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Ear and Sinus Infections

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Patients with acute infectious diseases of the ear and the sinuses frequently present to an emergency department or urgent care center. The majority of patients with these complaints have uncomplicated infectious problems that will resolve with relatively straightforward treatment. It must be noted that not all patients with an earache and a draining ear have acute infectious otitis media nor do those who have headache and a runny nose necessarily have a bacterial infection in their sinuses. This chapter uses classic acute infectious otitis media and sinusitis as the basic clinical disease process to which others are compared and contrasted. This will provide the foundation from which the more complicated aspects of the infectious diseases of these organ systems may be recognized and treated. Table 1 is a summary of the entities discussed in this chapter.

EAR INFECTIONS

Infectious Otologic Clinical Syndromes

Ear Pain

Pain secondary to primary infections of the auricle, external canal, and middle ear is usually so severe that it frequently will be the chief complaint motivating a patient to seek medical care in an emergency setting. Examination of the ear usually will reveal the infectious problem causing the pain. These primary infections are discussed later in this chapter.

Lesions of the tonsil, the base of the tongue, the epiglottis, and the temporal mandibular joint commonly present as otalgia (Fig. 1). Infections in these areas rarely have ear pain as their sole presenting symptom but may have it

TABLE 1. *Acute infections with ear pain or sinusitis*

Infection	Signs and symptoms	Antibiotic	Dosage	Comment
Cellulitis/ perichondritis	Erythema, swelling, exposed cartilage	Dicloxacillin, erythromycin	Cephalexin 250–500 mg q8h PO for 10 days	External drainage of pus, follow-up 24–48 hours, IV antibiotics if no improvement
Acute otitis externa (bacterial)	Moisture-associated, erythema external canal, possible swelling and pus, pain on pinna traction	Broad-spectrum ototopical otic drops (cortisporin otic suspension)	2 gtt tid for 5–7 days	Use wick if canal is swollen shut
Fungal otitis externa	Less painful than bacterial otitis externa	Clotrimazole or n-cresyl acetate		Hyphae seen in canal
Malignant otitis externa	Immunocompromised, 3/5 signs (see text)	Ticarcillin or piperacillin + tobramycin, tobramycin + ceftazidime in penicillin allergy, ototopical drops	IV for 6–8 weeks	Admit
Myringitis granulosa	Sensation of hearing loss, occasional drainage, granular TM bumps, suppuration	None	—	—
Acute infectious otitis media	TM red, dull, bulging, nonmobile, perforation possible, fever	Amoxicillin	40 mg/kg/day ÷ tid PO for 10 days	14-day follow-up
		Erythromycin-sulfa	50/150 mg/kg qid,	
		Trimethoprim-sulfamethoxazole	8/40 mg/kg/day ÷ bid	
		First-generation cephalosporin	40 mg/kg/day ÷ tid	
		Second-generation cephalosporin	Dose varies	
Persistent middle ear effusion	Fussy child, TM red or brown, TM not bulging, afebrile	—	—	Chronic process requires scheduled ENT follow-up

as an associated complaint, which can lead to some confusion in the diagnosis. Adult epiglottitis with its normal oral pharyngeal examination is a prime example.

Hearing Loss and Vertigo

Conductive hearing loss is a common complaint and finding in infectious otologic pathology presenting to an emergency department. Infection of the ear affects hearing through swelling and transudation of either the external canal or the middle ear space. It can be diagnosed using at least a 256-Hz, preferably a 512-Hz, tuning fork to perform a Weber's test (placing the vibrating tuning fork in the middle of the forehead). The patient will hear the sound best on the side of the conductive hearing loss. The Rinne test (comparing bone to air by placing the vibrating fork on the mastoid and then presenting the tines approxi-

TABLE 1. *Continued*

Infection	Signs and symptoms	Antibiotic	Dosage	Comment
Chronic infectious otitis media				
Without cholesteatoma	Chronic symptoms, no systemic response	Ototopical drops	Topical	ENT referral, evaluate for CNS complications
With cholesteatoma	Chronic symptoms, TM thickened, perforated, ossicles calcified or necrotic, drainage (recurrent), cholesteatoma possible, confirmed by CT	Ototopical drops	Topical	ENT referral, evaluate for CNS complications
Mastoiditis	Antecedent otitis media, erythema, swelling or postauricular abscess	Cefuroxime + metronidazole	100 mg/kg/day ÷ q6–8h, 30 mg/kg/day ÷ q6h	Hospitalization, ENT consult, + CT scan
Sinusitis	Nasal congestion, facial pain, headache, tenderness over sinus, fever	Amoxicillin	40 mg/kg/day ÷ tid PO for 10 days	Topical decongestion; consider admission if patient toxic or with frontal or sphenoid sinusitis
		Erythromycin-sulfa	50/150 mg/kg qid, 8/40 mg/kg/day ÷ bid	
		Trimethoprim-sulfamethoxazole	40 mg/kg/day ÷ tid	
		First-generation cephalosporin	Dose varies	
		Second-generation cephalosporin		
Chronic infectious sinusitis	Complaints chronic, nasal congestion, facial pain, afebrile	None needed acutely	—	CT scan, allergy evaluation, ENT referral

mately 2 cm away from the external canal) will demonstrate that sound heard through bone stimulation is perceived better than sound heard through air conduction.

Infectious nerve hearing loss may result either from a primary infection (mumps, measles, varicella-zoster, syphilis) of the cochlear-vestibular apparatus or secondary to bacterial or viral central nervous system (CNS) infection, such as meningitis. The patient may present with hearing loss alone, hearing loss with vestibular symptoms, or any combination of these two associated with other CNS manifestations of infection. If the hearing loss is unilateral, the Weber's test will lateralize away from the affected ear toward the "good" cochlea. The Rinne test will show air conduction greater than bone conduction.

If the vestibular system is involved, the patient may present with disabling vertigo, nausea, and vomiting as the primary complaints.

The Red Tympanic Membrane

Infectious entities causing abnormalities of the tympanic membrane (TM) are listed in Table 2.

Acute Infectious Pathology

The normal TM is so thin that the middle ear space can be seen directly through it. Infection

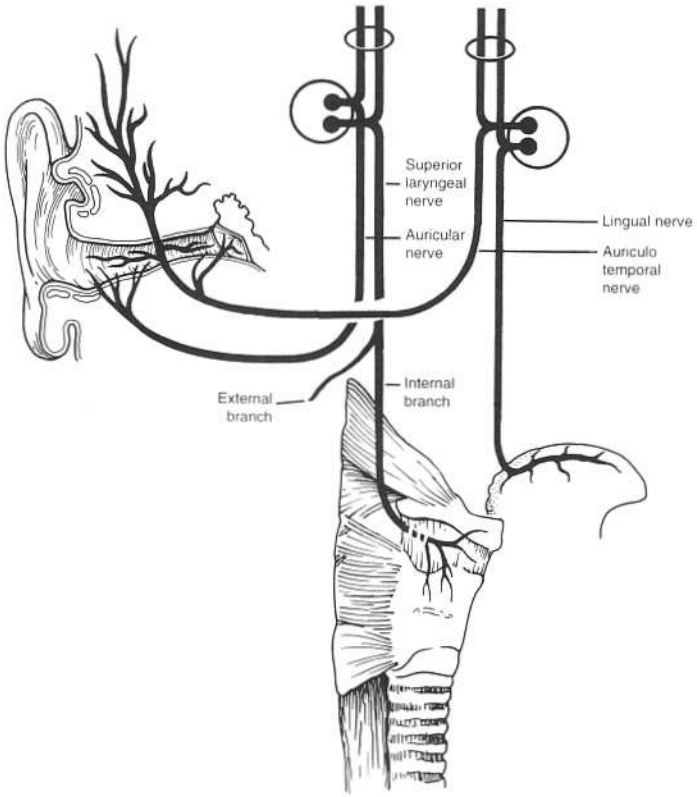


FIG. 1. Nerve paths from the tongue and the pharynx by which a lesion may cause ear pain. (Adapted with permission from MacComb WS, Fletcher GH. *Cancer of the Head and Neck*, 1967:223.)

of the mucosa of the middle ear causes immediate erythema and edema of the drum. Early infections may cause only hyperemia of the TM with little loss of landmarks. If the infection is allowed to progress, the tissues of the drum will weep fluid directly into the external canal without any evidence of a perforation.

Acute Noninfectious Pathology

The TM may become erythematous after closed head injury and acute or chronic eustachian tube dysfunction or barotrauma. The patient usually will not have any associated systemic infectious signs, such as fever or an elevated white blood cell count.

The "No Landmark" Ear Drum

Commonly emergency department physicians will see a patient with a possible infectious problem related to the ear and not be able to recognize landmarks on the TM. This is usually either an acute infectious otitis media, in which the drum is so swollen without a perforation that no landmarks can be recognized, or a total TM perforation, in which the edges of the drum are not easily seen and one is looking at the medial wall of the middle ear, which is

TABLE 2. *Infectious differential diagnosis of the abnormal tympanic membrane*

Finding	Myringitis granulosa	Bullous myringitis	Acute infectious otitis media	Persistent middle ear effusion	Chronic infectious otitis media	
					Without cholesteatoma	With cholesteatoma
Fever	No	No	Yes	No	No	No
Time course	Acute	Acute	Acute	Chronic	Chronic	Chronic
Tympanic membrane	Red, granular bumps	Vesicles	Red, bulging, wet	Red to normal	Thickening	Pus
Perforation	No	No	Maybe	No	Yes	Yes
Drainage	No	No	Maybe	No	Maybe	Yes
Hearing loss	No	No	Yes*	Maybe	Yes	Yes
Etiology	Unclear	Mycoplasma	Bacterial, *S. pneumoniae, M. catarrhalis*	Bacterial, viral	Aerobic, anaerobic bacteria	Aerobic, anaerobic bacteria
Treatment	None	None	Antibiotics, see Table 1	ENT follow-up	Otologic drops, ENT follow-up	Otologic drops, ENT follow-up

*Rarely measured.

made up primarily of the bulging basilar turn of the cochlea. In chronic infectious otitis media (which is discussed below), there will be no systemic signs of infection.

The Draining Ear

The patient who presents to the emergency department with an infectious otologic problem frequently will have drainage and pain as the presenting complaints (Table 3). These symptoms may represent acute external otitis, necrotizing external otitis media, acute infectious otitis media, chronic infectious otitis media with or without a cholesteatoma, or even mastoiditis.

The first differential sign that should be noted is fever. This sign will often separate chronic infectious otitis media from external otitis, acute otitis media, and mastoiditis, since the patient with the chronic process rarely has systemic toxicity. The presence of a large perforation will confirm the diagnosis of chronic infectious otitis media. Pain on auricular mo-

TABLE 3. *The draining ear*

Finding	Chronic otitis media	Acute otitis media	Mastoiditis	External otitis	Malignant external otitis
Fever	−	+	+	±	±
Malaise	−	+	+	±	+
Immune problem	±	±	±	±	+
Auricular tenderness	−	−	±	+	+
Postauricular abscess	−	−	+	−	−
Swollen external canal	−	−	±	+	+
Perforation	+	+	+	−	−

tion may be present in both otitis externa and mastoiditis but does not occur in acute infectious otitis media. A significant portion of otitis externa patients will not have fever or systemic toxicity, so they can be differentiated, leaving only the febrile external otitis and mastoiditis patients. Granulation tissue in the canal in a diabetic patient may point to malignant external otitis. Those patients with a postauricular abscess plus mastoiditis can then be identified. The external otitis patient will have a swollen external canal but an intact drum. The mastoiditis patient without a postauricular abscess will usually have a tympanic perforation or at least swelling of the posterior canal wall.

The Auricle

Cellulitis and perichondritis should be considered together because their presentations are similar and they are frequently difficult to differentiate. Historically, cellulitis may have a more rapid onset, and there may be no antecedent trauma, as is frequent in perichondritis. Pain, tenderness, and fever are common to both. Erythema, swelling, and tenderness of the auricle are seen in each (Fig. 2). Evidence of injury with exposed cartilage points to perichondritis. Practically, though, initial treatment consists of 10 days of oral antibiotics covering both *Staphylococcus* and *Streptococcus* organisms. Any drainage or possible fluctuant areas should be cultured if noted. *Pseudomonas* species may be seen in perichondritis but should not be treated without culture identification (1). If there is no history or evidence of cartilaginous trauma, decrease in swelling, tenderness, and erythema should be evident in 24 to 48 hours. If that is not the case, otolaryngology consultation and possible hospitalization for intravenous (IV) antibiotic therapy should be considered.

FIG. 2. Perichondritis of the auricle. Swelling and erythema of the upper two-thirds of the auricle as seen in perichondritis. (Reprinted with permission from Hawke M, Jahn AF. *Diseases of the Ear: Clinical and Pathologic Aspects.* Philadelphia: Lea & Febiger, 1987:Fig. 1.69.)

The External Canal

General Considerations

The external canal is a bony and cartilaginous tube that is covered by a relatively thin layer of skin. Any swelling secondary to inflammation will fill the external canal, partially or completely obliterating any opening. Because there is little loose tissue, the swelling causes significant tension and therefore pain. Often the swelling is so severe that the TM cannot be visualized, which raises the question of whether there is an associated middle ear infection. Although otorrhea may be present, it is rare for acute or chronic infectious otitis media with perforation to have an associated external otitis; therefore, systemic treatment for an acute middle ear infection usually is unnecessary. Ototopical antibiotic drops usually are adequate for treatment of infections of the ear canal. These include polymyxin, a neomycin and hydrocortisone combination

(Cortisporin Otic), and dilute acetic acid or boric acid solutions.

Otitis Externa

Bacterial Infection

Bacterial infection of the external canal skin appears to have a significant correlation with moisture (swimming or humidity) and increased temperature. Incidence statistics show a clear increase during the summer months and in swimmers (1). Pain, fullness, itching, and hearing loss are frequent complaints, depending on the degree of swelling of the external canal skin. Physical findings can range from mild erythema of the skin to marked swelling with pus, completely closing the external canal (Fig. 3). The pinna usually is normal and, most important, causes significant pain when gently pulled. Treatment is local, consisting of any of the broad-spectrum otitic drops (2 drops tid for 5 days). Suspensions are less irritating than solutions. Topical medication will not work if the canal is swollen shut. Under those circumstances, a wick soaked with topical medication must be placed. Either a handmade cotton or manufactured wick (Otowick) can be used. Placement can be extremely painful to the patient, but the use of injectable local anesthesia is more painful than the wick placement itself.

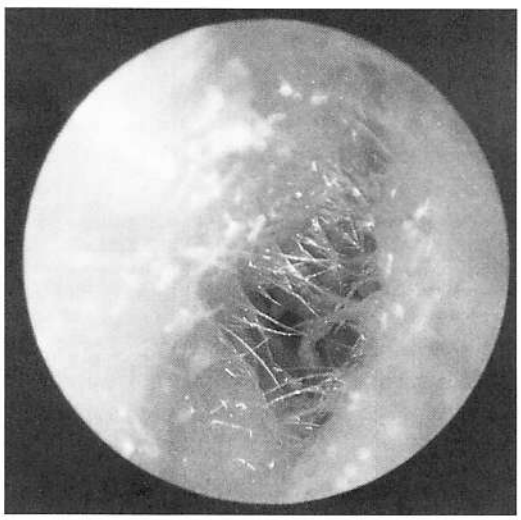


FIG. 3. Acute external otitis. Marked swelling and erythema of the skin of the external canal, which may be so marked that the TM cannot be visualized. Also note clear, watery discharge. (Reprinted with permission from Hawke M, Jahn AF. *Diseases of the Ear: Clinical and Pathologic Aspects.* Philadelphia: Lea & Febiger, 1987:Fig. 2.51.)

Fungal Infection

Although much less common than bacterial otitis externa, fungal external otitis may present similarly. Most often the external canal skin is not completely swollen shut and usually is not as painful. Unaided visual examination often will reveal white or colored hyphae in the canal. Clotrimazole (Lotrimin) or M-cresyl acetate should be prescribed. This is a local infection that rarely needs systemic antifungal agents for elimination.

Malignant External Otitis (Necrotizing Otitis Externa)

Malignant external otitis is an unusual, life-threatening disease that may present similarly to uncomplicated bacterial external otitis; the patient has pain, ear fullness, and drainage. The patient, however, will usually have some sort of immunocompromised status, such as diabetes (80%) (2), and at least three of the five signs and symptoms of necrotizing otitis externa: (a) persistent external otitis, (b) granulation tissue in the external auditory canal, (c) computed tomography (CT) scan confirmation of osteomyelitis of the external auditory canal, mastoid air cells, or skull base, (d) cranial nerve involvement, and (e) isolation of *Pseudomonas aeruginosa* from a culture of ear drainage (3). Although it is not likely that culture results showing *Pseudomonas* species would be available to the emergency department physician, the history and the physical signs in this specific patient population should create a high index of suspicion for a diagnosis of malignant external otitis. This is a life-threatening infection that

requires hospitalization, IV antipseudomonal antibiotic therapy for 6 to 8 weeks, and occasional surgical debridement of the temporal bone. Even newer broad-spectrum oral antibiotics are not appropriate for treating this very ill population.

The Tympanic Membrane

Myringitis Granulosa

Myringitis granulosa is a presumed infectious disease of the TM that presents with ear pain, a sensation of hearing loss, and occasional drainage. Examination reveals multiple small, red, granular bumps on the TM with surrounding or overlying suppuration. No perforation will be noted, the hearing will be normal, and the TM will move normally on pneumatic examination. The etiology of this condition is not clear. Its clinical importance lies in confusing it for acute or chronic infectious otitis media. Any of the ototopical drops are adequate treatment for this problem.

Bullous Myringitis

Bullous myringitis, which often is due to *Mycoplasma* bacteria, presents with the rapid onset of ear pain. There may be no associated systemic symptoms, or the patient may have symptoms of upper respiratory infection (URI). Clear vesicles are seen on the drum. Incising the vesicles with a needle usually will resolve the symptoms but requires an operating microscope. Systemic antibiotic therapy is not indicated for isolated myringitis, but ototopical antibiotics may prevent otitis externa (4).

The Middle Ear Cavity

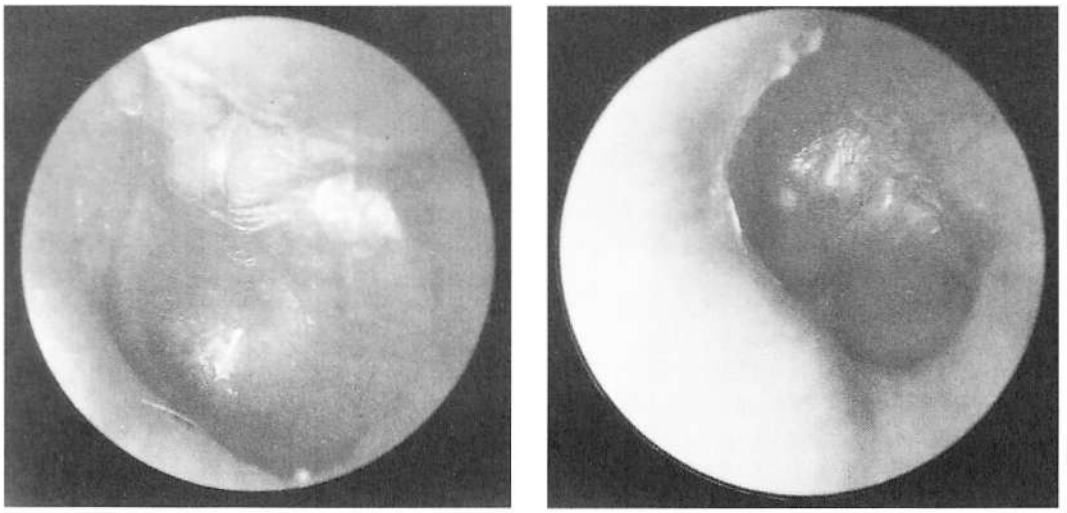
Acute Infectious Otitis Media

Otitis media is the most common bacterial infection of infancy. One-third of infants will have an attack of otitis media by 12 months of age, 90% of all children will have at least one episode of acute infectious otitis media by age 7 years, and 75% of this population will have had at least three or more episodes (5). Otitis media becomes less common after 10 years of age. The cornerstone of this disease is a bacterial insult resulting in an acute inflammatory response. Pathologically this response is represented by such histologic findings as polymorphonuclear white cell accumulation, vascular dilation, and transudation of fluid. Eustachian tube dysfunction predisposes children to these pathologic changes.

The diagnosis in the adult is relatively straightforward. The patient will complain of ear pain, fever, and possibly hearing loss, ear popping or fullness, tinnitus, or vertigo. Symptoms of a URI also may be present. The clinical symptoms in older children may be similar, but symptomatology is more subtle in infants and young children. Although rubbing or pulling of the ears may be present, this is not a reliable sign. Other symptoms are nonspecific, including irritability, crying, sleep problems, change in feeding, vomiting, and diarrhea. An antecedent URI is common. Fever may not be present and when present is generally low grade (6). The presence of otitis media does not rule out more serious illness such as bacteremia, pneumonia, or meningitis, and approximately 5% of infants with otitis media and temperatures higher than 39°C have bacteremia (7).

