tissue sampling
    - Recommended prior to endoluminal or surgical therapy to define anatomy: hernias, diverticula, etc.
  - *Endoscopy*
    - Allows for assessment severity of disease
      - Esophagitis
      - Strictures/stenosis
      - Barrett's esophagus
    - Evaluation for alternate diagnoses
      - Eosinophilic esophagitis
      - Malignancy
      - Gastric outlet obstruction
  - *Gastric-emptying study*
    - Evaluation for delayed gastric emptying
  - *Ambulatory reflux monitoring*
    - Documents acid exposure and regurgitation events
      - Documents the presence of disease in patients without esophagitis
    - Correlates symptoms with reflux episodes
    - Documents (in)effectiveness of medication
  - *Esophageal manometry*
    - Not required for diagnosis or evaluation of refractory symptoms but necessary prior to consideration of endoluminal or surgical treatment for reflux
    - Documents adequate esophageal motility
    - Detects early achalasia, disorder often confused with GERD
- Nonpharmacologic intervention
  - *Endoluminal therapy*
    Two currently FDA-approved therapies are Stretta and transoral incisionless fundoplication (TIF). Short-term results are promising but long-term efficacy remains to be seen [3].
    - Stretta
      - Applies radiofrequency energy to the esophagus causing inflammation and fibrosis resulting in decreased tissue compliance and lower esophageal sphincter relaxation

- TIF
  - Endoscopically creates a reflux barrier similar to traditional surgical fundoplications.

– *Surgery*
Shown to be equally effective as medical management in the appropriately chosen patient population for the control of GERD symptoms [2]

- Fundoplication
  - Recreates and/or augments the physical barrier at the lower esophagus
  - Exact type of surgery dependent upon surgeon expertise and patient characteristics

Bibliography

1. Committee, C. f. The role of endoscopy in the management of GERD. Gastrointest Endosc. 2015;81(6):1305–10.
2. Committee SG. Guidelines for surgical treatment of Gastroesophageal Reflux Disease (GERD). Retrieved 9 Apr 2016, from Society of America Gastrointestinal and Endoscopic Surgeons. 2010, Feb www.sages.org.
3. Katz PO, Gerson LB, Vela MF. Guidelines for the diagnosis and management of gastroesophageal reflux disease. Am J Gastroenterol. 2013;108:308–26.
4. McGuigan JE, Belafsky PC, Fromer L, Mccarthy D, Nostrant T, Postma GN, et al. Review article: diagnosis and management of night-time reflux. Aliment Pharmacol Ther. 2004;20(9):57–72.
5. Locke RG III, Talley NJ, Fett SL, Zinsmeister AR, Melton JL III. Prevalence and clinical spectrum of gastroesophageal reflux: A population-based study in olmsted county, minnesota. Gastroenterol. 1997;112:1148–1456.
6. Tran T, Lowry A. M, EL-SERAG H. B. Meta-analysis: the efficacy of over-the-counter gastro-oesophageal reflux disease therapies. Aliment Pharmacol Ther. 2007;25(2):143–153.
7. Tytgat GN. Review article: treatment of mild and severe cases of GERD. Aliment Pharmacol Ther. 2002;16(4):73–8.
8. Worrell SG, Greene CL, DeMeester TR. The state of surgical treatment of gastroesophageal reflux disease after five decades. J Am Coll Surg. 2014;219(4):819–30.

Chapter 12
Dyspnea

Michael Marchick

12.1 Introduction

Dyspnea is defined by an official statement of the American Thoracic Society as "a subjective experience of breathing discomfort that consists of qualitatively distinct sensations that vary in intensity" [1]. A wide variety of patient complaints can therefore fall under the umbrella of dyspnea, including shortness of breath, chest tightness, and a sense of impending doom [2]. The associated differential diagnosis ranges from minor self-limited etiologies to the immediately life-threatening. Unfortunately, there is often poor correlation with the severity of self-reported severity of dyspnea and the seriousness of the underlying etiology [3].

12.2 Differential Diagnosis

- Cardiopulmonary
  - Acute coronary syndrome (ACS)
  - Airway obstruction
    - Airway infections (epiglottitis, retropharyngeal abscess)
    - Anaphylaxis
    - Angioedema
    - Foreign body
    - Hemorrhage

M. Marchick, MD
Emergency Medicine, UF Health Shands Hospital,
1600 SW Archer Road, Gainesville, FL 32610, USA
e-mail: marchick@ufl.edu

B. Desai, A. Desai (eds.), *Primary Care for Emergency Physicians*,
DOI 10.1007/978-3-319-44360-7_12

  - Arrhythmia
  - Asthma
  - Chronic obstructive pulmonary disease (COPD)
  - Malignancy
  - Pericardial disease (effusion, pericarditis, tamponade)
  - Pleural effusion
  - Pneumonia
  - Pulmonary embolus (PE)
  - Pulmonary edema/congestive heart failure (CHF)
  - Spontaneous pneumothorax
  - Valvular dysfunction
- Abdominal
  - Ascites
  - Obesity
  - Pregnancy
  - Referred pain
- Hematologic/metabolic
  - Acute chest syndrome
  - Anemia
  - Carbon monoxide poisoning
  - Diabetic ketoacidosis
  - Fever
  - Organophosphate poisoning
  - Renal failure
  - Salicylate overdose
  - Sepsis
  - Thyrotoxicosis
- Neuromuscular
  - Botulism
  - Cerebrovascular accident
  - Guillain-Barre syndrome
  - Myasthenia gravis
- Psychiatric
  - Hyperventilation
  - Somatization
- Traumatic
  - Cardiac tamponade
  - Flail chest
  - Hemothorax
  - Pneumothorax
  - Pulmonary contusion
  - Rib fractures

12.3 History

When obtaining the history in a patient presenting with dyspnea, the clinician should focus not only on obvious respiratory symptoms but also on symptoms referable to interrelated organ systems, particularly the cardiovascular and renal systems. Neurologic, gastrointestinal, and psychiatric conditions can also lead to chief complaint of dyspnea. Any history of recent traumatic injury should also be elicited. With regard to the past medical history, focus on preexisting cardiopulmonary conditions as well as smoking history, occupational exposure history, and risk factors for ACS and PE (discussed below).

- Temporal course of symptoms
  - Abrupt worsening (*red flag): PE, flash pulmonary edema, spontaneous pneumothorax
  - Gradual onset: pneumonia, CHF exacerbation, anemia
- Triggers of dyspnea
  - Allergens
  - Environmental pollutants: asthma, COPD exacerbation
  - Exertion: ACS, CHF
  - Paroxysmal nocturnal dyspnea and orthopnea: CHF
- Wheezing
- Stridor
- Cough
- Quality
  - "Barking": Croup
  - Sputum: COPD +/− acute exacerbation, pneumonia
  - Hemoptysis (*red flag) tuberculosis, malignancy, or PE
- Associated symptoms
  - Abdominal pain
  - Anxiety/depression
  - Ascending muscle weakness: Guillain-Barre
  - Chest pain (*red flag)
  - Edema of the face, abdomen, or legs: renal failure, liver failure, CHF
  - Unilateral leg swelling (*red flag): venous thromboembolism
  - Fever
  - Leg pain (*red flag): venous thromboembolism
  - Weight gain: renal failure, CHF
  - Weight loss: malignancy, thyrotoxicosis

12.4 Physical

In any patient with a complaint of dyspnea, it is necessary to immediately evaluate for signs of impending respiratory failure.

*Red flags for impending respiratory failure:

- Accessory muscle use
- Paradoxical respiration
- Bilateral absent breath sounds
- Stridor
- Altered mental status
- Tripoding
- Inability to handle secretions

Once the need for emergent airway or respiratory interventions has been excluded, the exam can turn to a more systematic approach to gather further clues to the etiology of dyspnea.

- Oropharyngeal exam
  - Oropharyngeal edema/obstruction (*red flag)
  - Respiratory exam
- Barrel chest: COPD
- Pursed-lip breathing
- Auscultation:
  - Absent breath sounds (*red flag)
  - Decreased breath sounds:
    - Bilateral (*red flag): severe asthma or COPD exacerbation, bilateral pleural effusions
    - Unilateral: PTX, unilateral effusion
    - Wheezes: asthma, CHF, COPD
    - Crackles/rales: PNA, CHF
  - Stridor (*red flag): croup, retropharyngeal abscess, anaphylaxis
- Cardiovascular exam
  - Jugular venous distention (*red flag): CHF, tension pneumothorax, pericardial tamponade
  - Muffled heart sounds (*red flag): pericardial effusion with or without tamponade
  - S3 or S4: heart failure
- Abdominal exam: evaluate for intra-abdominal pathology which may irritate the diaphragm or cause apparent respiratory difficulty.
  - Distention
  - Tenderness

- Dermatologic exam
  - Clubbing
  - Cyanosis
  - Dependent edema: CHF, renal failure

12.5 Specific Relevant Etiologies

12.5.1 Asthma

The prevalence of asthma among Americans is approximately 8 %, with slightly higher rates among children. In 2008, approximately half of patients with asthma reported having at least one attack [4], making asthma exacerbations one of the most frequent emergency department diagnoses. While the details of the pathophysiology are complex, the condition is ultimately the result of an abnormal inflammatory response resulting in airway obstruction due to smooth muscle constriction, airway edema, and accumulation of secretions. Exacerbations are typically associated with dyspnea, chest tightness, wheezing, and/or cough. In addition to standard historical data mentioned above, clinicians should inquire regarding potential triggers of symptoms, as well as any history of intensive care unit admissions, history of intubation, and personal best measures on peak expiratory flow (PEF) testing.

Labs and imaging are of limited value in patients with a known history of asthma presenting with an uncomplicated exacerbation. The severity of illness and response to treatment is best assessed by clinical judgment and PEF testing, which is widely available in EDs and primary care offices, in contrast to spirometry. Values obtained by individuals can be compared to age, sex, and height-matched predicted values to monitor the severity of an exacerbation as well as the response to treatment. Severe exacerbations are typically defined by a PEF reading <40 % of personal best or predicted, with mild exacerbations by a reading ≥70 % of these values. Moderate severity exacerbations comprise the remainder.

Inhaled β-agonists are a cornerstone of asthma therapy. These agents relax bronchial smooth muscle, resulting in bronchodilation. Albuterol, a short-acting, rapid-onset, β-2 selective agent, is typically the drug of choice for acute exacerbations. Albuterol can be administered via metered dose inhaler (with or without a spacer device) or via nebulization. In the hospital setting, treatments are often delivered via nebulizer and can be given continuously or on a periodic basis. In the home setting, for children older than 1 year of age, there is no apparent benefit to nebulized therapy versus a metered dose inhaler with spacer. Levalbuterol does not appear to convey any benefit with respect to safety or efficacy compared with albuterol, but does cost significantly more. Combination of inhaled ipratropium with albuterol may reduce the need for hospitalization in patients with severe exacerbations. Systemic β-agonists do not appear beneficial compared with inhaled agents [5].

Early administration of systemic corticosteroids (40–60 mg prednisone PO for adults or 1–2 mg/kg prednisolone PO for children, or 1 mg/kg methylprednisolone

IV) reduces the rate of hospitalization. Patients are typically prescribed a non-tapering course of a total of 3–10 days of 40–60 mg prednisone daily (1–2 mg/kg prednisolone in children) once discharged from the ED or inpatient hospitalization. Prednisolone is preferred in children due to its palatability relative to prednisone.

Magnesium administration (25–75 mg/kg in children, up to 2 mg IV in adults) may be considered in severe exacerbations. Methylxanthines, such as theophylline, have a narrow therapeutic index and are generally avoided. Heliox has not proven to be effective in improving relevant clinical end points.

Long-acting β-agonists, such as salmeterol and formoterol, are indicated for maintenance therapy in patients with persistent asthma (generally defined as symptoms typically present for >2 days/week). These agents should not be used as an alternative to short-acting β-agonists in patients with acute exacerbations. Commercially available preparations of long-acting agents often also contain an inhaled corticosteroid (examples include budesonide/formoterol, mometasone/formoterol, and salmeterol/fluticasone).

Leukotriene-modifying agents, such as montelukast, zafirlukast, and zileuton, are useful as a potential alternative to inhaled corticosteroids in patients with persistent asthma. These agents are not indicated for use in acute exacerbations.

Patients with continued symptoms and/or a PEF <40 % following initial ED treatment should be admitted to the hospital for continued care. Patients with a sustained good response to therapy (PEF ≥70 % and improved symptoms) can generally be discharged. In the remainder of patients, a case-by-case decision should be made, with careful consideration of the severity of patient's prior exacerbations, comorbidities, access to primary care, and social supports. In discharged patients, a continued course of systemic steroids is indicated as noted above. Ensure that adequate supplies of short-acting β-agonist are available, give complete return precautions, and ensure prompt follow-up with the patient's PCP. In patients with a history of persistent asthma, consider addition of an inhaled steroid if not already utilized [3, 5].

12.5.2 COPD Exacerbations

The prevalence of COPD among American adults is approximately 6 %, with approximately 19 % of those affected reporting at least one ED visit or hospitalization in the previous year [6]. COPD is classically divided into chronic bronchitis and emphysema, although significant overlap exists between these forms (chronic obstructive asthma and bronchiectasis are also considered forms of COPD). Chronic bronchitis is defined clinically as the presence of a productive cough for at least 3 months in two consecutive years without an alternative explanation. Emphysema is defined by the destruction of distal airways, including bronchioles and alveoli, without evidence of fibrosis. In each form, there exists persistent airflow limitation and an abnormal inflammatory response. Smoking is by far the leading risk factor, although α-1 antitrypsin deficiency and exposure to occupational and environmental

Table 12.1 Severity of airflow limitation according to GOLD criteria

Severity	FEV_1 (% predicted)
Mild	≥80 %
Moderate	50–79 %
Severe	30–49 %
Very severe	<30 %

pollutants are also associated. At the time of diagnosis, patients typically report chronic and progressive dyspnea and cough with or without sputum production. The formal diagnosis of COPD is made by spirometry based on a post-bronchodilator forced expiratory volume in 1 s/forced vital capacity (FEV_1/FVC) of <0.70. Further stratification of the severity can be made by the GOLD criteria (Table 12.1) [7].

COPD exacerbations are characterized by an acute change in sputum character or an increase in dyspnea, cough, or sputum volume. A host of causes exists, with viral and bacterial infections by far the most common triggers. Environmental respiratory irritants, PE, pneumothorax, CHF, β-blocker use, and cold weather have also been implicated. Frequently, no cause is identified.

12.5.3 Chronic Management

The acute management of COPD exacerbations is quite analogous to that of asthma exacerbations. A key distinguishing factor is the relative lack of acute reversibility of airflow obstruction, limiting the utility of PEF measurement and reassessments in these patients.

Supplemental oxygen should be administered to maintain saturations of at least 90 %. Additional doses of short-acting β-agonist, combined with an inhaled anticholinergic agent (albuterol/ipratropium) beyond the patient's baseline regimen, are generally indicated. Oral steroids such as prednisone (or methylprednisolone IV in patients unable to tolerate PO) are also of benefit. A key distinction from acute asthma management is the role of antibiotics. As bacterial infection (particularly *Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Moraxella catarrhalis*) is commonly associated with the development of COPD exacerbations, patients with increased sputum production and/or moderate to severe exacerbations should typically be prescribed antibiotics. There is no clear data to support the use of any particular agent. Antimicrobial choice should therefore be guided by local resistance patterns to the above pathogens. Commonly utilized antibiotics include amoxicillin ± clavulanate, doxycycline, azithromycin, and respiratory fluoroquinolones. The optimal duration of antimicrobial treatment is also unclear [8]. Intravenous magnesium can also be considered in severe exacerbations, although evidence supporting its use is lacking. Long-acting inhaled anticholinergics (tiotropium and aclidinium) and inhaled corticosteroids (beclomethasone, budesonide, ciclesonide, fluticasone, and mometasone, often combined with long-acting β-agonists as described in the asthma section), while beneficial in chronic management of COPD, are not of benefit in the setting of an acute exacerbation [9].

Patients with marked worsening from baseline (such as new or increasing oxygen requirements), those who fail to improve (or worsen) despite the above interventions, and those with significant comorbidities or inadequate social support (including poor access to medications or primary care follow-up) should be admitted. If the decision is made to discharge a patient with a COPD exacerbation, ensure adequate supplies of albuterol with or without ipratropium are available to the patient, as well as sufficient amounts of supplemental home oxygen, and any additional medications prescribed for COPD by their primary care physician. A several day course of oral steroids is generally indicated, analogous to asthma management. If indicated, prescribe an appropriate antibiotic regimen. Finally, counsel patients who continue to smoke regarding the role of smoking cessation in the chronic management of COPD, as it is the only intervention shown to favorably alter the course of the disease [9].

12.5.4 Hyperventilation

Hyperventilation refers to minute ventilation in excess of metabolic needs. Anxiety disorders, including panic disorder, are commonly associated. The diagnosis should be one of exclusion, after careful consideration of the numerous potentially life-threatening etiologies of dyspnea noted above. Patients may report associated feelings of impending doom, palpitations, paresthesias, presyncope, chest pain, diaphoresis, and carpopedal spasm. An inciting distressing event may also be reported. Once emergent diagnoses have been excluded, a small dose of a benzodiazepine, such as lorazepam may be considered for resolution of an acute episode. Rebreathing of CO_2 (such as via a paper bag) was once widely advocated as a remedy for acute hyperventilation. However, due to concerns regarding potential hypoxemia, this treatment modality should be avoided [10]. If episodes are recurrent, treatment of any underlying anxiety disorder should be the focus of therapy as outlined in the Chap. 26 Anxiety.

12.5.5 Croup

Inflammation of the larynx and subglottic airway due to viral or bacterial illness resulting in stridor and cough is termed croup. Parainfluenza viruses are the most common culprit, although respiratory syncytial virus and influenza viruses are also implicated. Children between the ages of 6 months and 3 years are most commonly affected, with peak incidence in late fall, early winter, and spring. The course of illness typically begins with several days of URI symptoms such as congestion, rhinorrhea and fever, followed by the onset of the typical barking cough, stridor and hoarseness, which usually abates within 48 h. Young children are much more severely affected due to narrow caliber of the airway in this age group, whereas

adults typically only experience the hoarseness attributed to "laryngitis." Treatment for all pediatric patients presenting with croup includes immediate assessment of the ability to protect their airway, followed by administration of oral steroids. Dexamethasone (0.6 mg/kg, max 10 mg) is the preferred agent given its prolonged half-life. Oral administration is as effective as parenteral and has the advantage of being less alarming to a child than an injection, which could induce further respiratory difficulty. In patients with moderate (stridor at rest, frequent barking cough, chest wall retractions) to severe croup (respiratory distress, marked retraction, inspiratory and expiratory stridor), nebulized epinephrine should be administered. If clinical response is noted, patients should be observed for approximately 4 h as the potential for rebound symptoms exists. Patients with persistent increased work of breathing or stridor at rest, as well as those requiring more than two doses of epinephrine should be admitted.

12.5.6 Pulmonary Embolism

Pulmonary embolism (PE) is a feared cause of dyspnea with a significant burden of mortality. It has been estimated that up to 100,000 Americans die each year from PE [11], and fear of missing the diagnosis fuels a significant number of evaluations for the condition. The presentation varies from sudden cardiac death to incidentally diagnosed PE in virtually asymptomatic patients. The most common symptoms include dyspnea, pleuritic chest pain, leg pain/swelling, and cough (with or without hemoptysis). The onset of symptoms is often rapid, although significant variation exists. Tachycardia, hypoxemia, tachypnea, and leg tenderness and swelling may be noted on physical exam. Risk factors for PE include the components of Virchow's triad: venous stasis (prolonged immobilization), thrombophilia (e.g., malignancy, inherited thrombophilias, use of exogenous estrogens), and vascular endothelial injury.

CT pulmonary angiography (CTPA) is the most commonly used imaging study to confirm the diagnosis of PE, although ventilation/perfusion scanning is utilized in some pregnant patients, as well as in patients with contrast allergies and renal insufficiency. The sensitivity and specificity of CTPA are excellent. However, CTPA does expose patients to a significant amount of radiation and nephrotoxic contrast dye. Therefore, appropriate selection of patients for this testing is key. The pulmonary embolism rule-out criteria (PERC rule) identifies patients for whom no further testing is indicated. To fulfill the rule, the clinician must estimate a low pretest probability of PE (<15 %), and the patient must fulfill all of the criteria listed below [12]:

- Age <50
- Pulse rate <100
- Room air oxygen saturation ≥95 %
- Absence of hemoptysis
- No use of exogenous estrogens
- No history of venous thromboembolism

- No surgery or trauma in the past 4 weeks
- Absence of unilateral leg swelling

In patients for whom PE cannot be excluded by the PERC rule, an estimation of pretest probability should be made, either by clinician gestalt or by a structured decision rule such as the Wells score [13]. Patients with a high pretest probability (e.g., Wells score >4 [14]) should proceed directly to a definitive imaging study, while those with a low pretest probability are appropriate for D-dimer testing as a first step. Those patients with a D-dimer below the test threshold are considered to have PE effectively excluded, while others should proceed to definitive imaging.

Currently, treatment of confirmed PE generally includes hospitalization and initiation of anticoagulation with heparin, low-molecular-weight heparin, or fondaparinux. Patients are then typically transitioned to oral anticoagulants such as warfarin, rivaroxaban, apixaban, or edoxaban. Hypotensive patients without contraindications should be strongly considered for thrombolysis. Thrombolysis in patients with evidence of right-ventricular strain (hypokinesis or dilatation, troponinemia, or brain natriuretic peptide elevation) is controversial.

12.5.7 Pneumonia

Approximately four million cases of community-acquired pneumonia (CAP) are diagnosed per year [15]. Many additional patients are diagnosed with healthcare-associated pneumonia (HCAP) or hospital-acquired pneumonia. The first two entities are of particular relevance to emergency and outpatient-based primary care physicians. Cough is present in nearly all patients, with dyspnea, sputum production, and chest pain also frequently reported. Elderly patients often report fewer or atypical symptoms, so a lower threshold to evaluate for pneumonia is warranted in this population. On physical exam, fever, tachycardia, tachypnea, hypoxemia, rales, wheezes and/or diminished lung sounds may be appreciated. A chest X-ray should be obtained in patients with clinical evidence of pneumonia, and strictly speaking, a demonstrable infiltrate must be present to make the diagnosis [16].

HCAP is defined as a pneumonia diagnosis in a patient who has been hospitalized for two or more days within the past 90 days, is a resident of a nursing home or long-term care facility, has received care at a hemodialysis clinic in the past 30 days, or has received wound care, IV chemotherapy, or other IV infusions in the past 30 days [17]. Outpatients with none of these features are classified as having CAP. The distinction is important, as the potential for multiple drug-resistant bacterial infection is quite significant in patients with HCAP.

Common identified etiologies of CAP include *Mycoplasma pneumoniae*, *Chlamydophila pneumoniae*, *S. pneumoniae*, *H. influenzae*, and respiratory viruses, such as influenza, adenovirus, and respiratory syncytial virus. Atypical pathogens such as *Mycoplasma* and *Chlamydophila* are commonly associated with milder disease (“walking pneumonia”) outbreaks in crowded facilities such as college

dormitories. Chest X-ray in these patients often reveals an interstitial pattern. Tuberculosis is commonly associated with upper lobe involvement on chest X-ray, cavitating lesions, and mediastinal lymphadenopathy. *Legionella* should be considered in patients with high fever, gastrointestinal symptoms, relative bradycardia, smoking history, and underlying lung disease. Urine antigen testing can be used to identify infected patients.

Several methods exist to risk stratify disposition decisions for adult patients with community-acquired pneumonia. The Pneumonia Severity Index, which has been extensively studied and externally validated, stratifies patients into one of five risk classifications with 30-day mortality risks ranging between 0.1 % and 27.0 % [18]. A number of elements of historical and laboratory data are required, and the calculation can be somewhat cumbersome, so the use of an online calculator is typically employed to aid in appropriate classification. A simpler alternative method is the CURB-65 rule, which stratifies patients on the basis of confusion, urea (BUN >20 mg/dL), respiratory rate > = 30/min, BP (systolic <90 or diastolic ≤60 mmHg), and age ≥65 years. Reported 30-day mortality rates range from 0.7 to 57 % in patients with 0–5 factors, respectively [19]. As is the case with any decision rule, clinical judgment must always supersede. Hypoxic patients, particularly those with an oxygen saturation <92 %, should be strongly considered for admission regardless of results of formal risk stratification results [20]. In the pediatric population, infants under the age of 3–6 months and children with significant cardiopulmonary comorbidities, respiratory distress, hypoxemia, toxic appearance, and poor feeding should generally be hospitalized.

If treating as an outpatient, typical regimens for CAP adults include a macrolide such as azithromycin or a respiratory fluoroquinolone such as levofloxacin. If significant local resistance of *S. pneumoniae* to macrolides exists, macrolide monotherapy should not be used. Atypical pathogens are uncommon in children beyond infancy and less than 5 years old; therefore, high-dose amoxicillin is the typical first-line coverage. The increasing prevalence of atypical infections in older children results in macrolides being an appropriate empiric therapy for this population. Patients hospitalized for CAP are typically started on either a respiratory fluoroquinolone or the combination of a parenteral third-generation cephalosporin (such as ceftriaxone) as well as a macrolide. Patients requiring ICU-level care are generally empirically started on more aggressive antibiotic regimens. Regardless of age group or disposition decision, it is always wise to consider the possibility of influenza pneumonia and possible treatment with oseltamivir. If suspicion for tuberculosis exists, place the patient in respiratory isolation, admit the patient, and obtain acid-fast stains of the sputum. Patients with healthcare-associated pneumonia should be admitted and placed on broad-spectrum antibiotics as the potential for resistant bacterial infection is quite significant. Treatment should include coverage for *Pseudomonas aeruginosa* as well as MRSA.

Discharged patients should have close follow-up arranged, and any worsening or failure to improve within 48–72 h should prompt consideration of hospital admission and broadening of antibiotic coverage.

References

1. Parshall MB, Schwartzstein RM, Adams L, Banzett RB, Manning HL, et al. An official American Thoracic Society statement: update on the mechanisms, assessment, and management of dyspnea. Am J Respir Crit Care Med. 2012;185:435–52.
2. Banzett RB, Schwartzstein RM. Dyspnea: don't just look, ask! Am J Respir Crit Care Med. 2015;192:1404–6.
3. Marx JA, Hockberger RS, Walls RM. Rosen's emergency medicine – concepts and clinical practice. 8th ed. Philadelphia: Elsevier Saunders; 2013.
4. Centers for Disease Control and Prevention. Vital signs: asthma prevalence, disease characteristics, and self-management education: United States, 2001–2009. MMWR Morb Mortal Wkly Rep. 2011;60:547–52.
5. Pollart SM, Compton RM, Elward KS. Management of acute asthma exacerbations. Am Fam Physician. 2011;84:40–7.
6. Centers for Disease Control and Prevention. Chronic obstructive pulmonary disease among adults – United States, 2011. MMWR Morb Mortal Wkly Rep. 2012;61:938–43.
7. Global strategy for the diagnosis, management and prevention of COPD, global initiative for chronic obstructive lung disease (GOLD). 2016. http://www.goldcopd.org/guidelines-global-strategy-for-diagnosis-management.html.
8. Vollenweider DJ, Jarrett H, Steurer-Stey CA, Garcia-Aymerich J, Puhan MA. Antibiotics for exacerbations of chronic obstructive pulmonary disease. Cochrane Database Syst Rev. 2012;12:CD010257.
9. Evensen AE. Management of COPD exacerbations. Am Fam Physician. 2010;81:607–13.
10. Callaham M. Hypoxic hazards of traditional paper bag rebreathing in hyperventilating patients. Ann Emerg Med. 1989;18:622–8.
11. Horlander KT, Mannino DM, Leeper KV. Pulmonary embolism mortality in the United States, 1979–1998: an analysis using multiple-cause mortality data. Arch Intern Med. 2003;163:1711–7.
12. Kline JA, Courtney DM, Kabrhel C, Moore CL, Smithline HA, et al. Prospective multicenter evaluation of the pulmonary embolism rule-out criteria. J Thromb Haemost. 2008;6:772–80.
13. Wells PS, Anderson DR, Rodger M, Ginsberg JS, Kearon C, et al. Derivation of a simple clinical model to categorize patients probability of pulmonary embolism: increasing the models utility with the SimpliRED D-dimer. Thromb Haemost. 2000;83:416–20.
14. van Belle A, Büller HR, Huisman MV, Huisman PM, Kaasjager K, Christopher Study Investigators, et al. Effectiveness of managing suspected pulmonary embolism using an algorithm combining clinical probability, D-dimer testing, and computed tomography. JAMA. 2006;295:172–9.
15. Halm EA, Teirstein AS. Management of community-acquired pneumonia. N Engl J Med. 2002;347:2039–45.
16. Mandell LA, Wunderink RG, Anzueto A, Bartlett JG, Campbell GD, Dean NC, Dowell SF, File Jr TM, Musher DM, Niederman MS, Torres A, Whitney CG, Infectious Diseases Society of America, American Thoracic Society. Infectious Diseases Society of America/American Thoracic Society consensus guidelines on the management of community-acquired pneumonia in adults. Clin Infect Dis. 2007;44:S27–7.
17. American Thoracic Society, Infectious Diseases Society of America. Guidelines for the management of adults with hospital-acquired, ventilator-associated, and healthcare-associated pneumonia. Am J Respir Crit Care Med. 2005;171(4):388–416.
18. Fine MJ, Auble TE, Yealy DM, Hanusa BH, Weissfeld LA, Singer DE, Coley CM, Marrie TJ, Kapoor WN. A prediction rule to identify low-risk patients with community-acquired pneumonia. N Engl J Med. 1997;336:243–50.
19. Lim WS, van der Eerden MM, Laing R, Boersma WG, Karalus N, Town GI, Lewis SA, Macfarlane JT. Defining community acquired pneumonia severity on presentation to hospital: an international derivation and validation study. Thorax. 2003;58:377–82.
20. Majumdar SR, Eurich DT, Gamble JM, Senthilselvan A, Marrie TJ. Oxygen saturations less than 92% are associated with major adverse events in outpatients with pneumonia: a population-based cohort study. Clin Infect Dis. 2011;52:325–31.

Chapter 13
Hemoptysis

Julie Estrada, Bobby Desai, and Alpa Desai

13.1 Introduction

Hemoptysis can be defined as the expectoration of blood that originates from the lower respiratory tract (trachea, bronchi, and bronchioles) [1]. The majority of cases of hemoptysis seen in the emergency department involve trace losses of blood and are not life-threatening. Less than 5 % of patients will present with massive hemoptysis which is generally defined as 200–1,000 mL of blood loss over a 24 h period. Massive hemoptysis can cause hemodynamic changes, results in inadequate gas exchange, and reaches a mortality rate of 80 % [2]. Therefore, massive hemoptysis requires prompt intervention to ensure airway protection and adequate ventilation.

Care must also be given to determining other potential sources of bleeding. Bleeding from the nasopharynx, oropharynx, or gastrointestinal system can often mimic true hemoptysis as blood can sometimes contaminate the tracheobronchial tree.

Hemoptysis is rare in the pediatric population, but causes can include lower respiratory tract infections, irritation from aspiration of foreign bodies, congenital heart disease, and bronchiectasis (typically associated with cystic fibrosis).

13.2 Pathophysiology

The blood supply to the lungs is made up primarily by two arterial vascular systems:

- The bronchial arteries carry oxygenated blood to the lung parenchyma. These arteries branch from the aorta and are at systemic pressure.

J. Estrada • B. Desai, MD, MEd (✉)
Emergency Medicine, University of Florida, Gainesville, FL, USA
e-mail: julieestrada@ufl.edu; bdesai@ufl.edu

A. Desai, DO
Community Health and Family Medicine, University of Florida, Newberry, FL, USA
e-mail: desaas@shands.ufl.edu

B. Desai, A. Desai (eds.), *Primary Care for Emergency Physicians*,
DOI 10.1007/978-3-319-44360-7_13

- The pulmonary arteries are responsible for bringing deoxygenated blood to the lungs for gas exchange and are under much lower pressures.

Approximately 90 % of massive hemoptysis arises from the bronchial arteries, while 5 % originates from the pulmonary arteries [3]. Episodes of low-volume hemoptysis tend to arise from capillaries that are disrupted due to trauma, inflammation, infection, or even infarction.

13.3 Risk Factors

- Smoking
- Age greater than 40
- Anticoagulant use
- Asbestos exposure
- Travel
  - Increased risk of developing pulmonary embolism
  - Exposure to tuberculosis

13.4 Differential Diagnosis

- Pseudohemoptysis
  - Nasopharyngeal bleeding
  - Oropharyngeal bleeding
  - Gastrointestinal bleeding
- Tracheobronchial sources
  - Bronchitis – account for up to half of cases of hemoptysis
    - Acute
    - Chronic
  - Bronchiectasis
  - Cystic fibrosis
  - Neoplasm
    - Small cell carcinoma
    - Squamous cell carcinoma
    - Bronchial adenoma
    - Metastatic disease
    - Carcinoid tumor
    - Kaposi's sarcoma

  - Traumatic injury to the airway
  - Foreign body
  - Broncholithiasis
- Parenchymal sources
  - Pneumonia
    - *Staphylococcus*
    - *Legionella*
    - *Pneumococcus*
    - *Klebsiella*
  - Tuberculosis – accounts for approximately 8 % in the United States but remains the most common cause in third world countries [4].
  - Viral
    - Varicella
    - Influenza
  - Fungal
    - *Aspergillus*
    - *Coccidioides*
    - *Histoplasma*
    - *Blastomyces*
  - Pneumonitis
    - Inhalational
    - Chemical
  - Lung abscess
  - Lung contusion
  - Goodpasture syndrome
  - Wegener granulomatosis
- Vascular sources
  - Congestive heart failure
  - Arteriovenous malformation
  - Aortopulmonary fistula
  - Aortic aneurysm
  - Pulmonary embolism
  - Pulmonary hypertension
  - Endocarditis
  - Mitral stenosis
- Coagulation sources
  - Von Willebrand disease
  - Thrombocytopenia

  - Hemophilia
  - Antiplatelet or anticoagulant use
  - Disseminated intravascular coagulation
- Other sources
  - Iatrogenic
    - Tracheostomy/intubation complications
    - Post bronchoscopy
    - Post biopsy
    - Pulmonary artery catheter
  - Autoimmune
    - Behcet's disease
    - SLE
    - Polyarteritis nodosa
  - Ectopic endometrial tissue
  - Trauma
  - Illegal drugs
    - Marijuana
    - Cocaine
  - Idiopathic

Conditions which are more likely to cause massive hemoptysis:

- Tuberculosis
- Neoplasm
- Lung abscess
- Bronchiectasis
- Cystic fibrosis

13.5 History

Care must be taken to differentiate between hemoptysis and hematemesis [5].

Hemoptysis	Hematemesis
History	History
Absence of nausea/vomiting	Nausea/vomiting
Associated hypoxia	Hypoxia less common
Lung disease	Gastrointestinal and/or hepatic disease
Sputum appearance	Sputum appearance
Frothy or clotted	Coffee ground

Hemoptysis	Hematemesis
Bright red or pink	Dark red, brown, or black
Laboratory studies	Laboratory studies
Alkaline pH (>7)	Acidic pH (<7)

Additional pertinent historical features include:

- Amount of blood expectorated [6]
  - Mild hemoptysis: less than 30 mL in 24 h
  - Moderate hemoptysis: between 30 and 200 mL in 24 h
  - Massive hemoptysis: greater than 200 mL in 24 h
- Prior episodes of hemoptysis
  - Known parenchymal disease such as chronic obstructive pulmonary disease, recurrent pneumonia, and bronchiectasis
- Symptoms suggestive of infection (fever, chills, night sweats)
  - Pneumonia, bronchitis, upper respiratory tract infection, and sinusitis
- Weight loss
  - HIV, tuberculosis, malignancy, and bronchiectasis
- Tobacco use
  - Primary lung malignancy
- Dyspnea on exertion, orthopnea, and paroxysmal nocturnal dyspnea
  - Congestive heart failure and mitral valve stenosis
- Pleuritic chest pain or recent immobilization
  - Pulmonary embolism or infarction
- Vomiting, melena, alcohol abuse, and chronic or excessive NSAID use
  - Gastric ulcer, gastric varices, and Mallory-Weiss tear
- Hematuria
  - Goodpasture syndrome
- Hemoptysis associated with menses
  - Pulmonary endometriosis
- Travel history
  - Tuberculosis, fungal, or parasitic infections
- Anticoagulant use
- Recent percutaneous or transbronchial procedures

13.6 Physical Examination

The physical examination may provide insight on the cause and location of the bleeding

- Vital signs
  - Fever, hypo-/hyperthermia, and elevated respiratory rate should alert the physician to possible infectious etiology.
  - Tachycardia
    - Appropriate fever response
    - Compensatory mechanism in maintaining perfusion in response to intravascular volume deficits and vasodilation as seen in sepsis
  - Hypoxia on pulse oximetry may be an indication of massive hemoptysis and should prompt immediate intervention.
- Head, Ears, Eyes, Nose, and Throat
  - Focus on alternative sources of bleeding.
  - Saddle nose and septal perforation should heighten suspicion for Wegener's granulomatosis.
  - Stridor in a child may be suggestive of aspiration of a foreign body.
- Cardiovascular
  - Rumbling diastolic murmur suggestive of mitral stenosis.
  - New murmur in addition to fever could suggest endocarditis with resulting septic pulmonary emboli.
  - S3 and JVD suggestive of congestive heart failure.
- Pulmonary/Chest
  - Evaluate for signs of consolidation, wheezing, rales, and trauma.
- Gastrointestinal
  - Vomiting, abdominal tenderness, and ascites
- Extremities
  - Unilateral swelling may be seen with deep vein thrombosis and/or pulmonary embolus.
- Integumentary
  - Clubbing suggests lung malignancy or chronic obstructive disease.
  - Cutaneous purpura or ecchymosis may suggest a blood dyscrasia or anticoagulant use.

13.7 Diagnosis

For patients that present with hemoptysis, the initial evaluation of choice is chest radiography.

If the chest radiograph is *normal* and the patient is at risk for malignancy, consider computed tomography and bronchoscopy.

If the chest radiograph is *normal* and the patient is a smoker, computed tomography should be considered.

If the chest radiograph is *abnormal*, computed tomography should be obtained to delineate the abnormality.

In addition, multidetector computed tomography prior to embolization can reveal the source of hemoptysis.

13.8 Management Approaches

13.8.1 Massive Hemoptysis

Initial management entails maintaining airway patency, localizing the source, and controlling the bleeding. In massive hemoptysis, the threat is asphyxiation, not exsanguination. If intubation is necessary, a large-bore endotracheal tube (8 mm in diameter or greater) is preferred in order to allow for fiber-optic evaluation. If the source is known, the patient can be placed in the lateral decubitus position toward the site of bleeding [7]. Early bronchoscopy is the procedure of choice as it facilitates localization and possible intervention. Rigid bronchoscopy is preferred as it allows for more effective suctioning. If bronchoscopy is unsuccessful, interventional angiography is the next choice in therapy. These patients are typically admitted to the hospital – even in an intensive care setting. Consultation with a pulmonologist is advisable as well. Lastly, percutaneous embolization can be used to slow or stop the hemorrhage before potential surgery.

13.8.2 Non-massive Hemoptysis

A stable patient with non-massive hemoptysis should undergo initial evaluation with chest radiography. The most common cause of negative chest radiography in a patient with acute non-massive hemoptysis is bronchitis. Low-risk patients may be treated on an outpatient basis with close primary care follow-up and antibiotics if indicated [8]. If the hemoptysis persists or is unexplained, outpatient pulmonology evaluation may be needed. In patients with a mass found on chest radiography or in patients with negative radiography who are high risk for lung malignancy, outpatient bronchoscopy is indicated.

References

1. Jeudy J, Khan AR, Mohammed TL, et al. ACR appropriateness criteria hemoptysis. J Thorac Imaging. 2010;25(3):W67–9.
2. Wong ML, Szkup P, Hopley MJ. Percutaneous embolotherapy for life-threatening hemoptysis. Chest. 2002;121(1):95–102.
3. Yendamuri S. Massive airway hemorrhage. Thorac Surg Clin. 2015;25(3):255–60.
4. Tintinalli JE, Stapczynski JS. Tintinalli's emergency medicine: a comprehensive study guide. New York: McGraw-Hill; 2011. 1 online resource (xl, 2120 pages).
5. Harrison TR, Braunwald E. Hemoptysis. In: Harrison's principles of internal medicine. 15th ed. New York: McGraw-Hill; 2001. p. 203–6.
6. Sood R, Mukhopadhyaya S. Approach to a patient with haemoptysis and a normal chest X-ray. J Indian Acad Clin Med. 2002;3:14–22.
7. Jean-baptiste E. Clinical assessment and management of massive hemoptysis. Crit Care Med. 2000;28(5):1642–7.
8. Bidwell JL, Pachner RW. Hemoptysis: diagnosis and management. Am Fam Physician. 2005;72(7):1253–60.

Chapter 14
Abdominal Pain

Tara Dyson

14.1 Introduction

Abdominal pain is one of the most common complaints in the emergency department (ED), comprising approximately 5–10 % of total visits [1–3]. The differential diagnosis is broad and challenging, and about a fourth of these patients seen in the ED with abdominal pain are either discharged or admitted with undifferentiated abdominal pain. This poses a challenge for both the primary care and specialist physician, as well as for the patient.

For the most part, acute and life-threatening causes of abdominal pain are addressed during the ED visit. Most patients seen in outpatient settings would either have chronic or recurrent abdominal pain or in follow-up for acute abdominal pain. In this chapter, we will limit our discussion to chronic and recurrent abdominal pain.

14.2 Special Populations

These groups of patients may need special attention because of the broader differential diagnosis and because they may have atypical signs and symptoms of abdominal pain and thus a greater potential for serious complications [4].

14.2.1 Women

- Nonpregnant women presenting with lower abdominal pain should prompt the physician to assess for underlying pelvic disease including:
  - Infectious etiologies (i.e., sexually transmitted infections)
  - Dysmenorrhea

T. Dyson, MD
Division of Gastroenterology, Department of Internal Medicine,
PO BOX 100277, Gainesville, FL 32610-0277, USA
e-mail: dysont@ufl.edu

B. Desai, A. Desai (eds.), *Primary Care for Emergency Physicians*,
DOI 10.1007/978-3-319-44360-7_14

  - Endometriosis
  - Gynecological malignancies

- Pregnancy-related complications may include:
  - Ectopic pregnancies.
  - Preeclampsia.
  - Placental rupture.
  - Pregnancy should *always* be considered especially in women of the childbearing age.

- Postmenopausal women.
  - Malignancy [4]
    - For example, endometrial carcinoma

14.2.2 Older Adults

- A number of older adults have underlying chronic diseases such as atherosclerosis, arthritis, and long-standing autoimmune disease, and because of the chronicity and slow progression of these chronic diseases, older patients may have atypical and very subtle signs and symptoms as compared to the younger patients [5].
- This makes the examining physician vulnerable to missing severe, life-threatening disease.
- It is important to determine the patient's risk factors and to be vigilant identifying subtle disease.
- Common serious events may include:
  - Ruptured abdominal aortic aneurysm
  - Mesenteric ischemia
    - Both acute and chronic
  - Myocardial infarction
  - Malignancies

14.2.3 Immunocompromised and Immunosuppressed Patients

- Similar to older adults, these patients may also present with subtle signs and symptoms.
- Immunocompromised patients include:
  - Immune deficient
  - HIV

  - Diabetics
  - Patients on dialysis
- Immunosuppressed patients include:
  - Patients undergoing chemotherapy
  - Patients on short- or long-term immunosuppressants)
- This group of patients may not be able to illicit an appropriate response to infections or inflammation [6].

14.3 Evaluation

Initial evaluation involves a thorough history and physical examination which will eventually help the examining physician determine the severity and acuteness of the disease.

- Patients that present with intense, persistent abdominal pain, extreme tenderness on exam, and features of hemodynamic instability would be concerning for an acute abdomen (i.e., appendicitis, peritonitis, mesenteric ischemia, aortic dissection, etc.) and would need emergent treatment.
- Patients that present with fevers, gastrointestinal bleeding, anemia, weight loss, and signs of malnutrition or dehydration may still have an organic etiology of pain and need an urgent – not emergent –evaluation.
- A pitfall is not fully examining the patient.
  - Since abdominal wall pain or examination of certain skin lesions (i.e., herpetic lesions) should be assessed during the initial visit as these conditions can be easily managed and unnecessary and expensive work-up avoided.

Once initial evaluation has been performed and the differential diagnosis narrowed, subsequent work-up may be necessary. This may potentially involve obtaining certain labs, imaging, endoscopic evaluation, and potentially even surgical procedures.

14.4 Differential Diagnosis

	Clinical features	Comment(s)
Epigastric pain		
Acute myocardial infarction	May be associated with shortness of breath and exertional symptoms	Elderly patient with risk factors for coronary artery disease. EKG may be consistent for ST elevation myocardial infarction

	Clinical features	Comment(s)
Acute pancreatitis	Acute onset, severe epigastric pain radiating to the back	Associated with elevated lipase acutely
Chronic pancreatitis	Epigastric pain radiating to the back. Calcifications and atrophy may be seen on imaging	Associated with pancreatic insufficiency. Some may have diabetes. Lipase may not be elevated in cases of chronic pancreatitis
Peptic ulcer disease	Persistent, sharp stabbing pain. Usually associated with food intake	Associated with NSAID use, *H. pylori* infection
Gastroesophageal reflux disease	Heartburn, usually associated with food intake	
Gastritis/gastropathy	Abdominal discomfort/pain, heartburn, nausea, vomiting, and hematemesis	Variety of etiologies including alcohol, NSAIDs, *H. pylori*
Gastroparesis	Nausea, vomiting, abdominal pain, early satiety, postprandial fullness, and bloating	Most causes are idiopathic, due to diabetes, hypothyroidism, or postsurgical in nature. Also seen in Parkinson's disease
Left upper quadrant pain		
Splenomegaly	Pain may radiate to left shoulder. May have palpable spleen on examination	Multiple etiologies
Splenic infarct	Severe LUQ pain. Splenomegaly	Associated with hypercoagulable states and embolism
Splenic abscess	Associated with fever and LUQ tenderness	Common complication after splenic infarction
Splenic rupture	May complain of LUQ, left chest wall, or left shoulder pain that is worse with inspiration	Most often associated with trauma
Right upper quadrant pain		
Biliary colic	Intermittent, intense, dull discomfort	Associated with gall bladder or biliary disease
Acute cholecystitis	Intense pain and sometimes with fever. May have a (+) Murphy's sign	May see pericholecystic fluid and gallstones on ultrasound
Acute cholangitis	Fever, jaundice, RUQ pain	Associated with biliary duct infection or obstruction. Patients are typically very ill
Sphincter of Oddi dysfunction	Intermittent biliary-type pain. May occasionally have elevated lipase and amylase	Biliary-type pain without other apparent causes
Acute hepatitis	RUQ pain with fatigue, malaise, nausea, vomiting, and anorexia. Patients may also have jaundice	Variety of etiologies include hepatitis A, alcohol, and drug induced

	Clinical features	Comment(s)
Perihepatitis (Fitz-Hugh-Curtis syndrome)	Pain associated with coughing or breathing. Referred pain to the right shoulder	Usually in women with gonorrhea or chlamydia infection
Liver abscess	Fever and abdominal pain. Transaminitis is common	Risk factors include diabetes, underlying hepatobiliary or pancreatic disease, or liver transplant
Budd-Chiari syndrome	Associated with signs of portal hypertension, without cirrhosis. Hepatosplenomegaly	From occlusion of the hepatic vein
Portal vein thrombosis	Associated with signs of portal hypertension	Most commonly associated with cirrhosis
Lower abdominal pain		
Appendicitis	Severe, mostly localized RLQ pain. Associated with leukocytosis, anorexia, vomiting	Emergent surgical evaluation is necessary
Diverticulitis	Constant pain, common in the LLQ (sigmoid colon)	Inflammation of a diverticulum
Epiploic appendagitis	Acute or subacute abdominal pain	Benign and self-limited condition of epiploic appendages
Nephrolithiasis	Severe flank pain, but may have back, flank, or abdominal pain	Causes symptoms as stone passes from renal pelvis to ureter. Pain may shift from flank to groin
Pyelonephritis	Associated with dysuria, frequency, urgency, hematuria, fever, chills, flank pain, and costovertebral angle tenderness	Associated with pyuria. Computed tomography may show inflammatory stranding around the affected kidney
Acute urinary retention	Present with lower abdominal pain and discomfort; inability to urinate	Associated with urinary obstruction. Drainage of the bladder is typically curative
Cystitis	Associated with dysuria, frequency, urgency, and hematuria	
Inflammatory bowel disease	Chronic abdominal pain, associated with diarrhea and bloody stools. Crohn's patient may have fistula formation or bowel obstruction	May be from Crohn's disease or ulcerative colitis
Infectious colitis	Diarrhea as the predominant symptom but may also have associated abdominal pain, which may be severe	
Abdominal wall pain		
Abdominal wall hernias	A small subcutaneous mass may be felt in the midline (epigastric hernia) or through the Spigelian fascia (Spigelian hernia)	

	Clinical features	Comment(s)
Surgical scars	Pain around previous surgical scar, usually at the edge of the scar	May be seen on either incisional scar or from laparoscopy (insertion point of trocar)
Abdominal wall endometriosis	Common in women with previous cesarean section	
Radicular pain	Disease involving T7–T12 nerve roots. Common in patient with diabetic neuropathy and spinal cord disease	Exam the spine and back. Tricyclic drugs may be beneficial
Costochondritis	Tenderness in the lower chest or xiphoid. Associated with nausea and even vomiting when pressure is applied to the painful site	
Rectus sheath hematoma	Hematoma or ecchymosis may be seen in the anterior abdomen. May have a history of trauma to the abdomen	Common in patient with bleeding dyscrasia or on anticoagulation
Irritable bowel syndrome	Abnormal stool frequency (≤3 bowel movements per week or >3 bowel movements per day), abnormal stool form (lumpy/hard or loose/watery), defecation straining, urgency, or a feeling of incomplete bowel movement, passing mucus, and bloating	Patients must not have rectal bleeding, nocturnal or progressive abdominal pain, weight loss, anemia, elevated inflammatory markers, or electrolyte abnormalities
Functional dyspepsia	Postprandial fullness, early satiety, epigastric pain or burning for the last 3 months with symptom onset at least 6 months before diagnosis, *and* exclusion of other causes of dyspepsia with EGD and other testing	Cause is unclear. May be associated with gastric motility and compliance, visceral hypersensitivity, *H. pylori*, altered gut microbiota, and psychosocial dysfunction

14.5 Physical Examination

The physical examination must always be comprehensive. Subtle behavioral signs alone may help the physician guide his/her approach to examining the patient and arriving with an accurate diagnosis.

- Vital sign assessment
  - The presence of fever should alert the physician to a possible infectious etiology.
  - Tachycardia may have multiple etiologies including:
    - An appropriate response to fever

    - The potential use of medications or drugs, including over-the-counter medications and illicit substances
    - Active pain
  - Pulse oximetry.
    - May alert the clinician to the presence of an underlying pulmonary pathology
- Behavior
  - Comfortable
    - Benign or functional disease
  - Lethargic
    - The use of narcotics, marijuana, other sedating medications
  - Restlessness
    - Associated with withdrawal symptoms
  - Guarded
    - Seen in visceral disease or peritonitis.
    - Laying still may indicate that the patient may have peritonitis; any movement may cause pain.
- Neurologic assessment should focus on:
  - Level of alertness
    - Sedation and agitation may be associated with drugs.
  - Encephalopathy
    - Underlying metabolic or drug-related issues
- Cardiovascular and pulmonary examination
  - Crackles and rales may indicate underlying pneumonia.
- Gastrointestinal examination
  - Auscultation
    - Bowel sounds are absent in ileus and high-pitched in obstruction.
    - Friction rub may be seen with splenic infarct.
  - Palpation
    - Pain to light touch can be seen in peritonitis.
    - Movement may worsen pain in patients with peritonitis.
  - Percussion
    - Tympany signifies bowel distention.

    - Dullness may signify organomegaly or mass.
    - Ascites may have shifting dullness.
  - Special maneuvers (Carnett's, psoas, Murphy's, etc.)
  - Rectal/pelvic examination (rectal mass/strictures, ectopic pregnancy, salpingitis, tubo-ovarian abscess)

14.6 Diagnostic Work-Up

The diagnostic work-up will necessarily be dependent upon the clinical picture of the patient. Not all tests will be of value in all patients.

- Laboratory tests may include:
  - CBC, BMP, liver panel, lipase
  - Antibodies (tissue transglutaminase, etc.)
  - Pregnancy test
    - Mandatory for all women of childbearing age
  - Urinalysis
- Radiologic tests may include:
  - X-rays
  - CT scans
  - MRI
- Endoscopy – typically reserved for outpatient settings, unless the patient presents with significant hematemesis and abdominal pain, in which case it becomes an urgent test.
  - EGD
  - Enteroscopy
  - Colonoscopy
  - ERCP
- Laparoscopy/surgery.
  - For examination of extraluminal disease and for obtaining biopsies
- Special testing including:
  - Nuclear testing
  - Motility testing
  - Paracentesis
  - Trigger point injections

14.7 Management

Due to the wide nature of abdominal pain etiologies, management will solely depend upon the presentation of the patient. Emergency department management will necessarily focus on the acute treatment of pain which may include modalities not typically used in the primary care setting (e.g., opioids).

- Medical
  - Pain control may include:
    - Heating pad
    - Medications including:
      - NSAIDs
      - Acetaminophen
      - Opioids
      - Muscle relaxants
  - Pain modulators
    - Tricyclic antidepressants
    - Selective serotonin reuptake inhibitors (SSRIs)
    - Serotonin-norepinephrine reuptake inhibitors (SNRIs)
  - Symptom relief
    - Antidiarrheals
    - Laxatives
    - Antiemetics
    - Proton-pump inhibitors
    - Histamine-2 blockers
- Endoscopic
  - Celiac plexus injections
  - Endoscopic retrograde cholangiopancreatography with decompression
  - Botox injections
- Surgical
  - Placement of pain-modulating devices
- Physical therapy
- Psychiatric/behavioral modalities

References

1. Brewer BJ, Golden GT, Hitch DC, et al. Abdominal pain. An analysis of 1,000 consecutive cases in a University Hospital emergency room. Am J Surg. 1976;131:219.

2. Powers RD, Guertler AT. Abdominal pain in the ED: stability and change over 20 years. Am J Emerg Med. 1995;13:301.
3. Sanson TG, O'Keefe KP. Evaluation of abdominal pain in the elderly. Emerg Med Clin North Am. 1996;14:615.
4. Kamin RA, Nowicki TA, Courtney DS, Powers RD. Pearls and pitfalls in the emergency department evaluation of abdominal pain. Emerg Med Clin North Am. 2003;21:61.
5. de Dombal FT. Acute abdominal pain in the elderly. J Clin Gastroenterol. 1994;19:331.
6. Thuluvath PJ, Connolly GM, Forbes A, Gazzard BG. Abdominal pain in HIV infection. Q J Med. 1991;78:275.

Chapter 15
Irritable Bowel Syndrome

Elvira Mercado

15.1 Introduction

Irritable bowel syndrome (IBS) is the most common functional bowel disease. It affects up to 20 % of the population. It is, however, difficult to estimate prevalence as many patients do not seek medical care [1].

Patients with IBS present to the emergency department primarily for abdominal pain. This is a chronic, relapsing disease. It is imperative that emergency medicine physicians properly consider red flag symptoms but use judicious discretion to decrease unnecessary testing and imaging. The yearly cost of IBS is estimated at $20 billion with direct cost being highest in the initial diagnosis of the disease [2].

IBS is a clinical diagnosis characterized by abdominal pain or discomfort with alteration in bowel habits. Multiple comorbidities are often associated with IBS such as fibromyalgia, chronic fatigue syndrome, and chronic pelvic pain. Patients with IBS will often present to emergency department with other coexisting symptoms due to associated conditions [3].

Current recommendations from the American Gastroenterological Society and American College of Gastroenterologist are to diagnose IBS without alarm features by symptom-based guidelines such as the Rome Criteria. There is no pathognomonic test or symptom for IBS [2].

15.2 Diagnostic Criteria

15.2.1 Rome III Criteria

Recurrent abdominal pain or discomfort (usually crampy and dynamic) at least 3 days per month in the past 3 months with two or more of the following:

- Improvement with defecation

E. Mercado, MD
Department of Community Health and Family Medicine, UF Health Shands, Gainesville, FL, USA
e-mail: elvy@ufl.edu

B. Desai, A. Desai (eds.), *Primary Care for Emergency Physicians*,
DOI 10.1007/978-3-319-44360-7_15

- Onset with change in bowel frequency
- Onset with change in stool consistency

Symptom onset should be at least 6 months prior to formal diagnosis [1, 4].
Irritable bowel syndrome is classified into the following subgroups:

- Irritable bowel syndrome constipation predominant (IBS-C)
- Irritable bowel syndrome diarrhea predominant (IBS-D)
- Irritable bowel syndrome mixed (IBS-M)

Without alarm symptoms, appropriate workup in the emergency department includes CBC, ESR, and CRP. In diarrhea-predominant disease, celiac testing with endomysial antibodies or tissue transglutaminase is recommended as well. Age-appropriate colorectal cancer screening should be obtained in the outpatient setting [5]. Biomarkers are becoming available for diagnostic use but their validity and clinical significance are still undetermined [6].

Although the cause of IBS is felt to be multifactorial, the following factors are felt to contribute to the disease symptomatology [3]:

- Altered pain perception
- Altered brain-gut interaction
- Increased intestinal permeability
- Visceral hypersensitivity

15.3 Differential Diagnosis

Many symptoms of IBS are overlapping with other GI diseases [7]. It is important to screen for red flag symptoms to appropriately identify other conditions.

Differential diagnosis:

- Carcinoid tumor
- Celiac disease
- Colorectal cancer
- Diverticular disease
- Drug use (opiates, antidepressants, calcium channel blockers)
- Gastrointestinal infection (small bowel bacterial overgrowth, *Giardia*, *Clostridium difficile*)
- Thyroid disease
- Inflammatory bowel disease (IBD)
- Ischemic colitis
- Lactose intolerance

15.4 History and Physical Examination

In the emergency department, it is critical to obtain a complete history and physical examination to rule out other conditions. This information will allow for a confident clinical diagnosis.

The history should also uncover any potential risk factors for IBS [1, 3].

- Female sex
- Lower socioeconomic status
- Peak age 20–50
- Comorbid psychiatric conditions
- History of abuse (physical, sexual, emotional)
- Psychosocial stressors
- Food intolerances
- Enteric infections

It is imperative to screen for any red flags indicative of organic pathology [8, 9].

- Anemia (cancer, IBD)
- Chronic severe diarrhea particularly with nighttime symptoms (cancer, infection, IBD)
- Family history of GI disease (cancer, IBD, celiac disease)
- Signs of bleeding (cancer, polyps, IBD, ischemic colitis)
- Fever (infection, IBD)
- Weight loss (cancer, IBD)
- Onset after age 50 (cancer)
- Palpable mass on exam (cancer)
- Elevated inflammatory markers (IBD, infection)

Any presence of red flags needs to be further evaluated. In the emergency room setting, investigation of red flag symptoms is important to ensure proper diagnosis.

In the absence of alarm symptoms, ultrasound, endoscopy, or stool testing is not recommended and leads to increased healthcare costs [5, 10]. Once red flags are eliminated and preliminary labs are negative, reassurance should be provided [10].

15.5 Treatment of IBS

Once a diagnosis is made, there are several indicated treatments for IBS that can be initiated by emergency room physicians. Treatment of IBS varies on the individual and their symptoms. Treatment will usually encompass a combination of lifestyle and diet modification, medications, and behavioral interventions [3].

Therapies include:

1. Education
   - Improve understanding of disease, pathophysiology, and recurrence.
   - Establish goals of IBS therapy as control of symptoms and improvement of quality of life (not to cure all symptoms).
2. Diet modification
   - Avoid trigger foods and common inciting foods (lactose, gluten).
   - Consider low fermentable oligosaccharide, disaccharide, monosaccharide, and polyol (FODMAP) diet.
3. Behavioral and alternative therapies
   - Cognitive behavioral therapy
   - Hypnosis
   - Biofeedback
   - Stress reduction
   - Peppermint oil
   - Acupuncture
4. Medication management should include:
   - Careful consideration of current comorbidities.
   - Consideration of medication interactions and side effects.
   - Starting medications at a low dose.
   - Providing a short supply until a primary care physician or gastroenterologist reevaluates the patient.
   - Avoiding use of narcotics, which can cause dependence and worsen IBS symptoms.

15.6 Pharmacologic Treatment

Many medications are currently indicated in treatment of IBS. Treatment is guided by predominant symptoms and severity of symptoms. Symptomatic treatment can be initiated in the emergency department. Other therapies can also be initiated if symptom-based control has failed. Therapies should undergo a trial of at least weeks.

15.6.1 Over-the-Counter Fiber or Laxatives

Fiber and osmotic laxatives are first-line agents used to manage constipation in IBS. Long-term uses of these medications are effective and critical in symptomatic relief. These supplements should be started at low dose and titrated to desired stool consistency.

Fiber/laxatives
Psyllium or other soluble fiber
Polyethylene glycol

Pitfalls
1. Nonsoluble fiber and polyethylene glycol can worsen bloating, gas, and cramping.
2. Wheat bran should be avoided.
3. Require self-titration and adjustment if diarrhea occurs.
4. May not improve pain of IBS.
5. Cost of over-the-counter medication not covered by insurance.

15.6.2 Over-the-Counter Antidiarrheal Medications

Loperamide is the first-line agent in management of diarrhea in IBS. Other antidiarrheal medications are not currently recommended for IBS. Loperamide works in multiple mechanisms including inhibition of peristalsis, prolonging gut transit, and reduction of fecal volume [3].

Pitfalls
1. Can lead to constipation
2. May not improve pain and global symptoms of IBS
3. Cost of over-the-counter medication not covered by insurance

15.6.3 Antispasmodics

Antispasmodics are used as needed or prophylactically preprandial to control abdominal pain, diarrhea, bloating, and urgency. This is achieved through smooth muscle relaxation.

Antispasmodics
Hyoscyamine
Dicyclomine
Methscopolamine

Pitfalls
1. Can worsen constipation dominant disease
2. Caution with anticholinergic side effects (fatigue, dry mouth, dizziness)
3. Caution with sedation
4. Caution use in elderly and cognitively impaired

15.6.4 Prosecretory Agents

Two agents are currently FDA approved for IBS-C patients. Lubiprostone is a chloride channel activation increasing intestinal fluid secretion. Linaclotide is a novel agent that reduces activation of visceral pain fibers.

FDA approved prosecretory agents
Lubiprostone
Linaclotide

Pitfalls

1. Lubiprostone can cause dose-dependent nausea and should be taken with food.
2. Linaclotide causes diarrhea and should be given 30–60 min prior to breakfast to limit diarrhea.
3. Costly and poor formulary coverage.

15.6.5 Antidepressants

Antidepressants are becoming more standard in care of IBS given their effects on pain perception, mood, and motility. The two classes of agents mostly used and proven to be effective are tricyclic antidepressants (TCAs) and selective serotonin reuptake inhibitors (SSRIs). These agents are recommended in moderate to severe IBS and have been shown to effectively reduce abdominal pain of IBS.

Antidepressants
TCAs (nortriptyline, amitriptyline)
SSRIs (citalopram, paroxetine)

Pitfalls

1. May take weeks and several dose titrations to achieve maximum effect.
2. Caution with TCA dose-dependent anticholinergic side effects (dry mouth, urinary retention, constipation, sedation, weight gain).
3. Caution with TCA-induced QT prolongation (may need EKG to assess prior to starting).
4. SSRIs can cause sexual dysfunction, agitation, nausea, drowsiness, and diarrhea.
5. Caution with use of SSRIs and other serotonergic medications that may increase risk of serotonin syndrome.

15.7 Medications to Avoid

Although therapeutic medication treatment is important, it is also the key to avoid medications that can possibly exacerbate IBS symptoms. A risk-benefit analysis should be performed prior to use of the following over-the-counter and prescription therapies [3]:

Over the counter

- Antihistamines
- Calcium
- Iron
- Magnesium
- Nonsteroidal anti-inflammatory agents
- Wheat bran

Prescription drugs

- Antibiotics
- Some antidepressants
- Antiparkinson medications
- Antipsychotics
- Calcium-channel blockers
- Diuretics
- Metformin
- Narcotics
- Sympathomimetics

A trial of medication discontinuation may be beneficial if it is believed to be exacerbating symptoms of IBS. Patients with IBS often report multiple medication intolerances.

References

1. Sayuk GS, Gyawali CP. Irritable bowel syndrome: modern concepts and management options. Am J Med. 2015;128:817–27.
2. Burbige EJ. Irritable bowel syndrome: diagnostic approaches in clinical practice. Clin Exp Gastroenterol. 2010;3:127–37.
3. Chey WD, Kurlander J, Eswaran S. Irritable bowel syndrome a clinical review. JAMA. 2015;313(9):949–58.
4. Ford AC, Moayyedi P, Lacy BE, et al. American College of Gastroenterology Monograph on the management of irritable bowel syndrome and chronic idiopathic constipation. Am J Gastroenterol. 2014;109:s2–S26c.
5. National Institute for Health and Care Excellence. Irritable bowel syndrome in adults: diagnosis and management (online). 2015. Available from http://guidance.nice.org.uk/CG61.

6. Chira A, Dumitrascu DL. Serum biomarker for irritable bowel syndrome. Chijul Med. 2015;88(3):258–64.
7. Wilkins T, Pepitone C, Biju A, Schade R. Diagnosis and management of IBS in adults. Am Fam Physician. 2012;86(5):419–26.
8. Hadley SK, Gaarder SM. Treatment of irritable bowel syndrome. Am Fam Physician. 2005;72(12):2501–8.
9. Viera AJ, Hoag S, Shaughnessy J. Management of irritable bowel syndrome. Am Fam Physician. 2002;66(10):1867–75.
10. Graham L. ACG releases recommendations on the management of irritable bowel syndrome. Am Fam Physician. 2009;79(12):1108–17.

Chapter 16
Bloating

Bobby Desai and Alpa Desai

16.1 Introduction

Bloating is a bothersome complaint that is commonly reported to physicians; it ranks second to only abdominal pain in frequency of complaint to physicians [3]. It is estimated that around 11 % of the general population report frequent bloating [1], while in another study, 31 % of patients met the Rome I criteria for functional bloating [5]. In fact, 90 % of patients with a diagnosis of irritable bowel syndrome will report symptoms of bloating [6]. Unfortunately, bloating is a nonspecific term that relates to the patient's subjective sensation of their abdominal distention, fullness or excessive gas, and the physician's objective determination of true abdominal distention [2]. Furthermore, abdominal bloating is present in multiple functional abdominal disorders which include both non-emergent and emergent processes. When bloating is not part of another functional gastrointestinal process, it is included in the Rome III criteria as "functional bloating" [source]. The Rome III criteria for functional bloating include the repeated feeling of bloating or a noticeable abdominal distention for at least 3 days per month, onset of symptoms for at 6 months prior to diagnosis along with the presence of symptoms for at least 3 months, and inability to diagnose dyspepsia, irritable bowel syndrome, or another functional gastrointestinal disorder [7].

Since the natural history of bloating is poorly understood, diagnosis of this entity is sometimes difficult to the inability of the clinical to identify measureable parameters for assessment and grading, and, in many instances, bloating lacks a clear pathophysiologic explanation and a singular management strategy which makes the entity a quandary for physicians. Unfortunately, bloating can cause significant patient distress and concern which may prompt a subsequent presentation to the emergency department (ED). In fact, in those patients who did not have a diagnosis of irritable bowel syndrome, three-fourths described their symptoms as moderate to severe, and 50 % reduced their daily activities due to the symptoms of bloating [8].

B. Desai, MD, Med (✉)
Emergency Medicine, University of Florida, Gainesville, FL, USA
e-mail: bdesai@ufl.edu

A. Desai, DO
Community Health and Family Medicine, University of Florida, Newberry, FL, USA
e-mail: desaas@shands.ufl.edu

B. Desai, A. Desai (eds.), *Primary Care for Emergency Physicians*,
DOI 10.1007/978-3-319-44360-7_16

16.2 Pathophysiology

The pathophysiology of this disorder is complicated and relies on the understanding on a multitude of factors. These include the natural production of gas within the gastrointestinal tract (GIT), the role of gut microflora, transit time of intestinal contents, and, finally, neuronal functional with the GIT [9]. Furthermore, there are different theories on bloating etiology that range from an increase in luminal contents ranging from fat, liquids, gas, and stools to an impairment of abdominal emptying to an alteration in intra-abdominal volume displacement (also known as the abdomino-phrenic theory) or increased intestinal stimuli due to the use of specific treatments or medications. These include antibiotics, probiotics, prokinetic agents, medications for spasm and gas reduction, and tricyclic antidepressants [4].

16.2.1 Intestinal Gas Production

The normal individual has approximately 100–200 cc of gas within the gastrointestinal tract, which increases after eating, especially in the colon. Stomach distention and corresponding small bowel stimulation subsequent to eating can accelerate the transit of gas; lipids, however, can cause the retention of gas, especially within the early small bowel [10, 11]. Gas production in the colon occurs primarily through bacterial metabolism, especially those foods that are not completely digested within the small intestine, including sugars such as lactose and fructose [14]. Other foods such as legumes and complex carbohydrates are metabolized within the colon. Additional sources of gas within the GI tract include the swallowing of air, ingestion of carbonated beverages, and acid/alkali neutralization in the upper GIT [12, 13]. Furthermore, the small intestine readily absorbs carbon dioxide which may be promptly consumed by some colonic bacterial species which also consume hydrogen. Thus, the total amount of gas in the intestine is ever in flux due to the normal passing of flatus; healthy patients tolerate intestinal gas easily due to the efficient evacuation.

Even though an excessive volume of intestinal gas has been proposed as a mechanism for bloating and distention, the vast majority of studies do not support this theory [15]. Infusion of a large amount of gas into the intestinal tract of healthy volunteers produced only small changes in abdominal girth, whereas patients with IBS show large changes in abdominal girth even in the absence of gas infusion [17]. While it is known that increased gas volumes are present in IBS patients compared to controls, there is a poor correlation between intra-abdominal gas contents and bloating [16]. Therefore, it has been theorized that the impaired transit of gas or an abnormal distribution of gas may be more problematic.