The physical examination in adults and older children generally is easier because the ear canal is wider than it is in young children. If cerumen obscures the TM, it must be removed with a swab or a curette or by irrigation. Depending on what point in the infection the patient is examined, the physician may see a reddish, dull, nonmoving drum or a bulging, wet, red TM, which on occasion will have ruptured, resulting in pus draining from the ear (Fig. 4).

The most common organisms that cause acute otitis media are *S. pneumoniae* (60%), nonencapsulated *Haemophilus influenzae* (15%–20%), and *Moraxella catarrhalis* (5%–15%). Approximately 30% of *Haemophilus* organisms and more than 80% of *Moraxella* organ-



A B

FIG. 4. Acute infectious otitis media. (A) Early acute infectious otitis media showing mild erythema and minimal bulging of the TM. (B) Full-blown acute infectious otitis media with classic bulging, wet, red TM. Note the white purulent fluid that can be seen accumulating behind the drum in the lower portion of the middle ear. (Reprinted with permission from Hawke M, Jahn AF. *Diseases of the Ear: Clinical and Pathologic Aspects.* Philadelphia: Lea & Febiger, 1987:Fig. 3.37.)

isms produce beta-lactamase, which inactivates many penicillin and cephalosporin antibiotics. In recent years, pneumococci have become increasingly resistant to penicillins, oral cephalosporins, and erythromycin and sulfonamides. Currently, 5% to 30% of pneumococci in certain regions may be resistant to antibiotics commonly used to treat otitis media.

The TM should be assessed for color, transparency, position, landmarks, and mobility. Normally, the TM is pearly gray to pink. Although crying may increase TM injection, this is generally bilateral. Redness without other TM abnormality may be early evidence of infection. Inflammation decreases transparency of the TM, so myringitis (inflammation of the TM) may cause opacity. Alternatively, pus may be seen through the uninflamed TM and is diagnostic of otitis media. Landmarks include the light reflex of the anterior inferior quadrant and the ossicles, the manubrium of the malleus of which is most easily seen. The light reflex results from the normal shine of a normal TM and so may be lost in otitis media. The malleolus may not be seen secondary to opacity of the TM or bulging of the external eustachian canal from effusion. Negative ear pressure will cause retraction of the ear drum over the malleolus. Effusion will also decrease TM movement on insufflation.

A change in color or transparency with a bulging TM and decreased mobility is 95% predictive of acute, purulent otitis media (8). In patients under 4 months of age, the TM is duller, with diminished light reflex and decreased mobility.

The aminopenicillins are still effective and are the initially recommended therapeutic modalities in this disease (amoxicillin, 40–50 mg/kg/day divided tid) in spite of the recent emergence of penicillin-resistant *Streptococcus pneumoniae*. Ten days of therapy is usually adequate. Symptomatic improvement should be seen within 72 hours. If there is no improvement in 3 days, therapeutic failure at 10 days, penicillin allergy, or a high local incidence of ampicillin-resistant organisms, antibiotics that resist beta-lactamase, such as trimethoprim-sulfamethoxazole, erythromycin and sulfamethoxazole, amoxicillin/clavulanate, or loracarbef

(or other second-generation cephalosporin) would be appropriate (9). Cefaclor is not recommended by many pediatricians because of its poor activity clinically against *Haemophilus influenzae* and the incidence of serum sickness as a side effect. There is no single drug of choice in this disease, since social, epidemiologic, and economic factors must all be considered in the treatment decision.

Ceftriaxone in a single dose of 50 mg/kg IM or IV has been shown to be effective for otitis media and should be considered in infants whose parents' ability to comply with oral regimens or follow-up is questionable (10).

Even with effective antibiotic treatment, at least one-third of children develop chronic serous ear effusions. Infants should be reexamined at 3 weeks after the acute episode. However, parents should be instructed to return if there is a recurrence of symptoms of pain or fever or new signs, such as ear drainage.

Recurrent infections often are caused by the same organisms sensitive to the initial antibiotic. Nasal decongestants may help nasal stuffiness, but they have no effect on the outcome of the ear infection. Myringotomy is rarely indicated in acute infectious otitis media except in cases of an immunocompromised host, when accurate bacterial identification is mandatory. A child under 2 weeks of age with otitis media must undergo a septic workup and be admitted for observation. Extreme caution must be used in a child less than 8 weeks of age to rule out systemic illness. Cultures in these infants may yield *Escherichia coli*, *Klebsiella* species, *P. aeruginosa*, and other gram-negative bacteria (11). All patients require follow-up in 10 to 14 days. The natural history for the patient treated for otitis media is given in Fig. 5.

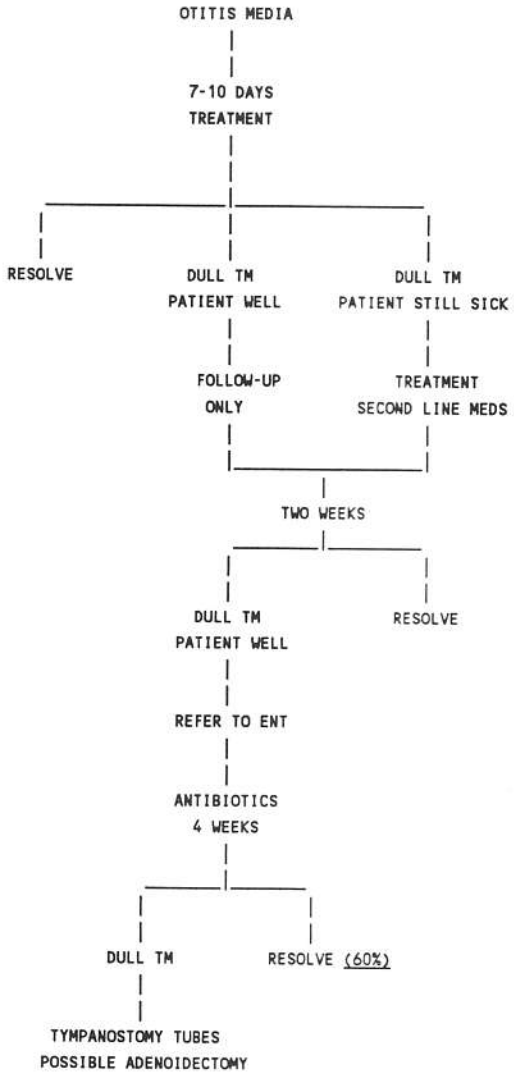


FIG. 5. Common clinical course of an otherwise healthy child presenting with an initial episode of acute infectious otitis media.

The Draining Tympanostomy Tube

One of the most common medical procedures performed in the United States is placement of tympanostomy tubes. Up to 21% of children will have at least one episode of otorrhea while tubes are in place (12). The child with a draining ear in the presence of a tympanostomy tube may have pain or discomfort but will rarely have fever or other systemic signs. No definitive studies have been accomplished defining the best treatment for this condition, except for the study by Balkany et al (13) showing that ototopical drops decrease the incidence of otorrhea in the immediate postoperative period. After that time, a child seen in an urgent context probably should be treated with oral antibiotics similar to those used to treat acute otitis media.

Persistent Middle Ear Effusion

Often a child will present pulling on the ear, being fussy, or crying who on physical examination will have a red- or brownish-appearing TM. The drum will not be wet or bulging but may be retracted. A conductive hearing loss will be present. The child will be afebrile, and if obtained, the white blood cell count will be normal.

Although commonly diagnosed and treated as if it were acute infectious otitis media except for pain medication, persistent middle ear effusion does not require an emergency therapeutic intervention. Effusion may persist for up to 6 weeks after an episode of acute otitis media in up to 48% of cases (14). Treatment with antibiotics is recommended only if an associated sinusitis is suspected or the child's history of otitis media is severe enough that tympanostomy tubes are being considered (15). A bacterial process has been implicated in about 50% of these situations (16). Initiation in an emergency situation of any antimicrobial treatment for what is, in effect, a chronic process will not improve immediate outcome. Elective otolaryngology follow-up is required for long-term management, which may include antibiotic therapy.

Chronic Infectious Otitis Media Without Cholesteatoma

The word *chronic* can be related to either time or pathology. Many physicians use chronic as if it refers solely to time. This usage, however, does not give a complete picture of chronic infectious otitis media. Histologically the chronic inflammatory response includes fibrosis, necrosis, hyalinization, calcification, and hypertrophy, to name a few. On examination of an ear in which there is a chronic inflammatory response, the clinician may see evidence of all these processes. Clinically there would be a perforation and thickening of the middle ear mucosa (Fig. 6). Calcified and necrotic ossicles also may be evident. Drainage from either a long-standing bacterial infection or an acute exacerbation of the chronic process may be evident. Although the patient may have pain, it is unlikely that there will be any systemic response, such as fever or an elevated white blood cell count. What the examiner is looking at is, in effect, a walled-off abscess cavity. The bacteriology is that of a chronic infection, including both aerobic and anaerobic organisms such as *Pseudomonas* species, *Staphylococcus aureus*, and *Bacteroides* species (17). Both children and adults may present to the emergency department with a painful perforated draining ear, simulating an acute infectious otitis media. Questioning will most commonly elicit previous episodes of drainage over months or even years. The physical findings will be as just described, and the patient rarely will have any evidence of systemic toxicity. The acute drainage is merely an exacerbation of a chronic process. Emergency treatment requires little other than ototopical antibiotic drops, pain medication, and elective otologic referral. Systemic antibiotics are not indicated except occasionally in

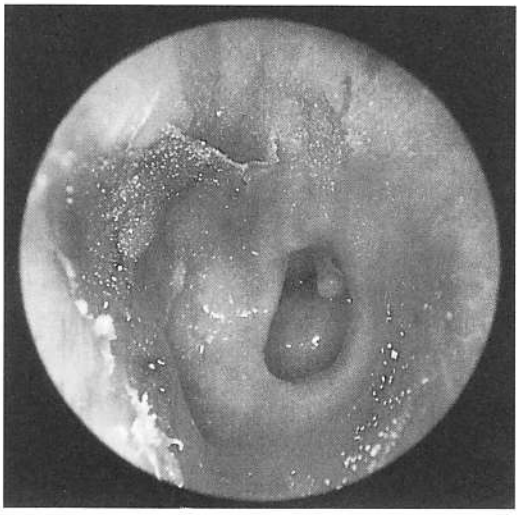


FIG. 6. Chronic infectious otitis media. The surface of the drum and deep meatus is coated with a thick, brown, mucopurulent discharge. Through a posterosuperior perforation, the head of the stapes is visible. The long process of the incus has undergone resorption and hence is not seen. (Reprinted with permission from Hawke M, Jahn AF. *Diseases of the Ear: Clinical and Pathologic Aspects.* Philadelphia: Lea & Febiger, 1987:Fig. 3.119.)

conjunction with definitive surgical therapy. These patients should be referred to an otolaryngologist.

Chronic Infectious Otitis Media with Cholesteatoma

A cholesteatoma is an epithelial cyst that has grown into the middle ear or mastoid (Fig. 7). Its danger lies in its acting as a growing, frequently infected benign "tumor" in a closed space adjacent to many vital structures, such as the meninges, the eighth cranial nerve, the cochlea, and the semicircular canals. Diagnosis is difficult in the emergency department environment, since the patient usually will present with a painful ear full of pus. Microscopic otologic examination frequently is required to make the clinical diagnosis. Radiologic evaluation, usually a CT scan, is used for confirmation rather than as a primary diagnostic tool. A defect in the drum containing epithelial debris supports the diagnosis of cholesteatoma. Emergency treatment is similar to that of chronic infectious otitis media without cholesteatoma. If a diagnosis of cholesteatoma is made in an emergency department situation, the physician should have a greater index of suspicion for the more serious complications of chronic infectious otitis media and look for the signs and symptoms described in the following sections. If the patient's only complaints and findings are a painful draining ear with some hearing loss, then elective otolaryngology referral is indicated.

Mastoiditis

Mucosal

The middle ear space is directly connected to the mastoid through a space in which lie the heads of the incus and the malleus. This passage leads directly to the antrum within the mastoid and from there connects to the remaining mastoid air cells (Fig. 8). All these contigu-

FIG. 7. Cholesteatoma. The white cyst visible behind the intact tympanic membrane is part of a cholesteatoma that extends around the ossicles. (Reprinted with permission from Hawke M, Jahn AF. *Diseases of the Ear: Clinical and Pathologic Aspects.* Philadelphia: Lea & Febiger, 1987:Fig. 3.163.)

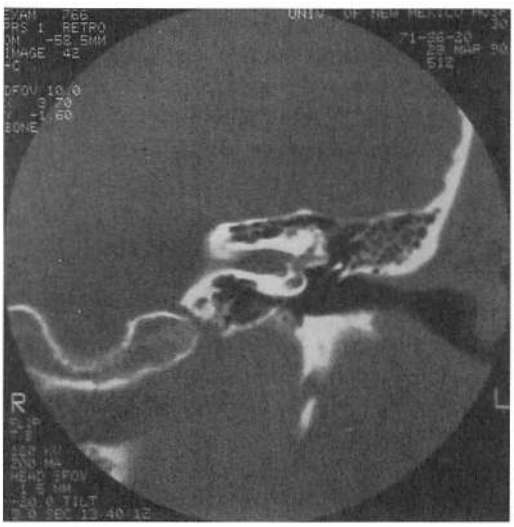


FIG. 8. Connection between the middle ear and the mastoid. Coronal CT scan of the temporal bone.

ous chambers are mucosally lined. Any acute infection of the middle ear space commonly will involve the mucosa of part or all of the mastoid, thus giving rise to the term *mastoiditis*. This is *not* the bony-mastoid infection that has such serious consequences. Such an infection may be confused with bony osteitic mastoiditis if an x-ray is obtained that shows some fluid in the mastoid air cells and is returned to the emergency department with a radiologic diagnosis of "mastoiditis" (Fig. 9). Unless there is objective evidence of bony osteitic mastoiditis, such as a postauricular abscess (the auricle will be pushed forward by a fluctuant mass overlying the mastoid, as in Fig. 10), or radiologic evidence of breakdown of mastoid bony septa (coalescent mastoiditis), the clinician most likely is dealing with a straightforward acute otitis media with some mastoid mucosal extension. Treatment is outpatient oral antibiotic therapy, as previously described.

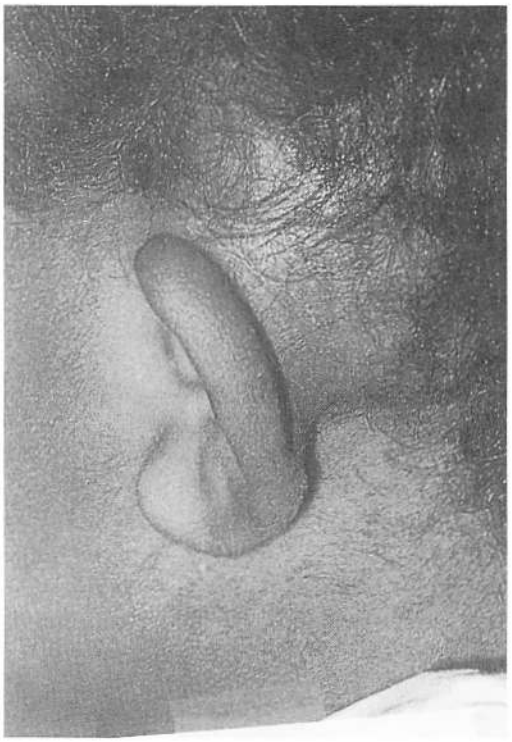


FIG. 10. Postauricular subperiosteal abscess secondary to mastoiditis. (Reprinted with permission from Hawke M, Jahn AF. *Diseases of the Ear: Clinical and Pathologic Aspects.* Philadelphia: Lea & Febiger, 1987:Fig. 3.52.)

Osteitis

A small percentage of children and occasionally adults will develop a true osteitis secondary to an acute infectious otitis media. Before the antibiotic era, 10% of children with acute infectious otitis media went on to develop bony mastoiditis. Today large urban medical centers rarely see more than 5 to 10 cases per year. Clinically these cases are characterized by a history of recently acquired or even treated acute otitis media. The illness persists, and the patient may become increasingly toxic. Physical findings will include a red and bulging TM or posterior canal wall, and the drum may be perforated with pus present. There often will be postauricular erythema, swelling, or a frank abscess (see Fig. 10). Radiologic evaluation (CT scan) should confirm that there is bony septal breakdown within the mastoid. The bacteriology of mastoiditis includes the standard organisms found in acute infectious otitis media plus additional aerobic species, including *S. aureus* and *Proteus mira-*

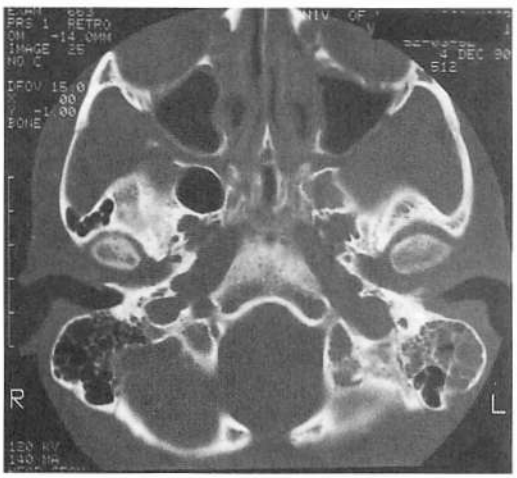


FIG. 9. Mucosal mastoiditis. CT scan of the mastoid air cells showing fluid secondary to acute infectious otitis media but no evidence of the bony septal destruction.

bilis, and various anaerobic organisms, such as *Bacteroides*, *Clostridium*, and *Fusobacterium* species (18). This disease requires antibiotic treatment in the hospital and both pediatric and otologic consultation. Prior to culture results, antibiotic treatment should include cefuroxime plus metronidazole. Once identification of the offending agent(s) has been made, therapy should be continued for 4 to 6 weeks, as in any bone infection.

Cochlear-Vestibular Infections

Viral Infections

Patients with acquired mumps, measles, and varicella-zoster may develop an acute neurosensory hearing loss during their illness, causing them to seek urgent medical evaluation. In measles and mumps, the infection may affect both the cochlea and the vestibular systems, although hearing loss is more common. The hearing loss in mumps is unilateral in 80% of cases (19). Varicella-zoster (herpes zoster oticus) produces painful auricular vesicles, followed shortly by a facial nerve paralysis. Auditory and vestibular symptoms are much less common in this infection. There is no effective treatment for any of these infections. Therapy with antivertiginous medications, such as meclizine or scopolamine dermal patches (Transderm-Scop), may provide symptomatic relief.

Bacterial Infection

Primary cochlear-vestibular bacterial infections do not occur. Secondary involvement of the inner ear is seen in meningitis and rarely may be associated with an acute or chronic infectious otitis media.

Syphilis

Congenital syphilis may cause neurosensory hearing loss early (0–3 years) or cause it to develop in the second or third decade (20). Clinically the hearing loss develops gradually and should be an uncommonly seen chief complaint in an emergency department. On the other hand, patients with acquired syphilis may develop nerve hearing loss during the second stage of the disease (20). This loss is sudden, may be bilateral, and usually has no associated vestibular symptoms. Diagnosis is made by identifying the high-risk patient and performing the appropriate clinical and serologic evaluation for secondary syphilis. This is one of the few neurosensory hearing losses that may respond to medical treatment, including antisyphilitic antibiotics and systemic steroids.

Infectious CNS Complications of Otologic Infection

Meningitis

Meningitis is the most common CNS complication of both acute and chronic infectious otitis media (18). (For symptoms and diagnosis of meningitis, see Chaps. 7 and 29.) Otologic findings may be anything from an acute infectious otitis media to a cholesteatoma. Because any patient with an ear infection may present with fever and headache, the emergency physician must not overlook the possibility of CNS involvement (see Chaps. 7 and 29).

Brain Abscess

Brain abscess is the second most common intracranial infectious problem secondary to suppurative ear disease. Up to 25% of all brain abscesses arise secondary to otologic infection (21). As in meningitis, the practitioner must have a relatively high index of suspicion for intracranial pathology in the patient presenting with headache and a draining ear. Most commonly, brain abscess is associated with chronic infectious otitis media with or without cholesteatoma. Radiologic evaluation confirms the diagnosis (see Chap. 29).

Sigmoid Sinus Thrombosis

Sigmoid sinus thrombosis is an extremely uncommon disease. Like brain abscess, it is associated with a chronic infectious otologic problem. It also may present with CNS-associated symptoms such as headache, fever, nausea, and vomiting. There may be septic embolic phenomena manifested by fever spikes, which may be helpful in suspecting the diagnosis. Cranial nerve involvement (IX, X, XI) and papilledema often are late clinical signs. A CT scan or magnetic resonance imaging is indicated if this diagnosis is suspected.

SINUSITIS

Uncomplicated Acute Infectious Sinusitis

Clinical Presentation and Evaluation

Most adults with uncomplicated acute infectious sinusitis will present with some history of nasal congestion from either a URI or a worsening of an acute allergic problem. Other patients will have nasal polyps, nasal packing, an infected tooth root abutting the maxillary sinus, or barotrauma as a predisposing condition. Symptoms begin with facial pain, headache, rhinorrhea, and then fever. The pain may be stabbing or achy and a feeling of fullness noted. Bending, coughing, or straining may increase the pain. The location of the pain will be variable depending on the sinus or sinuses involved but often has some anatomic relationship to the infected cavity. Sinusitis occurs in order of frequency in the maxillary, ethmoidal, frontal, and sphenoidal sinuses. The pain of ethmoidal or sphenoidal sinusitis is vague.

Physical examination will reveal mucopurulent nasal drainage, but drainage may be prevented by completely blocked ostia. Tenderness may be elicited over an infected maxillary or frontal sinus. Facial and orbital swelling or skin erythema may occur but are not routine in the average case. Careful rhinoscopy with suction and magnification may reveal pus coming from the middle meatus, supporting the diagnosis of purulent sinusitis. This kind of nasal examination, however, rarely is available in an emergency department situation.

The history along with mucopurulent nasal drainage, fever, facial pain, and tenderness provides the database on which a presumptive diagnosis of sinus infection can be made. The remaining evaluation question is whether a radiologic examination is necessary. In an otherwise healthy adult presenting with a typical history and physical examination, sinus radiographs usually will add little to the diagnosis while adding significantly to the cost of the medical encounter. Diagnostic radiology becomes more relevant in the minority of these "typical" patients if, after a reasonable therapeutic intervention, they do not have resolution of their disease. X-rays in acute sinusitis will demonstrate an air-fluid level or complete opacification of the involved sinus. In chronic sinusitis, mucosal thickening or complete opacification will be found.

Children present less typical patterns than adults. Their "cold" is usually somewhat more severe than normal, with higher temperatures that do not resolve in the usual 5 to 7 days. They may not complain of pain but will usually have some facial swelling or erythema. Continued URI symptoms in a child beyond 10 days should alert the clinician to the possibility of a paranasal sinus infection, as up to 75% have bacterial sinusitis (22). An x-ray demonstrating complete sinus opacification or an air-fluid level is useful in making the diagnosis in children more than 2 years old (23).

Nasal Congestion and Facial Pain

Nasal congestion from any cause can precipitate midfacial pain and even tenderness that is often diagnosed as acute bacterial sinusitis, resulting in systemic antibiotic therapy. There is a group of patients who present this way but who have few systemic signs of toxicity, such as fever or an elevated white blood cell count. They usually have only obstruction of the os-

teomeatal complex with some air resorption and pressure in the maxillary sinus. Left untreated, they may go on to develop an acute bacterial infection. At this stage, all the patient may need is decongestion of the nasal mucous membranes and opening of the osteomeatal complex to allow air to flow into the maxillary sinus and restore pressure equalization. Oral and, more important, topical nasal decongestion should be used initially. Long-acting topical nasal spray should be used three times per day. The medication should be given in each nostril, then 3 to 5 minutes later applied again, aiming each time for the inner canthus of the eye. Within 24 to 48 hours, this procedure will often open up the obstruction to the osteomeatal complex and eliminate the symptoms and may prevent the development of an acute bacterial sinusitis. The nasal spray usually can be discontinued in 3 to 5 days (24).

Pathophysiology and Microbiology

Acute infectious sinusitis in an otherwise healthy patient is most commonly caused by obstruction of the osteomeatal complex (Fig. 11), which refers to an area in the anterior portion of the middle meatus into which the maxillary, frontal, and anterior ethmoidal sinuses drain. This area is normally quite narrow, so any inflammation can easily cause obstruction of the ostea of these sinuses and result in acute infectious sinusitis. Either an acute viral rhinitis or a noninfectious inflammatory event such as allergic rhinitis can precipitate the infection. Up to 0.5% of patients with viral URIs may develop a sinusitis (25). Since adults may develop two to three and children six to eight colds per year, sinusitis is a common diagnosis in an emergency outpatient setting (26). In adults, *H. influenzae* and *S. pneumoniae* are the most common organisms associated with this infection (65%). *Neisseria* species, *Streptococcus pyogenes*, alpha-hemolytic streptococcus, *Streptococcus viridans*, *Pseudomonas* species, *S. aureus*, Enterobacteriaceae, and viruses such as rhinovirus and influenza A also have been isolated (27,28). Children have a similar pattern of offending organisms except for the finding that *Moraxella catarrhalis* has been noted in up to 19% of antral isolates (29,30).

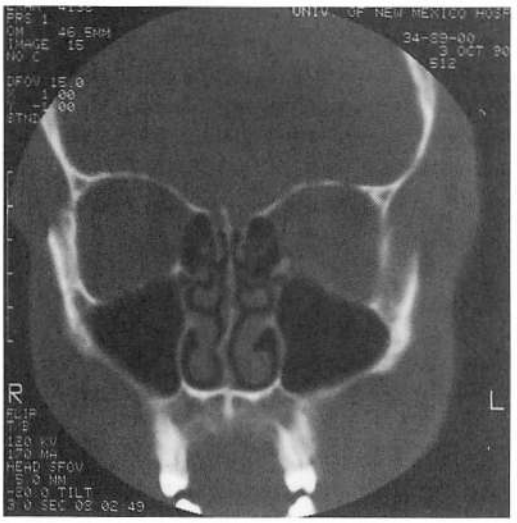


FIG. 11. CT scan of osteomeatal complex.

Maxillary Antrum

The maxillary antrum, which lies lateral to the nasal cavity and under the eye, may be the most commonly infected of the paranasal sinuses. It has been the most studied, since it is the most accessible. It is the only sinus that does not have a dural surface adjacent to it; therefore, its infections are rarely life-threatening. Ten to fifteen percent of maxillary sinusitis cases are of dental origin (31). The maxillary antrum sinus is present from birth but is not clinically significant until the child is 1 to 2 years old (32). When this sinus is infected, the patient will experience facial or upper maxillary dental pain. The sinus will be tender to palpation. Diagnostic aspiration can be performed with an 18-gauge trocar passed through the medial wall of the nose. It is a relatively easy procedure for an otolaryngologist to perform, even in an emergency department setting.

The Ethmoidal Sinus

The ethmoidal sinus, or labyrinth (since it is not a single cavity but is made up of 3–15 separate cells), lies between the orbit and the nasal cavity. Its roof is the floor of the anterior cranial fossa, and its anterior and posterior margins are marked by the frontal and sphenoidal sinuses, respectively. Because the roof of the sinus (fova ethmoidalis) is quite thick and the medial wall (lamina papyracea) so thin, most complications of ethmoidal infection manifest within the orbit. The ethmoidal sinuses cannot be palpated from the face, so tenderness is not an associated physical finding in this disease. Pain is orbital or retro-orbital and will be vague in nature. Periorbital erythema and swelling frequently may be seen. This sinus complex is present from birth and though less commonly infected then the maxillary, it is usually the origin of orbital complications of sinusitis in both children and adults (33).

The Frontal Sinus

The frontal sinus, which develops late in childhood or early adolescence and is often absent in many patients, lies above the orbit and forms a portion of the anterior wall of the anterior cranial fossa. When the sinus is infected, the pain is frontal, and tenderness can be elicited easily. Palpation should be accomplished just below the brow and above the medial canthus because the wall of the sinus is thinnest at that point. The entire posterior wall of the sinus, which can be several square centimeters, is dural lined, putting anterior cranial fossa structures at risk for directed extension infectious complications.

The Sphenoidal Sinus

The sphenoidal sinus, which is rarely solely infected, lies at the posterior superior border of the nasal cavity and has medial dural borders with the middle cranial fossa. Pain is rarely localizing and may be occipital when it does occur. Palpation is impossible. Radiologic evaluation is necessary for any accurate diagnosis of sphenoidal sinusitis. It, along with the frontal and ethmoidal sinuses, does not easily lend itself to diagnostic aspiration.

Treatment

The antibiotic used should cover the most common organisms associated with maxillary sinusitis. Amoxicillin, ampicillin, and trimethoprim-sulfamethoxazole have all been shown to be approximately 80% effective in eliminating bacteria from an infected maxillary antrum (26). The role of oral and topical decongestants in this disease is not clear. Symptomatic relief from the nasal congestion may be obtained from these agents. If obstruction of the osteomeatal complex is a primary etiologic factor in acute bacterial sinusitis, then relieving that obstruction should be a therapeutic aim. Topical decongestants can provide the most immediate effect on that obstruction. However, no controlled prospective study has proved that such therapy either negatively or positively affects the outcome of this disease. Until those data are forthcoming, topical and oral decongestants should be part of the medical therapy, so increased patient comfort may be achieved. Initiation and management of an uncomplicated maxillary or ethmoidal sinusitis is appropriate in the urgent care setting. Acute frontal sinusitis, though less common, has been reported to have intracranial complications in up to 17% of cases in children and adolescents (34). Adults also have a significant risk of intracranial problems (35). Outpatient treatment as previously described and follow-up of a reliable patient who has no severe toxicity, swelling, or erythema over the frontal sinuses are possible. However, any patient who deviates from this clinical picture probably should be referred to otolaryngology for admission and IV antibiotic therapy.

Complicated Acute Infectious Sinusitis

The Immunocompromised Patient

Microbiologic findings in complicated acute infectious sinusitis do not follow typical patterns. *Streptococcus*, *S. aureus*, *Staphylococcus epidermidis*, and assorted gram-negative bacilli have been found to be associated with sinusitis in diabetics (36). No good data are available for patients with acquired immunodeficiency syndrome (AIDS). In these situations, accurate cultures are necessary. Cultures must be obtained from antral puncture because it has been shown in both children and adults that there is no correlation between nasal cultures and sinus aspirations (37,38). Close follow-up is mandatory, and hospitalization or parenteral antibiotic therapy should be considered.

Nosocomial Sinusitis

Devices or packing placed intranasally during hospitalization may lead to sinusitis secondary to nosocomial bacteria such as *P. aeruginosa*, *E. coli*, *Klebsiella pneumoniae*, and *Bacteroides melaninogenicus* (39). Should a patient who has been recently discharged from the hospital present with an acute sinusitis with a history of such nasal manipulations, then sinus aspiration is indicated to accurately define the microbiology for proper antibiotic therapy.

Orbital Complications of Sinusitis

The orbital septum is a connective tissue structure that extends from the orbital periosteum to the tarsal plates. It provides a protective barrier that helps prevent many of the lid and conjunctival inflammatory diseases from extending posteriorly to involve the orbit. The periosteum of the orbit provides a similar barrier for the rest of the intraorbital contents. That periosteum overlies the lamina papyracea of the ethmoidal sinuses. In addition to the lamina papyracea being extremely thin, there are natural dehiscences in it that allow direct infectious access to the orbit (40). Orbital complications of sinusitis occur most frequently in children, and up to 75% of these infections appear to develop from an ethmoiditis (41). Any patient with a suspected orbital complication of an acute infectious sinusitis should have a CT scan performed to define the extent of the disease. Orbital and periorbital cellulitis are discussed in Chapter 33.

Frontal Bone Osteomyelitis

Although quite rare today, a patient with frontal bone osteomyelitis (Pott's puffy tumor) occasionally presents to an emergency department. An aggressive or untreated frontal sinusitis may involve the overlying bone. The skin over the sinus becomes swollen and erythematous. There may be abscess formation under the periosteum of the frontal bone. Bilateral periorbital inflammation is often present. CT evaluation is necessary for delineation of the extent of the disease. Hospitalization for medical and surgical therapy is indicated.

CNS Complications of Sinusitis

The walls of both the frontal and sphenoidal sinuses are dural lined, leading to the potential for the CNS spread of infection from these sinuses. Meningitis and subdural empyema are potential sequelae of sinus infection. The patient with frontal or sphenoidal sinusitis must undergo a careful neurologic evaluation for CNS infection. Close follow-up of patients with frontal or sphenoidal sinusitis is mandatory.

Chronic Infectious Sinusitis

Persons with chronic infectious sinusitis may not be seen in the urgent care setting per se because their symptomatology is rarely acute. The patient complains of long-standing nasal congestion and/or obstruction. Headache or facial pain also may be present over several months or even years. Physical examination would reveal an afebrile patient in no severe distress. Nasal polyps, mucosal congestion, or

septal deviation may be present. Workup in a routine ambulatory setting would include a CT scan of the sinuses plus allergy evaluation if warranted by the history. The bacteriology of chronic infectious sinusitis includes beta-hemolytic streptococci, *S. aureus*, and *H. influenzae* plus several strains of anaerobic organisms, such as *Bacteroides* and *Fusobacterium* species (42). Treatment is directed at correcting the long-underlying problem by allergy management, correction of a deviated septum, surgically opening up an obstructed osteomeatal complex, and appropriate antibiotics.

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33
Ocular Infections

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To adequately evaluate ophthalmic complaints and manage them appropriately, it is necessary to first master the basics of ocular anatomy and the eye examination. The first portion of this chapter covers these points for the primary care physician as well as a review of the red eye. The second portion of the chapter is dedicated to ocular infections.

ANATOMY

The anatomy of the eye is illustrated in Fig. 1. The eyelids are composed of an anterior skin-muscle lamella and a posterior tarsoconjunctival lamella. The eyelids are "separated" from the orbit by a layer of connective tissue called the orbital septum. It extends from the bony orbital rim superiorly and inferiorly to the upper and lower lids. There are six extraocular muscles that control movement of the globe: four rectus muscles and two oblique muscles. The superior, inferior, and medial recti, as well as the inferior oblique, are innervated by the third cranial nerve. The lateral rectus is innervated by the sixth cranial nerve and the superior oblique muscle is innervated by the fourth cranial nerve. The levator muscle elevates the upper eyelid and is innervated by the third cranial nerve. Closure of the lids is controlled by the orbicularis, which is innervated by the seventh cranial nerve. Sensation is supplied by the fifth cranial nerve. Tears are produced by the lacrimal gland located laterally just beneath the

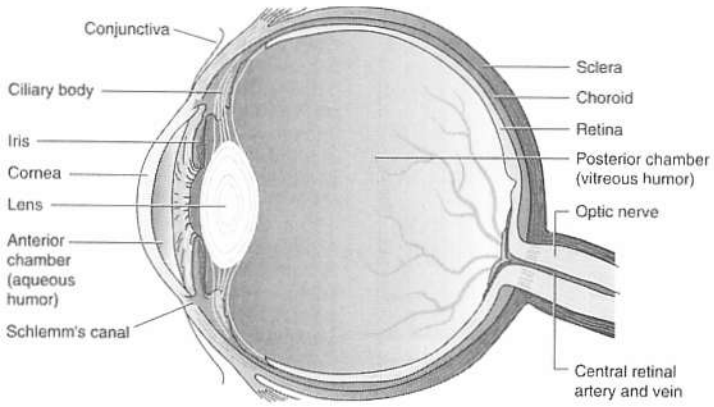

FIG. 1. Anatomy of the eye.

superior orbital rim. The tears drain medially through the upper and lower puncta of the lids into the lacrimal sac, which lies posterior and inferior to the medial canthal tendon. Tears then leave the sac via the lacrimal duct, which exits beneath the inferior turbinate in the nose.

The globe is covered anteriorly by a thin layer of connective tissue called conjunctiva, which composes the posterior layer of the lids and extends anteriorly to the cornea. The transparent cornea transmits and refracts light and is continuous with the white sclera or "wall of the eye" posteriorly. The junction of the cornea and sclera is called the limbus.

The anterior chamber contains aqueous humor and lies between the cornea anteriorly and the iris posteriorly. The pigmented iris controls the amount of light entering the eye. The anterior chamber angle refers to the junction between the cornea and the iris and houses the trabecular meshwork through which aqueous exits the eye. Posterior to the iris is the ciliary body, which produces aqueous. The lens also lies posterior to the iris and is held in place by zonules.

The sclera is lined interiorly by the choroid, which supplies blood to the outer layers of the retina. The retina lies on top of the choroid and constitutes the innermost layer of the globe. The posterior chamber is filled with a gel-like substance called vitreous. The macula is the portion of the retina that is responsible for detailed central vision. The anterior edge of the retina in the region of the limbus is called the pars plana. Retinal impulses are transmitted from the retina to the optic chiasm via the optic nerve. The optic disk is the anterior aspect of the optic nerve visible within the eye. It is made up of axons of the ganglion cell layer of the retina. The central retinal artery and vein travel within the optic nerve to provide the vascular supply to the inner retinal layers.

EXAMINATION OF THE EYE

History

Much of the history is the same as in any clinical situation. Onset of symptoms, duration, severity, medications, past medical history, and so on are all pertinent. Particular attention should be paid to the severity of visual loss and whether the visual loss was sudden or gradual in onset. Sudden severe visual loss generally heralds a more urgent condition than gradual visual loss. Pain, redness, discharge, and history of previous ocular conditions or surgery are also important issues to pursue. A history of trauma or exposure to others with similar symptoms may provide important clues regarding .

Examination

All patients with ocular complaints must have their visual acuity documented. Visual acuity is a measurement of the smallest object a per-

son can identify at a given distance from the eye. A visual acuity of 20/20 is considered "normal." Thus, a visual acuity of 20/60 indicates that the patient can recognize at 20 feet a symbol that a person with normal acuity could recognize at 60 feet. Visual acuity is usually tested using a Snellen chart at a distance of 20 feet. Patients should wear their distance prescription (eyeglasses or contact lenses) if available. Test each eye separately. If the vision is worse than 20/30, it should be tested with the patient looking through a pinhole, which can be used over the patient's glasses. For patients who cannot see the Snellen letters, determine whether they can count fingers and at what distance. If not, test if they can detect hand motion. If not, test if they can detect light. Confrontational visual field testing may also be indicated by the history.

The external examination can be performed with a penlight and includes evaluation of the lids, conjunctiva, pupils, and extraocular movements. Note the distribution of conjunctival injection if present, and use the swinging flashlight test to check for a Marcus Gunn pupil (afferent pupillary defect).

Many emergency departments are equipped with a slit lamp, which should be used if available. The slit lamp examination is used to evaluate the cornea, anterior chamber, lens, and intraocular pressure (IOP). Alternatively, a Wood's lamp can be used to check for corneal staining, and a Schiötz tonometer or Tono-Pen can be used to measure IOP. To check for corneal staining, a drop of anesthetic and some fluorescein dye are placed in the eye, and the cornea is examined with a Wood's lamp or cobalt blue light.

Finally, a funduscopic examination using the direct ophthalmoscope should be performed. Attention should be directed to the clarity of the view, optic disk, blood vessels, and macula. On occasion, indirect ophthalmoscopy by an ophthalmologist may be required to view the peripheral retina.

THE RED EYE

Primary care physicians frequently encounter patients with a red eye. Although there are many possible etiologies (Table 1), it is fairly easy to narrow the differential diagnosis by performing a focused history and ocular examination. Several causes of red eyes can be managed in the emergency room. Most important is to realize how urgent a particular condition is and when referral to an ophthalmologist is indicated.

A red eye occurs when there is injury to or inflammation of the anterior aspect of the globe or orbit. The inflammation may be localized to the eyelid, orbit, conjunctive, sclera, cornea, or anterior chamber and iris. Not all causes of red eye are infectious in origin. The infectious entities will be covered in more detail later in the chapter.

Adnexal Disease

Adnexal disease refers to pathology associated with the eyelids, lacrimal system, and orbit.

TABLE 1. *Causes of red eye*

Common causes	Less common causes
Eyelid problems (stye, chalazion, cellulitis, blepharitis)	Corneal ulcer
Conjunctivitis (infectious, allergic, toxic)	Keratoconjunctivitis sicca
Pterygium	Herpes simplex or herpes zoster ophthalmicus
Subconjunctival hemorrhage	Contact lens overwear
Corneal abrasion	Episcleritis/scleritis
Corneal foreign body	Hyphema
Iritis	Endophthalmitis
Chemical injury	Lacerated or ruptured globe
	Carotid cavernous fistula
	Acute angle-closure glaucoma

Cellulitis, dacryocystitis, dacryoadenitis, styes, chalazia, and blepharitis are all examples of adnexal disease causing a red eye and are discussed in detail later in the chapter. In addition, eyelid carcinomas, thyroid disease, orbital inflammatory pseudotumor, and orbital vascular lesions can also cause a red eye.

Subconjunctival Hemorrhage

Subconjunctival hemorrhage occurs when a conjunctival blood vessel ruptures, causing a sudden bright red eye. The appearance is striking and often alarms the patient. There usually are no associated symptoms such as blurred vision or discomfort. Examination with a penlight reveals a deep red hemorrhage under the conjunctiva that may be severe enough to cause elevation of the conjunctiva. Subconjunctival hemorrhages are often associated with a Valsalva's maneuver such as coughing or sneezing, or they may be spontaneous. Other associated disorders include hypertension and blood dyscrasias. The work-up consists only of a blood pressure check. Recurrent hemorrhages require further investigation to rule out blood dyscrasias. There are no visual sequelae of a subconjunctival hemorrhage, and there is no treatment other than to reassure the patient that the blood will resolve within 2 weeks.