16.2.2 Impairment of Gas Transit

Studies have shown that patients with IBS have abnormalities in intestinal transit which may cause the symptoms of gas and bloating. Patients with IBS-related constipation have an increased prevalence of abdominal distention and bloating. Furthermore, in the study of gas infusion noted above, patients with IBS experienced more during the infusion than healthy volunteers. Furthermore, in another study of IBS patients subsequent to gas infusion, 90% of IBS patients developed intestinal gas retention compared to control subjects [17]. The amount of abdominal distension correlated with gas retention in these patients. IBS patients also had impaired gas clearance from both the small intestine and proximal (not distal) colon [18].

16.2.3 Impaired Evacuation

The ineffective evacuation of gas results in gas retention and the symptom of pain with abdominal bloating. Specific patients, especially those with IBS, constipation, and functional bloating, have difficulty effectively evacuating gas and more likely to develop symptoms of abdominal distention [19].

16.2.4 Other Potential Causes

These include the unconscious changing of body position to a more lordotic one, abnormal abdominal-diaphragmatic reflexes where the diaphragms of specific patients descend while the ventral muscles relax which leads to a subsequent abdominal girth [20], and abnormal sensation or perception where patients with IBS are more sensitive to stretch and distention [21].

16.3 Risk Factors

- Females
  - Especially during menstruation
- Younger age
- Presence of functional gastrointestinal disorder
  - Irritable bowel syndrome
  - Dyspepsia

- Higher Somatic Symptom Checklist score
- History of cholecystectomy
  - In terms of functional bloating
- History of hysterectomy
  - In terms of functional bloating
- History of abdominal surgery
  - For organic bloating

16.4 Differential Diagnosis

- Abnormal colonic transit
- Acute or subacute bowel ischemia
- Aerophagia
- Anorexia and bulimia
- Bacterial overgrowth in the small bowel
- Celiac disease
- Changes or disturbances to colonic microflora
- Constipation
  - Acute or chronic
- Dietary
  - Increased carbohydrate intake
  - Consumption of nonabsorbable sugars

    Sorbitol
  - Lactose intolerance
  - Intolerance to fructose
  - Sensitivity to gluten
- Disorders of the pelvic floor
- Diverticulosis
  - Small or large bowel
- Enteropathogenic infections
- Functional bloating
- Functional dyspepsia
- Gastroparesis
- Gastric outlet obstruction
- Irritable bowel syndrome

- Malabsorptive conditions
- Neoplastic disorders
- Organic causes of small bowel motility dysfunction
  - Systemic lupus erythematosus
  - Amyloidosis
  - Neurofibromatosis
  - Parkinson's disease
  - Diabetes mellitus
  - Scleroderma
  - Thyroid disorders, especially hypothyroidism
  - Muscular dystrophies

16.5 History

Many patients that present to the emergency department with complaints of bloating do so due to the uncomfortable sensation of the distended abdomen. However, though bloating can cause significant patient distress, it typically represents a benign condition. Critical historical features to ascertain are:

- The time and duration of symptoms
  - Acute nature of condition
    - For an acutely distended abdomen, the physician must consider a potential surgical etiology.
  - Acute on chronic nature
    - These patients typically have a known condition such as IBS that has caused bloating in the past, but their current presentation to ED is due to an acute deterioration.
- Diet
  - Association of symptoms with specific foods including:
    - Dairy products
    - Fruits (fructose)
    - Sucrose-containing products
    - Fatty foods
    - Beans
    - Hx of drinking carbonated beverages
- The presence of associated gastrointestinal problems
  - Irritable bowel syndrome
  - Gastroparesis

- The presence of significant abdominal pain
  - The clinician must determine if there are palliating features such as relief with the passage of flatus.
  - Significant abdominal pain with the presence of bloating and vital sign abnormalities may be concerning for an acute surgical emergency.
- Prior diagnosis of functional gastrointestinal disorders
  - Bloating is one of the most common and bothersome complaints in patients with several functional disorders, including irritable bowel syndrome, functional dyspepsia, and functional constipation [4].
- History of psychiatric conditions
  - Anxiety
  - Depression
- History of metabolic disorders
  - Diabetes mellitus
  - Hypothyroidism
- History of:
  - Unintentional weight loss.
  - Anemia
    - The above two may be a sign of a malabsorptive process.
  - Nocturnal gastrointestinal symptoms.
  - Vomiting.
  - All may suggest an organic cause of bloating.
- Medication history
  - May identify agents that slow gastrointestinal transit time or alter motility
- Family history may be important including:
  - History of celiac disease
  - Inflammatory bowel disease
  - Intolerance to lactose

16.6 Physical Examination

Examine the patient closely for:

- Evidence of weight loss
- Anemia
  - Conjunctival pallor
  - Pale skin

- Anxiety
- Hyperventilation with subsequent aerophagia
- Liver and spleen enlargement
- Evidence of intestinal obstruction
  - Hyperactive bowel sounds
  - Tympany on percussion
- Evidence of ascites
- Attempt to examine patient in the supine and standing positions
  - This may confirm the distention by removing exaggeration of lumbar lordosis with its consequent abdominal protuberance.

16.7 Diagnosis

Since a formal diagnosis is out of the purview of the emergency physician, the evaluation of life-threatening processes is paramount. Thus, the workup initiated in the ED for bloating will entirely depend on the clinical scenario. If the bloating is acute, an evaluation of a potentially surgical etiology must be considered. If acute on chronic, typically a careful history and physical examination will elucidate the potential cause. For these conditions, appropriate follow-up will be paramount.

16.8 Introduction to Treatment

Treatment of bloating is highly individualized and typically proceeds in a stepwise fashion. First is the identification of the main symptoms: abdominal distention, bloating, or both, which may provide insight into the underlying pathophysiology of the condition. From this point, the clinician has some options, including:

16.8.1 Education

1. Dietary modifications: Those patients who ingest dairy, fructose, and fiber, for example, should be counseled to limit intake of foods with these substances, since they readily ferment within the colon. Patients should be counseled to eliminate one at a time in order to identify the offending agent.
2. Exercise: Exercise and the limitation of recumbent periods has been shown to increase intestinal gas clearance and reduce symptoms of bloating [22].

16.8.1.1 Pharmacologic Treatment

Most nonprescription-based treatment options have not been well studied but may be of some benefit in specific patient population.

16.8.2 Over-the-Counter Medications

Simethicone – an antifoaming agent which alters elasticity of gas bubbles and improves the passage of flatus – has improved symptoms in some patients.

Bulking agents, including psyllium, have been used as first-line agents for patients with IBS and chronic constipation and has been shown to decrease the frequency and severity of bloating if taken for an extended period of time, typically months.

16.8.3 Probiotics

These are live microorganisms that may offer a health benefit when consumed in adequate amounts. These too have not been studied in-depth. Some probiotics, including those with *Lactobacillus acidophilus* and *Bifidobacterium lactis*, in those patients with nonconstipated functional bowel disorders improved bloating severity over an 8-week period [23].

16.9 Introduction to Prescription Medications

Caution is urged when considering these medications, and appropriate follow-up and consideration of consultation should be made prior to prescribing them.

16.9.1 Antibiotics

Rifaximin is a gut-selective antibiotic which is not absorbed systematically and has been well studied for the treatment of bloating. Patients who received this antibiotic (400 mg three times a day for 10 days) reported improvement in both IBS symptoms and bloating compared to placebo.

16.9.2 Antispasmodics

Since these medications are used to control abdominal pain in patients with IBS, it seems intuitive that these medications may be used to treat symptoms of bloating. However, since they relax smooth muscle, they may have the potential to further

cause gas accumulation in the GI tract as well as delay transit of gas through the GI tract. Therefore, their routine use is not recommended.

16.9.3 Tricyclic Antidepressants

This class of medication (desipramine) is used to treat functional abdominal pain and has been shown to be superior to cognitive behavioral therapy in those patients as well as improvement in bloating.

16.9.4 Prokinetic Agents: Neostigmine

This medication is a potent cholinesterase inhibitor that is typically used while in the hospital under the supervision of a specialist, which limits its utility.

16.9.5 Prokinetic Agents: Metoclopramide

This medication is a dopamine antagonist that is approved for the treatment of diabetic gastroparesis. Patients with this condition commonly have symptoms of bloating. This medication can be tried, but there are no studies that prove its benefit.

16.9.6 Osmotic Laxatives

Polyethylene glycol is a nontoxic, water-soluble substance that is absorbed in trace amounts and has been approved for the short-term treatment of chronic constipation. The usage of this medication has also been shown to relieve bloating as well.

References

1. Szarka L, Levitt M. Belching, bloating and flatulence. Am Coll Gastroenterol, 2006.
2. Azpiroz F, Malagelada JR. Abdominal bloating. Gastroenterology. 2005;129(3):1060–78.
3. Rao SSC. Belching, bloating, and flatulence. Postgrad Med. 1997;101(4):263–78.
4. Iovino P, Bucci C, Tremolaterra F, Santonicola A, Chiarioni G. Bloating and functional gastrointestinal disorders: where are we and where are we going? World J Gastroenterol: WJG. 2014;20(39):14407.
5. Drossman DA, Li Z, Andruzzi E, et al. U.S. householder survey of functional gastrointestinal disorders. Prevalence, sociodemography, and health impact. Dig Dis Sci. 1993;38:1569–80.
6. Houghton LA, Lea R, Agrawal A, Reilly B, Whorwell PJ. Relationship of abdominal bloating to distention in irritable bowel syndrome and effect of bowel habit. Gastroenterology. 2006;131:1003–10.

7. Longstreth GF, Thompson WG, Chey WD, Houghton LA, Mearin F, Spiller RC. Functional bowel disorders. Gastroenterology. 2006;130:1480–91.
8. Sandler RS, Stewart WF, Liberman JN, Ricci JA, Zorich NL. Abdominal pain, bloating, and diarrhea in the United States: prevalence and impact. Dig Dis Sci. 2000;45:1166–71.
9. Lacy BE, Gabbard SL, Crowell MD. Pathophysiology, evaluation, and treatment of bloating: hope, hype, or hot air? Gastroenterol Hepatol. 2011;7(11):729.
10. Serra J, Azpiroz F, Malagelada JR. Gastric distension and duodenal lipid infusion modulate intestinal gas transit and tolerance in humans. Am J Gastroenterol. 2002;97:2225–30.
11. Harder H, Serra J, Azpiroz F, Malagelada JR. Reflex control of intestinal gas dynamics and tolerance in humans. Am J Physiol Gastrointest Liver Physiol. 2004;286:G89–94.
12. Levitt MD. Volume and composition of human intestinal gas determined by means of an intestinal washout technic. N Engl J Med. 1971;284:1394–8.
13. Lasser RB, Bond JH, Levitt MD. The role of intestinal gas in functional abdominal pain. N Engl J Med. 1975;293:524–6.
14. Perez F, Accarino A, Azpiroz F, Quiroga S, Malagelada JR. Gas distribution within the human gut: effect of meals. Am J Gastroenterol. 2007;102:842–9.
15. Accarino A, Perez F, Azpiroz F, Quiroga S, Malagelada JR. Intestinal gas and bloating: effect of prokinetic stimulation. Am J Gastroenterol. 2008;103:2036–42.
16. Koide A, Yamaguchi T, Odaka T, et al. Quantitative analysis of bowel gas using plain abdominal radiograph in patients with irritable bowel syndrome. Am J Gastroenterol. 2000;95:1735–41.
17. Serra J, Azpiroz F, Malagelada JR. Impaired transit and tolerance of intestinal gas in the irritable bowel syndrome. Gut. 2001;48:14–9.
18. Hernando-Harder AC, Serra J, Azpiroz F, et al. Colonic responses to gas loads in subgroups of patients with abdominal bloating. Am J Gastroenterol. 2010;105:876–82.
19. Passos MC, Serra J, Azpiroz F, Tremolaterra F, Malagelada JR. Impaired reflex control of intestinal gas transit in patients with abdominal bloating. Gut. 2005;54:344–8.
20. Accarino A, Perez F, Azpiroz F, Quiroga S, Malagelada JR. Abdominal distention results from caudo-ventral redistribution of contents. Gastroenterology. 2009;136:1544–51.
21. Bouin M, Plourde V, Boivin M, et al. Rectal distention testing in patients with irritable bowel syndrome: sensitivity, specificity, and predictive values of pain sensory thresholds. Gastroenterology. 2002;122:1771–7.
22. Villoria A, Serra J, Azpiroz F, Malagelada JR. Physical activity and intestinal gas clearance in patients with bloating. Am J Gastroenterol. 2006;101:2552–7.
23. Ringel-Kulka T, Palsson OS, Maier D, et al. Probiotic bacteria lactobacillus acidophilus NCFM and bifidobacterium lactis Bi-07 versus placebo for the symptoms of bloating in patients with functional bowel disorders: a double-blind study. J Clin Gastroenterol. 2011;45:518–25.

Chapter 17
Constipation

Alpa Desai and Bobby Desai

17.1 Introduction

Constipation is a problem affecting between 12 and 19 % of people within the United States [1] and is described as infrequent defecation and/or difficulty passing stool [2]. This condition is more common in certain groups including women, children, and the elderly [3]. The Rome criteria define functional constipation as including two or more of the following: At least 25 % of defecation involves straining, lumpy or hard stool, sensation of incomplete evacuation, sensation of anorectal obstruction/blockage, or manual maneuvers to facilitate defecation, and there are fewer than three defecations per week [4]. Additionally, loose stool are rarely present without use of laxatives, and there are insufficient criteria for irritable bowel syndrome.

17.2 Primary Constipation

Normal transit constipation – functional constipation – most common.

- Stool passes through the colon at a normal rate.

A. Desai, DO (✉)
Community Health and Family Medicine, University of Florida, Newberry, FL, USA
e-mail: desaas@shands.ufl.edu

B. Desai, MD, Med
Emergency Medicine, University of Florida, Gainesville, FL, USA
e-mail: bdesai@ufl.edu

B. Desai, A. Desai (eds.), *Primary Care for Emergency Physicians*,
DOI 10.1007/978-3-319-44360-7_17

Slow transit constipation – prolonged delay of passage of stool through the colon [5].

- Patients complain of abdominal bloating and infrequent bowel movements [6].
- Theorized to be due to abnormalities of myenteric plexus, defective cholinergic innervation, and anomalies of the noradrenergic neuromuscular transmission system [5].

Anorectal dysfunction – inefficient coordination of the pelvic musculature in the evacuation mechanism [7].

- Patients complain of feeling of incomplete emptying, sensing obstruction, or a need for digital manipulation.
- Usually is an acquired behavioral disorder.

17.3 Secondary Constipation: Wide Array of Sources [8–10]

Endocrine/metabolic disorders:

- Hyperparathyroidism
- Diabetes mellitus
- Uremia
- Hypercalcemia
- Hypothyroidism

Myopathic conditions:

- Amyloidosis
- Myotonic dystrophy
- Scleroderma

Neurologic diseases:

- Autonomic neuropathy
- Cerebrovascular disease
- Hirschsprung's disease
- Multiple sclerosis
- Parkinson's disease
- Spinal cord injury/tumor

Psychological conditions:

- Anxiety/depression
- Somatization

Structural abnormalities:

- Anal fissure/stricture or hemorrhoid
- Colonic stricture
- Inflammatory bowel disease

- Obstructive colonic mass
- Rectal prolapse or rectocele

Others:

- Irritable bowel syndrome
- Pregnancy
- Medications:

  Antacids
  Anticholinergics
  Antidepressants
  Antihistamines
  Calcium channel blockers
  Clonidine
  Diuretics
  Iron
  Levodopa
  Narcotics
  NSAIDs
  Opioids
  Psychotropics
  Sympathomimetics

17.4 Diagnosis [11]

Labs

- CBC
- BMP: calcium, creatinine levels
- TSH

Colonoscopy or sigmoidoscopy in patients 50 years of age or older

17.5 Treatment

Nonpharmacologic treatments [12]:

- Bowel training – encourage patients to attempt defecation in the morning, shortly after awakening, when the bowels are more active, and 30 min after meals to use the gastro colic reflex.
- Dietary fiber intake – recommended 20–35 g daily – increases by 5 g daily until reached daily recommended intake. Increase fiber rich foods: bran, fruits, vegetables, nuts, and prune juice [9].

- Fluid intake – decreased intake may result in fecal impaction [13].
- Regular exercise
  - Biofeedback/pelvic floor retraining – useful for anorectal dysfunction

Pharmacologic treatment [14]:

- *Bulk laxatives*: Citrucel, FiberCon, and Metamucil absorb water from the intestinal lumen to increase stool mass and soften stool consistency.
- Work best for patients with functional normal transit constipation.
- Improve symptoms of constipation such as stool consistency and abdominal pain.
- Side effects include bloating and increased gas production.
- *Emollient laxatives* – stool softeners: Docusates lower surface tension, thereby allowing water to more easily enter the bowel.
- Not as effective as bulk laxatives but useful in patients with painful defecation conditions such as hemorrhoids or anal fissures.
- *Osmotic laxatives* – Milk of Magnesia, magnesium citrate, Miralax, and lactulose hyperosmolar agents which use osmotic activity to result in secretion of water into the intestinal lumen.
- Monitor for electrolyte imbalances such as hypokalemia and hypermagnesemia.
- Use with caution in congestive heart failure and chronic renal insufficiency patients.
- *Stimulant laxatives* – Dulcolax, Senna, tegaserod, castor oil, bisacodyl increase intestinal motility and secretion of water into the bowel.
- May cause abdominal cramping as a result of increased peristalsis.
- Do not use in patients with suspected bowel obstruction.

References

1. Higgins PD, Johanson JF. Epidemiology of constipation in North America: a systematic review. Am J Gastroenterol. 2004;99:750–9.
2. American College of Gastroenterology Chronic Constipation Task Force. An evidence-based approach to the management of chronic constipation in North America. Am J Gastroenterol. 2005;100 Suppl 1:S1–4.
3. Lindsley CW. The top prescription drugs of 2011 in the United States: antipsychotics and antidepressants once again lead CNS therapeutics. ACS Chem Neurosci. 2012;3:630–1.
4. Longstreth GF, Thompson WG, Chey WD, et al. Functional bowel disorders. Gastroenterology. 2006;130:1480–91.
5. Rao SS. Constipation: evaluation and treatment. Gastroenterol Clin N Am. 2003;32:659–83.
6. Koch A, Voderholzer WA, Klauser AG, Muller-Lissner S. Symptoms in chronic constipation. Dis Colon Rectum. 1997;40:902–6.
7. Rao SS, Welcher KD, Leistikow JS. Obstructive defecation: a failure of rectoanal coordination. Am J Gastroenterol. 1998;93:1042–50.
8. Prather CM, Ortiz-Camacho CP. Evaluation and treatment of constipation and fecal impaction in adults. Mayo Clin Proc. 1998;73:881–6.

9. Thomas DR, Forrester L, Gloth MF, Gruber J, Krause RA, Prather C, et al. Clinical consensus: the constipation crisis in long-term care. Ann Long-Term Care. 2003;11(Suppl):3–14.
10. Schiller LR. Constipation and fecal incontinence in the elderly. Gastroenterol Clin N Am. 2001;30:497–515.
11. Locke III GR, Pemberton JH, Phillips SF. American gastroenterological association medical position statement: guidelines on constipation. Gastroenterology. 2000;119:1761–6.
12. Rao SS, Sadeghi P, Beaty J, Kavlock R, Ackerson K. Ambulatory 24-h colonic manometry in healthy humans. Am J Physiol Gastrointest Liver Physiol. 2001;280:G629–39.
13. Wrenn K. Fecal impaction. N Engl J Med. 1989;321:658–62.
14. Hsieh C. Treatment of constipation in older adults. Am Fam Physician. 2005;72:2277–84. 2285.

Chapter 18
Diarrhea

Bobby Desai and Alpa Desai

18.1 Introduction

Acute diarrhea is a common problem encountered by physicians and has a range from presentations, from benign (though concerning to the patient), to conditions that may be life threatening in those at the extremes of age, who are immunocompromised, or who have concurrent gastrointestinal system issues. Diarrhea is a significant public health issue; there are hundreds of millions of episodes of acute diarrhea yearly that result in nearly a million hospitalizations and 6000 deaths per year in this country alone. Of these, there are approximately 50 million cases of foodborne diarrheal illnesses every year, resulting in 128,000 hospitalizations and 3000 deaths [1]. In those countries that are underdeveloped, diarrheal illnesses are even more significant since due to the lack of medical care, these illnesses may have a greater morbidity and mortality. In these locales, episodes of acute diarrhea are typically related to contaminated water and food supplies.

Acute diarrhea can be defined as an increase in one's stool frequency up to three more times per day or greater than 200 g of stool per day up to 14 days [2]. Subacute and chronic diarrheas are defined as diarrhea lasting from 14 to 30 days, respectively.

B. Desai, MD, Med (✉)
Emergency Medicine, University of Florida, Gainesville, FL, USA
e-mail: bdesai@ufl.edu

A. Desai, DO
Community Health and Family Medicine, University of Florida, Newberry, FL, USA
e-mail: desaas@shands.ufl.edu

B. Desai, A. Desai (eds.), *Primary Care for Emergency Physicians*,
DOI 10.1007/978-3-319-44360-7_18

18.1.1 Pathophysiology

Diarrheal syndromes can be divided into two distinct categories: inflammatory and noninflammatory conditions. Inflammatory etiologies typically are caused by invasive pathogens or may be toxin induced, whereas noninflammatory diarrhea is typically viral but can be caused by bacteria or parasites. Inflammatory syndromes, due to mucosal disruption, may be bloody and cause fever and more severe abdominal pain. Noninflammatory conditions typically cause an increased secretion into the bowel lumen causing more profuse nonbloody watery diarrhea. These patients may have abdominal cramping, but not typically severe in nature. Furthermore, they may present with nausea and vomiting. Noninflammatory syndromes are typically milder than inflammatory syndromes. Finally, inflammatory conditions will typically have fecal leukocytes on laboratory evaluation.

18.2 Risk Factors for Diarrhea

- Extremes of age
  - Elderly
  - Infants
- Traveler's disease
- Immunosuppressed
  - HIV
  - Those on chronic steroids
  - Those undergoing chemotherapy
- Patients in close contact with others who have an infectious diarrheal etiology
  - Day care
  - Cruise ships
  - Sick contacts
  - Military
- Exposure to contaminated food or water
- Exposure to inadequate sewage disposal
- Specific medical conditions
  - Gastrointestinal etiologies
    - Irritable bowel syndrome
    - Crohn's disease
    - Ulcerative colitis
    - Mesenteric ischemia

    - Colorectal cancer
    - Malabsorption
    - Short bowel syndrome
    - Bowel obstruction
    - Overflow incontinence secondary to chronic constipation
  - Endocrine etiologies
    - Diabetes mellitus
    - Adrenocortical insufficiency
    - Hyperthyroidism
    - Carcinoid tumor
  - Lactose intolerance
  - Celiac disease
  - Chronic alcohol use
    - This is typically chronic.
  - Opioid withdrawal
    - May be acute
- Medications and drugs
  - Antibiotics
    - Especially broad spectrum
  - Anti-inflammatory agents
    - NSAIDs
  - Any medications with magnesium
    - Antacids
      - May be calcium based as well
  - Alcohol
  - Chemotherapy
  - Colchicine
  - Laxatives
  - Other less common
    - ACE inhibitors
    - Cholesterol-lowering medications
    - Lithium
    - Proton pump inhibitors
- Pregnancy
- Fecal-oral sexual contact

18.3 Differential Diagnosis

Since the differential diagnosis of diarrhea is extremely broad, only representative examples have been listed.

18.3.1 Acute

- Infectious diarrhea
  - Viral
    - *Rotavirus*
      - Incidence has decreased with adoption of the *Rotavirus* vaccine.
    - *Norovirus*
      - Implicated in many cruise ship outbreaks
    - The vast majority of acute diarrheal illnesses are due to a viral etiology.
  - Bacterial
    - *Salmonella*
    - *Shigella*
    - *E. coli* O157:H7
    - *Vibrio*
    - *Yersinia*
    - *Listeria*
    - *Clostridium difficile*
      - Typically antibiotic induced
        - In general, antibiotics alter gut flora allowing *C. difficile* to propagate unchecked.
    - Bacterial cultures are positive in only 1–5 % of cases [3].
  - Protozoal
    - Giardia
      - Consumption of untreated water while camping
      - May also have weight loss
  - Fungal
- Foodborne illness with representative examples
  - Raw or undercooked beef
    - Shiga toxin-producing *E. coli*

    - *Staphylococcus aureus*
    - *Clostridium perfringens*
    - *Salmonella*
    - *Listeria*
    - *Bacillus cereus*
    - *Yersinia*
  - Undercooked pork
    - *Staphylococcus aureus*
    - *Clostridium perfringens*
    - *Salmonella*
    - *Listeria*
    - Shiga toxin-producing *E. coli*
    - *Bacillus cereus*
    - *Yersinia*
  - Undercooked poultry
    - *Staphylococcus aureus*
    - *Clostridium perfringens*
    - *Salmonella*
    - *Listeria*
    - *Campylobacter*
  - Fried rice
    - *Bacillus cereus*
  - Seafood, including raw shellfish
    - *Vibrio parahaemolyticus*
    - *Vibrio cholera*
  - Raw milk
    - *Listeria*
    - Shiga toxin-producing *E. coli*
    - *Salmonella*
    - *Campylobacter*
- Fecal-oral sexual contact
  - *Salmonella*
  - *Shigella*
  - *Campylobacter*
  - Protozoal disease
- Day care
  - *Rotavirus*
  - *Shigella*
  - *Cryptosporidium*

- Medication induced
  - *See above*
- Travel to endemic country
  - Most common is enterotoxigenic *E. coli*.
  - Other common pathogens may be the etiologic agent as well.

18.3.2 Chronic

- HIV infection or immunosuppression (may be acute as well)
  - *Isospora*
  - *Listeria*
  - *Mycobacterium avium-intracellulare* complex
  - *Cryptosporidium*
  - CMV
- Pregnancy
  - *Listeria*
- Alcohol abuse
- Irritable bowel syndrome
  - The most common cause of functional diarrhea in the developed world
- Malabsorption of bile acids
- Malabsorption of carbohydrates
- Celiac disease
- Crohn's disease
- Ulcerative colitis
- Congenital syndromes
- Neuroendocrine tumors
- Gastric bypass
- Mesenteric ischemia
- Short bowel syndrome
- Tropical sprue
- Whipple's disease
- Hepatobiliary disorders
- Pancreatic exocrine insufficiency
- Neoplasia
  - Colon cancer
  - Lymphoma
- Radiation colitis

18.3.2.1 Common Complaints and Red Flags of Acute Diarrhea

- Acute abdominal pain
  - Typically crampy in nature and benign
  - More severe pain or pain out of proportion to the clinical gestalt requires a more thorough investigation.
  - Can typically present with any etiology of diarrhea.
- Acute rectal pain or signs/symptoms of proctitis
  - Syphilis
  - Gonorrhea
  - Chlamydia
  - Herpes simplex virus
  - May have other more common etiologies including:
    - *Salmonella*
    - *Shigella*
    - *E. histolytica*
- Fever
  - Typically is moderate to severe diarrhea of an invasive pathogen
  - May also have bloody diarrhea
  - Is quite contagious
  - Etiologies include:
    - *Salmonella*
    - *Shigella*
    - *Campylobacter*
    - *Yersinia*
- Nausea and vomiting
  - Typically occurs with "traveler's-type" diarrhea
  - May include abdominal cramping and tenesmus
  - Etiologies include:
    - Toxin-based etiologies
      - Enterotoxigenic *E. coli*
    - *Salmonella*
    - *Campylobacter*
    - *Vibrio*
    - Viral-based etiologies
      - *Rotavirus*
      - *Norovirus*
- Bloody stools

  - Etiologies include:
    - *Salmonella*
    - *Shigella*
    - *Campylobacter*
    - *Yersinia*
    - Shiga toxin-producing *E. coli*
      - Classically has abdominal pain with no fever
- "Rice-water stools"
  - *Vibrio cholera*
- Diarrhea with a potentially common exposure source
  - Within 6 h
    - *Staphylococcus*
    - *Bacillus cereus*
      - May have vomiting
  - After 8 h
    - *Clostridium perfringens*

18.4 Common Complaints and Red Flags of Chronic Diarrhea

- Watery, fatty stools
  - Typical of malabsorptive causes
- Bloody, pustular stools (may be intermittent for weeks to months, as opposed to several days for acute diarrhea)
  - Typical of inflammatory causes
- Important to know that categories may overlap
- Weight loss
- Lymphadenopathy

18.5 History

When assessing a patient with a diarrheal illness, a careful history and physical examination are paramount in determining potential etiologies of the illness. In addition to a review of current medical and surgical history, specific questions that should be asked include:

- What is the usual frequency and consistency of bowel movements?
- What is the current frequency and consistency of bowel movements?
- Is there blood or mucus in the stool?
- Have there been changes to diet?
  - Any over-the-counter supplements?
- Has there been travel abroad?
- What foods worsen the diarrhea?
  - Dairy products
  - Fatty foods
- Is the patient on any new medications?
- Is the patient pregnant?
  - Concern for *Listeria* which may prompt further consultation with obstetrics
- Is anyone else affected?
- Has there been a documented fever?

18.6 Physical Examination

The physical examination should focus on potential emergency conditions for the reported diarrhea and specifically for evidence of severe dehydration or possibly a sepsis picture.

- Vital signs and general assessment
  - The presence of fever or hypothermia should alert the physician to a possible infectious or inflammatory etiology.
  - Tachycardia may have multiple etiologies including:
    - An appropriate response to fever
    - Dehydration
  - An abnormal respiratory pattern may also be indicative of severe dehydration [16].
  - Evidence of weight loss
    - Typically more chronic in nature
    - Consider malignancy in this population
  - Skin findings of:
    - Jaundice
      - Hepatobiliary disorders

    - Dermatitis herpetiformis [4]
        - An itchy, blistering rash present in 15–25 % of patients with celiac disease
    - Evaluation of skin turgor
        - Decreased turgor is another sign of dehydration.
- Neurologic assessment should focus on:
    - Level of alertness
        - Patients at the extremes of age may have lethargy, obtundation, or even be comatose depending on the degree of illness.
- Head, eyes, ears, nose, and throat examination
    - Note moistness of mucous membrane.
    - Any evidence of sunken eyes
        - Most likely due to dehydration
    - Evidence of episcleritis
        - Found in inflammatory bowel disorders
    - Evidence of exophthalmia
        - Found in hypothyroidism
- Cardiovascular and pulmonary examination
    - Note capillary refill time.
        - Delayed capillary refill may indicate dehydration and decreased peripheral perfusion.
    - Orthostatic vital signs
        - Abnormalities also may indicate dehydration status.
- Gastrointestinal examination [5]
    - If vomiting is present, determine contents (e.g., blood, bile, clear contents).
    - Exposure of the abdomen is important to assess for:
        - Evidence of surgical scars
        - Evidence of bloating
    - The presence or absence of abdominal pain
        - Evaluate for the presence of a surgical etiology of pain.
    - Assess for evidence of masses
        - Neoplasia

  - Assess bowel sounds
    - Hyperactive bowel sounds may indicate hypermotility.
  - Rectal examination is important to assess for:
    - Evidence of fistula
      - Crohn's disease
    - Fecal occult blood testing
  - Consider anoscopy if available
    - May detect impacted stools which may be the cause of paradoxical diarrhea which is stool leakage around the impacted stool
    - May detect ulcerations
- Psychiatric evaluation
  - An overall general assessment may be important if a functional cause of diarrhea is considered.

18.7 Evaluation of Diarrhea

Since most diarrhea is of a watery, benign nature, diagnostic testing is rarely required and is typically reserved for those patients who present with a more severe clinical picture, including moderate to profound dehydration, fever, bloody stools, or a significant past medical history (HIV, immunosuppression, uncontrolled diabetes mellitus, etc.).

18.7.1 Specific Tests

1. Fecal occult blood testing
   (a) A rapid and inexpensive test whose utility is improved when used in combination with a positive stool lactoferrin or stool leukocyte test. If these are positive, the likelihood of the presence of an inflammatory diarrhea is more common [6].
   (b) A pitfall of fecal occult blood testing is overreliance on the test, since its sensitivity and specificity is only 71 % and 79 %, respectively [7].
2. Lactoferrin and leukocytes
   (a) A pitfall of stool leukocyte testing is the lack of standardization of laboratory interpretation. Coupled with the potential difficulty in handling specimens and the wide variability and sensitivity and specificity, this test has limited utility [8].

(b) Lactoferrin is a more sensitive and specific method for assessment since it is a marker for leukocytes that is present when cells are damaged. It increases in the setting of bacterial infections [9]. It has replaced fecal leukocytes as the ideal method for the screening of leukocytes when indicated [10].

3. Stool cultures

(a) A pitfall of stool cultures is overreliance on the test. Stool culture in every case of diarrhea is expensive and not helpful in the emergency department. However, obtaining stool cultures in those patients who have tests revealing the presence of leukocytes or who have grossly bloody stools increases the likelihood of a positive result [11].
(b) Typically, stool cultures should be reserved for those patients who present with a severe clinical picture, including those with significant dehydration, grossly bloody stools, signs or symptoms of an inflammatory disease, prolonged symptoms (3–7 days), or a history of immunosuppression (cancer, HIV) [12].

4. Testing for ova and parasites

(a) Like stool cultures, it is expensive and inefficient for those patients presenting the ED without historical features that warrant testing.
(b) Historical features that *do* warrant testing include [13]:

(i) Persistent diarrhea of a weeks' duration
(ii) Travel and/or camping in mountainous regions

1. Drinking untreated water in those areas

(iii) Infants in a day care setting
(iv) Patients with AIDS
(v) Men who have intercourse with men
(vi) Bloody diarrhea with a lack of fecal leukocytes
(vii) Diarrheal outbreaks in the community

5. Testing for *Clostridium difficile*

(a) Routine testing for *C. difficile* toxins is not indicated unless [14, 15]:

(i) The patient has unexplained diarrhea after 3 days of hospitalization.
(ii) The patient is currently on antibiotics.
(iii) Diarrhea is present 1 month after antibiotic discontinuation.
(iv) Consideration of *C. difficile* should also be made 2–3 months after the discontinuation of antibiotics.
(v) The patient has significant comorbidities as detailed previously.

6. Routine blood and urine testing

(a) Used on a case-by-case basis to evaluate for specific abnormalities.
(b) K+, BUN/creatinine ratio, and urine specific gravity assessment may be of benefit.

(c) A complete blood count, albumin level, erythrocyte sedimentation rate, liver function testing, and thyroid-stimulating hormone level may be of benefit especially in chronic diarrhea.

(i) Evidence of iron deficiency anemia may indicate celiac disease.

7. Other tests

(a) Endoscopy

(i) Of limited utility in the emergency department
(ii) May be considered by primary care providers if the diagnosis is not clear after ineffectual treatment, persistent symptoms, and unremarkable blood and stool tests
(iii) May be of more benefit in chronic diarrheal states and the diagnosis of noninfectious etiologies of acute diarrhea (e.g., inflammatory bowel disease, cancer, etc.)

(b) Computed tomography (CT)

(i) Of little benefit in those patients without abdominal pain, mild diarrhea, and unremarkable vital signs.
(ii) Of greater utility in those patients in which an underlying surgical emergency is considered (e.g., obstruction).
(iii) CT should be also considered if the clinician suspects an underlying significant GI bleed.

18.8 Introduction to Treatment

The initial stabilization begins with the assessment of airway, breathing, and circulation. The physician must simultaneously manage volume resuscitation with diagnostic modalities in order to determine the medical or potential surgical cause of the diarrhea.

1. Rehydration

(a) Emergent rehydration is a priority of the emergency physician.
(b) Oral rehydration is typically preferable to intravenous solutions

(i) Pitfall: May be difficult to adequately provide ongoing oral rehydration to patients in the ED
(ii) If given, oral rehydration should have a mixture of salt and glucose with a reduced osmolarity:

1. This decreases output of stool and frequency of emesis and has been shown to decrease the need for intravenous hydration [17].
2. It also does not increase hyponatremia [18].

3. A solution that the emergency physician can prescribe when sending patients home consists of 1.5 teaspoon of salt, 6 teaspoons of sugar, and 1 l of water

   (a) This is strong enough to replace the electrolyte loss but not overwhelm the bowels with a hyperosmolar solution which may worsen the diarrhea.

(c) The best options for the rehydration of children at home are Pedialyte, Gatorade, Infalyte, or the World Health Organization oral rehydration solution.
(d) If oral rehydration is not feasible, intravenous hydration is required:

Pitfalls

1. The use of high sugar drinks such as fruit juices or sodas will potentially worsen diarrhea and should not be used.
2. "Clear" liquids (e.g., water, chicken broth) should not be used as well since they are hypoosmolar and are inadequate for the replacement of sodium, potassium, and bicarbonate:

   (a) This will potentially cause hyponatremia.

2. Early feeding

   (a) Early feeding decreases the permeability of the intestines that is cause by infection, typically improves overall nutrition in diarrheal states, and decreases the duration of illness [19].
   (b) Pitfall: The commonly used "BRAT" (bananas, rice, applesauce, and toast) diet has limited evidence to support its routine use [20].
   (c) Pitfall: Patients typically are instructed to avoid solid foods for 24 h, and this also is not helpful [21].

3. Prevention

   (a) Factors that are important for the prevention of diarrheal illness include proper handwashing, overall good hygiene, and access to clean water.

18.9 Pharmacologic Treatment

There are several medications for the emergency physician to consider, but care must be taken to choose those medications that will have a benefit.

18.9.1 Antibiotics

Since most diarrhea is viral and typically self-limited, routine antibiotic use is not indicated for those with watery diarrhea and who are not ill appearing on

assessment. A common pitfall is the inappropriate prescribing of antibiotics which may lead to several consequences. First, the eradication of normal gut flora may lead to colonization with *C. difficile* which in turn may prolong the disease. In addition, antibiotics may lead to the delay in the excretion of organisms such as *Salmonella*, thus potentially causing a carrier state. Furthermore, the production of certain toxins, including Shiga toxins produced by organisms such as Shiga toxin-producing *E. coli*, may be enhanced. In this case, there is an increased risk of hemolytic-uremic syndrome. Finally, long-term consequences of inappropriate antibiotic administration also include potential development of antibiotic resistance.

Consideration of antibiotic use should be made for those patients who are immunocompromised, have severe illness, including sepsis, and those patients older than 65 years of age. Diarrhea lasting for 10–14 days or greater warrants testing and potential treatment for protozoal etiologies.

Specific therapy is listed in Table 18.1 and 18.2.

Table 18.1 Antibiotic choices for specific bacterial agents

Agent	Brief clinical tidbits	Treatment
Campylobacter, *Shigella*, *Salmonella*, *Yersinia*	Invasive diarrhea characterized by fever, bloody stools	TMP/SMX or fluoroquinolone for 3 days (may reduce duration of shedding); see below for more information regarding *Campylobacter*
Enterotoxigenic *E. coli*, *Campylobacter*, *Salmonella*, *Shigella*, *Aeromonas*, *Vibrio*, *Bacteroides*	Traveler's diarrhea: watery diarrhea, ± abdominal cramps, N/V, tenesmus	TMP/SMX or fluoroquinolone for 3 days may reduce duration of illness by 2–3 days
Shigella	Severe dysentery with fever, bloody diarrhea	TMP/SMX or fluoroquinolone two times a day for 3 days
Vibrio cholera	"Rice-water stools," severe diarrhea	Doxycycline 300 mg × 1 dose, or TMP/SMX two times a day for 3 days or fluoroquinolone × 1 dose
Campylobacter	Associated with Guillain-Barre, inflammatory bowel disease and reactive arthritis. Causes acute watery diarrhea	Azithromycin 500 mg a day for 3 days
Salmonella	Treat specific patient populations (see below)	TMP/SMX or fluoroquinolone two times a day for 5–7 days
Listeria	Treat specific patient populations (see below)	Ampicillin
C. difficile	See below	Metronidazole 500 mg orally three times a day for 10–14 days or vancomycin 125 mg orally four times a day for 10–14 days

TMP/SMX trimethoprim/sulfamethoxazole

Pitfalls

1. Treating patients who have bloody diarrhea with abdominal pain and no fever and relevant history of eating raw ground beef or seed sprouts with antibiotics:
   (a) There is a risk of hemolytic-uremic syndrome.
   (b) Treatment with antibiotics does not improve O157 illness.
2. Treatment of *Yersinia*. These patients present with "pseudoappendicitis" with right lower quadrant abdominal pain along with the watery diarrhea. Treatment is only required for immunocompromised patients.
3. Routine treatment of *Salmonella*. These patients may present acutely ill with fever and muscle aches and pains, and it typically is human-to-human transmission. Treatment is recommended for those patients who are:
   (a) Immunocompromised
   (b) Are at the extremes of age
   (c) Have persistent, severe diarrhea
   (d) Have severe illness, including sepsis
   (e) Have a history of valvular heart disease
4. Routine treatment of *Listeria*. Unless pregnant or immunocompromised – in which case the disease may be severe – patients typically have mild systemic symptoms.

Table 18.2 Antibiotic choices for specific protozoal agents

Agent	Brief clinical tidbits	Treatment
Giardia	Persistent diarrhea after exposure to untreated water; relapses may occur due to misdiagnosis	Metronizadole 750 mg three times a day for 7–10 days
Entamoeba histolytica	Severe disease may result in hepatic abscess	Metronizadole 750 mg three times a day for 5–10 days in addition to paromomycin 25–35 mg/kg in three divided doses for 5–10 days
Cryptosporidium	Therapy may be required in immunosuppressed patients but may not be necessary in immunocompetent patients with mild disease and those patients with HIV who have a CD4 count >150 cells/mm^3	Nitazoxanide 500 mg two times a day for 3 days for severe disease
Cyclospora or *Isospora*	Requires longer therapy in HIV or immunosuppressed	TMP/SMX two times per day for 7–10 days (2–4 times per day for 10–14 days for HIV or immunosuppressed)
Microsporidia	In patients with AIDS, highly active retroviral therapy may be adequate to eradicate intestinal disease	Albendazole 400 mg two times per day for 3 weeks

18.9.2 Treatment of Clostridium Difficile

This organism deserves special mention as it is one of the more serious causes of diarrhea affecting patients. Prior antibiotic use (including fluoroquinolones, clindamycin, penicillins, and cephalosporins) eliminates normal gut flora allowing for colonization by *C. difficile*. The diarrhea caused by *C. difficile* is severe, with up to 15 bowel movements per day with associated abdominal pain and cramping. Many patients may have a systemic reaction, with fever, tachycardia, and tachypnea depending on level of dehydration and overall illness. Complications of *C. difficile* infection include toxic megacolon, which is an abnormal dilation of the colon that is seen on imaging studies. Those patients who do not respond to medical management may require urgent surgical consultation due to the high risk of perforation.

18.9.3 Antidiarrheal Agents

Antimotility agents that are readily available over the counter, such as loperamide, may reduce the duration of diarrhea by 24 h, for those patients with nonbloody stools. For those patients with a diagnosis of Traveler's diarrhea, an increased chance of cure may be obtained at 1–2 days in conjunction with antibiotics [22]. Loperamide in combination with simethicone has been shown to be of benefit in acute nonspecific diarrhea associated with intestinal gas-related discomfort compared with either alone [23]. Bismuth subsalicylate is an effective alternative in patients with a fever and bloody diarrhea. It significantly reduces the number of unformed stools and improves symptoms [24].

Some antidiarrheal agents
Loperamide
Loperamide/simethicone
Bismuth subsalicylate

Pitfalls

1. Use of loperamide in bloody or potentially invasive/inflammatory diarrhea. This may prolong the duration of the illness and cause worsening of the patient's condition.

18.9.4 Probiotics

This class of over-the-counter medication is believed to work by immune system stimulation and competition for binding on the epithelial cells of the intestine. The use of probiotics in children has shown to reduce duration and severity of diarrhea by up to 1 day.

Pitfalls

1. Different strains of probiotics may have different clinical effects.

18.9.5 Zinc

Zinc has been shown in studies done in China that it too may reduce the duration of diarrhea in children less than 5 years of age. These studies showed a reduction in morbidity as a result of zinc supplementation for patients less than 5 [25]. The World Health Organization supports zinc treatment of diarrhea in low- to middle-income countries.

Pitfalls of Anticipatory Guidance

1. Providing guidance for all patients who present to the ED is paramount for their understanding of the diarrheal disease process.
2. Patients must be informed that this process may take from 3 to 5 days to fully run its course.
3. Return precautions of fever, bloody diarrhea, signs of significant dehydration (e.g., decrease in urine output), and lethargy should be provided.
4. Instructions on proper handwashing and good hygiene should be given.

References

1. Thielman NM, Guerrant RL. Acute infectious diarrhea. N Engl J Med. 2004;350:38–47.
2. Scallan E, Hoekstra RM, Angulo FJ, et al. Foodborne illness acquired in the United States. Emerg Infect Dis. 2011;17(1):7–15.
3. Guerrant RL, Van Gilder T, Steiner TS, et al. Practice guidelines for the management of infectious diarrhea. Clin Infect Dis. 2001;32:331–50.
4. Rodrigo L. Celiac disease. World J Gastroenterol. 2006;12(41):6585–93.
5. Guerrant RL, Van Gilder T, Steiner TS, Infectious Diseases Society of America, et al. Practice guidelines for the management of infectious diarrhea. Clin Infect Dis. 2001;32(3):331–51.
6. Thielman NM, Guerrant RL. Clinical practice. Acute infectious diarrhea. N Engl J Med. 2004;350(1):38–47.
7. Ilnyckyj A. Clinical evaluation and management of acute infectious diarrhea in adults. Gastroenterol Clin N Am. 2001;30(3):599–609.
8. Gill CJ, Lau J, Gorbach SL, Hamer DH. Diagnostic accuracy of stool assays for inflammatory bacterial gastroenteritis in developed and resource-poor countries. Clin Infect Dis. 2003;37(3):365–75.
9. Chen CC, Chang CJ, Lin TY, Lai MW, Chao HC, Kong MS. Usefulness of fecal lactoferrin in predicting and monitoring the clinical severity of infectious diarrhea. World J Gastroenterol. 2011;17(37):4218–24.
10. Hayakawa T, Jin CX, Ko SB, Kitagawa M, Ishiguro H. Lactoferrin in gastrointestinal disease. Intern Med. 2009;48(15):1251–4.
11. Talan D, Moran GJ, Newdow M, EMERGEncy ID NET Study Group, et al. Etiology of bloody diarrhea among patients presenting to United States emergency departments: prevalence of *Escherichia coli* O157:H7 and other enteropathogens. Clin Infect Dis. 2001;32(4):573–80.

12. Bauer TM, Lalvani A, Fehrenbach J, et al. Derivation and validation of guidelines for stool cultures for enteropathogenic bacteria other than *Clostridium difficile* in hospitalized adults. JAMA. 2001;285(3):313–9.
13. DuPont HL. Guidelines on acute infectious diarrhea in adults. The practice parameters committee of the American College of Gastroenterology. Am J Gastroenterol. 1997;92(11):1962–75.
14. Centers for Disease Control and Prevention. Vital signs: preventing *Clostridium difficile* infections. MMWR Morb Mortal Wkly Rep. 2012;61(9):157–62.
15. Siegel DL, Edelstein PH, Nachamkin I. Inappropriate testing for diarrheal diseases in the hospital. JAMA. 1990;263(7):979–82.
16. Steiner MJ, DeWalt DA, Byerley JS. Is this child dehydrated? JAMA. 2004;291(22): 2746–54.
17. Hahn S, Kim Y, Garner P. Reduced osmolarity oral rehydration solution for treating dehydration due to diarrhoea in children: systematic review. BMJ. 2001;323(7304):81–5.
18. Alam NH, Yunus M, Faruque AS, et al. Symptomatic hyponatremia during treatment of dehydrating diarrheal disease with reduced osmolarity oral rehydration solution. JAMA. 2006;296(5):567–73.
19. Duggan C, Nurko S. "Feeding the gut": the scientific basis for continued enteral nutrition during acute diarrhea. J Pediatr. 1997;131(6):801–8.
20. Gadewar S, Fasano A. Current concepts in the evaluation, diagnosis and management of acute infectious diarrhea. Curr Opin Pharmacol. 2005;5(6):559–65.
21. De Bruyn G. Diarrhea in adults (acute). Am Fam Physician. 2008;78(4):503–4.
22. Riddle MS, Arnold S, Tribble DR. Effect of adjunctive loperamide in combination with antibiotics on treatment outcomes in traveler's diarrhea: a systematic review and meta-analysis. Clin Infect Dis. 2008;47(8):1007–14.
23. Hanauer SB, DuPont HL, Cooper KM, Laudadio C. Randomized, double-blind, placebo-controlled clinical trial of loperamide plus simethicone versus loperamide alone and simethicone alone in the treatment of acute diarrhea with gas-related abdominal discomfort. Curr Med Res Opin. 2007;23(5):1033–43.
24. Steffen R. Worldwide efficacy of bismuth subsalicylate in the treatment of travelers diarrhea. Rev Infect Dis. 1990;12 Suppl 1:S80.
25. Lamberti LM, Walker CL, Chan KY, et al. Oral zinc supplementation for the treatment of acute diarrhea in children: a systematic review and meta-analysis. Nutrients. 2013;5(11):4715–40.

Chapter 19
Hemorrhoids

Run Gan

19.1 Introduction

Hemorrhoids are a common cause of anal bleeding, itching, and pain. It is estimated that 4.4 % of the population has symptomatic hemorrhoids while the true prevalence of asymptomatic hemorrhoids is unknown [4]. Males and females are affected equally and peak around age 45–65 years old. Hemorrhoids are uncommon before age 20.

Hemorrhoids result from dilation of the veins around the anus and rectum. Internal hemorrhoids originate above the dentate line [1]. They are covered by columnar epithelium, which lacks pain receptors. They were classified into four grades based on the degree of prolapse [2].

- Grade I: No prolapse, just prominent blood vessels
- Grade II: Prolapse upon bearing down, but spontaneous reduction
- Grade III: Prolapse upon bearing down requiring manual reduction
- Grade IV: Prolapse with inability to be manually reduced

External hemorrhoids occur below the dentate. They have somatic innervations and thus are sensitive to pain and temperature [2].

19.2 Risk Factors for Hemorrhoids

- Prolonged straining
- Constipation

R. Gan, MD
Family Medicine, UF Health, Gainesville, FL 32669, USA
e-mail: rungan@ufl.edu

B. Desai, A. Desai (eds.), *Primary Care for Emergency Physicians*,
DOI 10.1007/978-3-319-44360-7_19

- Diarrhea
- Pregnancy
- Ascites
- Heavy lifting

19.3 Differential Diagnosis

- Anal fissure
  - Usually very painful with defecation
- Rectal cancer
  - May be accompanied by night sweats, weight loss, and abdominal pain with changes in stool caliber
- Condyloma
  - May bleed, sometimes large cauliflower growths
- Perianal abscess
  - Painful with fevers or chills
- IBD
  - Hematochezia and tenesmus
- Skin tags
  - Usually painless

19.4 Physical Exam

Physical exam should include abdominal examination, inspection of the perineum, digital rectal examination, and anoscopy.

- Vital sign assessment
  - Look for fevers which should alert physician to possible infectious cause such as perianal abscess.
  - Weight loss may suggest malignancy or IBD.
- GI Exam
  - Presence of abdominal pain or abdominal mass
- GU Exam

- Do external rectal exam in the prone or left lateral position to look for fissures, condylomas, and hemorrhoids, or skin tags, or neoplasms. Prolapsed internal hemorrhoids appear dark pink. Thrombosed external hemorrhoids are extremely painful and appear purplish. Perianal skin tags are nonpainful and are remnants of previous external hemorrhoids.
- Do internal exam with finger to feel for masses or abscess.
- Anoscopy should be performed to visualize any internal hemorrhoids which usually appear as dilated purplish veins, fissures, or neoplasms.
- Colonoscopy is recommended for the following groups [1]:
  - Patients who are 50 years or older and have not had a complete examination of the colon within the past 10 years
  - Patients who are 40 years or older and have not had a complete examination of the colon within the past 10 years and who have one first-degree relative in whom colorectal cancer or adenoma was diagnosed at age 60 years or younger
  - Patients who are 40 years or older and have not had a complete examination of the colon within the past 5 years and who have more than one first-degree relative in whom colorectal cancer or adenoma was diagnosed at age 60 years or younger
  - Patients with iron deficiency anemia

19.5 Labs

- Laboratory testing is not routinely recommended.
- However, if it is performed and anemia or iron deficiency is identified, sigmoidoscopy or colonoscopy evaluation should be considered.

19.6 Treatment

Treatment of hemorrhoids usually begins with conservative therapy. This includes drinking more fluids and eating more fiber. Toilet habits such as prolonged sitting and reading should be avoided.

19.6.1 Conservative Therapy

- Analgesics taken orally or topically can be helpful.
  - Pitfall: Narcotic medications may occasionally be required, but if prescribed, care must be given to also provide stool softeners and to instruct the patient to

take in more fluids since hard stools may cause more straining, exacerbating the symptoms.

- The primary goal of topical treatment is to control symptoms rather than cure the disease.
  - Other definitive treatments may thus be required.
- Many over-the-counter preparations (creams and suppositories) are available.
  - Pitfall: Strong evidence of the true efficacy of these medications is lacking.

19.6.1.1 Specific Medications

- Topical glyceryl trinitrate has been shown to alleviate hemorrhoidal symptoms with low-grade hemorrhoids and high resting anal canal pressures [5].
  - Pitfall: Patients may experience headaches during treatment.
- Application of nifedipine ointment to acute thrombosed external hemorrhoids.
  - Efficacy may be a consequence of their relaxation effect on the internal anal sphincter.
- Preparation H®
  - Contains 0.25 % phenylephrine, petrolatum, mineral oil, shark liver oil
  - Provides temporary relief of acute symptoms, including bleeding and defecation pain
- Low-dose hydrocortisone cream (Proctofoam HC®, which also contains pramoxine, an anesthetic) may improve itching and swelling.
- Sitz baths may improve anal hygiene, thus reducing irritation and pruritus.

If conservative therapy fails, next consider rubber band ligation, sclerotherapy, or surgery.

- Rubber band ligation is typically recommended as the first-line treatment in those with grade 1–3 disease [3]. It is a procedure in which elastic bands are applied onto an internal hemorrhoid at least 1 cm above the dentate line to cut off its blood supply. Within 5–7 days, the withered hemorrhoid falls off. Cure rate has been found to be about 87 % [4] with a complication rate of up to 3 %.

Rubber band ligation is less effective than surgery in preventing recurrence of hemorrhoids, but it is associated with less pain and complications. Surgical options should be considered for thrombosed hemorrhoids or nonretractable prolapsed internal hemorrhoids.

- Sclerotherapy involves the injection of a sclerosing agent, such as phenol, into the hemorrhoid. This causes the vein walls to collapse and the hemorrhoids to shrivel up. The success rate 4 years after treatment is about 70 % [4].

- Surgical excision remains a very effective approach for treatment of symptomatic hemorrhoids but is generally reserved for patients who failed an office-based procedure.

References

1. Dayton MT (senior editor), Lawrence PF, Bell RM, editors. Essentials of general surgery. 4th ed. Philadelphia: Williams & Wilkins; 2006. p. 329.
2. Beck DE. The ASCRS textbook of colon and rectal surgery. 2nd ed. New York: Springer; 2011. p. 175. ISBN 9781441915818.
3. Lorenzo-Rivero S. Hemorrhoids: diagnosis and current management. Am Surg. 2009;75(8):635–42.
4. Rivadeneira DE, Steele SR, Ternent C, Chalasani S, Buie WD, Rafferty JL, Standards Practice Task Force of The American Society of Colon and Rectal, Surgeons. Practice parameters for the management of hemorrhoids (revised 2010). Dis Colon Rectum. 2011;54(9):1059–64. doi:10.1097/DCR.0b013e318225513d.
5. Tjandra JJ, Tan JJ, Lim JF, Murray-Green C, Kennedy ML, Lubowski DZ. Rectogesic (glyceryl trinitrate 0.2%) ointment relieves symptoms of haemorrhoids associated with high resting anal canal pressures. Colorectal Dis. 2007;9:457–63.

Chapter 20
Acute Pelvic Pain

Joshua Gordon, Bobby Desai, and Alpa Desai

20.1 Introduction

Acute pelvic pain is commonly defined as pain in the pelvis or lower abdomen lasting less than 3 months [1]. Pelvic pain is a common complaint, and it is estimated that approximately one in seven women of reproductive age will be evaluated for pelvic pain [2]. The workup and diagnosis of acute pelvic pain can be challenging, because signs and symptoms are often nonspecific and insensitive. Also, the clinical presentation of each cause of pelvic pain may vary from patient to patient. Therefore, an effort must be made to keep a broad differential diagnosis when assessing patients with acute pelvic pain in order to consider both common causes, such as urinary tract infections and cervicitis, and also uncommon causes, such as heterotopic pregnancy and mesenteric adenitis. It is critical to quickly identify emergent conditions of acute pelvic pain such as ectopic pregnancy, ovarian torsion, and appendiceal perforation. The consequences of missing these diagnoses are high and may result in infertility or death.

20.2 Differential Diagnosis

The differential diagnosis of acute pelvic pain is vast and includes pathology from a wide variety of organ systems. The differential diagnosis of pelvic pain also varies with respect to the patient's age. Therefore, when constructing a differential

J. Gordon, MD (✉) • B. Desai, MD, Med
Emergency Medicine, UF Health Shands Hospital, Gainesville, FL, 32610, USA
e-mail: nodrog47@ufl.edu; bdesai@ufl.edu

A. Desai, DO
Community Health and Family Medicine, University of Florida, Newberry, FL, USA
e-mail: desaas@shands.ufl.edu

B. Desai, A. Desai (eds.), *Primary Care for Emergency Physicians*,
DOI 10.1007/978-3-319-44360-7_20

diagnosis for acute pelvic pain, it can be helpful to categorize conditions based on pregnancy status, organ systems, and the patient's age.

20.2.1 Obstetric

- Ectopic pregnancy
- Abortion
  - Threatened
  - Inevitable
  - Incomplete
  - Complete
  - Missed
  - Septic
- Normal labor
- Preterm labor
- Placental abruption
- Uterine abruption
- Incarcerated gravid uterus
- Postpartum endometritis
- Ovarian vein thrombosis
- Pubic symphysis separation
- Fertility treatment related
  - Heterotopic pregnancy
  - Ovarian hyperstimulation syndrome

20.2.2 Gynecologic (Reproductive Age)

- Vulva and vagina
  - Bartholin gland cyst/abscess
  - Vulvovaginitis
    - Bacterial vaginosis
    - Candida vaginitis
    - Trichomonas
    - Contact vaginitis
  - Imperforate hymen
  - Transverse vaginal septum
  - Vaginismus

- Cervix and uterus
  - Cervicitis
  - Endometriosis
  - Adenomyosis
  - Post-procedure endometritis
  - Intrauterine device (IUD) perforation
  - Leiomyoma
- Fallopian tube and ovary
  - Pelvic inflammatory disease
  - Salpingitis
  - Tubo-ovarian abscess
  - Ruptured ovarian cyst
  - Corpus luteum cyst
  - Ovarian torsion
  - Round ligament mass
    - Lipoma
    - Teratoma
- Endocrine
  - Primary dysmenorrhea
  - Mittelschmerz

20.2.3 Gynecologic (postmenopausal)

- Malignancy
  - Uterine
  - Ovarian
  - Endometrial
- Atrophic vaginitis
- Uterine prolapse
- Uterine fibroids

20.2.4 Gastrointestinal

- Appendicitis
- Diverticulitis
- Inflammatory bowel disease

- Mesenteric ischemia
- Irritable bowel syndrome
- Bowel obstruction
- Inguinal hernia
- Perirectal abscess
- Mesenteric adenitis

20.2.4.1 Genitourinary

- Cystitis
- Pyelonephritis
- Urolithiasis
- Interstitial cystitis
- Urethral diverticulum
- Urinary retention

20.2.5 Others

- Abdominal aortic aneurysm
- Aortic dissection
- Lead poisoning
- Abdominal wall pain
- Porphyria
- Sickle cell crisis
- Malingering
- Somatization disorder
- Idiopathic

20.3 History

A thorough history and physical examination is essential to the diagnosis of acute pelvic pain in the emergency department. Eliciting specific information about the patient's clinical situation and correlating that information with the patient's physical examination allow a physician to choose the appropriate laboratory testing and imaging modalities in order to identify the cause of that patient's pain.

Important historical features to elicit include:

- Location of pain
  - Where is the pain located?
    - Right-sided pelvic pain can be suggestive of conditions such as appendicitis.

    - Left-sided pelvic pain can be suggestive of conditions such as diverticulitis.
        - Beware that some conditions can present as either right- or left-sided pelvic pain, such as ovarian torsion, tubo-ovarian abscess, or ectopic pregnancy.
    - Suprapubic pain can be suggestive of conditions such as cystitis.
- Radiation of pain
    - Where does the pain radiate?
        - Pain that radiates to the groin can be suggestive of urolithiasis or ovarian torsion.
        - Pain that radiates to the back can be suggestive of aortic dissection.
        - Many conditions have pain that does not radiate.
- Quality of pain
    - What does the pain feel like? (pressure, sharp, crampy, burning, colicky, etc.)
        - Burning pain can be suggestive of cystitis.
        - Colicky pain can be suggestive of urolithiasis.
- Onset of pain
    - How long has the patient had the pain?
    - What was the patient doing when the pain first began?
    - Was the pain gradual or sudden in onset?
    - Since the pain began, has it gotten better, worse, or stayed the same?
- Timing/frequency of pain
    - When does the pain occur?
        - Pain that occurs during the middle of each menstrual cycle can be suggestive of Mittelschmerz.
    - How often does the pain occur?
        - Colicky pain that waxes and wanes can be suggestive of urolithiasis.
- Associated symptoms
    - A thorough review of systems allows a physician to identify associated symptoms in order to narrow the differential diagnosis. By recognizing patterns of symptoms, a physician can decide which diagnoses are more likely, guiding the physical examination, laboratory testing, and imaging orders. For example:
        - Right lower quadrant abdominal pain that is associated with a history of multiple sex partners, history of sexually transmitted diseases, and vaginal discharge is suspicious for tubo-ovarian abscess.
        - Right lower quadrant abdominal pain that is associated with anorexia, nausea, and vomiting is more suggestive of appendicitis.

- Previous similar symptoms
  - If a patient has had previous episodes similar to this current episode, it may change your differential diagnosis.
  - Be careful not to become biased if a patient states that he or she is having an episode of kidney stones because the patient has had similar previous episodes. It is important to keep the differential open in order not to miss other diagnoses that can present similarly, such as an abdominal aortic aneurysm or dissection.
- Aggravating factors
  - What makes the pain worse? (eating, positional activity, intercourse, urinating, etc.)
- Relieving factors
  - What makes the pain better? (positions, holding pressure, medications, etc.)
- Gynecologic history
  - Last menstrual period
    - Patients who have missed menstrual cycles can be pregnant and expands the differential diagnosis to include ectopic pregnancy, miscarriage, and more.
  - Menstrual cycle and deviations from normal menstrual cycle
    - Age of menarche?
    - Cycle length?
    - Amount of vaginal bleeding?
    - Pain with menses?
    - Age of menopause? (if applicable)
      - Vaginal bleeding in postmenopausal women can be suggestive of endometrial cancer.
  - Contraception use
    - Type of contraception? (oral, IUD, barrier, rhythm method, none)
  - Sexual history
    - Sexually active? If yes, with men or women or both?
    - Recent new sexual partners?
    - History of sexually transmitted infections?
      - A history of sexually transmitted infections can increase the likelihood of developing an ectopic pregnancy [3].
      - Pelvic inflammatory disease (PID) is generally thought to be a complication of sexually transmitted infections, but cases have been reported of virgin women being diagnosed with PID [4]. Therefore, pelvic inflammatory disease should still be considered as a possible cause of pelvic pain in all women, regardless of sexual history.

  - Cervical cancer screening
    - Human papillomavirus (HPV) vaccination?
    - Previous pap smears?
      - History of abnormal pap smears? If yes, what diagnostic procedures were performed?
    - History of cervical cancer?
  - Pregnancies and outcomes
    - History of pregnancies? If yes, how many?
    - Results of pregnancies? (miscarriage, termination, preterm, term, full term, natural birth, cesarean)
    - Complications during pregnancy or delivery?
    - History of fertility-enhancing medications?
- Past medical history
  - A thorough past medical history is an important component of the history. A patient's past medical history can place a patient at risk for certain conditions that cause pelvic pain. In some cases, the patient's pelvic pain is an acute exacerbation of the patient's chronic medical condition.
- Past surgical history
  - A thorough past surgical history is also an important component of the history. Previous surgeries can increase the likelihood of certain diagnoses. For example:
    - History of abdominal surgeries, even laparoscopic surgeries, increases the risk of bowel obstruction [5].
    - History of pelvic organ surgery increases the risk for ectopic pregnancy [6].
    - IUD insertion is associated with a sixfold increase in the risk of developing pelvic inflammatory disease for the next 20 days [7].

20.4 Physical Examination

The physical examination should focus on vital signs, an abdominal examination, and a pelvic examination, consisting of both a speculum examination and a bimanual examination.

Vital signs should be assessed as soon as possible. Fever can be suggestive of infectious etiologies. Hypotension and tachycardia in the setting of pelvic pain should raise concern for surgical emergencies such as ruptured ectopic pregnancy.

The abdominal examination is important in the diagnosis of acute pelvic pain. Abdominal rigidity, rebound, and guarding can be indicative of a surgical abdomen and may warrant prompt consultation.

Even though it may be uncomfortable in a patient experiencing pelvic pain, a pelvic examination must be performed in the setting of pelvic pain. The examination begins with inspection of the external genitalia for discoloration, rashes, swelling, or lesions. The urethra, Skene's glands, and Bartholin glands should be assessed for abnormalities. Next, a speculum is inserted to examine the vaginal walls for erythema, blood, lacerations, discharge, swelling, or foreign body. The blades of the speculum are then opened to evaluate the cervix for erythema, blood, ulcerations, and masses and to assess the os (open vs closed). The speculum is then removed, and a bimanual examination is performed to assess the cervix for motion tenderness; the uterus for size, position, and tenderness; and the adnexa for masses or tenderness.

Although it is an integral component in the evaluation of acute pelvic pain, it is also important to recognize the limitations of the pelvic examination. Physical examination findings tend not to be specific for only one etiology. For example, cervical motion tenderness has been shown to be nonspecific for pelvic inflammatory disease. Cervical motion tenderness has been found to be present in 25 % of cases of appendicitis and 50 % of cases of ectopic pregnancies [8]. The absence of physical examination findings does not necessarily exclude certain diagnoses. For example, a recent study demonstrated that the detection of adnexal mass in women with a known adnexal mass has been shown to have sensitivities as low as 16–28 % [9]. Examination findings also vary from provider to provider. A recent study using emergency medicine physicians showed that there was only agreement of positive findings during pelvic examination in 17–33 % of examinations [10].

20.5 Laboratory Testing

All women of reproductive age should be assessed with a beta-subunit of human chorionic gonadotropin (β-hCG) to determine if the cause of pelvic pain is related to pregnancy. The remainder of the laboratory testing should be guided by the differential diagnosis created from the patient's history and physical examination. Other commonly ordered tests include complete blood count with differential to assess for infectious etiologies and to establish a baseline hemoglobin and hematocrit, urinalysis with culture to assess for pyuria and hematuria, and nucleic amplification tests for the detection of chlamydial and gonorrheal infections. Cervical swabs and urine samples for nucleic amplification of chlamydia and gonorrhea have been shown to have similar sensitivities and specificities for detection of chlamydia and gonorrhea [11].

20.6 Imaging

When required, imaging modalities should be selected that offer the greatest diagnostic value but also the least amount of radiation exposure. Although computed tomography (CT) is the most sensitive imaging modality for patients with acute

pelvic pain, ultrasound examination is the preferred initial imaging modality for most patients with acute pelvic pain given its low cost, the absence of radiation exposure, and high sensitivity. A recent study demonstrated that using ultrasonography first, with CT only used when ultrasound results were negative or inconclusive, provided the best diagnostic value while subjecting the patient to the lowest amount of radiation possible [12].

Ultrasonography with Doppler venous flow is the imaging modality of choice when evaluating for ovarian torsion. Absent venous flow to the ovary on ultrasound is 99.6 % specific for ovarian torsion. However, due to the possibility of partial or intermittent torsion, venous flow to the ovary on ultrasound is only 72.1 % sensitive for excluding ovarian torsion. Flow to the ovary can be present even when torsion is present due to the dual blood supply of the ovary. Therefore, ovarian torsion cannot be reliably excluded by ultrasound if suspicion is high despite a normal ultrasound [13].

Ectopic pregnancy must be excluded in all pregnant patients presenting with pelvic pain. When the serum β-hCG is above the discriminatory zone of 1500 IU/L, a transvaginal ultrasound must be performed to confirm an intrauterine pregnancy. If an intrauterine pregnancy cannot be confirmed, an ectopic pregnancy should be suspected. A recently performed meta-analysis determined that an empty uterus on ultrasound is associated with a sensitivity of 81.1 % and a specificity of 79.5 % for ectopic pregnancy. Similarly, the sensitivities and specificities of the presence of a pseudo-sac are 5.5 % and 94.2 %, the presence of adnexal mass are 63.5 % and 91.4 %, and the presence of free fluid are 47.2 % and 92.3 %, respectively, for ectopic pregnancy. Therefore, ultrasound has strong specificity for diagnosing ectopic pregnancy but poorer sensitivity for excluding ectopic pregnancy [14].

20.7 Treatment

The treatment of acute pelvic pain varies with respect to its etiology.

Most women with acute pelvic pain and normal physical examination, laboratory studies, and imaging will improve without any intervention and can be safely discharged with follow-up with their primary care provider or obstetrician/gynecologist. Such disease etiologies include pelvic adhesions, endometriosis, and subclinical pelvic inflammatory disease [15].

20.7.1 Endometriosis

Endometriosis can be a cause of debilitating pelvic pain. Management consists of controlling the patient's pain and preventing flares. Mild to moderate pain can be treated with NSAIDs and estrogen-progesterone oral contraceptive pills. Severe symptoms that do not respond to these therapies can be treated with ovarian

suppression via gonadotropin-releasing hormone (GnRH) analogs. If a patient still experiences pain secondary to endometriosis, the definitive treatment is obliteration of the extrauterine endometrial tissue by laparoscopy [16].

20.7.2 Ovarian Cysts

Ruptured ovarian cysts are managed according to whether they are uncomplicated or complicated. Uncomplicated ovarian cyst rupture occurs when there is no hemodynamic instability, evidence of infection, or suspicion of malignancy and is managed conservatively with observation and pain control, including NSAIDs. Complicated ovarian cyst rupture occurs when there is hemodynamic instability, evidence of infection, or suspicion of malignancy and is managed with laparoscopy.