Corneal Abrasion

Corneal abrasion refers to an area where the corneal epithelium has been removed. Patients with a corneal abrasion generally give a history of trauma such as being poked in the eye or getting a foreign body in the eye. They complain of pain, photophobia, foreign body sensation, and tearing. There may be blurred vision if the abrasion involves the central cornea. Conjunctival injection will be more intense at the limbus (ciliary flush) as opposed to diffuse injection seen with conjunctivitis. The pupil is generally more miotic on the side with the abrasion. All patients with conjunctival injection should have their cornea checked for fluorescein staining. The diagnosis of an abrasion is made by placing fluorescein dye in the eye and looking for an area of fluorescence with a cobalt blue light or Wood's lamp. The underlying corneal stroma takes up fluorescein dye and fluoresces, whereas intact epithelium does not. A slit lamp may be needed to visualize smaller abrasions. Topical anesthetics such as proparacaine typically relieve symptoms dramatically and make examination easier but should only be used for diagnostic purposes. Never give a supply of topical anesthetic to patients because it is toxic to the corneal epithelium.

The treatment of a corneal abrasion is straightforward and can be handled in the emergency department. First, it is important to rule out the presence of a corneal foreign body or a corneal infection. A corneal infection has a white appearance when examined with a penlight or slit lamp due to the presence of white blood cells, whereas a simple abrasion appears clear.

A simple abrasion is treated with a cycloplegic agent, topical antibiotic, and pressure patch. A cycloplegic agent, such as cyclopentolate 1%, dilates the pupil and relieves ciliary muscle spasm causing pain. A topical antibiotic such as erythromycin ointment prevents infection, and a pressure patch may make the patient more comfortable. The patient is reexamined daily until the abrasion has resolved. If at any time there is evidence of infection indicating a bacterial keratitis, immediate referral to an ophthalmologist is indicated. A moderate abrasion should heal completely in 1 to 3 days. If a corneal foreign body is present and is superficial, it may be carefully removed at the slit lamp with a 25- or 27-gauge needle and then treated as a corneal abrasion.

Chemical Injury

Patients with chemical injuries typically call or come in stating that they have splashed or sprayed something in their eye. A chemical injury is a true ophthalmic emergency, and immediate irrigation is essential. If the patient calls in, have the patient irrigate the eye before

leaving for the hospital. In the emergency department, a quick pH check is helpful, followed by immediate irrigation with sterile isotonic saline. Topical anesthetic drops such as proparacaine will facilitate irrigation. Several liters per eye is required if the pH is abnormal. The fornices should be swept with a cotton swab and the lid flipped to check for particles. The pH should be neutral 30 minutes after discontinuing irrigation to ensure that there is no retained particulate matter. After lavage, examination for corneal staining or haziness can be carried out. Alkali is much more damaging to the eye than acid because it penetrates tissue more readily. Acids form a barrier of precipitated tissue that tends to limit further damage.

Mild chemical injuries resulting in small corneal epithelial defects may be treated as corneal abrasions. If corneal clouding or conjunctival blanching is noted, immediate referral to an ophthalmologist is indicated once the pH has been neutralized. Severe acid or alkali burns can lead to permanent visual loss or loss of the eye.

Iritis

Iritis or anterior uveitis is inflammation of the iris or anterior uveal tract. It is usually unilateral and often occurs spontaneously, although it may occur a few days after blunt traumatic injury. The differential diagnosis for iritis is long and includes autoimmune, infectious, and post-traumatic etiologies (Tables 2 and 3). Patients with iritis typically complain of blurred vision, pain, photophobia, and tearing. They typically do not complain of a foreign body sensation, however. Examination of a patient with iritis reveals a mild to severe decrease in visual acuity, miosis on the affected side, and ciliary injection. These signs and symptoms are nearly identical to those associated with corneal inflammation or injury. The important difference is the absence of corneal staining with fluorescein. The diagnosis of iritis is made by visualizing white blood cells floating in the anterior chamber with a slit lamp. Treatment of iritis consists of dilating drops to prevent scarring and topical corticosteroids and is best left to an ophthalmologist. Long-term topical steroid use may lead to cataracts or glaucoma. Iritis may be a benign, self-limited disease, as in post-traumatic anterior uveitis, or it may lead to severe recurrent episodes of inflammation with permanent visual loss.

TABLE 2. *Characteristics of selected causes of the red eye*

Iritis/keratitis	Conjunctivitis	Angle closure
Pain	Irritation	Severe pain
Photophobia	Discharge	Iritis
Ciliary injection	Diffuse injection	Corneal edema
Miosis	Lids stick	Fixed mid-dilated pupil
		Shallow anterior chamber

TABLE 3. *Differential diagnosis of iritis*

Infectious	Noninfectious
Trauma	Syphilis
Ankylosing spondylitis	Tuberculosis
Inflammatory bowel disease	Reiter's syndrome
Sarcoidosis	Lyme disease
Fuch's iridocylitis	Herpes simplex virus infection
Tumor	Varicella-zoster virus infection
Psoratic arthritis	Postoperative endophthalmitis
Lens-induced	
Ugh syndrome	
Behçet's foreign body sclerouveitis	

Acute Angle-Closure Glaucoma

Acute angle-closure glaucoma is uncommon, constituting 10% of all cases of glaucoma. It is caused by sudden and complete occlusion of the anterior chamber angle by iris tissue. Open-angle glaucoma, which is much more common, generally causes no symptoms. Acute angle-closure glaucoma is a true ocular emergency, as it can lead to complete visual loss in a matter of hours. Patients with acute angle-closure glaucoma complain of severe pain, blurred vision, and photophobia (see Table 2). There is frequently nausea and vomiting due to the

markedly elevated IOP. Examination reveals a hazy cornea due to edema caused by the rapid rise in IOP. The pupil is mid-dilated and fixed. The anterior chamber is shallow, and the IOP is very high (40–70 mm Hg).

Immediate referral to an ophthalmologist is indicated, and treatment with pressure-lowering agents may be started in the emergency department once the diagnosis is confirmed.

Infectious Causes of a Red Eye

Infectious causes of a red eye include infections of the orbit and eyelid, as well as infections of the globe, which are outlined below.

INFECTIONS OF THE ORBIT AND EYELIDS

There are many different presentations of orbital and periocular infections as well as a multitude of potential causative organisms. According to statistics gathered by the National Ambulatory Care Center in 1991, people who believed they or their children had eye infections sought treatment more often from family physicians and pediatricians than they did from ophthalmologists (1). It is beyond the scope of this chapter to cover the entire range of infectious orbital diseases. Rather, an overview of the most common and most important conditions is presented, emphasizing clinical presentation, evaluation, and management.

Preseptal Cellulitis

Preseptal (also called periorbital) cellulitis refers to infection of the soft tissues of the eyelids anterior to the orbital septum (Fig. 2). Induration and erythema of the eyelids may be extensive; however, the globe is generally not involved. Fever is unusual. Chemosis, pain on eye movement, and proptosis or other indicators of orbit involvement are not found. The primary causes of preseptal cellulitis are spread from skin or adjacent structures and trauma. Traumatic preseptal cellulitis is most commonly caused by *Staphylococcus aureus*. Other common causative organisms include *Streptococcus pneumoniae* and, in children, *Haemophilus influenzae*.

FIG. 2. Preseptal versus orbital cellulitis. Marked lid edema may be associated with either entity.

Bacteremia and meningitis are commonly seen in children with preseptal cellulitis; therefore, an evaluation for these processes should be done, guided by the child's clinical condition. Lumbar puncture should be performed only for clinical suspicion of meningitis. The blood, conjunctiva, and nasopharynx are cultured. Preseptal cellulitis without evidence of systemic illness can be treated on an outpatient basis, while the more ill child is hospitalized. Post-traumatic infections are treated with an antistaphylococcal agent (dicloxacillin, cephalexin, oxacillin, or nafcillin). Those that are an extension of sinusitis should receive a third-generation cephalosporin such as ceftriaxone.

Adults usually respond quickly to oral antibiotics and can be treated on an outpatient basis unless orbital involvement is in question. The antibiotic of choice in adults is a penicillinase-resistant penicillin such as oxacillin or dicloxacillin. Outpatients should be followed daily (2–4).

Orbital Cellulitis and Orbital Abscess

Orbital cellulitis is defined as infection of the soft tissue of the orbit posterior to the orbital septum. In contrast to patients with preseptal cellulitis, patients with orbital cellulitis often demonstrate fever, proptosis, chemosis, pain on eye movement, and limitation of ocular motility. Limitation of ocular motility may cause diplopia. Pupillary abnormalities and decreased vision are particularly ominous signs that indicate involvement of the orbital apex. Untreated orbital cellulitis can rapidly lead to loss of vision, loss of the eye, cavernous sinus thrombosis, and intracranial extension. For this reason, evaluation and therapy should be instituted without delay. The most common cause of orbital cellulitis in all age groups is sinusitis, observed in 60% to 80% of cases (5–8); therefore, an evaluation of the sinuses is indicated as part of the work-up. Other causes of orbital cellulitis include extension from periorbital tissues (teeth, lacrimal sac, lids), trauma, postsurgical infection, bacteremia, and endophthalmitis (2,3). The most common organisms are *S. aureus*, *Staphylococcus epidermidis*, *S. pneumoniae*, other streptococci, *H. influenzae*, and anaerobes (7,9–11). Polymicrobial infections are frequent. *S. aureus* is the most common cause of post-traumatic orbital cellulitis (3,4).

A diagnostic work-up includes computed tomography (CT) with axial and coronal views and cultures of blood and nasopharynx. Material from sites of traumatic injury associated with vegetable matter should be sent for fungal culture. A CT scan is important to rule out abscess or, in the case of trauma, intraorbital foreign bodies. Abscesses can occur in the subperiosteal space or within orbital soft tissues. Surgical drainage is usually required if an abscess is present, especially if there is no improvement within 48 hours of the institution of intravenous (IV) antibiotics.

Treatment includes admission and broad-spectrum IV antibiotics. There is some variation in the literature regarding antibiotic recommendations. However, IV therapy should consist of broad-spectrum coverage against staphylococci and *H. influenzae*, as well as anaerobes. Appropriate therapy for orbital cellulitis in adults pending culture results is IV administration of a third-generation cephalosporin plus a penicillinase-resistant penicillin (e.g., Nafcillin). Traditionally, high-dose IV penicillin with a penicillinase-resistant agent was employed (10,12). Patients may be discharged on oral antibiotics, and systemic antibiotics are administered for a total of 2 weeks or 7 days after defervescense (6).

Cavernous Sinus Thrombosis

Cavernous sinus thrombosis may result from posterior extension of an orbital cellulitis, sinusitis, or bacteremia. It is often accompanied by rapid progression of proptosis; bilateral involvement in most patients; cranial nerve palsies including cranial nerves III, IV, V, and VI; papilledema; and severe systemic illness. Sepsis, nausea, vomiting, and signs of meningitis or altered mental status may occur. An orbital apex syndrome, characterized by proptosis, optic neuritis, external and internal ophthalmoplegia, and neuralgia of the ophthalmic division of the fifth cranial nerve, is caused by inflammation around the optic foramen and superior orbital fissure (6). Emergent antibiotic administration and radiologic evaluation are mandatory.

Mucormycosis

Mucormycosis is the most common and most aggressive fungal disease of the orbit (2,13). This potentially lethal infection occurs in patients with uncontrolled diabetes and ketoacidosis and in immunocompromised patients. The fungal pathogen is from the class Zygomycetes and has broad, branching, usually nonseptate hyphae. The ubiquitous organisms enter the nose, invade the mucosa, and spread by vascular extension. Thrombosis, infarction, and tissue necrosis occur, forming a characteristic black eschar. These dark ischemic lesions

of the palate, nasal mucosa, and adnexa are virtually pathognomonic, and direct extension leads to involvement of the paranasal sinuses, orbit, meninges, and brain. Orbital involvement is associated with proptosis, abrupt visual loss, and ophthalmoplegia. A CT scan, ENT consult, and urgent biopsy should be obtained and the biopsy sent for frozen section, stain, and culture. Treatment requires correction of the underlying metabolic disorder and prompt wide local excision of involved tissues as well as systemic amphotericin B. The mortality rate has improved from 88% in 1961 to 15% to 34% in 1987 because of earlier diagnosis and amphotericin B (3,14).

Dacryocystitis

Acute dacryocystitis is an infection of the lacrimal sac. It results from stasis of tears in the sac due to a partial or complete stenosis of the nasolacrimal duct that drains the sac. Clinically, patients present with a painful erythematous mass just inferior to the medial canthal tendon. *Staphylococcus* and *Streptococcus* species are the most common pathogens. There is often a long history of epiphora (tearing) preceding the acute infection. Acute dacryocystitis may occur at any age, and etiologic factors include congenital nasolacrimal duct obstruction, trauma, surgery, sinusitis, and nasal polyps (see Chap. 12 for congenital nasolacrimal duct obstruction). Idiopathic acquired nasolacrimal obstruction is common in adults. Healthy adults are usually not febrile unless the infection progresses. Without treatment, the lacrimal sac may burst, spreading the infection to the lids or orbit or draining through the skin. Treatment may be done on an outpatient basis and includes oral antibiotics such as dicloxacillin or a broad-spectrum cephalosporin and warm compresses (3). Patients should be reevaluated in 24 hours. Once the infection subsides, persistent obstruction requires a dacryocystorhinostomy to prevent recurrent infection.

Dacryoadenitis

Dacryoadenitis is an inflammatory enlargement of the lacrimal gland. It is separated into acute and chronic forms because the clinical presentation, etiology, and treatment are different. Acute dacryoadenitis is an inflammation of the lacrimal gland caused by a viral infection (usually mumps), bacterial infection, or orbital pseudotumor. Although bacteria cause dacryoadenitis less commonly than viruses, *Staphylococcus* and *Streptococcus* are the most commonly associated bacteria. Patients generally present with painful unilateral erythema and swelling of the lateral one-third of the eyelid. Oral erythromycin, tetracycline, or a first-generation cephalosporin is recommended initial therapy until culture results are available.

Chronic dacryoadenitis is more common than the acute form and presents as a nontender enlargement of the lacrimal gland that has been present for more than 1 month. Persistent low-grade inflammation of the lacrimal gland after acute infection can last several months. Other etiologies include trachoma, syphilis, tuberculosis, sarcoidosis, and other systemic conditions (2).

Styes and Chalazia

A hordeolum is an acute suppurative inflammatory mass of the eyelids. It is often infectious, usually caused by *S. aureus* (3,15). An external hordeolum is commonly known as a stye and appears as a pustule on the outer surface of the lid margin. The lash follicle or glands of Zeis or Moll are involved with a focal abscess. Treatment includes warm compresses and topical antibiotic ointment. Systemic antibiotics are rarely used but may be needed for large lesions or cellulitis. If the stye persists, incision and drainage may be necessary.

A chalazion is a granuloma arising from an obstructed meibomian gland. Chalazia are usually not infectious and appear as a nontender mass involving the posterior aspect of the lid

(they generally "point" inward toward the globe). They are very common and often occur farther from the lid margin. There may be a secondary bacterial infection (internal hordeolum). Chalazia are treated with warm compresses or, if persistent, incision and drainage. Erythema and tenderness signify a secondary infection (usually *Staphylococcus*), for which systemic antibiotics are indicated.

Blepharitis

Chronic inflammation of the lid margin is one of the most commonly encountered diseases in ophthalmology. Blepharitis still poses a diagnostic and therapeutic challenge, since it can be very difficult to eradicate. The most common types of blepharitis are the staphylococcal and seborrheic varieties. Patients with staphylococcal blepharitis may be asymptomatic or may complain of burning, itching, and irritation. Chronic staphylococcal infection may cause flaking, loss of lashes, redness, styes, or secondary conjunctival and corneal inflammation. Patients with seborrheic blepharitis (overaction of sebaceous glands) also complain of burning and itching and large, yellow, greasy scales all visible loosely attached to the cilia. The treatment for both consists of applying topical bacitracin or erythromycin ointment to the base of the lashes and scrubbing the lid margin with diluted baby shampoo (15,16). Treatment is generally chronic, as the disease often persists throughout the patient's life.

Other infestations of the eyelids that should not be overlooked include viruses such as herpes simplex, herpes zoster, varicella, molluscum contagiosum, verrucae, and parasites such *Phthirus pubis*.

INFECTIONS OF THE GLOBE

Infections of the globe are generally classified according to the ocular structures involved (e.g., conjunctiva, cornea, vitreous, retina). Serious ocular infections carry the potential for rapid damage to the globe and permanent visual loss. It is therefore imperative to be able to distinguish serious infections from less threatening ones.

External ocular infections include infections of the conjunctiva and cornea (keratitis), which are very common causes of a red eye (see Table 2).

Conjunctivitis

Conjunctivitis is an inflammation of the conjunctiva, which is the mucous membrane covering the sclera anteriorly and the inner surface of the eyelids. Conjunctivitis is the most common cause of a red eye and involves vascular dilation, cellular infiltration, and exudation (Fig. 3). The diagnosis is based primarily on history and clinical presentation and, occasionally, on Gram's stain and culture. Acute conjunctivitis is characterized by a discharge, eyelids sticking together (worse in the morning), diffuse conjunctival injection, and a foreign body sensation of less than 4 weeks' duration. There may be edema (chemosis) of the conjunctiva or small subconjunctival hemorrhages. Typically, pain is minimal unless the cornea is involved. Vision is generally not affected. Depending on the etiology, the discharge may range from watery to frankly purulent. Symptoms often begin in one eye and spread a few days later to the contralateral eye.

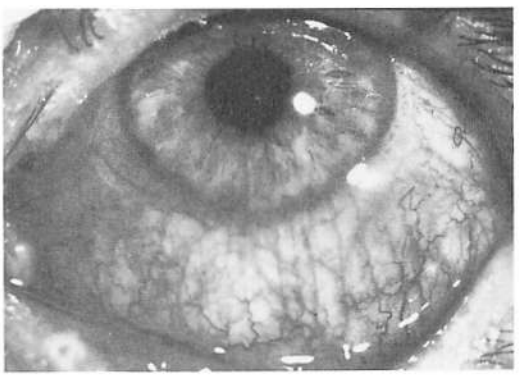

FIG. 3. Conjunctivitis. Marked diffuse conjunctival infection is evident. This patient presented with a watery mucoid discharge.

Table 2 contrasts conjunctivitis to other causes of the red eye.

Most cases of conjunctivitis are viral rather than bacterial. The clinical presentation generally fails to distinguish bacterial from viral etiologies. The discharge of viral conjunctivitis is often watery, while that of bacterial conjunctivitis is more purulent. Viral conjunctivitis is also often associated with preauricular adenopathy, a recent upper respiratory infection, or contact with someone with a red eye.

Viral conjunctivitis is most commonly caused by adenovirus and can produce a spectrum of findings ranging from a very mild follicular response of short duration to a more severe keratoconjunctivitis. Epidemic keratoconjunctivitis is associated with adenovirus 8 or 19 and typically produces a transitory keratitis. Picornavirus is less common and is associated with acute hemorrhagic conjunctivitis.

Acute bacterial conjunctivitis is also common. The most common organisms responsible are *S. aureus*, *S. pneumoniae*, and *H. influenzae* (especially in children).

Most cases of viral or bacterial conjunctivitis are self-limited, typically become worse for the first 4 to 7 days, and may not resolve for 2 to 3 weeks. Transmission is due to sneezing with direct droplet-to-eye contact, hand-to-eye contact, and contaminated water. Both viral and bacterial conjunctivitis are highly contagious, usually for 10 to 12 days from the onset of symptoms. Patients should avoid touching their eyes, shaking hands, sharing towels, and so on. They should not attend school or camp as long as their eyes are inflamed.

Since it is often impossible to distinguish bacterial from viral conjunctivitis clinically and because bacterial conjunctivitis is common and generally mild, cultures are often not taken. Typically, empiric topical therapy is started in addition to cool compresses. Agents that provide the most effective coverage include topical ciprofloxacin (Ciloxan) or trimethoprim sulfate–polymyxin B (Polytrim) drops qid for 5 to 7 days (17). Ciprofloxacin is generally reserved for more serious infections such as bacterial keratitis, however. Other commonly used topical antibiotics include gentamicin, tobramycin, sulfacetamide, erythromycin, and bacitracin. Neomycin-containing preparations should be avoided, as over 25% of the population is sensitive to this antibiotic. Further redness of the eye may result as a natural progression of the disease and is not an indication to stop antibiotics. Corticosteroids should not be instituted, and the eye should not be patched.

It is important to be aware that gonococcal conjunctivitis, if untreated, can rapidly lead to corneal ulceration or perforation. Gonococcal conjunctivitis is known for causing an extremely purulent discharge, marked lid edema, hyperemia, and chemosis. Any highly purulent conjunctivitis requires an immediate Gram's stain to rule out *Neisseria gonorrhoeae*. Patients with gonococcal conjunctivitis are admitted and treated with systemic and topical antibiotics.

Chronic conjunctivitis is defined as conjunctival inflammation that persists for longer than 4 weeks. The differential diagnosis is quite long and includes allergic, toxic, staphylococcal blepharoconjunctivitis, chlamydia, contact lens–related papillary conjunctivitis, molluscum contagiosum, chronic dacryocystitis, and Parinaud's oculoglandular conjunctivitis. Patients with chronic symptoms should be referred to an ophthalmologist. See Chapter 12 for conjunctivitis in the infant.