20.7.3 Infections

Infectious etiologies should be treated with antibiotics. The Centers of Disease Control and Prevention recommends empiric treatment of pelvic inflammatory disease with antibiotics that are effective against *N. gonorrhea* and *C. trachomatis* in women who are at increased of sexually transmitted infections or for whom clinical suspicion is high for pelvic inflammatory disease. The Centers for Disease Control and Prevention also recommends empiric treatment of all sexual partners during the 60 days preceding the patient's symptoms [17]. Vulvovaginal candidiasis is typically treated with topical antifungals such as clotrimazole and miconazole or with oral antifungals such as fluconazole. Bacterial vaginosis is typically treated with oral metronidazole or topical clindamycin. Trichomonas is typically treated with oral metronidazole. When prescribing metronidazole, it is important to educate the patient to avoid alcohol consumption due to its disulfiram reaction. Uncomplicated cystitis can be treated with several different medications. Nitrofurantoin can be given during pregnancy but should be avoided with renal impairment. Trimethoprim-sulfamethoxazole (TMP-SMX) should be avoided in pregnancy and when local resistance to TMP-SMX is >20 % [18]. Fluoroquinolones should be used sparingly due to the increased risk of tendon rupture.

20.7.4 Surgical Intervention

Surgical intervention, such as laparoscopy or laparotomy, is the treatment of choice for most emergent causes of acute pelvic pain. Laparoscopy is usually preferred over laparotomy due to shorter hospital stays, less postoperative pain, and faster recovery without sacrificing quality of treatment. However, laparotomy is preferred

in hemodynamically unstable patients. In many cases, laparoscopy is both therapeutic and diagnostic, such as in the setting of a nondiagnostic transvaginal ultrasound with concern for ectopic pregnancy. Laparoscopy is the definitive treatment for the removal of ectopic pregnancies, detorsion of ovarian torsion, and washout for ruptured tubo-ovarian abscess [19].

References

1. Kruszka PS, Kruszka SJ. Evaluation of acute pelvic pain in women. Am Fam Physician. 2010;82(2):141–7.
2. Jamieson DJ, Steege JF. The prevalence of dysmenorrhea, dyspareunia, pelvic pain, and irritable bowel syndrome in primary care practices. Obstet Gynecol. 1996;87(1):55–8.
3. Moore MS, Golden MR. Assessing trends in Chlamydia positivity and gonorrhea incidence and their associations with the incidence of pelvic inflammatory disease and ectopic pregnancy in Washington State, 1988–2010. Sex Transm Dis. 2016;43(1):2–8.
4. Cho HW, Koo YJ, Min KJ, Hong JH, Lee JK. Pelvic inflammatory disease in virgin women with tubo-ovarian abscess: a single-center experience and literature review. [published online ahead of print August 7, 2015]. J Pediatr Adolesc Gynecol. http://www.sciencedirect.com/science/article/pii/S1083318815002922. Accessed 31 Jan 2016.
5. Jafari MD, Jafari F, et al. Adhesive small bowel obstruction in the United States: has laparoscopy made an impact? Am Surg. 2015;81(10):1028–33.
6. Barnhart KT, Sammel MD, Garcia CR, et al. Risk factors for ectopic pregnancy in women with symptomatic first-trimester pregnancies. Fertil Steril. 2006;86(1):36–43.
7. Farley TM, Rosenberg MJ, Rowe PJ, Chen JH, Meirik O. Intrauterine devices and pelvic inflammatory disease: an international perspective. Lancet. 1992;339(8796):785–8.
8. Quan M. Diagnosis of acute pelvic pain. J Fam Pract. 1992;35(4):422–32.
9. Padilla LA, Radosevich DM, Milad MP. Accuracy of the pelvic examination in detecting adnexal masses. Obstet Gynecol. 2000;96(4):593–8.
10. Close RJ, Sachs CJ, Dyne PL. Reliability of bimanual pelvic examinations performed in emergency departments. West J Med. 2001;175(4):240–5.
11. Lunny C, Taylor D, et al. Self-collected versus clinician-collected sampling for Chlamydia and gonorrhea screening: a systematic review and meta-analysis. PLoS One. 2015;10(7):e0132776.
12. Laméris W, van Randen A, van Es HW, OPTIMA study group, et al. Imaging strategies for detection of urgent conditions in patients with acute abdominal pain: diagnostic accuracy study. BMJ. 2009;338:b2431.
13. Rostamzadeh A, Mirfendereski S, et al. Diagnostic efficacy of sonography for diagnosis of ovarian torsion. Pak J Med Sci. 2014;30(2):413–6.
14. Richardson A, Gallos I, et al. Accuracy of first-trimester ultrasound in diagnosis of tubal ectopic pregnancy in the absence of an obvious extrauterine embryo: systematic review and meta-analysis. Ultrasound Obstet Gynecol. 2016;47(1):28–37.
15. Harris RD, et al. Clinical outcome in female patients with pelvic pain and normal pelvic US findings. Radiology. 2000;216:440.
16. Fertil S. Treatment of pelvic pain associated with endometriosis: a committee opinion. Pract Comm Am Soc Reprod Med. 2014;101(4):927–35.
17. Centers for Disease Control and Prevention. Pelvic inflammatory disease (PID). 2015. Retrieved from http://www.cdc.gov/std/tg2015/pid.htm.
18. Gupta K. Emerging antibiotic resistance in urinary tract pathogens. Infect Dis Clin North Am. 2003;17(2):243–59.
19. Promecene PA. Laparoscopy in gynecologic emergencies. Semin Laparosc Surg. 2002; 9(1):64–75.

Chapter 21
Dysmenorrhea

Erich T. Wyckoff

21.1 Introduction

Dysmenorrhea is defined as painful menstruation occurring just before or during menses. It is the most common gynecologic condition among women of reproductive age [1]. In surveys, 50–90 % of reproductive-aged women worldwide describe experiencing painful menstrual periods [2]. Despite the prevalence of disease, many patients do not seek professional treatment. For clinical purposes, dysmenorrhea is further divided into primary and secondary dysmenorrhea. Primary dysmenorrhea refers to the presence of recurrent lower abdominal cramp/pain that occurs during menses in the absence of pelvic pathology. It normally occurs within the first 24 months of menarche [3]. Secondary dysmenorrhea has the same clinical features, but occurs in the setting of pelvic pathology that could be the origin of the patient symptoms such as endometriosis, adenomyosis, pelvic inflammatory disease, or uterine fibroids [3]. The most widely accepted explanation for the pathogenesis of primary dysmenorrhea is the overproduction of uterine prostaglandins (PG). Dysmenorrhea is responsible for considerable economic losses due to the costs of medication, medical care, and decreased productivity [4].

21.2 Risk Factors for Dysmenorrhea

- Primary dysmenorrhea
  - Age <30 years.
  - Body mass index <20 kg/m^2.

E.T. Wyckoff, MD
Obstetrics & Gynecology, University of Florida College of Medicine,
Gainesville, FL, 32610, USA
e-mail: wycket@ufl.edu

B. Desai, A. Desai (eds.), *Primary Care for Emergency Physicians*,
DOI 10.1007/978-3-319-44360-7_21

  - Smoking.
  - Menarche before age 12.
  - Longer menstrual cycles/duration of bleeding and irregular or heavy menstrual flow.
  - History of sexual assault [2].
  - Younger age at first childbirth and higher parity are associated with a reduced risk.

- Secondary dysmenorrhea

  - Onset of pain after age 25–30 and/or after years of normal cycles
  - Pain during menses that does not resolve after the first few days
  - Pain at times other than menses
  - Dyspareunia
  - Dyschezia
  - Progressively worsening symptoms [5]

Dysmenorrhea is positively associated with stress related to both work and general life, as well as with family history of dysmenorrhea [6].

Dysmenorrhea is inversely related to age, parity or number of live births, and oral contraception use [6].

21.3 Differential Diagnosis

- Mittelschmerz
- Endometriosis
- Adenomyosis
- Cervical stenosis
- Gastroenterologic causes
- Urinary tract infection
- Interstitial cystitis
- Pelvic congestive syndrome
- Leiomyoma
- Musculoskeletal causes
- Intrauterine device (IUD)
- Mullerian malformations
- Psychogenic disorders
- Miscarriage
- Ovarian cyst
- Ovarian torsion

21.4 Common Complaints and Red Flags

- Pain typically lasts 8–72 h [3].
- Pain is most severe during the first or second day of menses [5].
- Systemic symptoms: nausea, vomiting, diarrhea, fatigue, and insomnia [5].
- Patients with irritable bowel syndrome (IBS) are more likely to also experience dysmenorrhea [7].
- High rates of absenteeism from school/work, fatigue, and sleepiness may stem from inadequately treated dysmenorrhea.
- Red flags:
  - Pain lasting more than 72 h
  - Acute abdomen
  - Abnormal vital signs
  - Fever
  - Positive pregnancy test
  - Palpable abdominal or pelvic mass
  - Severe anemia

21.5 History

When evaluating for primary dysmenorrhea, it is important to differentiate primary dysmenorrhea from secondary amenorrhea in that secondary causes often require further evaluation. Severe pain sufficient to limit daily activities is considerably less common, affecting approximately 7–15 % of women [6].

Menstrual pain is referred to the abdomen in 70–90 % of patients and to the lower back in 40 % of patients [3].

Important historical features to elicit include:

- History of sexually transmitted infection
- Pregnancies
- Pelvic pathology
- Tobacco or alcohol use
- Sedentary lifestyle
- Family history of pelvic pain
- Dietary habits
- Menstrual history:
  - Age at menarche and at onset of menstrual pain
  - Menses: frequency, duration, and amount (heavy/moderate/light)
  - Regularity

  - Timing between onset of symptoms and menses
  - Severity and location of pain
  - Associated symptoms
  - Impact of symptoms on daily activities
  - Previous medication use (dose, duration, and efficacy)

21.6 Physical Examination

- Vital sign assessment (rule out)
  - Febrile
  - Hypotension
  - Tachycardia
- Abdominal exam (rule out)
  - Guarding/rebound tenderness
  - Abdominal mass
- Pelvic exam – if sexually active, has severe symptoms (grade 3) or has symptoms suggestive of pelvic pathology (rule out)
  - Cervix – mucopurulent discharge, friability, and cervical motion tenderness
  - Uterus – asymmetrical, enlarged, and exquisitely tender
  - Adnexa – fullness or masses and exquisitely tender
  - Products of conception in the vagina or protruding from the cervix
  - Vaginal wet mount/Gen-Probe/Pap smear when appropriate
- Urine HCG
- Urinalysis

While lab test, imaging studies, and laparoscopy are not mandatory to exclude secondary dysmenorrhea, they may be considered if pelvic disease is suspected [5] (Fig. 21.1).

21.7 Introduction to Treatment

Increased levels of circulating PGF2α and PGE_2 lead to increased myometrial contraction, vasoconstriction, and hypersensitization of pain fibers [3]. On account of the PG-based etiology of primary dysmenorrhea, the current most common pharmacologic treatment for dysmenorrhea is nonsteroidal anti-inflammatory drugs (NSAIDS) [9].

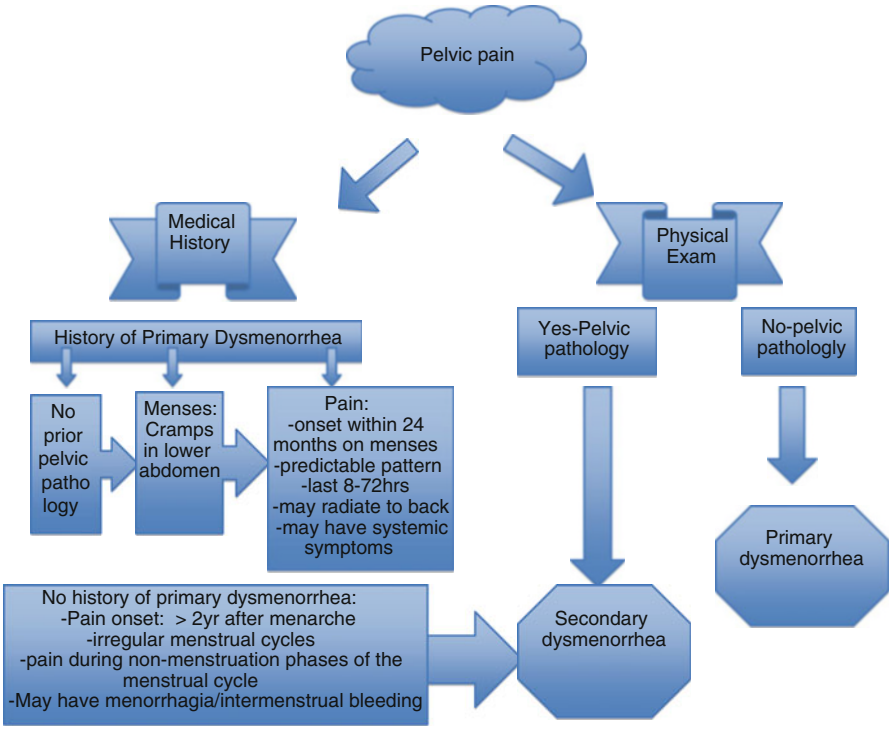

Fig. 21.1 Primary vs. secondary dysmenorrhea flow diagram. [3]

21.8 Pharmacologic Treatment

21.8.1 NSAIDS: First-Line Therapy

The various formulations of NSAIDS have comparable efficacy for dysmenorrhea, and pain relief is successfully achieved in 64–100 % on women [3]. *First-line therapy*:

Nonsteroidal anti-inflammatory drugs (NSAIDS)
Naproxen
Ibuprofen
Mefenamic acid
Diclofenac

Pitfalls

1. May increase risk of cardiovascular thrombotic events
2. May increase risk of stroke

3. May increase risk of gastric ulcer/bleeding and intestine perforation
4. Caution if asthmatic
5. Caution if dehydrated

21.8.2 Hormonal Contraceptives: Second-Line Therapy

Suppress ovulation and reduce thickness of the endometrial lining of the uterus, thereby reducing the volume of menstrual fluid [8]. This form of therapy includes hormonal intrauterine devices (IUDs) [3].

Pitfalls
1. Increase risk of thrombotic event
2. Increase risk of cardiovascular side effects with cigarette smoking
3. Contraindicated with known thrombophilias
4. Contraindicated if with migraine headaches with aura*
5. Contraindicated in estrogen- or progestin-dependent cancers
6. Caution if with hepatic disease

**IUDs are not contraindicated with migraine headaches with aura but should be used with caution.*

21.9 Nonpharmacologic Treatment

21.9.1 Abdominal Heat Wrap: First-Line Therapy

Found to be as effective as ibuprofen and more effective than acetaminophen in relieving dysmenorrheic pain [10]. It can decrease time to pain relief when used in combination with NSAIDs [5].

21.9.2 Exercise [5]

Pitfalls
1. Thermal skin damage when used inappropriately.
2. Exercise-induced injuries may occur.

21.10 Alternative Medicine

*Transcutaneous electrical nervous stimulation** [5]
*Acupuncture/Acupressure** [5]
*Low-fat diet** [5]
*Chinese herb preparations** [5]

**Limited evidence to support*

Pitfalls
1. Expensive
2. Controversial

References

1. Coco AS. Primary dysmenorrhea. Am Fam Physician. 1999;60:489–96.
2. Andersch B, Milsom I. An epidemiologic study of young women with dysmenorrhea. Am J Obstet Gynecol. 1982;144:655.
3. Lacovides S, Avidon I, Baker F. What we know about primary dysmenorrhea today: a critical review. Hum Reprod Update. 2015;21(6):762–78.
4. Lentz G, Lobo, Gershenson D, et al. Comprehensive gynecology. Philadelphia: Mosby Elsevier; 2012.
5. Roberts SC, Hodgkiss C, DiBenedetto A, Lee EC. Managing dysmenorrhea in young women. Nurse Practioner. 2012;37:47–52.
6. Ju H, Jones M, Mishra G. The prevalence and risk factors of dysmenorrhea. Epidemiol Rev. 2014;36:104–13.
7. Olafsdottir LB, Gudijonsson H, Jonsdottir HH, Bjornsson E, Thjodleifsson B. Natural history of irritable bowel syndrome in women and dysmenorrhea: a 10-year follow-up study. Gastroenterol Res Pract. 2012;21012:534204.
8. Ruoff G, Lema M. Strategies in pain management: new and potential indications for COX-2 specific inhibitors. J Pain Symptom Manage. 2003;25:S21–31.
9. Zahradnik HP, Hanjalic-Beck A, Groth K. Nonsteroidal anti-inflammatory drugs and hormonal contraceptives for pain relief from dysmenorrhea: a review. Contraception. 2010;81:185–96.
10. Akin MD, Weingand KW, Hengehold DA, Goodale MN, Hinkle RT, Smith RP. Continuous low-level topical heat in the treatment of dysmenorrhea. Obstet Gynecol. 2001;97:343–9.

Chapter 22
Diabetes

Ideen Zeinali, Bobby Desai, and Alpa Desai

(A) *Diabetes*

1. *Background*

   (a) *Epidemiology*: estimated overall prevalence of diabetes among adults in the USA ranges from 5.8 to 12.9 % but afflicts more than 20 % of the population over 60 years old [5]. Diabetes accounts for almost 14 % of US healthcare expenditures because of the associated microvascular disease (retinopathy, nephropathy, and neuropathy) and macrovascular disease (atherosclerosis) diabetes has. Half of these are related to complications, such as myocardial infarction (MI), stroke, end-stage renal disease (ESRD), retinopathy, and foot ulcers [2]:

      (i) Type II diabetes accounts for 90 % of cases of diabetes in the USA.

   (b) *Cost of diabetes* [3]:

      (i) Total cost of diagnosed diabetes in the USA in 2012 was $245 billion.
      (ii) Diabetes accounted for $176 billion for direct medical costs.
      (iii) $69 billion in reduced productivity.

I. Zeinali, MD (✉)
Emergency Department, UF Health-Shands, Gainesville, FL, USA
e-mail: i.zeinali2017@ufl.edu

B. Desai, MD, MEd
Emergency Medicine, University of Florida, Gainesville, FL, USA

A. Desai, DO
Community Health and Family Medicine, University of Florida, Newberry, FL, USA

B. Desai, A. Desai (eds.), *Primary Care for Emergency Physicians*,
DOI 10.1007/978-3-319-44360-7_22

2. *Pathophysiology*

   (a) *Type I diabetics (T1DM)* – unknown why this occurs but is considered an autoimmune disease where the immune system incorrectly targets the insulin-producing beta-cells in the pancreas, so there is an absolute lack of insulin production.
   (b) *Type II diabetics (T2DM)* – insulin production is present but is insufficient to meet the body's requirements as a result of end-organ insulin resistance. Essentially it is associated with slowly progressive beta-cell failure. By the time type 2 diabetes is diagnosed in patients, up to one half of their beta-cells are not functioning properly and that failure rate continues at ~4 % per year. Therefore, patients with T2DM often benefit from insulin therapy at some point after diagnosis [4].
   (c) *Environmental risk factors*:

      (i) T1DM – symptoms likely start in childhood or young adulthood.
      (ii) T2DM – likely secondary to environmental factors and can have a genetic component. Can occur at any age due to the fact that T2DM in children is increasing secondary to rising obesity epidemic, but most commonly occur during adulthood. T2DM, thought to be multifactorial, typically occurs in the setting of the metabolic syndrome, which also includes abdominal obesity, hypertension, hyperlipidemia, and increased coagulability [1].

   (d) *Major characteristics of diabetes mellitus*:

      (i) T1DM – first symptoms occur when blood sugar gets too high. Symptoms include thirst, hunger, fatigue, frequent urination, weight loss, and blurred vision. Very high blood sugar can cause rapid breathing, dry skin, fruity breath, and nausea.
      (ii) T2DM – symptoms may not show up for many years. Early symptoms can present as frequent infections, fatigue, frequent urination, thirst, hunger, blurred vision, and pain or numbness in the hands or feet.

3. *Diagnosis*

   (a) HgA1C > 6.5 % OR
   (b) Fasting plasma glucose ≥126 mg per dL
   (c) Random plasma glucose ≥200 mg per dL during an oral glucose tolerance test

(B) *Hyperglycemia* – condition of excessive amounts of glucose circulating in the plasma and is generally considered a level of > 200 mg/dL, unfasting. Signs and symptoms include frequent urination, increased thirst, and high levels of sugar in the urine. Two of the most serious acute metabolic complications of diabetes, aka hyperglycemic crises, are diabetic ketoacidosis (DKA) and hyperosmolar hyperglycemic state (HHS) [3].

1. *Diabetic ketoacidosis (DKA)* – accounts for more than 110,000 hospitalizations annually in the USA with mortality ranging from 2 to 10% [5]. Happens predominately in T1DM, but can occur in those with T2DM

   (a) *Definition*: it is a life-threatening condition that presents with uncontrolled hyperglycemia, metabolic acidosis, and increased total body ketone concentration. The condition results from an insulin deficiency, and in response, the body switches to burning fatty acids and produces acidic ketones (acetoacetate, ß-hydroxybutyrate). This leads to hyperglycemia-induced osmotic diuresis, which leads to dehydration if there isn't sufficient oral fluid intake, hyperosmolarity, and electrolyte losses. With diminished insulin action and hyperosmolarity, the utilization of potassium by skeletal muscles markedly diminishes leading to a decrease in intracellular potassium. However, there is a profound total body potassium deficiency since potassium is also lost via osmotic diuresis [7].

   (i) *Classification*

Criteria and classification of diabetic ketoacidosis (DKA)			
DKA	Mild	Moderate	Severe
Plasma glucose	>250 mg/dl	>250 mg/dl	>250 mg/dl
Arterial pH	7.25–7.30	7.00–7.24	<7.00
Serum bicarbonate	15–18	10–15	<10
Urine ketone	pos	pos	pos
Serum ketone	pos	pos	pos
Anion gap			
Serum osmolality	Variable	Variable	Variable
Mental status	Alert	Alert/drowsy	Stupor/coma

   (b) *Common causes*: infection, pancreatitis, cardiac event (i.e., MI), pregnancy, noncompliance with medication or inadequate insulin administration, new-onset diabetes, or cocaine use.

   (c) *Signs and symptoms*: dehydration, abdominal pain, nausea/vomiting, polydipsia, polyuria, and confusion. Often the patient can have a "ketotic" odor or "fruity" smell to their breath. Since the body is overwhelmed by the rising acidosis, the body tries to compensate via hyperventilation, which is why you may see the patient taking deep gasping breathing (Kussmaul respirations).

   (d) *Initial lab tests*: in order to make the diagnosis, the following are necessary – basic metabolic panel, serum osmolality, ß-hydroxybutyrate or urinalysis (to indicate ketones in the urine), CBC, and venous blood gas to get the pH.

   (e) *Treatment*

   (i) *Goals of treatment*:

1. Resuscitate the patient – average adult in DKA has a total body water shortage of about 6 L in addition to shortages of multiple electrolytes, such as sodium, potassium, chloride, phosphate, magnesium, and calcium [6]. Replace the fluid deficit within the first 24–36 h with the goal of 50% of volume replacement within the first 12 h.
2. Close the gap and normalize acidosis – this is primarily done with fluid hydration and starting an insulin drip:
   (a) Insulin drip: the potassium level should be >3.3 mEq/L before initiating insulin therapy. A Common form of practice is to start an intravenous regular insulin drip at 0.1 units/kg per hour. Blood glucose levels should be checked hourly, and when levels fall below 250 mg/dL, it may be appropriate to decrease the inulin infusion rate to 0.05 units/kg per hour and add dextrose (5–10%) to the IV fluids running. The insulin drip should be continued until the anion gap is closed, at which point the IV insulin should continue for 2 h more after initiation of subcutaneous insulin for people who are able to eat.
   (b) Bicarbonate use in DKA remains controversial. Prospective randomized studies have failed to show either beneficial or deleterious changes in morbidity or mortality with bicarbonate therapy in DKA patients with pH between 6.9 and 7.1 [3]. Current recommendations state no bicarbonate is necessary if pH is >7.0. However, if the pH remains <7.0 after the initial hour of hydration, it seems prudent to administer 1–2 mEq/kg sodium bicarbonate over the course of 1 h [14].
3. Resolution of DKA – plasma glucose <200 mg/dL, serum HCO3 >18 mEq/L, venous blood pH >7.3, and anion gap <12 [7]

(ii) *Complications*:

1. Hypoglycemia and hypokalemia – most frequent complications that can be prevented with insulin dose adjustment and frequent monitoring of glucose (hourly point of care glucose checks) and potassium levels (every 2–3 h).
2. Cerebral edema – it is the most dangerous complication seen from over-vigorous fluid resuscitation and is most commonly seen in children. Likely due to a combination of factors, dehydration, severe acidosis, and low carbon dioxide levels along with the increased level of inflammation and coagulation may, together with these factors, lead to decreased blood flow. The brain swells once fluid replacement has been started causing an increase in intracranial pressure and ultimately death [6].

3. Pulmonary edema – can develop from excessive fluid replacement, particularly in those with chronic kidney disease (CKD) or congestive heart failure (CHF).
4. Non-anion gap hyperchloremic acidosis – likely secondary to intensive administration of chloride-containing fluids (NaCl) and low plasma bicarbonate. This normally resolves and doesn't affect treatment course.

2. *Hyperosmolar hyperglycemic state (HHS)* – also known as hyperosmolar hyperglycemic nonketotic coma (HHNC). It is much less common than DKA, but confers a greater mortality (~15%) [14]. Prognosis is substantially worsened at the extremes of age and in the presence of coma and hypotension. More common in T2DM patients and process likely evolves over several days to weeks. Identified by marked elevation of blood glucose, hyperosmolarity secondary to high blood sugars causing severe dehydration, and little or no ketosis [14]. This is secondary to the fact that plasma insulin concentrations are inadequate to facilitate glucose utilization by insulin-insensitive tissues but adequate to prevent lipolysis and subsequent ketogenesis [14].

   (a) *Diagnosis*: blood glucose >600 mg/dL, pH >7.30, serum bicarb >15 mEq/L, small urine ketones, normal or slightly elevated ß-hydroxybutyrate, and serum osmolality >320 mEq/L; anion gap is variable. There are also several electrolyte losses: sodium, chloride, potassium, phosphate, calcium, and magnesium [13].
   (b) *Causes*: underlying infections are the most common, but other causes include noncompliance with medications, certain drugs that affect carbohydrate metabolism (i.e., corticosteroids, thiazides, and sympathomimetic agents like dobutamine or terbutaline), undiagnosed diabetes, substance abuse, and coexisting disease [14].
   (c) *Signs and symptoms* – polyuria, polydipsia, vomiting, dehydration, weakness, hypothermic secondary to vasodilation, clouding of sensoria, and finally coma [14]. Hypothermia and hypotension are poor prognostic signs.
   (d) *Treatment* – goals of treatment and management are the same as DKA listed above in regard to fluid resuscitation and insulin infusion. Patients in HHS have profound dehydration, up to an average of 9 L fluid deficit; however, frequent reevaluations need to be done in patients with either cardiac or renal compromise as to prevent iatrogenic fluid overload. In HHS, insulin therapy is continued with glucose levels being maintained around 250–300 mg/dL until mental obtundation and hyperosmolarity are resolved:

      (i) Complications of treatment – include hypokalemia, hypoglycemia, and cerebral edema

(C) *Hypoglycemia* – from physiological point of view, a detectable impairment of higher cerebral function has been demonstrated at plasma glucose levels of 54 mg/dL or less; however, counterregulatory responses to hypoglycemia have

been described at plasma levels between 65 and 70 mg/dL acting within a few minutes [9]. Hypoglycemia occurs when insulin deficiency in diabetics is overcorrected.

1. *Components of these counterregulatory responses*: suppression of insulin secretion from pancreatic B-cells, increase in glucagon secretion from pancreatic a-cells, increase in adrenomedullary epinephrine secretion, and increase in growth hormone and adrenocorticotropin secretion from the anterior pituitary [9].

   (e) In diabetic patients, these counterregulatory responses are decreased leading to more frequent episodes of hypoglycemia particularly in the setting of exogenous insulin administration.
   (f) Hypoglycemia is a more common problem in T1DM compared to T2DM secondary to impairment of the first and second counterregulatory mechanisms [9]. In T2DM, the counterregulatory mechanisms are preserved except for in the elderly (>65 years), it may be reduced, and thereby these patients are also more susceptible to hypoglycemia [9].

2. *Insulin overdose*: most insulin overdoses are intentional, but many are accidental. Symptoms include diaphoresis, chills, lightheadedness/dizziness, tachycardia, tremors, blurred vision, and tingling around the lips/mouth [11]. More severe symptoms include change in mental status, seizures, unconsciousness, and death. Initial emergency response is to inject glucagon (D50) and to have close/strict monitoring over glucose levels. If levels continue to drop, pt can be placed on a D5 infusion.

22.1 Treatment

1. *American Diabetes Association (ADA) recommendations*:

   (a) *Goals of treatment* – HgA1C <7.0 for many nonpregnant adult: recommendation based on pathophysiologic reasoning, expert opinion, and the reduction in microvascular complications shown in the UK Prospective Diabetes Study (UKPDS):

      (i) Recommends HgA1C <6.5 is reasonable for patients with a short duration of diabetes, a long life expectancy, and no significant cardiovascular disease.
      (ii) More stringent control of HgA1C <6.0 may be appropriate in T1DM and during pregnancy.
      (iii) Recommends HgA1C <7.5–8 is appropriate for patients with a short life expectancy, cardiovascular disease, two or more cardiovascular disease risk factors, or duration of disease of 10 years or more because of a lack of benefit and the potential for increased risk of mortality.

(b) The ADA recommends starting therapy with metformin first-line therapy to reduce microvascular complications, assist in weight management, reduce the risk of cardiovascular events, and reduce the risk of mortality in patients with type 2 diabetes mellitus.
(c) The ADA recommends medical nutrition therapy, preferably from a registered dietician, to be part of the treatment plan after pharmacotherapy is initiated.
(d) Self-monitoring of blood glucose levels is not recommended to guide treatment on its own secondary to meta-analysis of 12 RCTs found that it does not result in lower glycemic levels.
(e) Tight glycemic control for protection against cardiovascular disease (CVD) in diabetes has been established in the DCCT/Epidemiology of Diabetes Interventions and Complications (EDIC) study for T1DM [1]. However, the effect on CVD in patients with T2DM had either no effect or was associated with higher overall mortality [1].

2. *Oral hypoglycemics*: primarily used for maintaining glycemic control in T2DM

Hypoglycemic agents for treating type 2 diabetes mellitus

Medication	Examples	Mechanism of action	Average AIC reduction (%)	Potential adverse effects
Alpha-glucosidase inhibitors	Acarbose, Miglitol	Inhibit enzyme at intestinal brush border; slow absorption of carbohydrates	0.5–0.8	Flatulence, diarrhea, abdominal bloating
Biguanides	Metformin	Decrease hepatic glucose production; increase insulin sensitivity peripherally; and decrease intestinal absorption of carbohydrates	1.0–1.3	Nausea, diarrhea, abdominal bloating
Dipeptidyl-peptidase-4 inhibitors	Alogliptin, Linagliptin, Saxagliptin, Sitagliptin	Increase glucagon-like peptide-1; increase insulin secretion from B-cells; and decrease glucagon secretion from a-cells in the pancreas	0.5–0.9	Headache, pancreatitis
Glucagon-like peptide-1 receptor agonists	Albiglutide, Dulaglutide, Exenatide, Liraglutide	Increase insulin secretion from B-cells and decrease glucagon secretion from a-cells in the pancreas; suppress hepatic glucose production; delay gastric emptying	0.8–2.0	Nausea, vomiting, sense of fullness. Weight loss of 1–4 kg is likely. Pancreatitis

Hypoglycemic agents for treating type 2 diabetes mellitus				
Medication	Examples	Mechanism of action	Average AIC reduction (%)	Potential adverse effects
Meglitinides	Nateglinide, Repaglinide	Closes potassium channels in B-cells; stimulate release of insulin from the pancreas; Metabolized primarily by the liver	0.5–1.0	Hypoglycemia
Sodium-glucose cotransporter 2 inhibitors	Canagliflozin, Depagliflozin, Empagliflozin	Lower renal threshold for glucose and reduce reabsorption of filtered glucose from tubular lumen; increase urinary glucose excretion	0.5–0.9	Increased UTIs. Increased LDL. Weight loss of 0.7–3 kg is typical
Sulfonylureas	Glimepiride, Glipizide, Glyburide	Bind to potassium channels in B-cells; stimulate release of insulin from the pancreas	0.4–1.2	Hypoglycemia, weight gain
Thiazolindinediones	Pioglitazone, Rosiglitazone	Increase hepatic glucose uptake; decrease hepatic glucose production; increase insulin sensitivity in the muscle and adipose tissue	0.5–1.4	Weight gain, edema; contraindicated in patients w/ NYHA class III or IV CHF

(i) Regardless of the initial response to therapy, the natural history of most patients is for HgA1C to rise over time. Worsening beta-cell dysfunction with decreased insulin release as the primary reason for disease progression. However, decreased compliance with the dietary regimen may also contribute to progression [1]. T2DM often becomes insulin dependent to achieve acceptable glycemic control.

3. *Insulin*: natural hormone secreted by the B-cells of the pancreas that work in balance to maintain appropriate blood glucose levels. In T1DM, these cells are no longer functioning, and in T2DM, the body has become more resistant to insulin. There are different types of insulin, which are listed below, which are characterized based on how quickly they work, when they peak, and how long they last. ADA recommends TID monitoring with glucose checks.

   (a) *Rapid acting* – starts working ~15 min after injection and peaks at about 1 h. Will continue to work for 2–4 h [3].

(b) *Regular or short acting* – starts working ~30 min after injection and peaks at 2–3 h. It will continue to work for ~3 to 6 h. It is usually given before a meal in addition to the long acting [12].
(c) Long acting – starts working several hours after injection and lasts for ~24 h.
(d) Mixed – insulin is a mixture of NPH with either rapid-acting insulin or regular insulin in different ratios. Primarily written for patients who need a simple insulin treatment plan, primarily in those who are older, have diminished vision or trouble with dexterity, and have difficulty giving themselves multiple injections and with compliance [12]. Since short- and long-acting insulin is combined, only one injection is needed. Downside is the unpredictability in action and the inability to easily adjust the dosage for high or low glucose levels.
(e) *Intermediate acting* – starts working ~2 to 4 h after injection and will peak ~4 to 12 h later. Will continue to work for 12–18 h. Normally taken twice a day in addition to short-acting insulin [12] (Fig. 22.1).

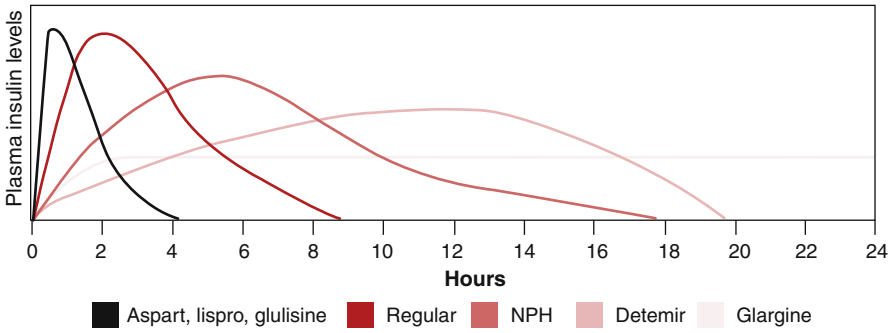

Fig. 22.1 Onset of action, peak, and duration of exogenous insulin preparations (adapted from Hirsch [16])

Pharmacokinetic profiles of insulin types			
Insulin type	Onset	Peak	Duration
Long-acting			
Detemir (Levemir)	3–4 h	6–8 h	6–23 h
Glargine (Lantus)	90 min	None	24 h
Intermediate-acting			
NPH (Humulin N)	1–2 h	4–10 h	>14 h
Short-acting			
Aspart (Novolog)	15 min	1–3 h	3–5 h
Glulisine (Apidra)	15–30 min	30–60 min	4 h
Lispro (Humalog)	15 min	30–90 min	3–5 h
Regular	30–60 min	2–4 h	5–8 h
Mixed			
NPH/Lispro or Aspart	15–30 min	Dual	14–24 h

(f) *Complications associated with insulin therapy*

(i) *Hypoglycemia*: iatrogenic hypoglycemia can occur secondary to multiple reasons including incorrect dosing of insulin, giving medication while fasting, exercising vigorously without snacking, etc. People are advised to ingest 15 g of a fast-digesting carbohydrate. Examples include raisins, soda, fruit juice, honey, and candy and symptoms tend to improve within 15 min [11].

(ii) *Pain*: associated with injection therapy and from glucose monitoring.

(iii) *Weight gain*: secondary to the anabolic effects of insulin, increased appetite, defensive eating from hypoglycemia, and increased caloric retention related to decreased glycosuria.

4. *Nonpharmacologic therapy in T2DM patients* – three major components: diet modification, exercise, and weight reduction. These changes are very important in improving glycemic control and can slow progression of impaired glucose tolerance to overt diabetes [1].

5. *Health maintenance*

(a) A1C every 3 months and make any therapy adjustments if A1C goals are not being changed within 6 months.

(b) Every year, measure lipid panel, liver function tests, urine albumin, and BMP.

(D) *Complications from diabetes* – there are microvascular and macrovascular complications associated with diabetes that can be delayed with interventions and the earlier diagnosis of diabetes.

1. *Macrovascular complications*

(a) Atherosclerosis – develops secondary to chronic inflammation and injury to arterial wall in response to endothelial injury and inflammation and oxidized lipids from LDL particles accumulating in the endothelial wall of arteries:

(i) *Peripheral arterial disease* – likely secondary to atheroma formation noted above along with increased platelet adhesion from impaired nitric oxide generation and increased free radical formation in platelets promoting aggregation and hypercoagulability [14].

(ii) *Cardiovascular disease [myocardial infarction (MI)]* – exact mechanism of how diabetes increases likelihood of atherosclerotic plaque formation is not clearly defined; however, rates for an MI were 1.8 times higher among adults with diabetes in 2010 [3]. Patients are more likely to be asymptomatic or have atypical symptoms than nondiabetic patients.

(iii) *Cerebrovascular accident (stroke)* – rates for stroke were 1.5× higher among adults with diabetes in 2010 [3].

(iv) *Screening* – don't perform exercise stress testing in asymptomatic patients because identifying asymptomatic disease or providing early intervention has not been proven to improve outcomes in this population. Perform annual assessment of risk criteria (blood pressure, fasting lipid panel, smoking history) to identify patients at high risk for coronary artery disease and would benefit from interventions, such as aspirin, ACE inhibitors, and statin therapy [1].

(v) *Treatment goals*

1. Hypertension: blood pressure goal <130/80 mmHg [3].
2. Lipid goals: lipid testing should be done annually. LDL goal <100 mg/dL (or <70 mg/dL in patients with overt CVD), HDL >50 mg/dL, and fasting triglycerides <150 mg/dL [14].
3. Aspirin therapy (81 mg) is indicated for prevention of CVD and should be used in patients >40 years old and in those 30–40 years old with other risk factors present.

(b) Routine foot examination should be conducted annually to conduct risk factors predicative of ulcers and amputation:

(i) Screen for peripheral vascular disease (PVD) by asking for history of claudication and assessing the pedal pulses. Consider obtaining an ankle brachial index (ABI) since many patients with PVD are asymptomatic.

(ii) Recommendations of prophylactic foot care: avoid going barefoot; test water temperature before stepping into a bath; trim toenails to shape of the toe and remove sharp edges with a nail file; wash and check feet daily; shoes should be snug and customized if feet are misshapen or have ulcers; socks should fit and be changed daily [1].

2. *Microvascular complications*

(a) *Retinopathy* – diabetes is the leading cause of new cases of blindness in adults ages 20–74, approximately 10,000 new cases every year [14]. There are several proposed pathological mechanisms by which diabetes may lead to retinopathy, such as aldose reductase, injury by glycoproteins, oxidative stress causing cellular injury from hyperglycemia, and growth factors (i.e., vascular endothelial growth factor [VEGF]) leading to hypoxia [15].

(i) *There are two different types*: background and proliferative

1. *Background* – includes features as small hemorrhages in the middle layers of the retina. They appear clinically as "dot hemorrhages." [14]. Hard exudates are caused by lipid deposition and occur at the margins of these hemorrhages. Microaneurysms occur and can be the first sign of retinopathy. Retinal edema can occur secondary to microvascular leakage [14].

2. Proliferative – characterized by the formation of new blood vessels on the surface of the retina and can lead to vitreous hemorrhage. White areas on the retina ("cotton wool spots") can be a sign of impending proliferative retinopathy [14]. If proliferation continues, blindness occurs via vitreous hemorrhage and retinal detachment from traction [14]:

(ii) Patients are also at increased risk for vision loss secondary to refractive errors (correctable visual impairment), cataracts, and glaucoma. Diabetes can also cause sudden changes in vision, such as blurriness or spots in the field of vision.

(iii) Annual evaluation of visual acuity in addition to dilated eye examinations for retinopathy to reduce injury risk and improve quality of life. T1DM should receive a comprehensive eye exam and dilation within 3–5 years after the onset of diabetes [14].

(iv) Reduce risk with and progression with good glycemic and BP control.

(b) *Nephropathy* – defined as proteinuria >500 mg in 24 h in the setting of diabetes [14]. This is likely preceded by lower degrees of proteinuria or microalbuminuria, which is albumin excretion of 30–299 mg/24 h [14]. Diabetes is the top cause of renal failure in the USA, accounting for 44 % of new cases in 2011, according to the National Institutes of Health. 10–40 % of people with T2DM develop kidney failure [10]. Pathological changes include increased glomerular basement membrane thickness, microaneurysm formation, mesangial nodule formation (Kimmelstiel-Wilson bodies), and other changes [14]:

(i) Screening for urinary albumin excretion – increased urinary protein excretion is the earliest finding of diabetic nephropathy. Persistent values between 30 and 300 mg/day in a patient with diabetes are indicative of diabetic nephropathy. Those with values >300 mg/day are considered to have clinical renal disease. Urine dipstick is a relatively insensitive marker for proteinuria since it won't detect protein until excretion exceeds 300–500 mg/day [1].

1. Screening for increased urinary albumin excretion can be deferred for 5 years after the onset of T1DM because it is uncommon before this time. Screening should begin at diagnosis in patients with T2DM because many have had diabetes for several years before diagnosis [1].

(ii) The effective therapy for diabetic nephropathy is to start a patient on an ACE (angiotensin-converting enzyme) inhibitor or ARB (angiotensin receptor blocker) medication. Treatment with an ACE inhibitor has been shown to decrease the risk of developing nephropathy and cardiovascular events in patients with T2DM [14]. Both medications have been shown to decrease the risk of

progression to macroalbuminuria in patients with microalbuminuria by as much as 60–70 % [14].

(c) Neuropathy – per the ADA, it is identified as the "presence of symptoms and/or signs of peripheral nerve dysfunction in people with diabetes after the exclusion of other causes" [3]. In 2005–2008, 4.2 million adults ages 40 years and older with diabetes had diabetic neuropathy [3]. Developing diabetic neuropathy is proportional to both the magnitude and duration of hyperglycemia. Precise nature of injury is not known, but likely related mechanisms include polyol accumulation, injury from AGEs, and oxidative stress [14]:

(i) Peripheral neuropathy can manifest in several different forms, including sensory, focal/multifocal, and autonomic neuropathies.
(ii) Patients experience a "burning, tingling, or electrical pain" or it may be described as a simple numbness. Pain may be worse at night. Physical exam reveals sensory loss to light touch, vibration, and temperature [14].
(iii) Amputations – ~60 % of nontraumatic lower limb amputations among people 20 years or older occur in people with diagnosed diabetes [3].
(iv) Treatment – there is no specific treatment for diabetic neuropathy, but there are many drugs available for symptomatic control and worsening of symptoms which can be prevented by improved glycemic control. Examples of medications available include gabapentin, pregabalin, amitriptyline, topiramate, SSRIs, etc.

References

1. McCullough D, Nathan D, Mulder J. Overview of medical care with people with diabetes mellitus. http://www.uptodate.com/contents/overview-of-medical-care-in-adults-with-diabetes-mellitus.
2. Loghami E. Diabetes mellitus: type 1 and type 2. http://www.epi.umn.edu/let/pubs/img/adol_ch14.pdf.
3. American Diabetes Association. www.diabetes.org/.
4. Ripsin C, Kang H, Urban R. Management of blood glucose in type 2 diabetes mellitus. Am Fam Physician. 2009;79(1):29–36.
5. Ford W, Self W, Slovis C, McNaughton C. Diabetes in the emergency department and hospital: acute care of diabetes patients. Curr Emerg Hosp Med Rep. 2013;1(1):1–9.
6. Diabetic Ketoacidosis. https://en.wikipedia.org/wiki/Diabetic_ketoacidosis.
7. Kitabchi A, Gosmanov A. Diabetic ketoacidosis. http://endoemergencies.org/sample-chapter.html.
8. Petznick A. Insulin management of type 2 diabetes mellitus. Am Fam Physician. 2011;84(2):183–90.
9. Liatis S, Katsilambros N, Kanaka-Gantenbein C, Makrilakis K, Tentolousi N. Diabetic emergencies: hypoglycemia caused by insulin. Nov 2012. http://www.diabetesincontrol.com/diabetic-emergencies-hypoglycemia-caused-by-insulin/.

10. The 7 most common diabetic-related emergencies. Jun 2008. http://www.health.com/health/condition-article/0,,20190740_3,00.html.
11. Story C, Pletcher P. Insulin overdose: signs and symptoms. http://www.healthline.com/health/diabetes/insulin-overdose#Symptoms4.
12. Hess-Fischl A. What is insulin? Important hormone allows your body to use sugar (glucose). http://www.endocrineweb.com/conditions/type-1-diabetes/what-insulin.
13. Stoner G. Hyperosmolar hyperglycemic state. Am Fam Physician. 2005;71(9):1723–30.
14. American Diabetes Association. Hyperglycemic crises in diabetes. Diabetes Care. 2004;27 suppl 1:s94–102.
15. Fowler M. Microvascular and macrovascular complications of diabetes. Clin Diabetes. 2008;26(2):77–82.
16. Hirsch IB. Insulin analogues. N Engl J Med. 2005;352(2):177.

Chapter 23
Back Pain

Grant Harrell

23.1 Introduction

Worldwide, back pain is one of the most frequent disorders people experience, with low back pain specifically accounting for the majority of complaints. In fact, the Global Burden of Disease study ranked low back pain as the number one cause of years lived with disability [1]. In the USA, back pain has a lifetime prevalence of close to 80 %, and spinal disorders are in the top five most common primary diagnoses for office visits [2].

For the emergency physician, acute back pain is of particular importance, although chronic back pain with an acute flare can precipitate an emergency department (ED) visit as well. While low back pain typically originates from the lumbosacral spinous structures and para-spinous musculature, thoracic back pain can often represent referred pain from the viscera and should warrant careful consideration.

Because back pain is so frequently encountered in both the primary care and ER setting, it poses a substantial financial burden on the healthcare system [3] and is a common source for unnecessary testing, referrals, and inappropriate prescribing practices. Therefore, differentiating back pain that is likely to spontaneously resolve versus that which may represent a more ominous underlying pathology is an important skill for patient care as well as for appropriate resource utilization.

G. Harrell, MD
Community Health and Family Medicine, UF Health Shands,
Gainesville 32610, FL, USA
e-mail: gharrell@ufl.edu

B. Desai, A. Desai (eds.), *Primary Care for Emergency Physicians*,
DOI 10.1007/978-3-319-44360-7_23

23.2 Risk Factors for Low Back Pain [4]

- Obesity
- Physical inactivity
- Age >30
- Work that requires heavy lifting, twisting, bending, or whole body vibration (e.g., truck driving)
- Arthritis
- Osteoporosis
- Pregnancy
- Stress or depression
- Bad posture
- Smoking

23.3 History and Differential Diagnosis

When obtaining history for low back pain with or without sciatica, it is particularly important to ask about red flag symptoms. The presence of red flag symptoms indicates the potential for a more complicated underlying pathology affecting the spine which will likely require additional workup with diagnostic imaging and lab tests. The following list represents historical items that are suggestive of a more serious underlying condition [5].

- Infection – 0.1 % of cases
  - Immunosuppression
  - Intravenous drug use
  - Prolonged corticosteroid use
  - Urinary infection
  - Fever
  - Pain not improved with rest or 6 weeks conservative measures
  - Unrelenting night pain
- Cancer – 0.7 % of cases
  - Prior history of cancer or current cancer diagnosis
  - Unexplained weight loss
  - Pain not improved with rest or 6 weeks conservative measures
  - Unrelenting night pain
- Spinal fracture (including osteoporotic compression fractures) – 4 % of cases
  - History of significant trauma
  - Prolonged corticosteroid use
  - History of osteoporosis especially in the setting of a fall or heavy lift
  - Elderly age group especially in the setting of a fall or heavy lift

- Cauda equina syndrome or severe neurologic compromise
  - Acute urinary retention or overflow incontinence
  - Loss of anal sphincter tone or fecal incontinence
  - Progressive motor weakness of lower extremities
  - Saddle anesthesia
- Inflammatory back pain (ankylosing spondylitis, reactive arthritis, psoriatic arthritis) – 0.3 % of cases
  - Progressive worsening of pain beyond 3 months.
  - Age less than 40.
  - Predominant morning stiffness.
  - Pain improves with activity.
  - Associated rash.
  - Other joint pains (especially of hands and feet).
  - Antecedent flu-like illness, STI, or gastrointestinal illness.
  - Eye complaints (painful, red eyes).

Although the above conditions are important to address in the history, almost 90 % of low back complaints are related to mechanical problems originating from damage to the musculo-ligamentous structures or intervertebral discs [5].

- Lumbar strain or sprain – ≥70 % of cases
  - Diffuse, nonspecific pain of lumbar muscles
  - Radiation to buttocks
  - Worsened with activity
  - Improvement with rest and/or OTC pain relievers
- Degenerative disc or facet disease – 10 % of cases
  - History of chronic back pain
  - Pain localized closer to lumbar spine but still nonspecific
  - Worsened with activity
  - Improvement with rest and/or OTC pain relievers
  - Age >50
- Herniated disc – 4 % of cases
  - Radicular pain (often radiates below the knee, but the specific pattern depends on the affected vertebral level).
  - Leg pain often exceeds back pain.
  - Age 20–50.
  - Unilateral leg weakness (especially activities involving dorsi/plantar flexion).
- Spinal stenosis – 3 % of cases
  - Aching in thighs or calves with walking (i.e., “neurogenic claudication”).
  - Pain is bilateral.

  - Exacerbated by spine extension (e.g., walking downhill or lying flat).
  - Alleviated by spine flexion (e.g., walking uphill or leaning on shopping cart).
  - Pain improved with sitting.

- Spondylolisthesis
  - Worsened with activity
  - Improvement with rest
  - Can present similarly to degenerative disc disease or spinal stenosis
  - Usually discovered on imaging

Non-spinal and visceral disorders account for about 2% of cases of back pain (although rarely as isolated back pain) [5], so although they are a relatively uncommon cause of back pain, they are important to recognize because they often require specific treatment.

- Prostatitis
  - Dysuria
  - Fever
  - Pain with sitting
  - Lower abdominal/pelvic pain
- Pelvic inflammatory disease
  - Vaginal discharge
  - Fever
  - History of STIs or multiple sex partners
  - Lower abdominal/pelvic pain
- Cystitis
  - Dysuria
  - Urinary frequency
  - Fever
- Endometriosis
  - Dyspareunia
  - Pain worse at time of menses
  - Lower abdominal/pelvic pain
- Pyelonephritis
  - Localized to unilateral flank/thoracic back
  - Fever
  - Nausea and/or vomiting
- Nephrolithiasis
  - Localized to unilateral flank/thoracic back
  - Nausea and/or vomiting

  - Hematuria
  - Radiation to ipsilateral inguinal region
- Aortic aneurysm
  - Epigastric pain
  - Age >65
  - History of smoking
- Aortic dissection
  - Ripping/tearing quality to pain
  - History of hypertension (HTN)
  - Anterior chest pain
- Pancreatitis
  - Pain worsens with eating.
  - Pain radiates from the epigastrium.
  - History of alcohol abuse, gallstones, or severe hypertriglyceridemia.
  - Localized to the mid back.
  - Nausea and/or vomiting.
- Peptic ulcer
  - Heavy NSAID use.
  - Pain worsens with eating.
  - Melena.
  - Pain radiates from the epigastrium.
  - Nausea and/or vomiting.
- Cholecystitis
  - Pain radiates to the right scapula.
  - Pain radiates from abdominal RUQ.
  - Nausea and/or vomiting.
- Shingles
  - Localized to unilateral flank/thoracic back in specific dermatome
  - Usually associated with blistering rash but pain can precede rash

While low back pain accounts for the majority of back pain syndromes, thoracic back pain is by no means rare. Fortunately, with few exceptions, low back pain and thoracic back pain share the same etiologies and therefore also share historical features that help formulate a differential. It should be noted that degenerative processes which commonly lead to lumbar pain are much less likely to occur in the thoracic spine, as opposed to fractures (especially compression fractures) which are relatively more common in the thoracic spine [6].

A final important aspect of the history for back pain is the possibility of opioid addiction. Some patients may be presenting with complaints of back pain as a way

to receive narcotic pain medications either for personal abuse or for resale illegally. Some prototypical warning signs of drug-seeking behavior are as follows [7]:

- Multiple visits for same complaint
- Inability to focus on anything other than the medicine
- Lost/stolen prescription
- "Doctor unavailable"
- No PCP contact
- "Allergic" to narcotic alternatives
  - NSAIDs
  - Muscle relaxants
  - Neuropathic agents
- Requests specific narcotics
- "Unbearable" pain
- Overly creative requests
- Upset when asked clarifying questions about pain
  - Location
  - Duration
  - Onset
- Night or weekend presentation

23.4 Physical Exam

For the emergency physician, the physical exam should focus on evaluating those conditions that may require urgent surgical consultation or systemic therapy (i.e., antibiotics).

- Vital sign assessment
  - Fever or hypothermia may suggest osteomyelitis, discitis, or spinal abscess.
  - Tachycardia and/or hypertension may correspond to the degree of pain.
    - Response may be muted if on beta-blocker therapy.
    - Patients in opiate withdrawal may present this way also.
- Musculoskeletal (MSK) and neurological exam [8]
  - Straight leg raise
    - Radicular pain in affected leg being raised at 60° or less suggests disc herniation.
  - Crossed straight leg raise
    - Radicular pain in affected leg while when contralateral (i.e., non-affected) leg is raised, it suggests disc herniation

  - Range of movement
    - Reduced flexion of the spine suggests ankylosing spondylitis.
    - Pain with prolonged extension of the spine (30 s) suggests spinal stenosis.
  - Palpation of the spine
    - Point tenderness over the spine suggests fracture, malignancy, or infection.
  - Strength testing
    - Weakness in the great toe, ankle, or quadriceps suggests disc herniation.
    - Diminished anal sphincter tone can be seen with cauda equine syndrome.
  - Reflexes
    - Hyporeflexia at Achilles or patellar tendons suggests nerve root involvement such as with disc herniation or spinal stenosis.
    - Hyperreflexia suggests spinal cord involvement such as with metastatic disease, infection, or fracture.
    - Positive Babinski test, lower extremity spasticity, or the presence of clonus indicates possible spinal cord involvement.
  - Sensation
    - Diminished sensation especially along a dermatome suggests nerve root impingement.
  - Gait assessment
    - Inability to heel walk suggests L5 nerve involvement.
    - Inability to toe walk suggests S1 nerve involvement.
- Skin exam
  - Blistering rash in dermatomal distribution suggests shingles.
- Abdominal exam
  - Palpation
    - Tenderness in the epigastrium or RUQ may suggest pancreatitis or cholecystitis, respectively.
    - Large pulsatile mass in periumbilical area suggests abdominal aortic aneurysm.
- Psychiatric evaluation
  - Assess for depression and/or anxiety
    - May indicate somatization of back pain

23.5 Diagnostic Workup

In general, acute low back pain and/or radiculopathy that does not present with red flag features (see section entitled "History and Differential Diagnosis") is a benign condition that will resolve with conservative measures and does not require imaging studies [9]. In fact, early imaging (before 6 weeks) has been shown to increase healthcare costs without providing any clear evidence of clinical benefit. Furthermore routine imaging in these situations has the potential to produce harm by exposure to radiation and/or additional unnecessary interventions [10]. The following tables from the American College of Radiology provide clinical scenarios that warrant imaging and specify which imaging tests are most appropriate for each scenario:

Scenario 1 Acute, subacute, or chronic uncomplicated low back pain or radiculopathy. One or more of the following: low-velocity trauma, osteoporosis, elderly individual, or chronic steroid use

Radiologic procedure	Rating[a]	Comments
X-ray lumbar spine	7	Initial study of choice especially in setting of osteoporosis or osteoporosis risk factors
CT lumbar spine without contrast	7	If suspicion for fracture is high and X-ray is negative
MRI lumbar spine without contrast	7	Can evaluate for ligamentous injury or in setting of worsening neuro deficit
Tc-99m bone scan with SPECT spine	3	Can uncover radiographically occult fractures
CT lumbar spine with contrast	3	
CT lumbar spine with/without contrast	1	
X-ray myelography and post-myelography CT lumbar spine	1	
X-ray discography and post-discography		
CT lumbar spine	1	

[a]Rating scale: 1, 2, and 3 usually not appropriate; 4, 5, and 6 may be appropriate; 7, 8, and 9 usually appropriate

Scenario 2 Acute, subacute, or chronic low back pain or radiculopathy. One or more of the following: suspicion of cancer, infection, or immunosuppression

Radiologic procedure	Rating[a]	Comments
MRI lumbar spine with/without contrast	8	Contrast helps for cancer patients suspected of epidural or intraspinal disease
MRI lumbar spine without contrast	7	Sufficient if low risk of epidural or intraspinal disease
CT lumbar spine with contrast	6	If MRI is contraindicated
CT lumbar spine without contrast	6	If MRI is contraindicated

Radiologic procedure	Rating[a]	Comments
X-ray lumbar spine	5	
Tc-99m bone scan with SPECT spine	4	Helps with anatomic localization if widespread metastasis is suspected
FDG-PET/CT whole body	4	Can help distinguish benign from malignant compression fractures but MRI is better
CT lumbar spine with/without contrast	3	If MRI is contraindicated
X-ray myelography and post-myelography CT lumbar spine	3	If MRI is contraindicated can be useful for anatomic localization and problem solving

[a]Rating scale: 1, 2, and 3 usually not appropriate; 4, 5, and 6 may be appropriate; 7, 8, and 9 usually appropriate

Scenario 3 Acute, subacute, or chronic low back pain or radiculopathy. Surgery or intervention candidate with persistent or progressive symptoms during or following 6 weeks of conservative management

Radiologic procedure	Rating[a]	Comments
MRI lumbar spine without contrast	8	
CT lumbar spine with contrast	5	If MRI is contraindicated
CT lumbar spine without contrast	5	If MRI is contraindicated
MRI lumbar spine with/without contrast	5	If MRI without contrast is nondiagnostic or if history of prior lumbar surgery
X-ray lumbar spine	4	Usually not sufficient by itself but can help with surgical planning
Tc-99m bone scan with SPECT spine	4	Useful for facet arthropathy or stress fracture
X-ray discography and post-discography		
CT lumbar spine	3	Controversial, but may be helpful in patients with pain >3 months

[a]Rating scale: 1, 2, and 3 usually not appropriate; 4, 5, and 6 may be appropriate; 7, 8, and 9 usually appropriate

Scenario 4 Low back pain or radiculopathy. New or progressing symptoms or clinical findings with history of prior lumbar surgery

Radiologic procedure	Rating[a]	Comments
MRI lumbar spine with/without contrast	8	Can differentiate disc from scar
CT lumbar spine with contrast	6	Useful in postfusion patients or when MRI is contraindicated or nondiagnostic
CT lumbar spine without contrast	6	Useful in postfusion patients or when MRI is contraindicated or nondiagnostic

Radiologic procedure	Rating[a]	Comments
MRI lumbar spine without contrast	6	Contrast is usually necessary
X-ray myelography and post-myelography CT lumbar spine	5	
X-ray lumbar spine	5	Flexion and extension views can be helpful
Tc-99m bone scan with SPECT spine	5	Helps detect and localize painful pseudarthrosis
X-ray discography and post-discography		
CT lumbar spine	5	
CT lumbar spine with/without contrast	3	

[a]Rating scale: 1, 2, and 3 usually not appropriate; 4, 5, and 6 may be appropriate; 7, 8, and 9 usually appropriate

Scenario 5 Low back pain with suspected cauda equine syndrome or rapidly progressive neurologic deficit

Radiologic procedure	Rating[a]	Comments
MRI lumbar spine without contrast	9	Use of contrast depends on clinical circumstances
MRI lumbar spine with/without contrast	8	Use of contrast depends on clinical circumstances
X-ray myelography and post-myelography CT lumbar spine	6	If MRI is contraindicated or nondiagnostic
CT lumbar spine with contrast	5	
CT lumbar spine without contrast	5	
X-ray lumbar spine	3	
CT lumbar spine with/without contrast	3	
Tc-99m bone scan with SPECT spine	2	

[a]Rating scale: 1, 2, and 3 usually not appropriate; 4, 5, and 6 may be appropriate; 7, 8, and 9 usually appropriate

23.6 Introduction to Treatment

While developing a comprehensive pain management strategy in an ED setting is likely to be unrealistic, the proper use of counseling and the prescribing of appropriate short-term pain medications is an important aspect of patient care and can help eliminate repeat visits to the ED. The treatment of complicated back pain due to serious underlying etiologies requires appropriate subspecialty consultation and is beyond the scope of this chapter; however the majority of back complaints do not fall into this category. The following section provides guidance on both non-pharmacologic and pharmacologic approaches to treatment:

- Patient education [4]
  - Reassure patients that acute back pain/radiculopathy is very common.
  - Fifty to 75 % of the time acute back pain/radiculopathy resolves spontaneously by 4 weeks.
  - Ninety percent of the time acute back pack pain/radiculopathy resolves spontaneously by 6 weeks.
  - Majority of patients with acute back pain/radiculopathy do not require surgery even when there is an acute disc herniation present.
- Activity level
  - Avoid bed rest and return to regular activities as tolerated as soon as possible.
    - Patients who return to normal activity have less pain and better functional recovery [11].
- Monitored exercise program
  - Evidence indicates only modest improvements in patients with chronic rather than acute back pain [12].
- Other physical modalities
  - Lack of evidence supporting electrical nerve stimulation, traction, ultrasound, interferential therapy, or short-wave diathermy – although all are considered safe.
  - Superficial heat application has some benefit in acute low back pain [13].
- NSAIDs [14]
  - Examples include ibuprofen, naproxen, meloxicam, ketorolac, and diclofenac.
  - Effective for short-term relief of acute and chronic low back pain.
  - No specific NSAID is clearly superior to another.
  - Cox-2 inhibitors have fewer GI side effects but should be used cautiously in patients with cardiovascular disease.
  - Use all NSAIDs cautiously in patients with history of renal impairment or peptic ulcer disease.
  - Common side effects include GI upset and rash.
- Acetaminophen
  - As effective as NSAIDs for acute low back pain, perhaps less effective in chronic low back pain [14]
  - Can be used as adjunctive therapy along with NSAIDs
  - Less renal and GI toxicity than NSAIDs
  - Caution in hepatic impairment

- Skeletal muscle relaxants [15]
  - Examples include cyclobenzaprine, tizanidine, methocarbamol, and baclofen.
  - Considered as a second-line treatment or as an adjunctive to NSAIDs or acetaminophen.
  - Evidence does support their effectiveness in nonspecific acute low back pain over placebo.
  - No specific muscle relaxant is clearly superior to another.
  - Work best within 1–2 weeks of presentation.
  - Use with caution in the elderly due to increased risk of falls.
  - Common side effects include sedation and dry mouth.
- Opioids
  - Examples include hydrocodone, oxycodone, morphine, and codeine.
  - No clear evidence of benefit over NSAIDs or acetaminophen in acute back pain [16].
  - Should be reserved for patients with severe acute back pain who have failed first- or second-line treatments.
  - Only short courses should be prescribed in the acute setting.
  - May have some benefit for chronic back pain in select populations.
  - Common side effects include sedation, constipation, nausea, and addiction.
- Anticonvulsants
  - Examples include gabapentin, pregabalin, and carbamazepine.
  - Some evidence of benefits for patients with acute or chronic radiculopathy [16].
  - Common side effects include sedation and dizziness.
- Corticosteroids
  - Examples include prednisone and solumedrol.
  - No evidence of any benefit in acute or chronic back pain [16].
  - Extensive side effect profile.

References

1. Murray CJ, Lopez AD. Measuring the global burden of disease. N Engl J Med. 2013; 369(5):448–57.
2. Centers for Disease Control and Prevention. National Ambulatory Medical Care Survey: 2010 summary tables. Atlanta: Centers for Disease Control and Prevention; 2011. Accessed at www.cdc.gov/nchs/data/ahcd/namcs_summary/2010_namcs_web_tables.pdf on 28 Dec 2015.
3. Martin BI, Deyo RA, Mirza SK, Turner JA, Comstock BA, Hollingworth W, et al. Expenditures and health status among adults with back and neck problems. JAMA. 2008;299:656–64.
4. Chou R, Cotton D, Rao JK, Taichman D, Williams S. In the clinic: low back pain. Ann Intern Med. 2014;160(11):ITC6. 2-14.
5. Kinkade S. Evaluation and treatment of acute low back pain. Am Fam Physician. 2007; 75(8):1181–8.

6. Ombrect L. Disorders of the thoracic spine: pathology and treatment. In: A system of orthopedic medicine. 3rd ed., Elsevier, Churchill Livingstone;2013. p. E169–84.
7. Vukmir RB. Drug seeking behavior. Am J Drug Alcohol Abuse. 2004;30(3):551–75.
8. Bickley LS, Szilagyi PG, Bates B. The musculoskeletal system. In: Bates' guide to physical examination and history taking. 8th ed. Philadelphia: Lippincott Williams & Wilkins; 2003. p. 520–22.
9. American College of Radiology. ACR appropriateness criteria. Low back pain. Reston: American College of Radiology; 2015. Accessed at https://acsearch.acr.org/docs/69483/Narrative/ on 5 Dec 2015.
10. Chou R, Qaseem A, Owens DK, Shekelle P. Diagnostic imaging for low back pain: advice for high-value health care from the American College of Physicians. Ann Intern Med. 2011;154(3):181–9.
11. Hagen KB, Hilde G, Jamtvedt G, Winnem M. Bed rest for acute low-back pain and sciatica. Cochrane Database Syst Rev. 2004;4:CD001254.
12. Hayden JA, van Tulder MW, Malmivaara AV, Koes BW. Metaanalysis: exercise therapy for nonspecific low back pain. Ann Intern Med. 2005;142:765–75.
13. Chou R, Huffman LH, American Pain Society. Nonpharmacologic therapies for acute and chronic low back pain: a review of the evidence for an American Pain Society/American College of Physicians clinical practice guideline. Ann Intern Med. 2007;147:492–504.
14. Roelofs PD, Deyo RA, Koes BW, Scholten RJ, van Tulder MW. Nonsteroidal antiinflammatory drugs for low back pain. Cochrane Database Syst Rev. 2008;23(1):CD000396.
15. van Tulder MW, Touray T, Furlan AD, Solway S, Bouter LM. Muscle relaxants for non-specific low back pain: a systemic review within the framework of the cochrane collaboration. Spine; 28(17), 1978-1992.
16. Chou R, Huffman LH, American Pain Society. Medications for acute and chronic low back pain: a review of the evidence for an American Pain Society/American College of Physicians clinical practice guideline. Ann Intern Med. 2007;147:505–14.

Chapter 24
Skin Problems

Gail A. Knight and Cheri N. Adgerson

24.1 Introduction

In 2010, 4.2 % of all emergency department (ED) visits were for rash or itching of which the differential diagnosis can be quite broad. Diagnosis and management are primarily based on history and physical exam. Laboratory and imaging workup can oftentimes be quite limited particularly in the ED as the diagnostic gold standard for most rashes or rashes of unclear etiology is skin biopsy. Indeed, this is a malady that is not going to be solved by the shotgun approach.

24.2 Differential Diagnosis

The differential diagnosis of skin conditions which may present to the emergency department is quite broad and may be stratified. This text will serve as a primer in more common acute and chronic conditions as well as the initiation of treatment from a primary care standpoint.

G.A. Knight, MD (✉)
Emergency Medicine, UF Health Shands Hospital, Gainesville, FL 32610, USA
e-mail: gferek@ufl.edu

C.N. Adgerson, MD
Dermatology, UF Health Shands Hospital, Gainesville, FL 32606, USA
e-mail: cadgerson@dermatology.med.ufl.edu

B. Desai, A. Desai (eds.), *Primary Care for Emergency Physicians*,
DOI 10.1007/978-3-319-44360-7_24

24.2.1 Dry Skin (Xerosis)

- Presentation
  - Intense itching particularly of anterolateral legs (shins) and can range in severity, affects the back, flanks, and abdomen, and usually spares the axilla, groin, and scalp
  - Mild to more severe changes: faint reticulate pinkness or fine cracks to deep redness and cracking
- Risk factors
  - Increased risk with age and more common in the elderly as is due to abnormal keratin production and lower amount of skin fatty acids
  - Worsened with cold, dry weather
- Diagnosis
  - Physical exam
- Treatment
  - Risk factor modification: humidity, indoors; avoid rough clothing and synthetic fibers, and avoid vasodilators if found to worsen itching (caffeine, alcohol, hot water).
  - Topical management: avoid topical anesthetic and antihistamines including topical corticosteroids for brief course. Apply menthol/camphor lotions, oatmeal baths, and pramoxine (PramaGel, Prax, Pramosone).
  - Oral antipruritic therapy.
    ASA, doxepin, antihistamines, and sedating vs nonsedating (hydroxyzine vs cetirizine)

24.2.2 Atopic Dermatitis

- Presentation
  - "The itch that rashes" with scratching
  - Red and edematous crusted exudates, scaling commonly on the face particularly the cheeks, scalp, extensor surfaces of arms and legs, and trunk in infants and toddlers
  - Older children and adults commonly present with rash to flexor surfaces of wrists, ankles, warm or sweaty fossae (antecubital, popliteal), hands, and anogenital region

- Usual onset within the first 24 months of life; 90 % of patients are diagnosed by age 5-years-old.