Bacterial Keratitis

Bacterial keratitis, often called bacterial corneal ulcer, refers to bacterial infection of the cornea. The surface epithelium, mechanical properties of the lids, and bioactive components of the tear film provide an effective barrier to infection. Infection may occur during trauma or when there is a break in the epithelium and may rapidly lead to ulceration of the epithelium and stroma with resultant scarring and loss of vision. Early treatment is essential to minimize loss of vision. If untreated, bacterial keratitis can rapidly lead to corneal perforation, endophthalmitis, and loss of the eye.

Patients with bacterial keratitis complain of foreign body sensation followed by increasing pain, photophobia, redness, and decrease in vision. There is marked inflammation of the conjunctiva with a ciliary flush. There may be tearing or purulent discharge. Frequently, there is associated lid edema, chemosis, and pupillary constriction. A hypopyon (pus in the anterior chamber causing a white fluid line) may be present. The hallmark of the condition, however, is a white infiltrate within the corneal stroma that looks like a "white spot" on the cornea (Fig. 4) and stains with fluorescein.

The principal organisms responsible for bacterial keratitis in the United States are *S. epidermidis*, *S. aureus*, and *Pseudomonas aeruginosa* (especially in contact lens wearers). Other less common etiologic organisms include *Enterobacter* species, streptococci, and *Corynebacterium* species. These organisms account for 89% of cases (18). It is important to note that not all keratitis is bacterial or even infectious. Fungi, *Acanthamoeba* (contact lens wearers), and herpes simplex can also lead to infectious keratitis.

Patients with suspected bacterial keratitis should receive an ophthalmology consult immediately. Prompt initiation of antibiotics is crucial, but only after the proper cultures have been taken. Corneal scrapings are performed at the slit lamp using a heat-sterilized Kimura spatula or a moistened calcium alginate swab (with thioglycolate or trypticase soy broth). In contact lens wearers, the contact lenses and case are also cultured. Routine cultures include blood agar, chocolate agar, Sabouraud's agar (fungi), and thioglycolate broth (aerobic and anaerobic). Slides are sent for Gram's stain (bacteria, fungi) and Giemsa stain (bacteria, fungi, *Acanthamoeba*).

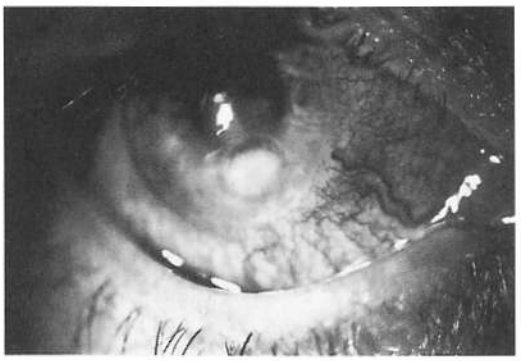

FIG. 4. Bacterial keratitis. There is marked conjunctival infection with a ciliary flush. A typical white corneal infiltrate is present.

In practice, very small peripheral corneal ulcers are not always cultured but are treated empirically, which is adequate 96% of the time (18). The severity of the infection as well as the probable reliability or compliance of the patient dictates whether hospitalization is necessary. If the lesion is small and peripheral and the patient compliant, treatment can be initiated with a topical fluoroquinolone such as ciprofloxacin on an outpatient basis with daily follow-up. For larger ulcers, fortified therapy using cefazolin (50 mg/mL) and either tobramycin or gentamicin (15 mg/mL) provides superior gram-positive coverage, especially for streptococci and enterococci. The antibiotics are formulated in fortified concentrations to provide the highest possible concentrations of antibiotic before toxicity is attained, so that bactericidal levels can be quickly established in the cornea. They are alternated every 30 minutes around the clock for 24 hours, with a gradual tapering over the ensuing days as therapeutic effect is observed. A topical cycloplegic is also initiated, and topical steroids may be added later to minimize the inflammatory response once the cornea has been sterilized. Subconjunctival and IV antibiotics are reserved for cases of impending perforation or when the infection extends into the sclera.

The principal risk factor for fungal keratitis among all age groups is corneal trauma, most commonly in healthy individuals with vegetable matter (e.g., wood chips, tree branch). The onset of a fungal corneal ulcer is very indolent, beginning 2 to 3 weeks after an injury. There is usually a recurrent erosion with a subepithelial infiltrate. The etiologic agents vary according to geographic location and climate, but worldwide the most common cause of keratomycosis is *Aspergillus* species (50%), then *Candida* species (25%), and then *Fusarium* (18).

Herpes Simplex

Herpes simplex virus is one of the most common and serious causes of ocular infection in the United States. Approximately 90% of the population carries antibodies to herpes simplex virus 1. It is second only to trauma as a cause of corneal blindness in this country (15). Primary infection usually occurs between ages 6 months and 5 years. Recurrent ocular herpes, however, is the presentation that usually confronts the clinician.

Symptoms are usually unilateral, and there is often a history of previous episodes of ocular herpes. As in other causes of keratitis, patients typically complain of unilateral redness, pain, photophobia, tearing, and decreased vision. There may be clear vesicles on the eyelids with an erythematous base.

Herpes simplex may cause conjunctivitis, corneal disease, or uveitis, but dendritic keratitis is the most frequent presentation. Dendritic keratitis is caused by virus multiplying in corneal epithelial cells. The characteristic branching "dendrite" stains brightly with fluorescein or rose bengal (Fig. 5). If the disease remains untreated, the branches coalesce, forming a map-shaped or "geographic" ulcer. In uncomplicated cases, these lesions may resolve spontaneously in several days or weeks, leaving an anesthetic cornea, with or without iritis. More severe cases may progress to disciform keratitis or necrotizing interstitial keratitis. Twenty-six percent of patients with a first attack of dendritic keratitis have a spontaneous recurrence within 2 years (18). Most cases of herpes simplex are diagnosed clinically and require no laboratory tests. Immediate ophthalmologic consult is mandatory.

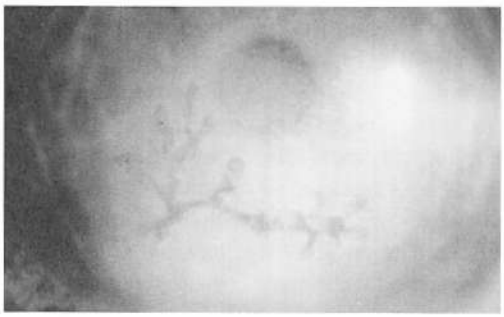

FIG. 5. Keratitis caused by herpes simplex virus.

Treatment depends on the disease process. For eyelid involvement, topical acyclovir administered three times a day to skin lesions may be helpful. Trifluorothymidine 1% drops (Viroptic) or vidarabine 3% ointment (Vira-A) five times a day is also recommended for 7 to 14 days. For corneal epithelial disease, Viroptic nine times a day is recommended as well as debridement of infected epithelium. For corneal stromal disease, a cycloplegic and Viroptic are often used with topical steroids; however, steroids are contraindicated in patients with epithelial disease. Topical acyclovir drops are not currently approved by the Food and Drug Administration in the United States.

Herpes Zoster Ophthalmicus

Herpes zoster (shingles) ophthalmicus is an infection that can involve every structure of the eye by varicella-zoster virus. The majority of infections occur in elderly or immunocompromised patients. Aging, malignancy, iatrogenic immunosuppression, acquired immunodeficiency syndrome (AIDS), local trauma, and irradiation predispose to zoster. The basic underlying mechanism of the disease is direct viral tissue invasion and vasculitis, which lead to tissue ischemia, inflammation, and necrosis. The ophthalmic division of the trigeminal nerve has three branches, namely, frontal, nasociliary, and lacrimal nerves. The frontal nerve is the most commonly affected (20) and supplies the skin of the upper eyelid, forehead, and superior conjunctiva. The nasociliary branch is the main sensory nerve to the globe and supplies the skin of both lids, conjunctiva, sclera, cornea, iris, choroid, and the skin of the tip of the nose. As a general rule, if the patient has vesicles on the tip of the nose (Hutchinson's sign), there is also ocular involvement. There may be a variety of ocular findings, and visual sequelae can be severe. Herpes zoster oph-

thalmicus can cause permanent scarring of the lids, extraocular muscle dysfunction, conjunctivitis, scleritis, keratitis, uveitis, iris atrophy, glaucoma, retinitis, optic neuritis, and postherpetic neuralgia. The keratitis (corneal involvement) may range from punctate lesions and pseudodendrites to disciform keratitis and neurotrophic corneal ulcers. Unlike herpes simplex, the pseudodendrites that involve the cornea are blunt-ended and stain mildly with fluorescein.

Adults with a moderate to severe skin rash present fewer than 3 to 5 days are treated with famciclovir, 500 mg PO tid for 7 to 10 days. If the condition is severe, admission for IV famciclovir is indicated. The role of systemic steroids in the treatment of postherpetic neuralgia is controversial. If there is involvement of the eyelids, conjunctiva, or tip of the nose or if there are ocular symptoms, referral to an ophthalmologist is indicated. Topical antibiotics are used for corneal and conjunctival involvement. Topical steroids and cycloplegics are often used for uveitis, and systemic famciclovir is indicated for retinitis, optic neuritis, or cranial nerve palsy.

Infectious Endophthalmitis

Endophthalmitis or infection within the globe is potentially the most devastating of ocular infections. It is a vision-threatening ocular emergency that warrants immediate and aggressive antibiotic therapy. Over the past decade, emergency department physicians and infectious disease specialists have become increasingly familiar with intraocular infections, particularly because of the rise in cataract surgery.

Endophthalmitis typically develops in three clinical settings: (a) postoperative, 79%, (b) post-trauma, 19%, or (c) endogenously by hematogenous spread of microbes to the eye, 4.4% (21). Endophthalmitis after intraocular surgery is the most common type. The incidence of endophthalmitis after cataract extraction is approximately 0.1% (22–24). There are approximately 1.5 million cataract surgeries performed each year in the United States. Therefore, there are approximately 1500 cases of postoperative endophthalmitis yearly, one half of which result in marked visual loss (22). Postoperative endophthalmitis may be subdivided into acute endophthalmitis, delayed-onset (>2 weeks postoperatively) endophthalmitis, and infections associated with conjunctival filtering blebs from glaucoma or cataract surgery. The organisms most commonly associated with endophthalmitis after surgical therapy are the same as those that colonize the lids, namely, *S. aureus*, *S. epidermidis*, several *Streptococcus* species, and assorted gram-negative rods (23). Coagulase-negative staphylococci and *S. aureus* account for approximately 80% of postoperative endophthalmitis. Endophthalmitis associated with filtering blebs is caused by streptococci in 57% of patients and *H. influenzae* in 23% (25).

Bacterial endophthalmitis following penetrating nonsurgical trauma is surprisingly rare. Retained intraocular foreign bodies appear to be important in endophthalmitis caused by *Bacillus cereus*. Gram-positive bacteria account for 60% to 90% of cases of post-traumatic endophthalmitis, with *B. cereus* and *S. epidermidis* being the two most common organisms. Gram-negative organisms and fungi account for fewer than 20% of cases in all series (23).

Endogenous endophthalmitis resulting from hematogenous spread of organisms from a distant focus is a rare event in the antibiotic era. Risk factors include bacteremia or fungemia from remote sites of infection such as infected heart valves, treatment with immunosuppressive drugs, chronic antibiotic therapy, indwelling catheters, IV drug abuse, and invasive surgical procedures. In endogenous endophthalmitis, organisms enter the retinal or uveal circulation and invade ocular tissue by crossing the blood-ocular barrier. A wide range of organisms, primarily gram-positive, have been reported to cause endogenous endophthalmitis. The most common organisms are *Streptococcus* species, *S. aureus*, *B. cereus*, and enteric gram-negative species (23,24). Compared with postoperative endophthalmitis, *S. epidermidis* is conspicuously absent.

In hematogenous fungal endophthalmitis, ocular involvement may be the first and only manifestation. *Candida albicans* is the most

common fungus reported, accounting for 75% to 80% of all fungal endophthalmitis. Endophthalmitis occurs in 10% to 37% of adult patients with candidemia (23). Periodic ophthalmic examinations are recommended for all patients with documented candidemia. Presence of ocular involvement with *Candida* is also used to document disseminated disease, particularly in neutropenic patients, because candidemia is not demonstrable in as many as 80% of immunocompromised patients with disseminated candidiases (23). The second most common cause of fungal endogenous endophthalmitis is *Aspergillus* species.

Patients with bacterial endophthalmitis typically present 1 to 4 days after surgery or penetrating trauma. The symptoms of endophthalmitis include moderate to severe pain, redness, and decreased vision. Patients typically have marked anterior chamber inflammation, often including a hypopyon (Fig. 6). A marked vitreous inflammatory cellular reaction is also frequently noted, and this may be accompanied by edema and opacification of the cornea as well as lid and conjunctival edema. In endogenous endophthalmitis, the findings are more marked in the posterior chamber initially, and fungal endophthalmitis generally has a more insidious presentation. Frequently, a vitreous haze is present, and fluffy white "balls" may be seen just anterior to the retina.

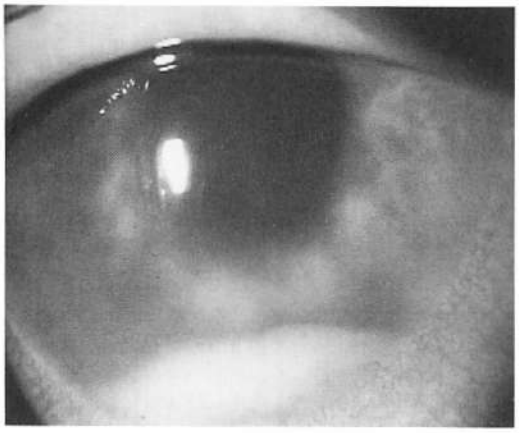

FIG. 6. Endophthalmitis. Ciliary flush of the conjunctiva is noted as well as a hypopyon. The view of the anterior chamber is hazy due to corneal edema.

Endophthalmitis is treated as a medical emergency, and immediate ophthalmologic consultation is obtained. Rapid diagnosis and initiation of therapy are crucial to salvage useful vision. Cultures of aqueous and vitreous humor are obtained, since organisms are frequently cultured from only one of these fluids when both are sampled. Patients must have blood cultures and a work-up for source of infection as well. Intravitreal aspirates are obtained either with a cutting vitrectomy instrument or by a 23-gauge needle passed through the sclera at the pars plana. The fluid is sent for Gram's stain and inoculated directly onto culture media.

Intravitreal antibiotics are administered empirically immediately after aspirates are taken. The bacteria themselves may release toxins and enzymes that destroy the retina, and the host inflammation can also compromise delicate intraocular structures. Antibiotic therapy for endophthalmitis is most effective in preserving vision if initiated during the first few hours following early detection before there is substantial tissue destruction.

The appropriate use of different treatment modalities is controversial. Intravitreal antibiotics are considered the therapy of choice; however, some ophthalmologists advocate vancomycin and amikacin, whereas others prefer a combination of vancomycin, ceftazidime, and dexamethasone. The use of intravitreal steroids is controversial. In addition, it is widely recognized that, although effective, aminoglycosides carry the risk of retinal toxicity. Most ophthalmologists also recommend subconjunctival and topical antibiotics. Recently, the Endophthalmitis Vitrectomy Study found that IV antibiotics were of no added benefit and that immediate vitrectomy was of substantial benefit in patients with light perception vision only (26).

Fungal endophthalmitis is currently treated with intravitreal and IV amphotericin B.

The virulence of the infecting organism appears to be the most important factor determining outcome. Endophthalmitis associated with *S. epidermidis* typically has the best visual out-

come. Results with *S. aureus* are more variable. Streptococci, *B. cereus*, gram-negative organisms, and fungi are all associated with a poor prognosis for vision (23).

Cytomegalovirus Retinitis

Cytomegalovirus retinitis is the most common ocular infection in patients with AIDS. It may also occur in patients with bone marrow and organ transplants and in neonates. Cytomegalovirus retinitis is a congenital or acquired disease that produces progressive retinal deterioration, with marked decrease in visual acuity. See Chapter 15 for diagnosis and management of cytomegalovirus retinitis.

Other Infections

There are a multitude of additional internal ocular infections that are beyond the scope of this chapter to cover in depth. They may cause uveitis, retinitis, or choroiditis depending on the pathogen. They include toxoplasmosis, histoplasmosis, and syphilis. Less common etiologies include onchocerciasis, cysticercosis, toxocariasis, tuberculosis, meningococcus, and rubella. It is perhaps most important for the primary care physician to recognize the signs and symptoms of a potentially serious ocular infection so that prompt referral to an ophthalmologist can be arranged. Resolution of the disease process is then often the result of a collaborative effort between the ophthalmologist and the infectious disease specialist.

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34
Mouth and Salivary Gland Infections

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The oral cavity contains the dental apparatus and provides the terminal drainage point for the salivary glands. It is home to one of the most complex microbiologic habitats in the body. Over 200 bacterial species reside in the mouth (1). There may be up to 10^{11} organisms per gram of collected material in some locations in the mouth (2). Multiple excellent defense mechanisms prevent infections in the mouth and adjoining structures. These mechanisms include the epithelial barrier, the salivary flow, and the complex bacterial interaction of the resident organisms (3). Additional protection comes from specific host defenses that include both humoral and cellular immunity (4). Many of the oral, dental, and salivary gland infections that are commonly seen by physicians can be directly attributed to the breakdown of these protective barriers. Radiotherapy decreases vascular supply to oral tissue and permanently limits salivary flow. Poor dental hygiene can lead to peripical abscess, which in turn can invade the fascial spaces of the neck. Inadequate nutrition and hydration are prime etiologic factors in suppurative sialadenitis.

THE CLINICAL EVALUATION

History

As is true for any chief complaint, a good history must be obtained of any problem involving the mouth, the salivary glands, or the neck. Pain is probably the most common presenting symptom of infection, whether it originates on a mucosal surface, a dental structure, the neck,

TABLE 1. *Differential diagnosis by appearance of common oral mucosal lesions*

Clinical description	Differential diagnosis	Presenting symptoms	Distinguishing features	Diagnostic tests
White lesions	Pseudomembranous candidiasis	Burning	Plaques wipe off easily	Culture, smear
	Hyperplastic candidiasis	—	Adherent plaques	Biopsy
	Trauma—physical, chemical, electrical	Pain	History, sloughing	—
	Bacterial—staphylococcal, streptococcal, diphtheria, *Treponema pallidum*	Pain	Erythema, sloughing	Culture
	Benign keratosis	No pain	Homogeneous	Biopsy
	Dysplasia, squamous cell carcinoma	Mild or no pain	Irregular white patch	Biopsy
Red-white lesions	Candidiasis	Burning	Erythema, plaques	Culture, smear
	Mucositis	Burning, pain	Chemotherapy or irradiation	Culture, smear
	Bacterial—staphylococcal, streptococcal, others	Pain	Erythema, sloughing	Culture, smear
	Dysplasia, carcinoma	Minimal or no pain	Irregular red-white	Biopsy
Red lesions	Atrophic candidiasis	Burning	Depapillation of tongue, erythema	Culture, smear
	Mucositis	Burning	Chemotherapy or irradiation	Culture, smear
	Bacterial	Burning, pain	Erythema	Culture, smear
	Viral (CMV)	Burning	Mucosal erythema	Viral study
	Pemphigoid	Burning	Gingiva epithelial sloughing	Biopsy
	Lichen planus	Burning	Buccal mucosa, tongue	Biopsy
	Erythema multiforme	Burning	History, skin lesions	—
	Dysplasia, carcinoma	Minimal pain	Erythema	Biopsy

TABLE 1. *Continued*

Clinical description	Differential diagnosis	Presenting symptoms	Distinguishing features	Diagnostic tests
Vesicular lesions	HSV, primary	Pain, malaise	Lymphadenopathy, fever, gingival lesions	History, viral studies
	HSV, recurrent	Pain	Lip, gingiva	History
	Coxsackie	Pain	Oropharynx	History, viral studies
	VZV (chicken-pox)	Variable	Skin, oral mucosa	History, viral studies
	VZV (recurrent)	Pain	Neurologic pattern	History, viral studies
	Rubella	Systemic	Buccal mucosa	
HistoryUlcerative lesions	Viral	Pain	Round, 1–2 mm ulcers in clusters	Culture, viral studies
	Aphthous	Pain	Nonkeratinized mucosa, family history, regular borders	Biopsy
	Trauma	Pain	History, irregular borders	—
	Fungal—*Candida, Aspergillus,* others	Variable	Indurated ulcer	Culture, biopsy
	Malignancy	No or minimal pain	Indurated ulcer	History, biopsy
Pigmented lesions	Melanoma, vascular	No symptoms	Location, color	History, biopsy
Masses and tumors	Malignancy	Variable	Size, texture, base, location	History, biopsy

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or a salivary gland. A history of trauma, factors that elicit pain, previous dental care, and previous antibiotic use is essential.

Examination

The Mouth

Proper evaluation of the mouth requires a good light, tongue blades, and gloves for palpation. The light can be a bright flashlight, a head mirror, or a head lamp. All mucosal surfaces and dental structures should be examined carefully and palpated if necessary. Palpation can reveal a loose tooth, tenderness, or fluctuance. Tooth percussion may elicit pain in infected teeth. Oral carcinoma may present with a relatively short history, have some ulceration with secondary infection, and manifest as a painful lesion. Short-term antibiotic treatment may relieve some of the symptoms but will not eliminate the lesion. Close follow-up of intraoral lesions is necessary to ensure that a malignancy will not be missed. Oral mucosal lesions can be caused by a variety of infectious agents and present from plaque-like lesions to ulcerative or vesicular lesions that are red to white in color. Table 1 provides a differential diagnosis of oral lesions by appearance.

The Neck

Swelling of the neck or salivary glands is the most common sign of inflammation of these structures. Fluctuance is an easily identifiable sign that confirms an abscess. Absence of fluctuation in an inflammatory neck mass does not rule out this process. Either needle aspiration or radiologic evaluation may be required for diagnosis.

The Salivary Glands

Swelling and tenderness of the parotid and submandibular glands are the common manifestations of infection in these structures. Careful intraoral inspection may reveal pus coming from the major salivary duct openings opposite the second maxillary molar or in the anterior floor of the mouth. Obstruction of the ducts by a stone may prevent pus or saliva from flowing out of the ducts. This sign is difficult to observe with unaided vision.

THE MOUTH

Oral manifestations of infectious disease that does not arise from the teeth are heterogeneous. Viruses, particularly childhood enanthems, are frequent causes of oral lesions. Other viruses, such as as coxsackieviruses and herpes simplex virus, are also common. Oral lesions may be found in debilitated and immunosuppressed patients (candidiasis, gangrenous stomatitis). Sexually transmitted diseases are also considerations. Table 2 summarizes major infectious oral lesions by entity.

Noninfectious oral lesions also must be considered. Erythema multiforme may present with stomatitis, conjunctivitis, and rash. Stevens-Johnson syndrome is a severe form of erythema multiforme that involves mucous membranes extensively. Vesicles, bullae, and crusted or bleeding lesions may be noted about the mouth. This syndrome may be confused with severe herpetic gingivostomatitis, but lesions of Stevens-Johnson syndrome are more extensive. This is a medical emergency, and the patient should be hospitalized for observation.

Oral burns and other traumatic injuries must be considered and an appropriate history elicited. Burns can be caused by hot foods, caustic substances, or electrical injury (e.g., a child biting an electric cord). Angular cheilosis, painful fissures at the mouth corners, can result from iron deficiency anemia, candidiasis, or local allergic reactions. Systemic lupus erythematosus can lead to oral ulcers and petechiae. Mucositis is suspected in the patient undergoing chemotherapy or irradation. Malignancy must be considered in lesions slow to resolve (see Table 1).

TABLE 2. *Nonodontogenic mouth infectious lesions*

Disease	Symptoms or history	Description	Antibiotics	Comments
Herpangina	Sore throat, odontophagia; in children, refusal to eat	Vesicles on soft palate, tonsils, and uvula, up to 12 lesions	None	Symptomatic treatment
Hand-foot-and-mouth disease	Children: T = 38°–39°C, mouth pain, refusal to eat	Vesicles on buccal mucosa and tongue; peripheral rash	None	Symptomatic treatment
Herpes simplex virus infection	Generalized prodrome	Vesicles, extensive involvement of oral mucosa	None	Symptomatic treatment
Candidiasis	Infants, debilitated, immunosuppressed	Patchy white, plaques, erythema underlying plaques	Nystatin or clotrimazole troche	KOH stain of scraping
Gonorrhea	Recent antibiotic treatment, exposure to STD, fever, malaise	Pharyngitis with exudate	Ceftriaxone (see Chap. 28)	
Syphilis	Exposure to STD		Penicillin (see Chap. 28)	
Primary	Extragenital lesion rare	Chancre on lip, tongue, tonsil		
Secondary		Mucous patch, pharyngitis		Highly infectious
Recurrent aphthous stomatitis	Oral burning, extreme pain 10–20 years	Ulcers 2–5 mm, buccal and labial mucosa, yellow-gray with red margins	None	Anesthetic topical paste

Coxsackievirus Infections

Herpangina

The majority of cases of herpangina are caused by coxsackievirus A. The disease is often seasonal, seen frequently from June to October, with most cases being seen in children and adolescents. It appears less commonly in adults (5). The presentation is similar to any viral pharyngitis, with fever, malaise, and sore throat. Physical examination will show small vesicles anywhere on the mucosal surface of the mouth but most commonly on the structures of the faucial arch (tonsillar pillars and soft palate). These vesicles rupture within a day or two, leaving shallow ulcers. The disease is self-limited, requiring only supportive care. The lesions are painful, so herpangina should be suspected in young children who will not eat.

Hand-Foot-and-Mouth Disease

Coxsackievirus A is also responsible for hand-foot-and-mouth disease. In addition to the oral lesions, there are vesicles or papules on the hands and feet. Seventy-five percent of the cases will occur in children below the age of 4 years. The syndrome lasts 3 to 7 days (6).

Acute Lymphonodular Pharyngitis

Acute lymphonodular pharyngitis is much less common then the other coxsackievirus infections. The initial picture is similar to herpan-

gina as seen in children, with complaints of sore throat, fever, anorexia, and perhaps some lymphadenitis. The lesions consist of raised whitish-yellow nodules located on the posterior pharyngeal wall. There are no oral lesions present. Symptoms last 1 to 2 weeks (7).

Herpes Simplex

Primary herpetic gingivostomatitis is most often caused by herpes simplex virus type 1 (HSV-1) (8). The disease may be seen in all age groups, and the patient will present with a history of generalized prodromal symptoms of fever, headache, malaise, and possibly nausea, followed by development of oral lesions. Vesicles may be noted throughout the oral mucosa (Fig. 1), which, like those in cocksackievirus infections, will rupture and leave ulcers. They may coalesce into large lesions. The gingivitis is more pronounced in the herpetic infection and may be helpful in the differential diagnosis (5). In the healthy patient, supportive care is the only treatment.

FIG. 1. Herpes simplex vesicles on the hard palate. (Reprinted with permission from Bengel W. Differential *Diagnosis of Diseases of the Oral Mucosa.* Lombard, IL: Quintessence, 1989:219.)

Candidiasis

Thrush is a superficial infection of the mucosa of the oral cavity that results in patchy whitish plaques consisting of desquamated epithelial and leukocytic cells. The mucosa surrounding the plaques may be erythematous, and the plaques are easily dislodged. Since *Candida* is a normal inhabitant of the mouth, the infection is opportunistic. It is seen in all types of debilitated patients and may be secondary to antibiotic treatment for any manner of conditions. Several other infectious diseases appear clinically similar to oral candidiasis, so diagnosis depends on obtaining a history of prior antibiotic use or noting the characteristic oral lesions in a debilitated patient. Oral candidiasis is also seen in young children and may be associated with a candidal diaper rash. Breast-fed infants of mothers on antibiotics or with fungal infection in the nipples also may have thrush. Definitive diagnosis requires microscopic examination of the lesions and identification of the yeast. Oral topical nystatin or clotrimazole troches plus elimination of the predisposing factors will usually reverse acute oral candidiasis.

Sexually Transmitted Diseases

Gonorrhea

Gonococcal oropharyngitis is not physically distinguishable from any other acute bacterial oropharyngitis. The patient will present with sore throat, fever, and malaise. Oral examination will show erythema of the oropharynx and tonsils, with occasional mucopurulent exudate. Diagnosis depends on a good history to identify high-risk patients (e.g., homosexuals). Specific instructions to the laboratory requesting culture media (Thayer-Martin) to identify *Neisseria gonorrhoeae* are necessary to obtain accurate mi-

crobiologic identification. Treatment is a single dose of ceftriaxone, 250 mg IM (see Chap. 28).

Syphilis

Primary Syphilis

In primary syphilis, chancre is rarely seen anywhere in the oral cavity. Extragenital oropharyngeal syphilitic chancres were not uncommon many years ago, occurring most commonly on the lip but also on the tongue and the tonsil (9) (see Chap. 28).

Secondary Syphilis

Mucous patches may be seen anywhere in the oral cavity in secondary syphilis. These patches are considered the most highly infectious lesions of this disease (5). The examiner also may see what appears to be a prolonged pharyngitis, with the patient complaining of sore throat over a few weeks and physical examination showing only erythema of the oropharynx and tonsillitis. The appearance of secondary oral syphilis is not distinctive and, as in gonorrhea, requires a high index of suspicion to initiate the appropriate serologic testing to make the proper diagnosis.

Tertiary Syphilis

Gummatous lesions of the palate and tongue are almost never seen (see Chap. 28).

AIDS

There are no unique oral manifestations of AIDS. Patients with this disease will complain of oral masses and difficult, painful swallowing. They will present with oral candidiasis, hairy leukoplakia, herpes simplex, Kaposi's sarcoma, intraoral ulcers, and benign follicular lymphoid hyperplasia (10) (see Chap. 15). These problems rarely are the chief complaint of a patient with AIDS, so the systemic diagnosis usually will have been made. Oral candidiasis may be a marker for esophageal candidiasis (11). If the diagnosis is AIDS, appropriate referral is indicated.

Recurrent Aphthous Stomatitis

Recurrent aphthous stomatitis is a relatively common disease that is characterized by recurrent ulcers of the oral mucosa in patients who are otherwise well. Although commonly thought to be of viral etiology, it appears that this pathology is related to some form of an altered immune response rather than an overt infection (12,13). The disease often may start during adolescence with the individual noting some burning prior to the appearance of small, shallow, painful ulcers most commonly seen on the buccal and labial mucosa. The pharynx and the tonsils are not involved. The lesions are yellow-gray with raised red margins. Lymphadenopathy occurs only after secondary bacterial infection. There may be several lesions, which in most cases will resolve over several days. The patients are often uncomfortable, experiencing pain that may seem out of proportion to the observable lesions. Oral ulcers are also a feature of Behçet's disease, other features of which include genital ulcers and iritis. Treatment of aphthous ulcers is local, consisting of strict oral hygiene and medications containing steroids in a paste vehicle, such as Orabase. In more severe cases, tetracycline mouthwashes (250 mg in 5 mL of water qid) have been found to be helpful in decreasing the symptomatology and duration of the illness (14).

Symptomatic Relief of Oral Infections

Specific therapeutic intervention is the ideal way to deal with mucosal infections of the mouth. In addition, the following symptomatic measures should be considered for the immediate relief of the patient's symptoms:

1. *Diet.* A bland soft diet without a high salt or acid content is useful. Consultation with a dietitian is useful.

2. *Mouthwash.* Simple saline or bicarbonate solutions are helpful in removing thickened secretions or necrotic debris from mucosal ulcers. Hydrogen peroxide in addition to mechanical cleansing of the mouth may be useful in helping periodontal infections in which anaerobic organisms may be involved (15).
3. *Topical anesthetics.* Any of the topical anesthetic agents, such as viscous lidocaine or benzocaine, can provide short-term improvement of symptoms. This is especially useful if the patient's oral discomfort is interfering with fluid and caloric intake.
4. *Coating agents.* Preparations such as milk of magnesia, kaolin-pectin, and amphogel can be used as a palliative oral swish when mucosal ulcerations are present (7). A paste of kaolin-pectin, viscous lidocaine, and diphenhydramine elixir may be prepared.

INFECTIONS OF DENTAL AND CERVICAL SPACES

Odontogenic infections of the mouth are summarized in Table 3.

Dental Caries and Plaque

Dental caries and plaque, with their attendant microbiologic components, are the most common "infectious disease" that affects the mouth. Over 90% of the population of the United States have had dental caries at some time in their lives, and there are close to 500 million untreated cavities in any given year (16,17). Although caries may be considered less than life-threatening, they are responsible for a considerable amount of oral morbidity, which if severe enough can force a patient to seek help in an urgent care or emergency department facility. The primary complaint the patient will have is pain. Without proper radiologic and dental equipment, unavailable in most emergency departments, the diagnosis becomes one of exclusion. The emergency physician must rule out progression of the carious process to pulpitis or periapical abscess. If plaque and caries are left untreated, they become the basis for the more severe and often life-threatening fascial-space infections not uncommonly seen in emergency departments. The emergency physician must educate the patient to the need for dental follow-up and provide appropriate referral.

TABLE 3. *Odontogenic mouth infections*

Disease	Symptoms or history	Description	Treatment	Comments
Pulpitis/periapical abscess	Tooth pain	Localized gingival swelling, tooth percussion tenderness	Penicillin or erythromycin	Dental referral, abscess drainage
Simple gingivitis	Bleeding or pain on toothbrushing or eating	Diffuse gingival inflammation	Dental hygiene	
Acute necrotizing ulcerative gingivitis	Gum pain and bleeding, taste alteration, fever, malaise, lymphadenopathy	Shallow ulcers, gray membrane, fetid odor	Metronidazole or clindamycin or penicillin	Dental referral
Odontogenic cellulitis	History of dental infections, cheek pain and swelling	Buccal induration, tenderness, erythema	Penicillin or erythromycin	Dental referral, admission for extensive involvement

Pulpitis and Periapical Abscess

The pulp of the tooth is the central soft-tissue core containing connective tissue, blood vessels, and nerves, which enter through the apical foramen at the base of the tooth. This area can become infected when caries penetrate the center of the tooth or when periodontitis allows egress of bacteria to the pulp via the periapical foramen. Once pulpitis occurs, the infection can spread out of the apical foramen and form a periapical abscess (Fig. 2). The patient will complain of pain. The pain may be excruciating as pressure builds within the unyielding enamel and bone, or it may be aching if drainage is occurring. Examination may show gingival swelling and erythema adjacent to the affected tooth, and occasionally pus will be coming from the tooth socket (Fig. 3). Percussion of the tooth may elicit tenderness. Compression of the gum over the root tip may also elicit pain. A panorex x-ray may show the periapical bone erosion typical of this process.

Treatment consists of antibiotics such as penicillin or erythromycin for the penicillin-allergic patient and referral for dental evaluation within 24 hours. Tooth extraction or a root canal may be necessary. If an abscess can be identified on the gingiva, dental consultation (if available) may be obtained to drain the abscess, but care must be taken if an abscess exists to avoid dissemination of infection. Analgesia can be obtained with a regional dental block.

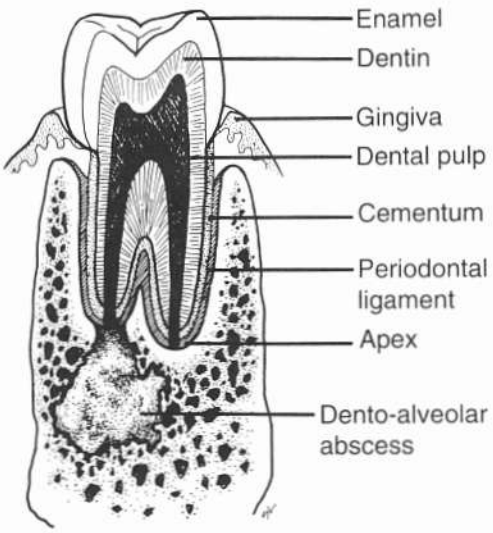

FIG. 2. Anatomy of a tooth with periapical abscess.

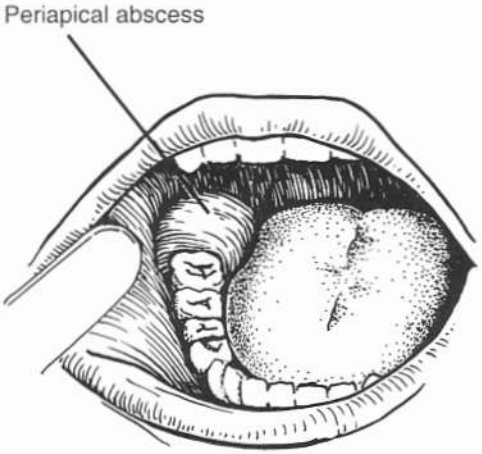

FIG. 3. Clinical appearance of periapical abscesss.

Periodontal Infection

Simple Gingivitis

Simple gingivitis is an almost universally common disorder (18,19) that manifests itself as either an acute or a chronic bacterial inflammation of the gingival tissue surrounding the teeth. Pain or bleeding on toothbrushing or eating may be noted. Over a long period of time, it can result in periapical bone loss and ultimately loss of teeth. There is rarely acute symptomatology associated with this condition. This process is not usually seen as a primary diagnosis in an emergency department. Treatment is improved dental hygiene.

Acute Necrotizing Ulcerative Gingivitis

Acute necrotizing ulcerative gingivitis (ANUG), also referred to as Vincent's angina, trench mouth, and pseudomembranous angina, is an

oral infection that appears rapidly over 24 to 48 hours. Its etiology is not absolutely clear, but there appears to be some relationship to stress (20). It is seen in all age groups and may be associated with any debilitating illness. The onset of pain is acute and may interfere with eating. Taste alterations are noted, and bleeding is common. There is a fetid odor accompanying the infection. The patient has systemic symptoms, including fever, malaise, and regional lymphadenopathy. The lesions seen are shallow ulcers, usually covered by a gray membrane, on the gingival and oral surfaces. Multiple organisms are responsible for ANUG, including streptococci, oral treponemes, *Bacteroides melaninogenicus*, and *Fusobacterium* species (21). Treatment includes local débridement, aggressive oral hygiene, and oral metronidazole, clindamycin, or penicillin. Decisions about hospitalization depend on the patient's circumstances rather than on the oral pathology.

Gangrenous stomatitis (noma) no longer occurs in the United States, but it does occur in debilitated, malnourished children elsewhere. Although similar to ANUG, it is more focal and destroys tissues deeper than the gingiva. The initial painful red spot or vesicle leads to a necrotic ulcer. Extension leads to cellulitis of the lips and cheeks. Admission and high-dose intravenous (IV) penicillin are required.

Cellulitis

The first manifestation of dental infection in the soft tissue of the face and neck is cellulitis close to the offending tooth. The microbiologic process (mixed aerobic and anaerobic), be it any of the previously discussed conditions, has moved from a localized dental infection to the tissues outside the oral cavity. The patient will present to the emergency department with the complaint of pain and swelling in the cheek or skin close to the mandible. The swelling may be intraoral or extraoral. The history should include questions about toothache, trauma, and difficulty with swallowing or talking. Physical examination will confirm the swelling, induration, and tenderness. There may be erythema of the skin, fever, and rarely mild trismus. The differential diagnosis includes nonodontogenic causes of lateral facial swelling. Trauma and trauma-related infections must be considered. In the young child, purplish swelling may indicate *Haemophilus influenzae* cellulitis. Parotitis and insect bite also must be considered. Management includes penicillin, pain medication, and dental referral. Definitive treatment involves tooth extraction or a root canal. If the patient is already on penicillin, then a change to include broader-spectrum antistaphylococcal and antianaerobic antibiotics would be appropriate. Attention to the treatment of this disease is important because the infection may be the precursor of more severe cervical fascial infections. If infection has spread beyond the buccal pouch, otolaryngologic, dental, or maxillofacial consultation is indicated. Intravenous antibiotics or surgical drainage may be necessary.

Infections of the Cervical Fascial Spaces

Most infections of the deep cervical fascial spaces are secondary to sublingual IV drug abuse, odontogenic infections, and oropharyngeal infections. The most common symptoms seen are pain, fever, swelling, dysphagia, and odynophagia (22). Table 4 summarizes these infections. Evaluation of the airway is the most important immediate initial assessment and should be made on any patient presenting with a serious head and neck space infection. Virtually any fascial-space infection or abscess discussed here can be complicated by airway obstruction. The security of the airway outweighs any choice of antibiotic or identification of the offending organism. Even if there is no airway compromise when the patient arrives in the emergency department, it can develop while the patient awaits evaluation by an emergency physician or is being seen by a specialty consultant. If there is any question about airway integrity on initial evaluation, the airway should be secured by whatever appropriate means are available, be it endotracheal intubation or by

TABLE 4. *Cervical fascial space infections*

Infection	Symptoms or history	Location	Antibiotic	Comments
Submandibular abscess	Antecedent dental infection	Mass below and adjacent to jaw	Metronidazole or ampicillin-sulbactam or cefoxitin or cefotetan	Admission, surgical drainage
Ludwig's angina	Antecedent dental infection	Cellulitis of entire submandibular space, floor of mouth elevated and tongue pushed up	Metronidazole or ampicillin-sulbactam or cefoxitin or cefotetan	Admission, surgical drainage
Masticator infection	Antecedent dental infection	Swelling external to posterior mandible	Metronidazole or ampicillin-sulbactam or cefoxitin or cefotetan	Admission, surgical drainage
Pharyngomaxillary (lateral pharyngeal)	Antecedent pharyngitis	Swelling behind posterior pillar of tonsil	Penicillin or metronidazole or clindamycin	Admission, surgical drainage
Peritonsillar infection	Antecedent pharyngitis	Swelling superior to tonsil, uvular deviation	Penicillin or erythromycin or cefazolin	Needle aspiration in emergency department
Anterior visceral space	Esophageal perforation, attempts to place IV in internal jugular vein	Swelling midline and perimidline of neck	Ampicillin-sulbactam or penicillin + metronidazole or cefoxitin or cefotetan	Admission, incision and drainage, may replace with surgical drainage
Retropharyngeal prevertebral	Antecedent pharyngitis, pharyngeal perforation	Erythema, swelling of posterior pharyngeal wall	Ampicillin-sulbactam or penicillin + metronidazole or cefoxitin or cefotetan	Secure airway first, lateral neck x-ray or CT scan, admission, incision and drainage, may replace with surgical drainage

surgical means. Tertiary care centers should not allow transportation of these patients to their facility unless airway protection is ensured.