– Appearance varies depending on chronicity

24.2.3 Types

- Acute atopic dermatitis – characterized by weeping, crusted lesions with associated vesicles
- Subacute atopic dermatitis – dry and scaly red plaques and papules
- Chronic atopic dermatitis – lichenification of skin, related to chronic scratching resulting in thick, scaly localized plaques often associated with hyperpigmentation or hypopigmentation
  - Risk factors
    - Family history
    - Associated conditions: allergic rhinitis and asthma
    - Worsening with rough clothing; chemical irritants; emotional stress; foods such as cow's milk, eggs, soy, wheat, fish, tree nuts, and peanuts; dust and molds; and cat dander
  - Diagnosis
    - Biopsy if uncertain or symptoms are refractory to treatment (particularly if disturbing sleeping, school or work function)
  - Management
    - General care
      - Same as xerosis, avoidance of environmental allergens and treat superimposed infection.
      - Dietary changes (elimination of common food antigens for short period for improvement) though controversial.
    - Medication
      - Topical steroids
        - Limited use for exacerbation; treat all palpable areas.
        - Ointment preferred to lotion as it moistens very dry skin and less likely for ointment base to act as allergen.
        - Mild flare: 3–4-day use.
        - Moderate flare: taper over 2 weeks.

  - Severe flare: high-potency topical steroids; avoid oral steroids in order to prevent rebound.
  - Hydrocortisone (0.5 %, 1 %, 2.5 %) for the face or groin.
  - Triamcinolone acetonide 0.1 % for the trunk and extremities.
- More treatments

  http://www.fpnotebook.com/Derm/Dry/AtpcDrmts.htm

24.2.4 Contact Dermatitis

- Most common dermatologic diagnosis
- Presentation
  - Elderly and very young are more affected though it occurs in all demographics.
  - Atopy represents an independent risk factor.

24.2.4.1 Irritant Contact Dermatitis

- Sharply demarcated area of marked erythema (EM) and burning pain on exposed skin often followed by pruritus with onset of symptoms minutes to hours after exposure.
- Often associated with pustular lesions (more common here than in allergic type).
- Numerous known irritants: strong and weak acids such as acids of vinegar, heavy metals, wet cement, rubbing alcohol, nail polish remover, and soaps.
- Remove offending stimulus and contaminated clothing, prolonged irrigation with water.
  - Risk factors
    - Exposure to specific agents
  - Diagnosis
    - Clinical
  - Treatment
    - Removal of offending agent
    - Antihistamines for itching
    - Topical corticosteroids (hydrocortisone) for small skin areas
    - Consideration of oral steroids for more severe reactions

24.2.5 Seborrheic Dermatitis

- Abnormal epithelial function leads to redness and scaling with component of fungal overgrowth when *Malassezia* species release enzymes that cause local skin inflammation and scaling and underlying red patches.
- Presentation
  - Itchy, oily, and scaling rash to the scalp and face particularly the nasolabial fold, midface, and eyebrows
  - Can have chest involvement and breast folds
  - May also be associated with blepharitis, otitis externa, and acne vulgaris
- Risk factors
  - Immunocompromised state (malignancy, AIDS)
  - Cold, dry environments
  - Sun exposure
  - Emotional stress
  - Also associated with stroke patients, epilepsy, and Parkinsonism and nutritional deficiency
- Diagnosis
  - Biopsy when unclear diagnosis
- Treatment:

Scalp

- Initial therapy: over-the-counter shampoo, coal tar-based shampoo, or selenium sulfide shampoo
- Antifungal shampoo (ketoconazole 2 % or ciclopirox 1 %)
- Clobetasol 0.05 % shampoo for moderate to severe cases (high-potency steroid)
- Betamethasone valerate 0.12 % foam or fluocinolone 0.01 % shampoo (medium-potency steroid)

Face and Body

- First line
- Antifungal against the inflammation-provoking *Malassezia* growth
  - Considered a maintenance medication
    - Ketoconazole 2 % cream or gel or foam
    - Ciclopirox 0.77 % gel or 1 % cream
    - Sertaconazole 2 % cream

- Topical corticosteroids and calcineurin inhibitors
  - Acute exacerbations and thus intended for short-term use
- Low-potency topical steroid
  - Hydrocortisone 1 % ointment or cream
  - Desonide 0.05 % foam, gel, lotion, cream, and ointment
  - Fluocinolone 0.01 % cream, solution, and oil
- Topical calcineurin inhibitors
  - Tacrolimus 0.1 % ointment
  - Pimecrolimus 1 % cream

24.3 Parasitic Infections

24.3.1 Scabies

- Resultant of mite bite and infestation and transmission by direct contact including fomite exposure
- Presentation
  - Severe and intense itching particularly worse nocturnally leading to very small red papule followed by vesicle and even pustule formation
  - Pathognomonic burrow occurs in 10–20 % of cases; these are short, wavy gray lines on the surface of the skin most easily seen in webspaces and flexion points (wrist and elbows).
  - Commonly involves the trunk, genitalia, gluteal crease, and areola of the breast
- Risk factors
  - Crowded spaces (shelters, nursing homes)
  - Poor hygiene or nutritional status
  - Young children
  - Homelessness
  - Dementia
  - Sexually transmitted diseases
- Diagnosis
  - Burrow ink test (BIT) – burrow ink test involves coloring the burrow with a washable marker; wash area and look for a marker to penetrate burrow and thus make them more evident.
  - Burrow scraping – apply mineral oil, scrape burrow on its long axis with #15 blade placed on a slide, and view under low-power microscope for mites, eggs, etc.

- Treatment
  - Consists of environmental and local infection control, as well as symptomatic management.
  - Be aware that pruritus can continue 2–6 weeks posttreatment; this improves as skin sloughs.
- Environmental control
  - Wash all clothing, bedding, etc. in hot water (at least 140 F).
  - Those which cannot be washed should be sealed in a plastic bag for 2 weeks.
- Infection control
  - First line: permethrin 5 % cream
  - Apply to the neck down including the perineum and crevices in adults. In children and the immunocompromised, also apply to the face and head.
  - Wash off after 8–14 h followed by another application in 1 week.
  - Second line: ivermectin 200 mcg/kg oral agent given once followed by repeat dose in 14 days
    - Used in patients who cannot apply permethrin cream or those refractory to first-line agent
  - Tertiary treatments
    - Eurax 10 % cream (can be used in infants and during pregnancy or lactation)
    - Precipitated sulfur 6 % in petroleum (no safety data available)
- Symptomatic control
  - See pruritus management.

24.3.2 Bedbugs

- Results from a hypersensitivity reaction due to a bite from parasite of Cimicidae family
- Releases an anesthetic in its saliva thus preventing host awareness of bite
- Presentation
  - Moderate to severe itching that starts as red papules with central clearing but can progress in later reactions to wheals and vesicles
  - Bites are in linear pattern, distribution to the face and neck, arms and legs, and back; spares popliteal fossa and axilla
- Risk factors and common offenders
  - Transmission via travel pieces like luggage, clothing, shoes, seams of furniture, bedding, and curtains

- Diagnosis
  - No specific testing.
  - Consider detection system such as carbon dioxide-emitting trap (parasite attracted to CO2), moat device, and dogs trained to detect bedbugs.
  - Examine seams of furniture, bedding, and curtains with magnifying glass for bedbug debris and exoskeletons.
- Treatment
  - Alternative: heat item >120 F for at least 30 min to kill bedbugs or cool to <23 F for 5 days or ≤15 F (this is instantly lethal to them).
  - Call an exterminator (many pesticides work, silica gel dust for safer alternative but should be handled by a professional or professional cleaning service for commercial grade steam clean).
- Management of bites
  - Avoid secondary infection such as cellulitis or impetigo by managing itching and excoriations.
  - See xerosis management as there is no specific treatment for these bites.

24.3.3 Pediculosis (Lice)

- Parasite that lives in clothing or bedding seams and transmitted by direct head-to-head contact or fomites.
- Itching results from allergic reaction to louse saliva.
- Body lice are associated with rickettsial infections and typhus.
  - Common head lice are not.
- Symptoms are commonly regional depending on whether head, body, or genital lice.
- Nits survive <2 days without host.
- Presentation
  - Itching associated with red papules, macules, and wheal.
  - Itching is localized such as in the scalp and behind the ears in head lice or diffuse.
- Risk factors
- Crowded living conditions with poor hygiene
- Children aged 3–11 years of age
- Brunette or red-haired people > blonde or black haired
- Diagnosis
  - Use magnifying glass to visualize lice (sesame seed-sized parasite).

  - Usually found <1 cm from the scalp often accompanied by lice eggs, i.e., nits, which are white grain-looking pieces attached to the hair shaft.

- Treatment
- Infection control
  - First line: permethrin 1 % (Nix) or pyrethrins (Rid) – over-the-counter shampoo followed by repeat treatment in 9–10 days
  - Second line: used in resistant cases. Permethrin 5 %, malathion 0.5 % (but must use lice comb), natroba 0.9 % (expensive and best result with nit combing), and Sklice
  - Third line: used if refractory to initial treatments. Lindane 1 % recommended for adults >50 kg only due to neurotoxicity, risk of seizures
- Environmental control
  - There are no proven treatment strategies; however, recommendations exist.
  - Machine wash all possible such as clothing and hats in hot water 122°F followed by hot dryer.
    - Unwashables are sealed in plastic bags for 2 weeks.
  - Combs and brushes are soaked in 130°F water for 15 min.
  - Treat household contacts that share bedding.
  - May return to school or work after treatment
    - Nits are not a contraindication to return to activities.

24.4 Fungal Disorders

24.4.1 Cutaneous Candidiasis

(a) Presentation

- Redness, pruritus, burning, and scaling in affected area, may be associated with foul odor.
- Look for secondary infection; consider superimposed bacterial infection or dermatophyte.
- Intertrigo: within skinfold such as axillae, inframammary folds, groin, and toe webspaces. Protracted rash can lead to fissured, macerated tissues. Candida is the most common offender; however, it can be caused by various bacteria.

(b) Risk factors

- Immunodeficiency, pregnancy, diabetes mellitus, geriatric population, and oral antibiotic use

(c) Diagnosis

- Depends on physical exam or if etiology is unclear particularly if refractory to initial antifungals
- KOH prep

(d) Treatment

- General care
  - Apply drying agent to decrease moisture in skinfolds.
  - Barrier ointments (petroleum jelly or zinc oxide) and agents to eliminate friction.
  - Wear light, breathable clothing.
- Medication
  - First line:
    - Clotrimazole, econazole, and oxiconazole (imidazole class). Cover *Candida* species and other fungal species. See Dermatophyte section.
    - Nystatin: useful against *Candida* species only.
  - Second line:
    - Broad-spectrum topical antifungals
    - Naftifine, terbinafine, ciclopirox, and butenafine
  - Third line:
    - Oral antifungals
    - Fluconazole 100–200 mg daily × 7 days

24.4.2 Dermatophytoses

(a) Presentation

- Depends on location of rash which is caused by *Trichophyton tonsurans* (>90 % cases), remainder by *Microsporum* species
- Tinea capitis: itching in setting of boggy, irritated lesion with fine scales and areas of well-circumscribed alopecia and posterior cervical adenopathy

(b) Risk factors or associations

- Low socioeconomic groups

(c) Diagnosis

- Clinical: If three or more criteria are met, commence empirical treatment.
  - Alopecia, scalp scaling, scalp pruritus, and occipital adenopathy

- KOH prep
- Hair fungal culture

(d) Treatment

- Clotrimazole, econazole, and oxiconazole (imidazole class).
- Azoles and allylamines may also be used.
- Tinea capitis must be treated orally with griseofulvin or terbinafine.

24.5 Disorders of Pigmentation

24.5.1 Melasma

- Pigmentation disorder of multiple etiologies is commonly associated with pregnancy, hyperthyroidism, liver disease, oral contraceptive use, and phototoxic reaction to certain medications (phenytoin).
- More common in females than males
  - Presentation
    - Flat brown hyperpigmented patches usually of symmetric distribution to the cheeks, forehead, and upper lip
  - Risk factors
    - Darkening with sun exposure
  - Diagnosis
    - No formal testing and clinical diagnosis
  - Treatment
    - Prevention
      - Avoid sun exposure or use high-SPF sunscreen in pregnancy-related melasma.
      - Pregnancy- and OCP-related melasma commonly improve spontaneously months after delivery or medication use, respectively.
    - Medication
      - Hydroquinone bleaching creams – require repeat use, should be used with sunscreen, can cause hypopigmentation particularly in darker-skinned individuals, and should not be used longer than 4 months at a time due to the risk of ochronosis. Examples: hydroquinone 2–4 % or tretinoin 0.05 %
      - Keratolytics – augments the use of bleaching creams to reduce pigment over months. Examples: tretinoin 0.055–0.1 % cream and adapalene 0.1–0.3 % gel
      - Chemical peel – performed by dermatologist. Example: glycolic acid peel

24.5.2 Vitiligo

- Autoimmune melanocyte destruction

(a) Presentation

- Well-defined white macules 0.5–5 cm
- Localized, segmental, generalized variants
- Commonly found on the face, around the eyes, neck, intertriginous folds and axillae, dorsal aspect of the hands, umbilicus, genitalia, and anus

(b) Risk factors

- Thirty percent of patients have a family history.

(c) Diagnosis

- Clinical diagnosis

(d) Treatment

- Medication management

– Topical steroids, for example, betamethasone 0.05 % ointment
– Anti-inflammatory topicals: tacrolimus 0.1 % ointment
– Concealers
– Depigmentation therapy – permanent depigmentation with monobenzone 20 % cream which usually requires 6–18 months to complete and indicated for patients with >40 % body surface area affected
– Surgical grafting for localized lesions
– Avoidance of sun exposure and strict use of screens to prevent skin cancers

24.6 Rheumatologic-Related Rashes

24.6.1 Erythema Nodosum

- Inflammation of subcutaneous fat related to cutaneous type IV delayed hypersensitivity reaction
- Multitude of possible causes though estimated 55 % cases are idiopathic

– Postinfectious (streptococcal pharyngitis, mycoplasma, coccidiomycosis, chlamydia)
– Drug reaction (sulfonamides, amoxicillin, OCPs, gold)
– Pregnancy
– Granulomatous disease (TB, sarcoidosis, IBS)

(a) Presentation

- Viral syndrome-type prodrome followed by rash 1–3 weeks later. Rash consists of painful, red lesions often on bilateral lower extremities. These nodules are warm and very tender and range in size 1–10 cm diameter and are quite dynamic in their course, changing from firm to fluctuant, followed by involution over a couple of weeks. Healing lesions often appear bruised. Affected areas do not scar or ulcerate.
- Bilateral pretibial areas are most common; however it also forms on extensor surface of the forearm, thighs, and trunk.

(b) Risk factors

(c) Diagnosis

- Clinical diagnosis, though in setting of atypical presentations, do skin biopsy
- Consideration for labs (CBC, ESR, CRP, PPD, ASO titer, stool studies if indicated) or imaging such as CXR

(d) Treatment

- Anti-inflammatories, NSAIDs
- Support stockings and leg elevation
- Oral steroids if no contraindication such as active infection on malignancy, for example, prednisone taper

24.7 Ulcerative-Type Rash

24.7.1 Pyoderma Gangrenosum

- Rare skin condition resultant from neutrophil release of degradative enzymes resulting in skin interruption

(a) Presentation

- Variegated in appearance ranging from ulcers to pustular lesions usually on the legs, buttocks, face, and abdomen related to minor skin trauma.
- Painful.
- Lesion later ulcerates with pustular or hemorrhagic base followed by thin scar formation.

(b) Risk factors

- Associated with Behcet's disease and Sweet's syndrome

(c) Diagnosis

- Clinical diagnosis

- Pathergy test: intentionally inducing minor skin trauma such as subcutaneous injection which is (+) if results in ulceration or pustule formation within 24–48 h

(d) Treatment

- Biopsy to rule out infection and malignancy
- Wound care
- Immunosuppressants in severe cases

24.8 Vesiculobullous Disorders

24.8.1 Dermatitis Herpetiformis

- Chronic condition may last for years; however, approximately one-third of patients can have remission spontaneously.

(a) Presentation

- Collection of grouped lesions of various appearances including red papules, vesicles, bullae, and wheals usually occurring in distribution of elbows and knees, buttocks, and shoulders; it rarely involves mucous membranes or palms and soles.
- Rash is preceded by sense of burning and itching.

(b) Risk factors and associations

- Gluten-sensitive enteropathy – rash is thought to represent untreated celiacs thus a complication.
- Various autoimmune disorders including systemic lupus erythematosus, Sjogren's syndrome, vitiligo, type I diabetes, and thyroid disorders.

(c) Diagnosis

- Biopsy with immunofluorescence.
- Consider CBC with differential for eosinophilia.
- Autoimmune titers for antinuclear antibody or antithyroid microsomal antibody.

(d) Treatment

- Medications: dapsone is first line; monitor CBC for hemolytic anemia (G6PD deficiency) or sulfapyridine.
- Dietary modification: gluten-free diet though improvement in symptoms was delayed by 6–12 months.

24.8.2 Bullous Pemphigoid

- Autoimmune skin condition with onset later in life, 60–80-years-old.
- Characterized by antibodies that attack subepidermal layer.
- Course is often self-limited.
- Rarely life-threatening.

(a) Presentation

- Starts as itchy urticarial-like exanthema primarily of the lower legs, groin, and flexor forearms but can also affect the abdomen. Lesion goes on to become large bullae over weeks to months containing clear or blood-containing fluid.
- Healing lesion does not form a scar.
- Rare mucous membrane involvement.

(b) Risk factors and association

- Advanced age
- Medications, i.e., Lasix

(c) Diagnosis

- Biopsy with immunofluorescence

(d) Treatment

- First-line therapy:
  - Oral prednisone
  - Steroid-sparing agent such as azathioprine, mycophenolate, and tetracycline that is then continued as prednisone is tapered.
  - Topical corticosteroids if localized lesion.
- Second-line therapy:
  - Methotrexate – in patients unable to tolerate prednisone
  - Tetracycline antibiotics and nicotinamide

24.8.3 Herpes Simplex Virus

- Classic presentation is grouped vesicles on an erythematous base.
- Diagnosis can be confirmed by viral culture, Tzanck smear, and DFA.
- Primary infections usually have a prodrome of fever, malaise, and +/− LAD.
  - Genital infections may be preceded by dysuria and cervicitis.

- Treatment
  - Prevention and treatment of secondary infections (i.e., staph)
  - Acyclovir
    - Primary: 400 mg PO TID for 10 days
    - Recurrence: 400 mg PO TID for 5 days

24.8.4 Erythema Multiforme

- An acute, self-limited hypersensitivity reaction to infections (HSV, mycoplasma) or drugs.
- Primary lesion is targetoid papule; atypical lesions and vesicles/bullae may occur.
- EM minor vs EM major
  - Involvement of mucosal surfaces
    - Ocular, oral, and genital erythema and/or ulcerations
  - Presence of systemic symptoms

24.8.5 Stevens-Johnson Syndrome and Toxic Epidermal Necrolysis

- A rare, acute, and life-threatening mucocutaneous reaction, most commonly drug related
  - Common etiology: sulfonamides, penicillins, allopurinol, carbamazepine, lamotrigine, phenytoin, barbiturates, NSAIDs, and antiretrovirals
- Prodromal symptoms include high fever, moderate to severe skin pain, anxiety, and asthenia.
- Skin tenderness precedes dusky erythema and progresses to flaccid bullae and large sheets of desquamation.
- Oral, ocular, and genital mucosa may show erythema, ulceration, and crusting.
- Treatment
  - Identification and cessation of causative agent
  - Supportive care
    - Monitoring of fluids and electrolytes
    - Temperature regulation
    - Prevention of infection
    - Dressings
  - +/− IVIg

24.9 Acneiform Rash

24.9.1 Acne Rosacea

- Also known as rosacea
- Associated with various ocular maladies, conjunctivitis, blepharitis, keratitis, corneal scarring, and eyelid inflammation

(a) Presentation

- Various subtypes of acneiform-type exanthema, four types and granulomatous variant
- Erythematotelangiectatic type: redness and telangiectasias to midface with flushing and facial pain with or without ophthalmologic involvement
- Papulopustular type: midface redness with pustular component
- Phymatous: characterized by thickened, irregular, and nodular skin to the nose, forehead, eyelids, chin, and ears due to hyperplasia of sebaceous glands

(b) Risk factors and triggers

- Sun exposure, heat, hot baths, and hot drinks
- Emotional stress
- Exercise
- Caffeine
- Alcohol use

(c) Diagnosis

- Clinical diagnosis. Based on the presence of central face dermatitis and at least one of four other qualities
- Transient flushing
- Telangiectasias
- Papule and pustule formation
- Persistent erythema

(d) Treatment

- Topical metronidazole
- Oral tetracyclines

24.9.2 Acne Vulgaris

- Retained sebum (fats and waxes) followed by obstruction of follicle outlet leads to comedone formation. This is a setup for bacterial growth of obstruction followed then by an inflammatory reaction leading to papules, nodules, and cysts.
- Affects the face, neck, chest, and back.

- Affects estimated 90 % of adolescents. Estimated that 5 % women and 1 % of men aged 40 will have acne.

(a) Presentation

- Obstructive acne
  - Black heads (open comedones) – combination of sebum, keratin, and melanin, rarely become inflamed.
  - White heads (closed comedones) – are closed comedones that result in blocked pilosebaceous ducts followed by inflammatory lesions; they are prime for anaerobic bacterial growth.
- Inflammatory acne
  - Progression of lesions does not necessitate going through all stages however.
  - Papules→ pustules→ nodules → cysts → scars

(b) Risk factors and associations

- More common in males than females during adolescence

(c) Diagnosis

- Clinical diagnosis
- Features determine severity ranging from comedonal, mild (comedones and papulopustular lesions), moderate (more papulopustular lesions than in mild), and nodulocystic (comedones, inflammatory lesions, nodules associated with scarring) acne.
- Send culture of wound in refractory cases.
- Send total and free testosterone, LH, and FSH levels in females with dysmenorrhea or hirsutism.

(d) Treatment

- General care
- Avoidance of triggers such as medications
  - Corticosteroids and androgenic steroids
  - OCPs
  - Phenytoin
  - Isoniazid
  - Lithium
- Oil-based cosmetics
- Mechanical stress (chin straps, ear guards, helmet)
- Emotional stress
- Environmental factors
  - Grease-laden places (fast-food cook, oil field, mechanic shop)

General Care

- Avoid squeezing lesions.
- Wash the face only one to two times daily.
- Change to water-based, oil-free cosmetics.
- Medication options.

Topical Medications

- Benzoyl peroxide 2.5 %, 5 %, and 10 % wash (over the counter). Recommended to start with lower strength as this is less drying but is as effective as more concentrated formulation. Use in the morning.
- Acne wash such as Cetaphil

Nighttime Comedolytic Therapy

- First line: topical tretinoin 0.025 %, 0.05 %, and 0.1 % or adapalene 0.1 % or Epiduo (adapalene with benzoyl peroxide) can cause redness.

Antibiotic Therapy

- Topical therapy
  Indicated for papular and pustular acne (mild to moderate)
- Morning
  - Clindamycin (Cleocin T)
  - BenzaClin (combination of clindamycin and benzoyl peroxide)
  - Erythromycin lotion 1.5 and 2 % (resistance common)
  - Tetracycline HCl lotion 2.2 mg/ml (resistance common)
  - Sulfur acne lotion 10% or sulfur 5 % with sodium sulfacetamide 10 %
  - If intolerant of above medications
- Nighttime
  - Comedolytic as above or if refractory, consider tazarotene 0.05 % gel or cream. Pregnancy category X.
- Oral therapy
- Indicated for moderate to severe acne in addition to above therapies
  - First line:
    - Doxycycline 50–100 mg once or twice daily
    - Tetracycline 250–500 mg once or twice daily
    - Erythromycin 1 g/day in divided doses
    - Bactrim DS twice daily if first line is not tolerated (caution SJS/TEN)
  - Second line:
    - Minocycline 50–200 mg/day in divided doses

- Isotretinoin
  - Indicated in refractory or cystic acne
  - Restricted medication

Special Cases

- For women currently taking OCPs, increase the estrogen component to at least 50 ug and decrease the progestin component (androgenic steroid).
- Consider starting spironolactone 50–200 mg tablet daily in women with hormonal-type acne or androgen hypersecretion syndromes.

Chapter 25
Insomnia

Bobby Desai and Alpa Desai

25.1 Introduction

Insomnia is the most common sleep problem reported worldwide. It is estimated that 25–35 % of adults have transient insomnia, while 10–15 % of adults suffer from chronic insomnia [1]. Most patients who suffer from insomnia may not seek medical treatment for this condition, especially in the emergency setting. However, patients with insomnia have been found to have more emergency department visits, laboratory tests ordered, and overall an increase in prescription medication use than those patients without insomnia [2]. Patients that present to the emergency department may have an underlying emergent pathology that potentially may require investigation.

Patients complaining of insomnia may experience a myriad of complaints, including difficulty falling asleep as well as maintaining sleep. They may also complain of sleep that is inadequate and nonrefreshing and may complain of waking up too early [3].

The distinction between acute and chronic insomnia is of particular importance to the emergency physician (EP). Acute insomnia of a few days' duration may be of a medical etiology (including medications), an acute stress reaction, environmental disturbances, as well as conditions that do not allow for the normal circadian rhythm to function such as jet lag or night shift workers [4].

Chronic insomnia may be defined as insomnia of greater than 3 weeks duration and may arise from a combination of factors, including chronic medical etiologies, psychiatric conditions, and drugs and alcohol.

B. Desai, MD, Med (✉)
Emergency Medicine, University of Florida, Gainesville, FL 32610, USA
e-mail: bdesai@ufl.edu

A. Desai, DO
Community Health and Family Medicine, University of Florida, Newberry, FL 32669, USA
e-mail: desaas@shands.ufl.edu

B. Desai, A. Desai (eds.), *Primary Care for Emergency Physicians*,
DOI 10.1007/978-3-319-44360-7_25

Careful assessment of this condition is imperative for the emergency physician as misidentification of insomnia as a "nonemergency" may lead to harmful interventions, including inappropriate medication prescriptions such as a benzodiazepine for those patients who have an underlying sleep apnea.

25.2 Risk Factors for Insomnia

- Family history of insomnia
- Female sex
  - Increasing prevalence in the years subsequent to menopause
- Employment as a shift worker
- Employment status in general
- Increasing age
  - Peaks in middle age (45–54 years)
  - Decreases when slightly older (65–85 years)
  - Increases again at ≥85 years

25.3 Differential Diagnosis

Common complaints and red flags of acute or transient insomnia:

- Acute pain – etiology needs to be carefully determined.
  - Control of pain may be therapeutic.
- Significant life stress – consideration of depression and suicidal ideation.
  - Any exacerbation of a psychiatric condition will need emergency department (ED) evaluation.
- Change in circadian rhythm.
  - Jet lag
  - Night shift workers
- Acute ingestion of drugs or alcohol.
  - Sequelae of overdose may need ED evaluation.
  - Withdrawal may cause insomnia as well.
- Acute medical condition.
  - Head and neck complaints
    - Viral syndrome

        - Upper respiratory complaints
    - Constitutional complaints
        - Fever
        - Night sweats
    - Cardiovascular complaints
        - Angina
        - Chest pain of any etiology
            - Pleurisy
    - Pulmonary complaints
        - Shortness of breath
            - Asthma
            - COPD
    - Gastrointestinal complaints
        - Vomiting and/or diarrhea
        - Abdominal pain
    - Genitourinary complaints
        - Urinary tract infection
- Acute exacerbation of a chronic medical condition
    - Neurologic
        - Seizures
        - Sleep-related movement disorders
            - Periodic leg movements
                - Common in those over 65
            - Restless legs syndrome
                - Uncomfortable or painful sensation of the legs that presents at rest and is alleviated by movement.
                    - Renal failure with uremia may underlie restless legs syndrome.
                    - Iron or folate deficiency may occasionally underlie restless legs syndrome.
    - Head and neck
        - Allergic rhinitis
    - Cardiovascular
        - Congestive heart failure

    - May be associated with paroxysmal nocturnal dyspnea which can be mistaken for insomnia
  - Pulmonary
    - Asthma
    - COPD
  - Gastrointestinal
    - Peptic ulcer disease
    - Gastroesophageal reflux disease
  - Genitourinary
    - Benign prostatic hypertrophy
  - Rheumatologic
    - Fibromyalgia
    - Arthritis
      - Osteoarthritis
      - Gout

  Common complaints and red flags of chronic insomnia [(*) = common]

- Chronic medical disorders
  - Asthma
  - Diabetes mellitus
    - Polyuria
  - Hypertension
  - Congestive heart failure
  - Nocturnal seizures
  - Thyroid disorders
    - Hyperthyroidism
  - Sleep apnea (*)
    - Consider in obesity or history of snoring
  - Nocturia (may be secondary to congestive heart failure)
  - Nocturnal enuresis
  - Dementia
    - These patients may be restless during the day causing wakefulness during the night.
  - Restless legs syndrome
- Normal physiology

  - Pregnancy
  - Elderly patients
- Psychiatric conditions
  - Mania
    - May be seen as a component of bipolar disorder.
    - Insomnia may be marked just before and after this phase.
  - Anxiety (*)
    - May have significant difficulty falling asleep
    - May have concomitant complaints
  - Schizophrenia
  - Depression (*)
    - These patients have a delayed onset of sleep with frequent periods of wakefulness, classically during the early morning.
  - Suicidal ideation
  - ADHD
- Ingestants
  - Caffeine use (*)
  - Alcohol use (*)
  - Over-the-counter medications
    - Diet pills, which may include pseudoephredrine and phenylpropanolamine
  - Nicotine
    - Patches
    - E-cigarettes
    - Cigarettes and cigars
- Withdrawal syndromes, specifically of intoxicants, medications, or drugs
  - Alcohol
  - Caffeine
  - β-blockers
  - Antidepressants
  - Sedative-hypnotics
  - Sympathomimetics
- Chronic use of therapeutic medications
  - β-blockers
  - β-agonists
  - Calcium channel blockers

- Decongestants
- Anticholinergics
- Thyroid hormones

25.4 History

When evaluating a patient with this complain, the emergency physician must be cognizant of the fact that insomnia can be defined as both a symptom and a syndrome [5]. Thus, the length of time insomnia has been occurring may be important when constructing a differential diagnosis and ultimate workup if needed.

Important historical features to elicit include:

- Duration of insomnia
  - Acute vs. chronic
- Presence of potential secondary factors (correlate with past medical history) [* = red flags]
  - Acute medical complaints (or acute exacerbations of chronic medical conditions)
    - Headache (*)
    - Shortness of breath (*)
    - Chest pain (*)
    - Signs and symptoms of diabetes mellitus (*)
      - Polyuria/polydipsia
    - Abdominal pain or discomfort (*)
    - Musculoskeletal pain or other musculoskeletal issue
      - Restless legs syndrome
    - Frequent awakenings
- Use of medications or drugs (including caffeine and alcohol)
  - Pitfall: Not inquiring about over-the-counter medications which may contain stimulants
- Psychiatric history
  - Exacerbations of chronic conditions
    - Depression
      - May occur especially in the elderly
    - Anxiety
    - Presence of suicidal thoughts (*)

- Acute psychosis
  - May be elicited from family members

25.5 Physical Examination

The physical examination should focus on potential emergency conditions for the reported insomnia.

- Vital sign assessment
  - The presence of fever or hypothermia should alert the physician to a possible infectious etiology.
  - Tachycardia may have multiple etiologies including:
    - An appropriate response to fever
    - The potential use of medications or drugs, including over-the-counter medications and illicit substances
  - Pulse oximetry
    - May alert the clinician to the presence of an underlying pulmonary pathology
- Neurologic assessment should focus on:
  - Level of alertness
  - Symmetry of motor and sensory examination
- Head, eye, ear, nose, and throat examination
  - Note evidence of trauma
  - Pupillary size and reactivity
    - May be important in evaluation for withdrawal or acute ingestions
- Cardiovascular and pulmonary examination
  - Specifically for acute conditions including angina, flash pulmonary edema, or exacerbations of asthma or COPD
- Gastrointestinal examination
  - Presence or absence of abdominal pain, vomiting, or diarrhea
- Psychiatric evaluation
  - Assessment for:
    - Depression screen
    - Suicidal thoughts
    - Anxiety or panic states

25.6 Introduction to Treatment

After evaluation and stabilization of emergency conditions, the emergency physician has several medication options to treat patients. These include:

1. Education
   (a) Educating patients on the types of behavior that disrupt sleep is important. Noting that activities other than sleeping (e.g., reading, eating, watching television, etc.) in their bed may disrupt their sleep cycle and thus should be avoided.
   (b) Stabilization of the sleep-wake cycle is also important.
      (i) Wake up at the same time every day.
      (ii) Minimize daytime napping.
2. Medication management introduction
   (a) Considerations for medication management include:
      (i) Using the lowest effective dose
      (ii) The use of the medication on an intermittent basis
      (iii) Prescription of enough medication for a short-term basis only
      (iv) Warning patient of the possibility of *rebound insomnia* following discontinuation
      (v) Consideration of chronic medical conditions that may increase the side effects of the prescribed medication – specifically the sedative side effects

25.7 Pharmacologic Treatment

While there are many medications that are FDA approved for insomnia, the emergency physician may not necessary need to have specific knowledge of each of the different medications in each class, but the EP should understand that each class of medications will have different indications for insomnia.

25.7.1 Hypnotics

The primary indication for the use of hypnotics is for the short-term management of insomnia, since prolonged use may lead to tolerance and dependence. These medications are useful for immediate symptom relief, and they have been shown to be effective in both the induction of sleep and the maintenance of sleep [6].

Side effects of these medications include anterograde amnesia and residual daytime sleepiness. These medications should be cautiously given to those patients

with an underlying cardiac or pulmonary condition. The dose provided is the single best predictor of the risk of side effects. Due to the risk of daytime sleepiness, short-acting medications are preferred.

FDA-approved sedative-hypnotics
Flurazepam
Quazepam
Triazolam
Estazolam
Temazepam

Pitfalls

1. Prescribing for pregnant patients.
2. Prescribing for poorly treated patients with sleep apnea.
3. Prescribing for patients with history of substance abuse.
4. Patients with underlying renal, hepatic, cardiac, or pulmonary disease have a higher risk of side effects and must be monitored more closely.
   (a) Includes respiratory depression in those predisposed
5. Withdrawal effects may present within a few hours of discontinuing a short-acting medication.
   (a) Symptoms include anxiety, depression, rebound insomnia, and nausea.

25.7.2 Antidepressants

These typically can be prescribed for those patients with insomnia that is associated with a psychiatric disorder [7]. Furthermore, in those patients with a history of substance abuse, consideration may be given to prescribing a short course of this class of medication, in consultation with the patient's primary care physician or psychiatrist, though it is important to remember that there is limited evidence for the effectiveness of treating insomnia in depressed patients [8].

Some antidepressants used for treatment of insomnia
Amitriptyline
Trazodone
Mirtazapine

Pitfalls

1. Have anticholinergic effects, beware of overdose
2. Have the effect of increased morning sedation
3. May have cardiac toxicity in overdose

4. May have sexual dysfunction
5. May worsen restless legs syndrome

25.7.3 Antihistamines

This class of medications antagonize H1 receptors and thus have sedative effects; in fact many over-the-counter agents include an antihistamine as an active agent. Routine use should be discouraged due to their minimal effectiveness in inducing sleep. They additionally cause a reduction in the quality of sleep and may cause excessive tiredness [9]. Furthermore, tolerance to these medications has been noted [10].