Head and neck abscesses are caused by a wide variety of aerobic and anaerobic organisms including streptococci, *Staphylococcus aureus*, and *Peptostreptoccus* and *Bacteroides* species (19). In children, *S. aureus* and *Bacteroides* organisms are the pathogens most commonly identified (23). Treatment of most of these infections requires hospitalization, IV antibiotics (penicillin and metronidazole or clindamycin, ampicillin–clavulanate, ampicillin-sulbactam, or chloramphenicol, if there is penicillin allergy) and surgical drainage. An understanding of the anatomy of the fascial spaces that may become infected is important in evaluating a patient presenting to the emergency department with this disease. Levitt's (24) anatomic division of the neck based on the hyoid bone provides an excellent way of approaching these compartments.

Spaces Above the Hyoid

The Submandibular Space

The submandibular space is divided by the mylohyoid into the sublingual (above) and submaxillary (below) spaces (Figs. 4 and 5). Because the submandibular space is adjacent to the mandible, it is most commonly infected secondary to dental infection. The space below the hyoid (submaxillary) is most commonly af-

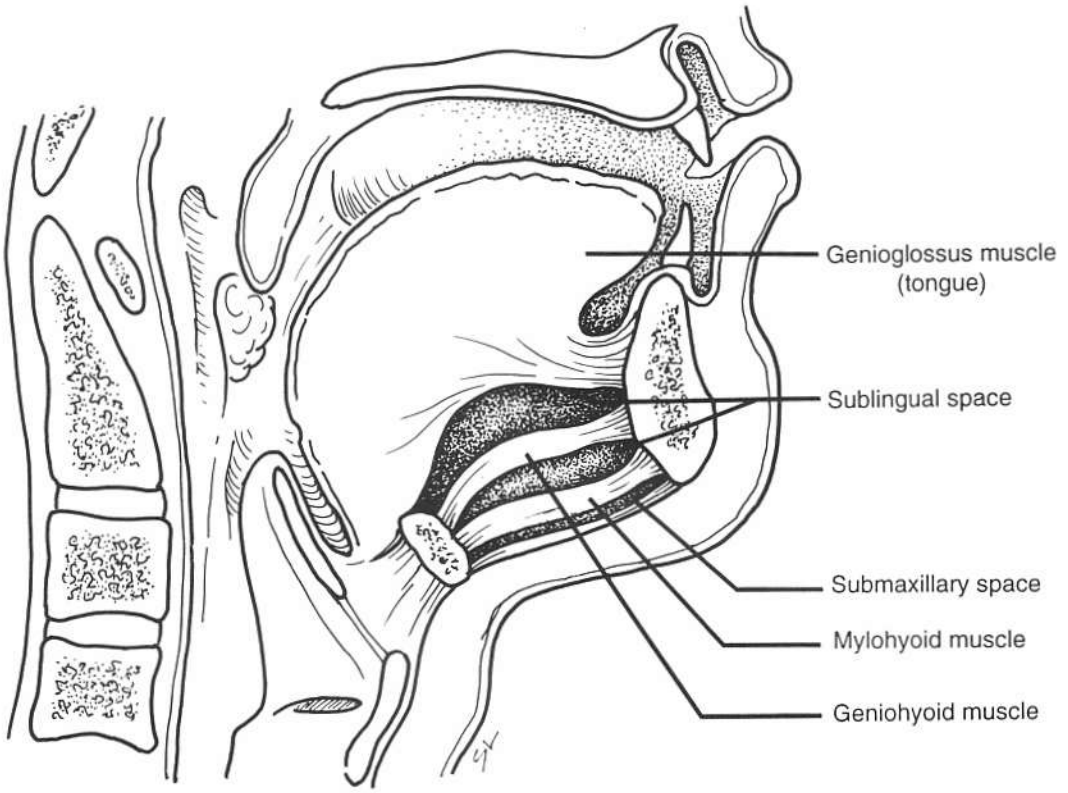

FIG. 4. Sagittal section through floor of mouth. Note submaxillary and sublingual spaces. (Adapted with permission from Paonessa DF, Goldstein JC. Anatomy and physiology of head and neck infections, infectious diseases of the head and neck. *Otolaryngol Clin North Am* 6:572, 1976.)

fected, representing approximately 25% of all neck infections seen in one study (19). The inflammation usually begins from one of the teeth, progresses to a cellulitis, and finally develops into a full-blown abscess. Physically the swelling is obvious just below and adjacent to the jaw. Fluctuation may be difficult to define initially because of the tenseness of the skin. A needle aspiration usually will confirm the diagnosis. This procedure can be performed by emergency department personnel, but if the result is positive, needle aspiration should not be used to empty the abscess cavity, since that could interfere with radiologic delineation of the disease. Hospitalization is indicated in all but the earliest of these infections. If the abscess is small and the patient is not toxic, then the abscess can be treated on an outpatient basis with needle aspiration and oral antibiotics (25). This therapy should not be attempted by anyone without significant experience in the treatment of fascial-space infections of the head and neck.

Ludwig's angina is an infection that, when advanced, involves the complete submandibular space affecting the area bilaterally above and below the mylohyoid (see Figs. 4 and 5). There is a cellulitis throughout the tissues with little or no frank pus encountered (26). Clinically there will be diffuse swelling of the entire submandibular space. The swelling will be woody without any apparent fluctuation. The mouth will be open with the tongue pushed up to the arch of the hard palate secondary to marked elevation of the floor of the mouth. The patient will have mild to severe respira-

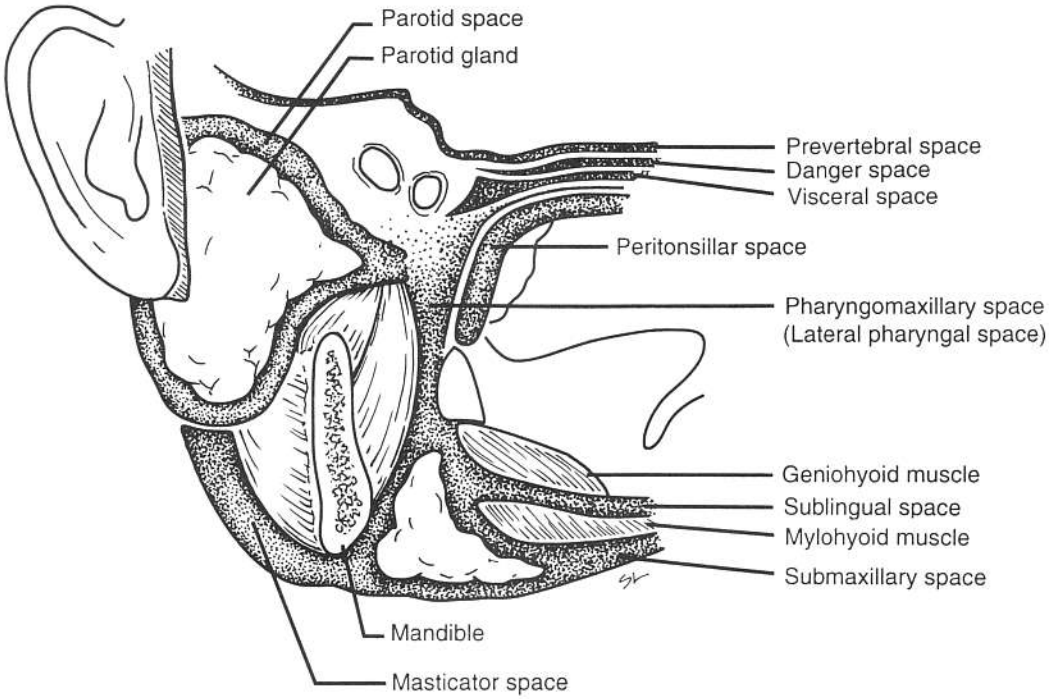

FIG. 5. Oblique section of the angle of the mandible. Note submandibular (sublingual and submaxillary) masticator and pharyngomaxillary spaces. (Adapted with permission from Paonessa DF, Goldstein JC. Anatomy and physiology of head and neck infections, infectious diseases of the head and neck. *Otolaryngol Clin North Am* 6:570, 1976.)

tory distress, have trouble handling secretions, appear toxic, and have a fever. Treatment includes hospitalization, securing an airway (usually a tracheotomy), IV antibiotics, incision, and drainage. On a rare occasion, this disease may be seen in a very early stage, in which the floor of the mouth is only mildly edematous, the tongue is not significantly raised, and there is no respiratory distress. Hospitalization for medical management and careful airway observation may suffice in that situation.

The Masticator Space

As indicated by its name, the masticator space involves all the muscles of mastication, including the masseter, the external and internal pterygoids, and the insertion of the temporalis on the mandible. The ramus and the posterior portion of the body of the mandible form the medial wall for this space (see Fig. 5). Dental infections are the antecedent cause for infection of the masticator space. Swelling is usually external, adjacent to the posterior body and the ramus of the mandible. Occasionally the infection may point intraorally if the medial portion of the space is involved. Trismus is prominent but airway obstruction uncommon, unless the patient does not seek treatment for several days. For most cases, hospitalization and surgical drainage are indicated. A small subset of the patients who have an early lateral abscess may be treated on an outpatient basis with needle aspiration, oral antibiotics, pain medication, and close follow-up (24). Referral to a head and neck specialist for this type of treatment is appropriate.

Infections of the pharyngomaxillary space (lateral pharyngeal) and peritonsillar space are discussed in Chapter 19.

Spaces Below the Hyoid

The Anterior Visceral Space

The anterior visceral space surrounds the central visceral structures of the neck, including the larynx, trachea, and thyroid gland, and lies against the anterior wall of the esophagus (see Fig. 5 in this chapter and Fig. 5 in Chap. 19). It extends from the top of the thyroid cartilage of the larynx down to the arch of the aorta. This space may become infected from an esophageal perforation or from attempts to utilize the internal jugular vein for IV drug use. Clinically there will be diffuse erythema, induration, and tenderness in the midline and perimidline of the neck. Dysphagia may be present because the larynx is mobile during deglutition. An abscess of this space is extremely dangerous because of its direct communication with the superior mediastinum. Hospitalization, IV antibiotics, and appropriate drainage are required.

Spaces Traversing the Entire Neck

The Carotid Sheath

The carotid sheath by itself rarely becomes solely infected except in IV drug users. It can, however, provide a pathway for infection from various other fascial spaces to spread throughout the neck (see Chap. 19).

The Superficial Cervical Space

There is a potential fascial space running from the mandible to the clavicle below the platysma. Isolated abscesses in this space are relatively rare and arise from suppurative breakdown of an infected lymph node or from neck trauma. Involvement more commonly is associated with other contiguous deep-space infections. Because of the superficial nature of this anatomic area, smaller abscesses occurring there lend themselves to outpatient medical and surgical treatment.

Retropharyngeal prevertebral space infections are discussed in Chapter 19.

SALIVARY GLANDS

Infections of the salivary glands are summarized in Table 5.

Acute Infectious Sialadenitis

Bacterial Infections

The parotid and the submandibular (submaxillary) major salivary glands are the likely sites of suppurative sialadenitis. In adults, the debilitated or postoperative state is the setting in which this disease is seen. Up to one-third of the cases of purulent sialadenitis are associated with a postoperative condition (28). Patients presenting to an emergency department are more likely to have dehydration or debilitation as their predisposing factors. Children with viral parotitis may also develop a secondary suppurative infection. In one study of 36 patients with septic parotitis, all the patients were over 77 years of age. Penicillin-resistant *S. aureus* was the most common organism recovered, followed by an occasional *Streptococcus viridans* (29). Clinical diagnosis is not difficult because the patient will present with swelling in the confines of the specific gland involved, usually the parotid. There will be pain, tenderness, erythema, and possible fever. Pus coming from the intraoral opening of the gland duct will confirm the diagnosis. If no purulence is seen, then gentle pressure exerted on the skin overlying the swollen gland, moving from posterior to anterior, may produce a positive result. Mortality from this disease has been reported, so aggressive therapy is indicated (28), usually hospitalization for treatment of the

TABLE 5. *Salivary gland infections*

Infection	Symptoms or history	Location and signs	Antibiotic	Comments
Acute infectious sialadenitis	Debilitated, post-operative, children with history of viral parotitis	Pain, tenderness, erythema, fever Parotid gland: swelling anterior and inferior to ear; Submandibular gland: swelling inferior to body of mandible	Dicloxacillin or cefazolin	Admission
Mumps	Malaise, fever	Pus from salivary gland, bilateral gonadal or CNS involvement	None	
Sialolithiasis	Symptoms with eating	Usually submandibular involved, may be pus from salivary duct, fewer systemic symptoms than acute sialadenitis	Dicloxacillin or cefazolin for bacterial superinfection	Stone visualized on x-ray, sialogogues
Recurrent parotitis in children	Onset <6 years	Periodic unilateral or bilateral swelling	Penicillin	No stone

debilitation and for IV antibiotics effective against penicillin-resistant *S. aureus*. Rarely an abscess developing from a suppurative parotitis will require drainage.

Viral Infections

Mumps (paramyxovirus) was once the most common viral infection seen in the United States, with almost all cases seen in children or adolescents. Since the development of a vaccine in 1967, the number of cases has been reduced by up to 90% (5). The disease is characterized by the sudden onset of bilateral parotid swelling associated with generalized malaise and fever. There may be associated gonadal and central nervous system involvement. Mumps used to be the most common cause of unilateral neurosensory deafness in children. Various other viruses, such as coxsackievirus A, parainfluenza, and echovirus, and lymphocytic choriomeningitis also have been implicated in viral parotitis. Treatment is supportive.

Sialolithiasis

Although not strictly an infectious disease, stones in the salivary glands quite frequently are complicated by secondary bacterial infection. Eighty to ninety percent of stones occur in the submandibular gland (30,31). Presentation includes acute swelling of the gland associated with severe pain. There may be a history of fluctuating swelling associated with eating. Systemic signs are variable, depending on whether secondary bacterial infection has occurred. An enlarged tender gland will be noted on examination. Intraoral examination of the duct orifice for pus will yield variable results depending on the completeness of the obstruction. Careful examination of the floor of the mouth by inspection or palpation may reveal a

stone in the Wharton's duct. Approximately 80% of these stones are radiopaque. A periapical or occlusal x-ray will pick up most submandibular stones, and oblique views of the mandible are useful for the parotid gland. Sialography is a somewhat complicated procedure that, in the author's experience, is not particularly valuable in making this diagnosis. Medical therapy includes antibiotics (antistaphylococcal) and sialagogues (hard candy) when no large stone is noted to stimulate salivation to wash the gland free of small minor stones. If a stone is large enough to be felt within the Wharton's duct, then referral for intraoral outpatient removal is indicated. Parenchymal stones may require surgical removal of the submaxillary or parotid glands.

Chronic Infectious Sialadenitis

There is an intimate relationship between sialolithiasis and chronic infectious sialadenitis. As previously noted, obstruction in the major salivary glands can lead to secondary bacterial infection. Often the secondary infection may resolve with antibiotic therapy, and the acute symptoms of swelling, pain, and tenderness may be relieved. If, however, the obstruction is not eliminated, then the process likely will be repeated in the future. This condition will evolve into a chronic bacterial process that eventually will be resistant to medical therapy and require the removal of the gland or stone for complete resolution.

Recurrent Parotitis in Children

Recurrent parotitis is an uncommon pediatric disease that usually starts in children under the age of 6 years and presents with periodic recurrent unilateral or bilateral parotid swelling. Pathophysiologically there is diffuse sialectasis through the affected gland. The exact etiology of this condition is unknown and is probably a noninfectious inflammatory response. There is often secondary infection during the acute exacerbations, which should be treated with antibiotics. The initial diagnosis cannot be differentiated from any other acute parotitis. It is only over time that the recurrent nature of the disease becomes apparent. Stones are not seen in this disease, and in most cases, the process ceases in the third decade of life. If the child is seen in an emergency department and the infection is the initial episode, treatment should include antibiotics (penicillin) and pain medication.

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35

Acute Undifferentiated Multisystem Infections

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This chapter focuses on a limited number of lethal and hazardous infectious disease in which the emergency medicine physician may perform the pivotal patient evaluation and intervention. These infections were chosen because each one can present as an undifferentiated febrile illness, especially early in the clinical course, and some may be rapidly fatal. Each of these classically involve more than one organ system. Typically, multisystem infections are due to organisms other than the usual pathogenic bacteria and are difficult to identify in the laboratory without special media or technique, which adds to the delay in or failure to

make the diagnosis. All share a problem that the diagnosis ultimately rests on clinical suspicion. All of the microorganisms responsible for the diseases in this chapter are present in the animal kingdom (see Chap. 10).

The emphasis will be on the clinical features and treatment approach most important in the emergency department setting. All require specific travel and exposure histories either to suggest or to exclude their diagnoses. Appropriate and timely chemotherapeutic intervention can be lifesaving in several of the infections. Considerable hazardous risk (particularly to medical personnel) may be associated with exposure to patients infected with these agents, and appropriate management of each infection requires the active participation of local, state, and possibly national public health organizations.

PLAGUE

Etiology and Epidemiology

Plague is a historic disease caused by the gram-negative bacillus *Yersinia pestis*, which continues to produce misery and death worldwide. Human infection results primarily from the bites of infected rodent fleas. The incubation period is 2 to 8 days following the flea bite, and the clinical course can be rapidly fatal without prompt, appropriate antimicrobial chemotherapy. Although plague is endemic in many areas of the world, especially Asia and Africa (see Fig. 1 and Chap. 17), it is predominantly restricted in North America to the southwestern United States. From 1944 through 1994, approximately 90% of the nearly 400 cases of human plague in the United States were reported from four western states (Arizona, California, Colorado, and New Mexico). During each successive decade of this period, the number of states reporting cases increased from three during 1944–1953 to 13 during 1984–1993, indicating the spread of human plague infection eastward to areas where cases previously had not been reported (1). The ecology and the epidemiology of plague transmission are complex, and they differ for the epidemic urban form and the sporadic sylvatic form of disease transmission.

In the United States plague is a seasonal zoonotic infection concentrated in endemic foci throughout the Southwest, with ground squirrels, prairie dogs, and wood rats being important natural reservoirs (2). Although human infection rarely results from direct contact with infected wild rodents or hares, approximately 85% of human infections are associated with bites of infected rodent fleas. Obvious groups at higher risk of exposure include hikers, hunters, and campers, but domestic pets can provide exposure risks by returning infected rodents or infected fleas to their owners, or by becoming infectious risks themselves (e.g., pharyngeal plague in domestic cats).