Antihistamines
Diphenhydramine
Doxylamine
Hydroxyzine

Pitfalls
1. Have anticholinergic effects, beware of overdose
2. May cause CNS depression

25.7.4 Melatonin

Melatonin is a hormone involved in sleep regulation that is produced by the pineal gland. It is typically used for insomnia produced by circadian rhythm changes, such as in shift workers or jet lag [11]. It is FDA approved for circadian rhythm sleep disorder in blind adults and children.

Pitfalls
1. High doses can actually disrupt sleep and cause headaches, fatigue, and dizziness.

25.7.5 Nonbenzodiazepine Hypnotics

These medications bind to specific benzodiazepine I receptors and typically have a less overall risk of adverse effects when compared with benzodiazepines. Of note, these medications are metabolized in the liver, and care should be given in prescribing to those patients with hepatic disease.

FDA-approved nonbenzodiazepine medications
Zolpidem
Zaleplon
Eszopiclone

Pitfalls

1. Care should be given to prescribing these medications to patients with underlying liver disease.

References

1. Benca RM. Diagnosis and treatment of chronic insomnia: a review. Psychiatr Serv. 2014;6(3):332–43.
2. Rosekind MR, Gregory KB. Insomnia risks and costs: health, safety, and quality of life. Am J Manag Care. 2010;16(8):617–26.
3. Kupfer DJ, Reynolds CF. Management of insomnia. N Engl J Med. 1997;336(5):341–6.
4. Rajput V, Bromley SM. Chronic insomnia: a practical review. Am Fam Physician. 1999;60(5):1431–8.
5. Walsh JK, Benca RM, Bonnet M, Buysse DJ, Hauri P, Kiley JP, Monjan A, Morin CM, Ricca JG, Rogus S, Roth T, Simon RD. Insomnia: assessment and management in primary care. Am Fam Physician. 1999; 59(11):3029–38.
6. Ramakrishnan K, Scheid DC. Treatment options for insomnia. Am Fam Physician. 2007;76(4):517–26.
7. Clark MS, Smith PO, Jamieson B. Antidepressants for the treatment of insomnia in patients with depression. Clinical Inquiries 2011 (MU).
8. Qaseem A, Snow V, Denberg TD, Forciea MA, Owens DK, Clinical Efficacy Assessment Subcommittee of American College of Physicians. Using second-generation antidepressants to treat depressive disorders: a clinical practice guideline from the American College of Physicians [published correction appears in Ann Intern Med. 2009;150(2):148]. Ann Intern Med. 2008;149(10):725–33.
9. NIH state-of-the-science conference statement on manifestations and management of chronic insomnia in adults. NIH Consens State Sci Statements 2005;22:1–30. http://consensus.nih.gov/2005/2005InsomniaSOS026main.htm. Accessed 20 Mar 2007.
10. Balter MB, Uhlenhuth EH. New epidemiologic findings about insomnia and its treatment. J Clin Psychiatry. 1992;53(suppl):34–9.
11. Buscemi N, Vandermeer B, Pandya R, Hooton N, Tjosvold L, Hartling L, et al. Melatonin for treatment of sleep disorders. Evid Rep Technol Assess (Summ). 2004;108:1–7.

Chapter 26
Anxiety

Kyle M. Iketani and Brandon R. Allen

26.1 Introduction

Generalized anxiety disorder (GAD) and panic disorder have a 12-month prevalence in US adults aging 18–64 of 2.9 % and a lifetime prevalence of 7.7 % and 4.6 % in women and men, respectively [1]. GAD can be difficult to diagnose even in the primary care setting. The setting of the emergency department lends itself to an increased difficulty of diagnosis due to symptom presentations that mirror GADs, including headache, abdominal pain, and shortness of breath. Diagnostic criteria for GAD as defined by the American Psychiatric Association in DSM-5 can be found in Table 26.1.

It is important to note that the diagnosis of GAD does not apply when the clinical picture can better be attributed to another diagnosis or when the clinical picture can be attributed to a medical condition, substance use, or substance withdrawal [1].

People with GAD have excessive anxiety about typical, everyday situations [1]. These patients may have a range of complaints including, but not limited to, sleep disturbances, restlessness, muscular tension, gastrointestinal complaints, and chronic headaches.

Panic disorder is defined as recurrent, unexpected panic attacks for more than 1 month in association with at least one of the following symptoms: persistent concern about having additional attacks, worry about the attacks' implications and/or consequences, or a significant change in behavior as a result of the attacks [4]. At least one comorbid axis I disorder, such as major depressive disorder or another

K.M. Iketani, MD
Emergency Medicine, University of Florida – Shands, Gainesville, FL 32610, USA

B.R. Allen, MD (✉)
Emergency Medicine, UF Health, Gainesville, FL, USA
e-mail: brandonrallen@ufl.edu

B. Desai, A. Desai (eds.), *Primary Care for Emergency Physicians*,
DOI 10.1007/978-3-319-44360-7_26

Table 26.1 DSM-5: generalized anxiety disorder criteria

(a) Excessive anxiety and worry (apprehensive expectation), occurring more days than not for at least 6 months, about a number of events or activities (such as work or school performance)
(b) The person finds it difficult to control the worry
(c) The anxiety and worry are associated with three (or more) of the following six symptoms (with at least some symptoms present for more days than not for the past 6 months). Note: Only one item is required in children
1. Restlessness or feeling keyed up or on edge
2. Being easily fatigued
3. Difficulty concentrating or mind going blank
4. With irritability
5. Muscle tension
6. Sleep disturbance (difficulty falling or staying asleep or restless unsatisfying sleep)
(d) The focus of the anxiety and worry is not confined to features of an axis I disorder, e.g., the anxiety or worry is not about having a panic attack (as in panic disorder), being embarrassed in public (as in social phobia), being contaminated (as in obsessive-compulsive disorder), being away from home or close relatives (as in separation anxiety disorder), gaining weight (as in anorexia nervosa), having multiple physical complaints (as in somatization), or having a serious illness (as in hypochondriasis), and the anxiety and worry do not occur exclusively during post-traumatic stress disorder
(e) The anxiety, worry, or physical symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning
(f) The disturbance is not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition (e.g., hyperthyroidism) and does not occur exclusively during a mood disorder, a psychotic disorder, or a pervasive developmental disorder

anxiety disorder, is commonly associated with panic disorder [5]. In addition, panic disorder is associated with low education and relatively low probability of working full time [5].

It is thought that anxiety is often a learned response to noxious or aversive stimuli [2]. Patients suffering from an anxiety disorder are more likely to have other medical issues, have longer hospital stays, undergo more procedures, incur higher health-care costs, suffer from failure at school or work, have lower-paying jobs, and are more often financially dependent on welfare [2]. In addition, anxiety disorders are the most prevalent mental disorder among American children and adolescents [3].

Identification of generalized anxiety disorder is of special importance to clinicians in both the emergent and non-emergent settings, because misdiagnosis can lead to costly, invasive, and/or harmful unnecessary medical testing.

26.2 Risk Factors for Anxiety

- A genetic component plays a role in anxiety disorders.

  - The greatest association is in panic disorder, generalized anxiety disorder, and phobias.

- Having involved, controlling, and rejecting parents
- Female
- Low education
- Low socioeconomic status
- Social disability
- Family history of anxiety disorders
- Introverted personality in early childhood
- Traumatic life events
- Sleep disturbances
- Poor physical health

26.3 Differential Diagnosis

Anxiety is a diagnosis of exclusion. A thorough medical evaluation is needed to both rule out and alleviate the patient's concern for serious medical conditions.

- Medication side effects
  - Beta agonists
  - Caffeine
  - Digoxin toxicity
  - Levodopa
  - Nicotinic acid
  - SSRIs
  - Steroids
  - Stimulants
  - Theophylline
  - Synthetic thyroid hormone
- Cardiovascular
  - Acute coronary syndrome
  - Congestive heart failure
  - Mitral valve prolapse
  - Dysrhythmias
  - Syncope
  - Hypertension
- Pulmonary
  - Asthma
  - Chronic obstructive pulmonary disease
  - Hyperventilation

  - Pneumonia
  - Pneumothorax
  - Pulmonary edema
  - Pulmonary embolus
- Endocrine
  - Hyperthyroidism
  - Hypothyroidism
  - Hyperadrenalism
- Gastrointestinal
  - Gastroesophageal reflux disease
  - Irritable bowel syndrome
  - Colitis
    - Typically of a chronic nature
  - Ulcers
- Neoplastic
  - Carcinoid syndrome
  - Pheochromocytoma
  - Insulinoma
- Neurologic
  - Parkinsonism
  - Encephalopathy
  - Restless leg syndrome
  - Seizure
  - Vertigo
  - Brain tumor
- Psychiatric
  - Affective disorders
  - Depression
  - Drug abuse
  - Drug dependence
  - Drug withdrawal
    - Both illicit and prescribed
- Other conditions
- Anaphylaxis
- Anemia
- Typically chronic

- Electrolyte abnormalities
- Occurring over time
- Porphyria
- Menopause

26.4 Types of Anxiety Disorders [2]

- Specific phobia
  - 18.4 % prevalence
  - Fear or anxiety about a specific object or situation (i.e., swimming in the ocean)
- Social phobia
  - 13.0 % prevalence
  - Fear or anxiety about one or more social situations where the individual is exposed to possible scrutiny by others
- Post-traumatic stress disorder
  - 10.1 % prevalence
  - Intrusive thoughts and flashbacks of traumatic events, avoidance of reminders, hypervigilance, and sleep disturbance leading to social and occupational dysfunction
- Generalized anxiety disorder
  - 9.0 % prevalence
  - At least 6 months of persistent and excessive anxiety and worry occur on most days, with difficulty controlling the worry
- Separation anxiety
  - 8.7 % prevalence
  - Excessive anxiety or fear concerning separation from those whom the individual is attached to
- Panic disorder
  - 6.8 % prevalence.
  - An abrupt surge of intense fear or discomfort that reaches a peak within minutes and can be recurrent and unexpected.
  - Dizziness, light-headedness, tingling sensations, and fear of dying are common.
- Agoraphobia

  - 3.7 % prevalence
  - Fear or anxiety about using public transportation, being in open or enclosed spaces, being in a crowd, or being outside alone
- Obsessive-compulsive disorder
  - 2.7 % prevalence
  - Recurrent intrusive thoughts and urges as well as repetitive mental and behavioral acts/compulsions that the individual feels driven to perform, causing anxiety and distress

26.5 History

The history plays a large part in the diagnosis of anxiety disorders. It is important to understand the timeline of worry and/or fear and elicit underlying causes for concern. Multiple negative work-ups for a concerning issue may indicate underlying anxiety. The Beck Anxiety Inventory (BAI) is a 21-question multiple choice self-reported inventory, designed for adolescents to the elderly, that helps measure a patient's severity of anxiety [6]. The test can be used to establish a baseline level of anxiety as well as to better understand posttreatment outcomes. Unfortunately, the inventory does not discriminate well between anxiety and depression [7].

26.6 Physical Exam

Physical exam may yield little in helping diagnose anxiety disorders, as there are many overlapping symptoms in signs with other disease states:

- CNS/PNS
  - Dizziness
  - Paresthesia
  - Light-headedness
  - Sweating
    - Clammy hands
- Cardiovascular
  - Palpitations
  - Tachycardia
  - Flushing
  - Pallor
  - Hot and cold spells

- Respiratory
  - Hyperventilation
  - SOB
  - Chest constriction/tightness
- Gastrointestinal
  - Dry mouth
  - Diarrhea
  - Upset stomach
  - Feelings of a lump in the throat
  - Nausea
  - Vomiting
- Urinary
  - Frequent urination
- Musculoskeletal
  - Muscle tightness/spasm
  - Back pain
  - Tremors
  - Headache
  - Weakness
  - Fatigue
  - Restlessness

26.7 Labs and Imaging

Laboratory tests are typically only useful in ruling out other medical conditions. A complete blood count, electrolyte panel (including glucose, creatinine, and calcium), liver panel, urine drug screen, and thyroid function tests may be useful in certain circumstances. Imaging is only necessary to interrogate lab abnormalities and rule out organ disease. Imaging may help lessen patient concerns of physical pathology.

26.8 Introduction to Treatment

There are several modalities aimed at treating anxiety disorders. These include education, psychotherapy, and pharmacotherapy. Combination therapy is often needed; however, any necessary treatment in addition to the acute phase warrants psychiatric consultation. A careful history should help to elucidate the cause of a patient's

anxiety. Any reversible causes should be treated accordingly. Medical conditions (i.e., hyperthyroidism) and medication side effects (i.e., theophylline toxicity) should be treated initially to see if anxiety resolves:

1. Education:
   (a) Patients should be counseled that anxiety is real and that they are not "crazy."
   (b) Providers should make patients aware of the different treatment modalities and refer them accordingly to appropriate services such as psychiatry, psychology, and other counseling resources.
2. Psychotherapy
   (a) Behavioral therapy and cognitive behavioral therapy have been demonstrated to be the most efficacious types of psychotherapy.
   (b) The outcome of treatment is determined by:
      (i) Severity of anxiety
      (ii) Level of functioning before symptoms onset
      (iii) Motivation for treatment
      (iv) Support system
      (v) Ability to comply with pharmacotherapy and psychotherapy
   (c) Cognitive behavioral therapy has been shown to be as successful as medication in generalized anxiety disorder.
3. Pharmacotherapy
   (a) Considerations for medication management include:
      (i) Using the lowest effective dose
      (ii) Intermittent vs long-term pharmacotherapy
      (iii) Quantity of medication being prescribed, especially out of the emergency department
      (iv) Warning patient of medication side effects and interactions

26.9 Pharmacologic Treatment

When considering pharmacologic agents, providers should choose based on the type of anxiety they are treating and whether it is acute vs chronic while understanding the side effects of different medications.

26.9.1 Antidepressant Agents

The drugs of choice in the treatment of anxiety disorders. Newer agents such as SSRIs have a safer adverse event profile and are easier to use than older agents like TCAs and MAOIs [8]. The American Psychiatric Association recommends SSRIs for panic disorder:

(a) SSRIs

SSRI	Pitfall(s)
Fluoxetine	May initially cause anxiety
Paroxetine	Partially sedating
Citalopram	QT prolongation
Escitalopram	Fewer drug interactions
Sertraline	Decreased libido, failure to ejaculate

(b) Alpha-2 antagonist

Alpha-2 antagonist	Pitfall(s)
Mirtazapine	Sedating, increased appetite, and weight gain

26.9.2 Benzodiazepines

These medications are effective anxiolytics for acute anxiety. They are fast in onset and can provide immediate relief. It is acceptable for an emergency physician to prescribe a short course of benzodiazepines upon discharge until the patient can be seen by a psychiatrist.

Selected benzodiazepines
Alprazolam
Clonazepam
Lorazepam

Pitfalls

1. These drugs should not be used for chronic therapy due to their abuse potential, side effect profile, and danger during withdrawal.
2. Side effects may include:
   (a) Anterograde amnesia
   (b) Daytime sleepiness
   (c) Decreased respiratory drive
3. The sudden stoppage of benzodiazepines can lead to:
   (a) Sleep disturbances
   (b) Irritability
   (c) Worsening anxiety
   (d) Panic attacks
   (e) Sweating
   (f) Confusion
   (g) Nausea

(h) Hallucinations
(i) Seizures
(j) Psychosis:

(i) All of these symptoms can be life-threatening.

4. Long-term benzodiazepine uses should be weaned rather than stopped suddenly.

References

1. Locke AB, Kirst N, Schultz CG. Diagnosis and management of generalized anxiety disorder and panic disorder in adults. Am Fam Physician. 2015;91(9):617–24.
2. Michels P, Steadman M. Anxiety disorders. In: Lebowitz H, editor. Current diagnosis & treatment: family medicine. 4th ed. New York: McGraw-Hill Education; 2015. p. 606–17.
3. Zarafshan H, Mohammadi M-R, Salmanian M. Prevalence of anxiety disorders among children and adolescents in Iran: a systematic review. Iran J Psychiatry. 2015;10(1):1–7. Pub med ID: 26005473.
4. Na HR, Kang EH, Lee JH, Yu BH. The genetic basis of panic disorder. J Korean Med Sci. 2011;26(6):701–10. http://dx.doi.org/10.3346/jkms.2011.26.6.701 PMCID: PMC3102861.
5. Tilli V, Suominem K, Karlsson H. Panic disorder in primary care: comorbid psychiatric disorders and their persistence. Scand J Prim Health Care. 2012;30(4):247–53. doi:10.3109/02813432.2012.732471. PMCID: PMC3520420.
6. Grant M. Beck anxiety inventory. http://webcache.googleusercontent.com/search?q=cache:F4qHbA5sI0MJ:www.manchester.gov.uk/download/downloads/id/19268/beck_anxiety_inventory_explanation.pdf+&cd=2&hl=en&ct=clnk&gl=us.
7. Muntingh AD, van der Feltz-Cornelis CM, van Marwijk HW, Spinhoven P, Penninx BW, van Balkom AJ. Is the beck anxiety inventory a good tool to assess the severity of anxiety? A primary care study in The Netherlands study of depression and anxiety (NESDA). BMC Fam Pract. 2011;12:66. doi:10.1186/1471-2296-12-66. PMID: PMC3224107.
8. Yates, W. Anxiety disorders. Medscape. November 3, 2015. http://emedicine.medscape.com/article/286227-overview. Accessed 14 Nov 2015

Chapter 27
Depression

J. Benjamin Barton and Brandon R. Allen

27.1 Introduction

Major depression affects more than 350 million people worldwide and is the leading cause of disability worldwide [1]. Mental health-related chief complaints make up about 3 % of the patients who present for care in the emergency setting, with depression being the most common mental health-related chief complaint [2]. It is estimated that up to 10 % of patients who present to primary care providers including the emergency department (ED) have major depression, and around half of these patients are undiagnosed [3].

It is well known that depressed patients may present with psychosomatic symptoms such as lower back pain or abdominal pain, but it is also important to know that depressed patients tend to have more medical comorbidities and are more likely to require admission to the hospital. More than 50 % of patients with major depression have at least one comorbid chronic medical condition, and depressed patients are more likely to suffer complications and present to the ED for these conditions [3]. Major depression is associated with numerous comorbidities and is associated with significantly increased rates of heart disease, diabetes, and stroke and with a decreased life expectancy [3–5].

While recognition of depression in the emergency department (ED) or an ambulatory care clinic can be challenging due to the large volume of patients and high acuity of patients being seen, it is nonetheless important. Recognition of depression

J.B. Barton, MD
Emergency Medicine, UFHealth Shands, Gainesville, FL 32608, USA
e-mail: johnbarton@ufl.edu

B.R. Allen, MD (✉)
Emergency Medicine, UF Health, Gainesville, FL 32610-0186, USA
e-mail: brandonrallen@ufl.edu

B. Desai, A. Desai (eds.), *Primary Care for Emergency Physicians*,
DOI 10.1007/978-3-319-44360-7_27

can save lives by prevention of suicide, improvement of quality of life for the patient and their family, as well as prevention of unnecessary health-care visits for somatic complaints related to their emotional problems [6].

27.2 Risk Factors for Depression

Lifetime prevalence of a major depressive episode is reported at around 15 % for the general population. Certain populations are known to be at an increased risk. Published risk factors for major depression include:

- Female gender
- Middle age (45–64 years old)
- Multiple medical comorbidities
- Assisted living or skilled nursing home residents
- Widowed, divorced, or never-married individuals
- Lesbian, gay, bisexual, or transgender sexual orientation

For emergency providers, the principal goal in identifying depression is to prevent depressed patients from harming themselves or others. The risk factors for depression and suicidality do not necessarily coincide. For example, females are more likely to be depressed, but males are more likely to succeed in committing suicide.

The following factors are known to be linked with greater risk of suicide and should be taken into consideration when deciding whether or not to admit a patient for depression/suicidality:

- Repeated suicide attempts
  - *Strongest* predictive factor of suicide risk! Especially if previous attempts were planned and lethal means were used
- Male gender
- Widowed, divorced, or separated individuals
- Relationship difficulties and conflicts
- Active unemployment
- Social isolation – lack of supportive family and friends
- Substance abuse
- Acute or chronic progressive illness
- No insight into condition
- Antisocial personality or disruptive behavior
- History of depression (unipolar or bipolar), schizophrenia, or panic disorder
- Family history of suicide
- Recent disciplinary action at school or work
- Lack of religious taboo against suicide
- Unstable or inappropriate affect

27.3 Diagnostic Criteria

The diagnosis of depression requires a total of five of the listed criteria below, but anhedonia or depressed mood must be one of the criteria:

- Depressed mood
- Anhedonia
- Significant weight change or appetite change (e.g., a change of more than 5 % of body weight in a month)
- Insomnia or hypersomnia
- Psychomotor agitation or retardation
- Fatigue or loss of energy nearly every day
- Feelings of worthlessness and excessive or inappropriate guilt nearly every day
- Diminished ability to think or concentrate, or indecisiveness, nearly every day
- Recurrent thoughts of death and/or suicide

The symptoms must cause significant distress or impairment and may not be due to the direct physiological effects of a substance or general medical condition [8].

An easy way to remember the symptoms described above is using the mnemonic SIG E CAPS:

- S – sleep disturbance
- I – interest loss (anhedonia)
- G – guilt
- E – energy loss
- C – concentration decreased
- A – appetite change
- P – psychomotor change
- S – suicide or death preoccupation

27.4 Differential Diagnosis

The differential diagnosis for major depression includes:

- Infection
  - HIV encephalopathy
  - Meningitis
  - Encephalitis
  - Lyme disease
- Substance abuse
  - Intoxication or withdrawal

- Medication side effects:
  - Steroids, antihypertensives, and hormone therapy are common causes.
- Trauma
- Intracranial mass
- CNS disorders
  - MS
  - Seizures
  - Alzheimer's
  - Microangiopathic lesions
- Metabolic disorders
  - Vitamin deficiencies – thiamine (B1), pyridoxine (B6), and cobalamin (B12)
- Endocrine disorders
  - Hypothyroidism
  - Cushing
  - Hyperparathyroidism
  - Addison disease
- Autoimmune disease
  - Systemic lupus erythematosus
- Obstructive sleep apnea
- Other psychiatric conditions
  - Bereavement
  - Bipolar depression
  - Schizoaffective disorder

27.5 History

Important clues from history known to increase a patient's likelihood of committing suicide include:

- Feelings of hopelessness, helplessness, and guilt.
- Few or weak reasons to live.
- Suicidal ideations that are frequent, intense, prolonged, and pervasive:
  - As opposed to ideations that are infrequent, transient, and low intensity
- Patient with an unambiguous or continuing wish to die:
  - As opposed to patients who had no previous wish to die or no ongoing wish to die after making an attempt

- Suicide plan is realistic.
- Patient has access to firearms.
- Planned suicide includes a situation where rescue is unlikely.

Depressed patients will likely present to the ED with other complaints, and many will have actual pathology, so a thorough history is essential.

27.6 Physical Exam

The diagnosis of depression is usually made with a thorough history, but physical examination of a depressed patient remains an important adjunct. A comprehensive physical exam will help in the investigation of somatic complaints and in evaluating other potentially more emergent issues that may present with the chief complaint of depression (i.e., ingestion, intoxication).

One vital aspect of the physical exam is the mental status exam which should include determination of:

- The patient's affect
- Appearance
- Orientation
- Attention
- Memory
- Speech pattern
- Level of consciousness
- Judgment
- Thought content

27.6.1 Red Flags of Depression

These physical exam red flags should alert you to consider more pressing diagnoses and consider medical/toxicologic explanations for a patient's depressed feelings:

- Abnormal vital signs
- Disorientation
- Clouded consciousness, comatose patients
- Focal neurologic deficits
- Recent memory loss, patient over 40 with no prior psychiatric disease
- Signs of trauma

Patients with any of these findings warrant further work-up. Helpful labs for these patients may include [7]:

- Complete blood count

- Serum electrolytes
- Creatinine
- Hepatic enzymes
- Thyroid-stimulating hormone level ± free T4
- Ethanol
- Urinalysis
- Pregnancy test
- Arterial blood gas
- Cerebrospinal fluid examination
- Electrocardiogram
- Computed tomography or magnetic resonance imaging of the brain
- Salicylate and acetaminophen levels in suicidal patients

27.7 Treatment

After properly recognizing a patient with depression in the ED or in the clinic, it can be challenging to provide proper treatment and disposition. An important first step is to stratify patients as high, moderate, or low risk for immediate harm to self and others.

High-risk patients are those who:

- Exhibit violent behaviors
- Exhibit agitation
- Endorse active suicidal or homicidal intent
- Require physical restraints
- Present after engaging in a possibly lethal suicide attempt (i.e., gunshot wound to the head or reported ingestion)

These patients will require admission to a psychiatric facility for inpatient psychiatric therapy, but only after medical clearance.

The following important steps should also be taken for all patients who are in the high-risk category:

- Disrobe, gown, and search the patient for potentially dangerous items.
- Determine if the patient needs to be detained for emergency evaluation.
- Treat medical conditions.
- Communication with the patient should be made while keeping the exit accessible for the provider, while avoiding excessive eye contact, and while using a nonthreatening voice.
- Enforce acceptable limits of behavior with the patient.

Any patient who is significantly agitated (shouting, cursing, threatening, physically aggressive, those attempting to elope) or who is noncompliant with the established limits may require sedation and/or restraints. There is potential for harm to

the patient with either sedation or restraints, so an effort should be made to de-escalate the situation verbally. If this is not successful, parenteral administration of sedation agents may be used. Benzodiazepines and antipsychotics (first and second generation) are considered first-line agents [9, 10]. The time to onset of sedation, dosing, and special considerations for benzodiazepines and antipsychotics are summarized in the table below.

27.7.1 Parenteral Options for Acute Agitation [9]

	Onset (min)	Initial dose (mg)	Considerations
Benzodiazepines			
Diazepam	30	5–10 IV	Avoid IM because of unpredictable absorption. Useful in the setting of alcohol withdrawal
Lorazepam	2–5 IV, 15–30 IM	1–2 IM/IV	–
Midazolam	120 IV, 240–300 IM	2.5–5 IM/IV	Higher risk of respiratory depression compared with lorazepam and diazepam
Antipsychotics			
Aripiprazole	60	9.75 IM	Relatively safe side effect profile
Haloperidol	1–2 IV, 30–60 IM	5 IM/IV	Risk for extrapyramidal symptoms. Higher risk of QT prolongation, particularly if given IV. IV haloperidol is an off-label route and requires careful monitoring for cardiac arrhythmias if used
Olanzapine	15–45	10 IM	Use with caution when given with benzodiazepines because of increased risk of cardiopulmonary depression
Ziprasidone	30–45	20 IM	Low risk of QT prolongation

Those patients who are stratified as low or moderate risk may not require admission for psychiatric treatment, but consultation with a psychiatrist is prudent especially for moderate-risk patients. Factors arguing for safe discharge of depressed patients include:

- Close follow-up with psychiatric consultant
- Good social support
  - Including someone willing to stay with the patient
- The absence of high-risk factors mentioned in the above risk factors and history sections

27.8 Other Treatment Modalities

Lifestyle changes, psychotherapy, and pharmacotherapy are all potentially effective treatment options for patients with a major depressive episode. Performance of psychotherapy is not appropriate for providers in the emergency setting because of the limited amount of time available. Patients who desire this treatment should be referred to providers who can provide these services. While emergency providers will not perform psychotherapy, it is important for them to know this is an effective option, particularly for patients with less severe depressive symptoms.

27.8.1 Lifestyle Changes

The following lifestyle changes have been shown to be efficacious in the treatment of depression:

- Exercise*
- Relaxation therapy*
- Change in diet away from calorie-rich and nutrition-poor foods
- Improved sleep hygiene
  - Including CPAP treatment for individuals with obstructive sleep apnea
- Decreased alcohol intake

* denotes lifestyle changes that have the best evidence of efficacy [11].

27.8.2 Pharmacotherapy

There is debate about whether patients with major depression should have pharmacologic therapy initiated in the emergency department. While over 60 % of prescriptions written for antidepressants are written by nonpsychiatrists [11], many emergency providers feel initiation of pharmacotherapy in the ED should only be carried out under the direction of a psychiatrist because of the significant risk of side effects, the long period of time before drugs become effective, and the lack of adequate follow-up.

However, there are some emergency providers who do not have access to an on-call psychiatric specialist and may want to initiate therapy in the emergency setting. When unsure about the need for initiation of pharmacotherapy, there are several decision aids which can be useful. One such aid is the PHQ-9 questionnaire which uses nine questions to come up with a score from 0 to 27 and gives the recommendation to initiate pharmacotherapy for any patient with a score equal to or greater than 15 [12].

When the decision is made to treat the patient, a second-generation antidepressant should be the first-line treatment. First-generation antidepressants (tricyclic antidepressants, monoamine oxidase inhibitors) and second-generation antidepressants (selective serotonin reuptake inhibitors, selective norepinephrine reuptake inhibitors, selective serotonin reuptake inhibitors, and atypical antidepressants) have been shown to be equally effective, but the side effect profile and overall safety relating to possible overdose make second-generation antidepressants a better option in most cases. Emergency providers should be familiar with these medications and their side effects because depressed patients may ingest these drugs during suicide attempts.

Classes of antidepressants		
Class	Mechanism of action	Important side effects
Tricyclic antidepressants (TCAs) – amitriptyline, clomipramine, desipramine, doxepin, imipramine, nortriptyline, selegiline, trimipramine	Block action of norepinephrine and serotonin at neural synapses	Long QT
	Has some antihistamine and antiadrenergic effects	Cardiotoxicity
		Arrhythmia
		Sedation
Monoamine oxidase inhibitors (MAOIs) – phenelzine, tranylcypromine	Inhibits degradation of monoamine oxidases which leads to increased levels of neurotransmitters in the neural synapses	Dietary interaction with tyramine-rich foods (aged cheese, wine) causes uncontrolled hypertension from adrenergic excess
		Insomnia
Selective serotonin reuptake inhibitors (SSRIs) – citalopram, escitalopram, fluoxetine, fluvoxamine, paroxetine, sertraline	Inhibits reuptake of serotonin, but not norepinephrine at the neural synapses	GI upset
		Sexual dysfunction
		Serotonin syndrome
		Increased suicidality in young adults aging 18–24 during initial treatment (generally the first 1–2 months)
Selective norepinephrine reuptake inhibitors (SNRIs) – desvenlafaxine, duloxetine, levomilnacipran, milnacipran, sibutramine, venlafaxine	Inhibits the reuptake of norepinephrine at the neural synapses	Same as SSRIs
		Increased blood pressure
		Anxiety
Atypical antidepressants – bupropion, mirtazapine, nefazodone, trazodone, vilazodone, vortioxetine	Variable effects on levels of dopamine, norepinephrine, and serotonin at neural synapses	Common effects include dry mouth, constipation, and light-headedness
		Trazodone – associated with priapism and arrhythmias
		Mirtazapine – arrhythmia
		Nefazodone – liver problems
		Bupropion – lowers seizure threshold

The side effect profiles of the individual drugs vary within the classes and can be tailored by the prescriber to meet the specific needs of patients. For example, patients with insomnia may benefit from trazodone as this medication causes increased somnolence. The following table contains other examples of important side effects of antidepressants.

Antidepressants and common side effects		
Side effect	Medications	Clinical pearl
Nausea and vomiting	Venlafaxine highest. Common in multiple antidepressants	Use extended-release formulation of venlafaxine to reduce nausea. Start at lower doses. Take with food
Diarrhea	Sertraline > paroxetine	Consider using in patients with constipation
Weight gain	TCAs/MAOIs, mirtazapine > paroxetine	Consider using in patients with anorexia or unintentional weight loss
Somnolence	Trazodone > mirtazapine	Use in patients with concurrent insomnia. Dose at night
Dizziness	Venlafaxine > sertraline, duloxetine	Consider bedtime dosing
Headache	Venlafaxine > bupropion, paroxetine, sertraline, escitalopram	
Sexual dysfunction	Sertraline > venlafaxine > citalopram > paroxetine	May require dose reduction or medication switch
	Bupropion and mirtazapine do not have this effect	
Insomnia	Bupropion > sertraline, fluoxetine, paroxetine, venlafaxine	Take in the morning
Smoking cessation	Bupropion superior	Bupropion proven to help patients quit smoking

Escitalopram and sertraline have been shown in multiple studies to be slightly more efficacious and easier tolerated than other second-generation antidepressants making them good first choices [13]. It is important to keep in mind the antidepressant effects of the medications often take several weeks, while the side effects may begin to manifest in only a few days. Side effects are the most common reason for discontinuation of pharmacotherapy, so patients need to be counseled about the common side effects prior to administration and the need to continue taking the medications if tolerable.

Pitfalls in treating depressed patients in the ED:

- Failure to ask about the patient's specific plan for committing suicide
  - Some physicians concerned asking the question may increase risk of later suicide – a sentiment which has been proven to be false [2].

- Patients who "only took a few pills" do not get a full work-up"
  - Do not believe patients when they tell you they only took a few pills or were only seeking attention – full work-ups are a necessity.
- Double-edged sword of somatic complaints
  - Do not ignore somatic complaints (chest pain, low back pain) as many depressed patients have true pathology.
  - But do not ignore the depression, and discharge suicidal patients after medical work-up.
- Failure to provide a sitter for depressed patients
  - Provide 1:1 sitters for all patients who may harm themselves or others during ED admission.
- Failure to diagnose major depression in patients with terminal conditions
  - Even patients with good reason to feel depressed should receive a full psychiatric and safety evaluation.

References

1. World Health Organization (WHO). Depression fact sheet N°369. Published October 2012. Retrieved from: http://www.who.int/mediacentre/factsheets/fs369/en/. Accessed Sept 2015.
2. Chang B. The depressed patient and suicidal patient in the emergency department: evidence-based management and treatment strategies. Emerg Med Pract. 2011;13(9):1–23.
3. Menear. The influence of comorbid chronic physical conditions on depression recognition in primary care: a systematic review. J Psychosom Res. 2015;78:304–13.
4. Katon WJ. Epidemiology and treatment of depression in patients with chronic medical illness. Dialogues Clin Neurosci. 2011;13:7–23.
5. Ford DE. Optimizing outcomes for patients with depression and chronic medical illnesses. Am J Med. 2008;121:S38–44.
6. Docherty JP. Barriers to the diagnosis of depression in primary care. J Clin Psychiatry. 1997;58:5–10.
7. Hoffman L. Emergency assessment and stabilization of behavioral disorders. In: Tintinalli's emergency medicine manual. 7th ed. Ch 185. McGraw-Hill; 2012. p. 910–13.
8. Diagnostic and statistical manual of mental disorders. 5th ed. (DSM-5). Washington, DC: American Psychiatric Association; 2013.
9. Deal N. Stabilization and management of the acutely agitated or psychotic patient. Emerg Med Clin North Am. 2015;33(4):739–52.
10. Gillies D. Benzodiazepines for psychosis-induced aggression or agitation. Cochrane Database Syst Rev. 2013;30:4.
11. Bentley SM. Major depression. Med Clin N Am. 2014;98(5):981–1005.
12. Kroenke K, Spitzer RL, Williams JBW. The PHQ-9: validity of a brief depression severity measure. J Gen Intern Med. 2001;16(9):606–13. doi:10.1046/j.1525-1497.2001.016009606.x.
13. Thronson LR. Psychopharmacology. Med Clin N Am. 2014;98(5):927–85.

Index

B. Desai, A. Desai (eds.), *Primary Care for Emergency Physicians*,
DOI 10.1007/978-3-319-44360-7