Clinical Features

Plague can present as any one or a combination of the following syndromes. Classic bubonic plague is described by sudden onset of fever, headache, and malaise, followed shortly by the appearance of extremely painful, localized lymphadenitis (bubo), usually in the groin (see Chap. 9). Gastrointestinal symptoms (including nausea, vomiting, abdominal pain, and diarrhea) are common. The liver and spleen may be tender. Bubonic plague can progress from first onset of symptoms to rapid clinical deterioration and death in 2 to 4 days. In septicemic plague, bacilli disseminate rapidly from the initial focus of infection, resulting in the syndrome of septicemic plague. Plague bacillus may be seen on the blood smear (Fig. 2). By definition, septicemic plague presents with fever and hypotension but without bubo (in contrast to bubonic plague with septicemia). The importance of this distinction is highlighted by the Hull et al. report (3), in which 25% of all cases of plague occurring in over a 5-year period in New Mexico (a state with a high prevalence of plague) were, in fact, the septicemic form. The case-fatality rate was 33%, even in a region where physicians are un-

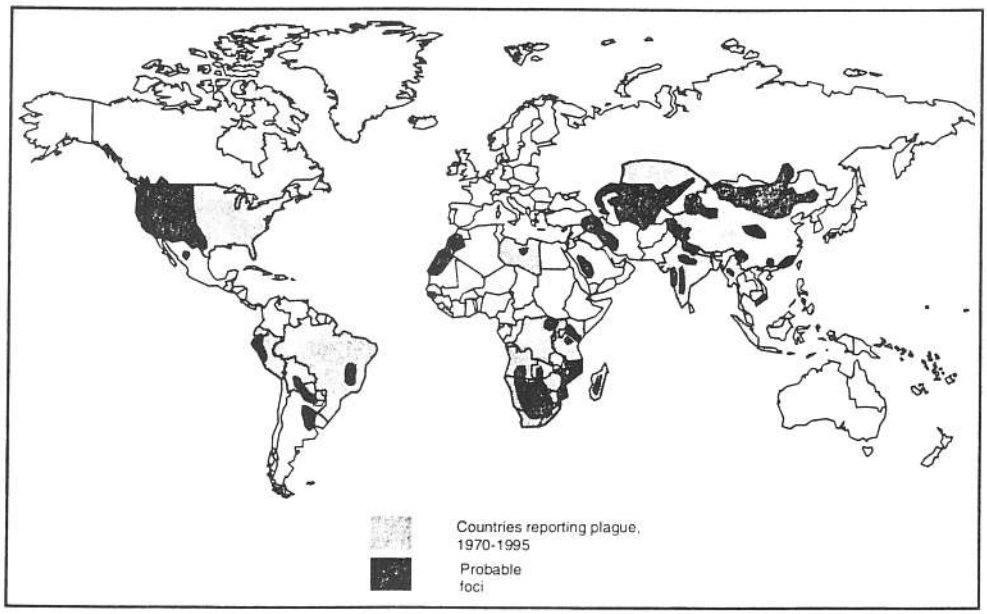

FIG. 1. World-wide distribution of plague. (Reprinted from Centers for Disease Control. Prevention of plague: Recommendations of the Advisory Committee on Immunization Practices (ACIP). *MMWR* 1996;45:2.)

usually sensitized to the possibility of human plague infections. A remarkable feature of plague sepsis is high-density bacteremia, which premorbidly can reach concentrations of thousands of bacilli per milliliter of blood. Hematogenous dissemination of organism to the lung with resultant secondary pneumonia (pneumonic plague) is associated with mortality in excess of 75% (4). Plague transmitted by aerosols is highly contagious. Individuals exposed to the pneumonic form reportedly have become ill and died of primary inhalation plague pneumonia, all in a single day (5). Unusual presentations of plague infections include vesicular eruptions, eschar or ecthyma gangrenosum associated with a bubo, and a meningeal form usually seen as a late complication of inadequately treated bubonic plague. Risk of transmission of plague to close contacts (family, medical personnel, and so forth) is related to exposure to respiratory aerosols from patients with pneumonia and to blood (through needlesticks or aerosols) in patients with high-density bacteremia.

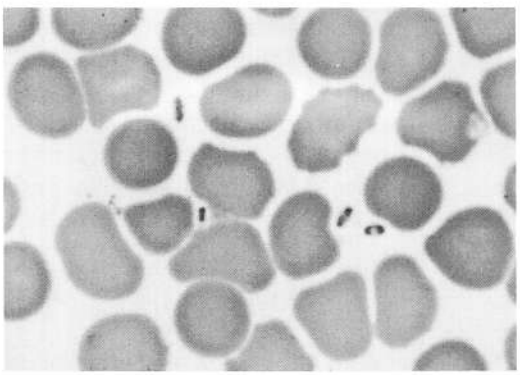

FIG. 2. Plague bacillus (*Yersinia pestis*) on blood smear.

From this discussion, it follows that the emergency medicine physician must consider the diagnosis of plague in certain clinical settings. If a patient has been in a plague endemic region (New Mexico, Arizona, Utah, Colorado,

or California) in the previous 10–14 days (during the seasonal period of March–November) and exposed to rodents or mammals, plague must be included in the differential diagnosis of the following clinical presentations:

- Sudden onset of fever and painful, localized unilateral lymphadenitis
- Clinical sepsis in an individual without obvious focus of infection or without underlying conditions predisposing to sepsis
- Any community-acquired, gram-negative pneumonia

Diagnosis

Plague should be suspected in febrile patients who have been exposed to rodents or other mammals in known endemic areas. The white blood cell count is elevated to 10,000–20,000 cells per mm^3 with many immature neutrophils. Occasionally a leukemoid reaction is seen with white blood cell (WBC) counts to 100,000 per mm^3. Toxic granulations of the WBCs are usually present. Disseminated intravascular coagulation is common as is elevation of liver function tests, particularly bilirubin and aminotransferases.

The bacteriologic diagnosis is made by smear and culture of a bubo aspirate, blood, sputum, or cerebrospinal fluid. Because the bubo does not contain pus it may be necessary to inject some sterile saline into it. Much care, including mask and gloves for the clinician, is required in obtaining the specimens owing to the high contagious nature of *Y. pestis*. A sample of the aspirate should be placed on microscopic slides and air dried. A Gram's stain and Wayson stain will reveal gram-negative coccobacilli. Serologic and passive hemagglutination tests, and cultures are also indicated (6).

Treatment

It should be emphasized that patients suspected of having plague with any evidence of pulmonary involvement should be placed in strict respiratory isolation.

The mortality is estimated to be 50% for untreated plague. Patients require intensive care unit (ICU) admission, usually for adjunctive supportive therapy. Emergent therapy is mandatory. Streptomycin 30 mg/kg body weight per day given in two divided daily doses intramuscularly (IM) for 10 days is the therapy of choice. Tetracycline in a dose of 2–4 g/day in four divided doses for 10 days is an alternative regimen for those who cannot take streptomycin or in whom oral therapy is preferred.

For septic patients or those with meningitis, intravenous (IV) chloramphenicol is recommended at a loading dose of 25 mg/kg of body weight, followed by 60 mg/kg/day in four divided doses. Completion of 10 days of therapy is important. *Y. pestis* resistance to these agents has not been reported, and there is no advantage in using combination therapy.

Local and state health offices should be notified immediately as plague is a reportable disease and to establish a contact surveillance system and to manage the chemoprophylaxis of possible contacts. Contacts of patients with pneumonic plague should receive prophylaxis with tetracycline 2 g/day for 3 to 5 days. Trimethoprim-sulfamethoxazole is acceptable for prophylaxis in children and pregnant women.

LYME DISEASE

Lyme disease is a multisystem tickborne zoonoses caused by *Borrelia burgdorferi*. In 1975 it was first recognized in Lyme, Connecticut. Lyme disease has now been reported in 47 states, Europe, the former Soviet Union, Asia, and Australia (7). Lyme disease is the most common vector-borne infection in the United States (8). Most U.S. cases occur between May and November in the Northeast, Wisconsin, Minnesota, Oregon, and California (Fig. 3).

Etiology

The spirochete, *B. burgdorferi*, is transmitted from deer and rodents as natural animal hosts to humans by the ixodid ticks. *Ixodid dammini (I. scapularis)* is the vector in the northeastern

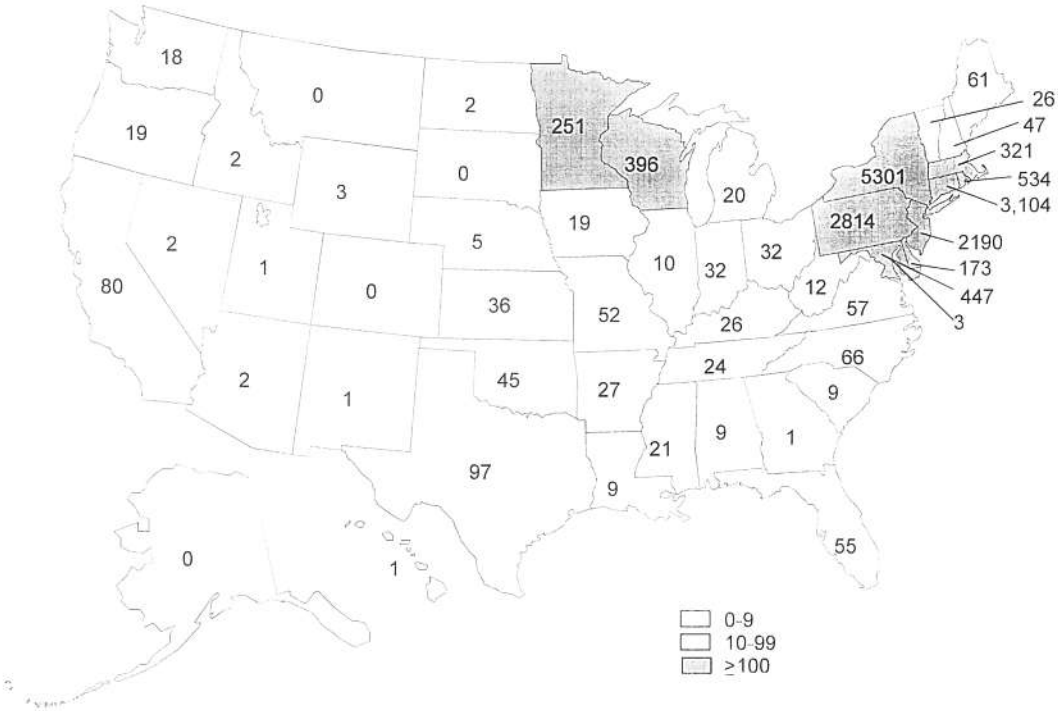

FIG. 3. Number of reported Lyme disease cases, by state, in the United States, 1996.

and midwestern regions of the United States; *I. pacificus* in the western region.

Clinical Features

Erythema chronicum migrans (ECM) is a primary annular erythematous lesion that is a clinically unique manifestation of Lyme borreliosis. It occurs from 3 to 32 days after the bite of ixodid ticks infected with *B. burgdorferi*, and it is observed in 5% to 75% of individuals subsequently diagnosed with Lyme disease. The lesion originates as a macule or papule at the site of the tick bite and evolves as an expanding, annular erythematous plague with central clearing. The most common sites are axillae, groin, and thigh. Within days of onset of ECM as many as half of patients develop secondary lesions that resemble but generally are smaller than the primary lesion. Even without specific antimicrobial therapy ECM and the secondary lesions fade in 3 to 4 weeks (range: 1 day to 14 months).

By convention, the manifestations of Lyme borreliosis have been divided clinically into three stages: early localized infection (stage 1), early disseminated infection (stage 2), and late persistent infection (stage 3). However, the progression and the clinical expressions of Lyme disease are highly variable. ECM represents early, localized (stage 1) infection, but widespread hematogenous dissemination can occur within days of onset, and frequently it is associated with an undifferentiated febrile illness syndrome, with varying and intermittent systemic symptoms (Table 1).

Weeks to months after onset of disease a second phase of illness may ensue, which can involve the central and peripheral nervous systems (15%–20% of patients will have frank neurologic abnormalities, including meningoen-

TABLE 1. *Early symptoms and signs of Lyme disease**

Symptoms	Percentage of cases	Signs	Percentage of cases
Malaise, fatigue, and lethargy	80	Erythema chronicum migrans	100*
Headache	64	Multiple annular lesions	48
Fever and chills	59	Lymphadenopathy	
Stiff neck	48	Regional	41
Arthralgias	48	Generalized	20
Myalgias	43	Pain on neck flexion	17
Backache	26	Malar rash	13
Anorexia	23	Erythematous throat	12
Sore throat	17	Conjunctivitis	11
Nausea	17	Right upper quadrant	
Dysesthesia	11	tenderness	8
Vomiting	10	Splenomegaly	6
Abdominal pain	8	Hepatomegaly	5
Photophobia	6	Muscle tenderness	4
Hand stiffness	5	Periorbital edema	3
Dizziness	5	Evanescent skin lesions	3
Cough	5	Abdominal tenderness	2
Chest pain	4	Testicular swelling	1
Ear pain	4		
Diarrhea	2		

*Erythema chronicum migrans was required for inclusion in this study.
Source: Rahn DW, Malawista SE. Lyme disease. *West J Med* 154:706–714, 1991.

cephalitis with superimposed cranial or peripheral neuropathy); the cardiovascular system (approximately 8% of patients will have cardiac abnormalities, most commonly fluctuating degrees of atrioventricular block); or the musculoskeletal system (about 60% of patients will suffer brief attacks of asymmetric, oligioarticular arthritis, primarily affecting the large joints, particularly the knee). Symptoms can persist for weeks to months and may resolve even without specific antimicrobial therapy.

Late persistent infections (stage 3), which can occur years after primary inoculation and ECM, may be associated with chronic arthritis (typically only one or a few large joints), chronic skin lesions (e.g., acrodermatitis chronic atrophicans), or uncommon, chronic neurologic syndromes (including demyelinating encephalopathy).

Diagnosis

Clinical and serodiagnosis are the mainstay of diagnosis of *B. burgdorferi* infections. The spirochete can be cultured from skin lesions (9). Serologic testing continues to be problematic in that methodology is both not standardized and insensitive early in infection. The enzyme-linked immunosorbent assay (ELISA) is used as a screening test for Lyme disease. IgM antibodies typically appear 2 to 4 weeks after the tick bite and peak at 6 to 8 weeks. IgG antibodies usually do not appear until 6 to 8 weeks and peak at 4 to 6 months. Ninety percent of untreated patients will have elevated IgG response to *Borrelia* (10). False-negative tests do occur but a far more important problem is the number of false-positive tests. Increased specificity is seen with the Western blot, the current "gold standard" for Lyme antibody detection (11). Other tests such as the polymerase chain reaction (PCR) may prove to be more accurate. Another shortcoming of serologic testing is that it cannot distinguish patients with active infections from those who have been previously exposed.

Treatment

Controversy continues concerning optimal treatment of Lyme disease. Most experts agree that early localized disease (erythema migrans)

should be treated with a 3- to 4-week course of either amoxicillin 500 mg PO tid or doxycycline 100 mg PO bid. Stage 2 disease can usually be treated by this same regimen. Those who fail or have meningitis or more severe disseminated disease can be given ceftriaxone 2 g/day IV for 3 to 4 weeks. Cefotaxime, 2 g q8h is equally efficacious. Penicillin at 20 million units/day IV is probably quite effective as well.

In patients whose only evidence for Lyme disease is a positive immunologic test, the risks for empiric, IV antibiotic treatment outweigh the benefits (12,13). Human vaccine trials are currently underway at the time of publication of this text.

BABESIOSIS

Babesiosis is a zoonotic infection of wild and domestic animals that only occasionally is seen in humans. This disease has been documented in many countries; in the United States, it is distributed along the northeast coast and nearby islands (i.e., Nantucket Island, Long Island, Fire Island) and has spread to upper Midwest and west coast. More than 100 documented human infections have been reported with cases being reported with increasing frequency. In healthy people, the infection is usually subclinical or typically mild but may be fulminant in asplenic or immunocompromised patients in particular (14).

Etiology

Members of the genus *Babesia* are called piroplasms because of the pear-shaped intraerythrocytic appearance of the dividing parasites. More than 100 species of *Babesia* exist. *B. microti* is the most common cause of infections in the United States; *B. divergens* is responsible for most of the infections in Europe.

Ixodes ticks transmit the disease to humans from rodents, primarily the white-footed deer mouse, the field mouse, rats, voles, and chipmunks. Following a bite by a *Babesia*-infected tick, the organism penetrates erythrocytes, resulting in hemolysis.

Cotransmission of the Lyme disease spirochete (*B. burgdorferi*) and *B. microti* or other piroplasms may occur inasmuch as they share a common tick vector (15,16). The presence of one disease should encourage the consideration of the others if clinically indicated.

Ehrlichia chaffeenis, the agent for human monocytic ehrlichiosis (HGE), is also found in the same ixodes ticks. Theoretically, co-infection with any combination of these three pathogens is possible.

Pathogenesis

The spleen and macrophage cellular immune functions appear to be important in the host defense to babesia infection. The sepsis syndrome from babesiosis is thought to be due to macrophage mediators, tumor necrosis factor (TNF) and IL-1.

Clinical Features

Cases reported in the United States tend to be less severe than those reported in Europe. However, the spectrum ranges from an occult disease with mild constitutional symptoms to life-threatening hemolytic disease. In adults, the incubation period is usually 1 to 3 weeks, but it can be as long as 3 months (17). Initially the symptoms are nonspecific with fever, rigors, headache, arthralgias, myalgias, and dark urine. Splenomegaly and hepatomegaly are occasionally seen, but lymphadenopathy is absent. There are no characteristic dermatologic manifestation of babesiosis, but if concurrent Lyme disease is present then erythema chronicum migrans may be noted. Hemolytic anemia is usually mild with 1% to 10% of the erythrocytes parasitized. Mild to moderate elevation in renal function and hepatic tests are common as is thrombocytopenia (18,19). The disease is much more serious and may be fatal in asplenic patients, aged individuals, immunocompromised patients or those co-infected with Lyme disease.

Diagnosis

Laboratory findings depend on the level of parasitemia. Anemia, thrombocytopenia, indirect hyperbilirubinemia, elevated liver function tests, and negative blood smears for piroplasms may be found in mild cases. Whereas, in severe cases, hemolytic anemia and hemoglobinuria are always evident. Microscopic examination of Giemsa- or Wright-stained thin or thick blood smears may show the parasitized erythrocytes resembling malaria.

An indirect immunofluorescent antibody (IFA) titer for *B. microti* is available. In most laboratories titers of more than 1:64 are considered consistent with *B. microti* infection. Detection of *B. microti* by PCR was first described in 1992 (20). PCR appears to be more sensitive and as specific as IFA for acute cases.

Treatment

Recommended therapy for human babesiosis is parenteral clindamycin 1200 to 2400 mg/day in three to four divided doses and quinine 650 mg q6–8h, both drugs for 7 to 10 days. Erythrocyte exchange transfusion has been used in seriously ill patients who do not respond to pharmacologic intervention.

HUMAN EHRLICHIOSIS

Two new tickborne zoonoses caused by infection with *Ehrlichia* species have recently emerged as serious illnesses. The first case of human monocytic ehrlichiosis (HME) due to *E. chaffeensis* in North America was reported in 1986. More than 400 cases of *E. chaffeenis* disease have been confirmed in 30 states from Texas and Oklahoma through the southeastern regions and along the eastern coastline to New Jersey. A few cases have also been reported in the Rocky Mountain west (21). A new species of *Ehrlichia*, *E. phagocytophilia,* that infects granulocytes rather than monocytes is associated with human illnesses in the upper midwestern United States. This infection has been named "human granulocytic ehrlichiosis" (HGE) (22).

Etiology

Ehrlichia are unique obligate intracellular bacteria taxonomically distinct from *Rickettsia* and *Coxiella*. *Ehrlichia* are small gram-negative organisms that form intracytoplasmic inclusions called morula in mononuclear cells (*E. equi*, *E. chaffeenis*) or granulocytes (*E. phagocytophila*,). Many other species exist, some of which may also cause disease in humans (23).

Clinical Features

The clinical course may vary from mild to fatal. Following a incubation period of a median of 7 days, patients may develop a multisystem illness starting with fever, (95%–100%), chills, headache (>80%), and myalgia similar to other tickborne febrile diseases. Gastrointestinal symptoms (anorexia, diarrhea), cough, and lymphadenopathy follow (24). Less than one half of patients develop a maculopapular or petechial rash. Complications include respiratory insufficiency, acute renal failure, central nervous system (CNS) abnormalities, gastrointestinal hemorrhage, and death (25,26). The mortality is 2%. Although clinically, RMSF and ehrlichiosis are indistinguishable, thrombocytopenia (50,000–14,000 platelets/mm^3) and leukopenia with elevated hepatic transaminases are more common and rash is less common with ehrlichiosis than with RMSF (27).

Diagnosis

Laboratory diagnosis is serologic using an indirect immunofluorescence assay (IFA). Cultivation of an *Ehrlichia* agent is currently difficult. A PCR method for detecting ehrlichial DNA in blood is now available and shows promise for rapid diagnosis (28). Giemsa-

stained thin blood smear may show morulae within granulocytes cells, suggesting the disease.

Differential Diagnosis

History of recent tick exposure/bite and acute onset of fever, headache, and myalgia suggest a rickettsial or ehrilichial disease as well as several other very serious diseases (Table 2 in this chapter and Table 4 in Chap. 10) (29,30).

Treatment

Tetracycline 500 mg qid or doxycycline 100 mg bid for 7 to 14 days provides successful treatment for human ehrlichiosis.

RICKETTSIOSES—SPOTTED FEVER GROUP

There are seven spotted fever group rickettsioses known to be pathogenic for humans, most are likely transmitted by tick bites. Increased incidence of spotted fever rickettsioses found in many parts of the world remind us to consider these diseases in travelers. However, from a more practical point of view, RMSF is the predominant infection in the United States that requires consideration by the emergency physician, and it will be discussed in this section. All of the spotted fever rickettsial infections have been successfully treated with antimicrobials indicated for RMSF.

Rocky Mountain Spotted Fever

Etiology

The etiologic agent of Rocky Mountain spotted fever is *Rickettsia rickettsii*, and the major vectors responsible for disease transmission in North America are the wood tick (in the Rocky Mountain states) and the dog tick (in the eastern and southern United States). Although reported from nearly all states, the highest prevalence of disease in recent years has been in Oklahoma and North and South Carolina. Between 1981 and 1992, the Centers for Disease Control collected and summarized 9223 cases of RMSF reported from 46 states. North and South Carolina, Oklahoma, and Tennessee accounted for 48% of the reports (31). Essentially all patients with RMSF present in the spring and summer months, and the highest age-specific incidence occurs in children (aged 5–10 years).

Clinical Features

After an incubation period of 4 to 10 days (median is 7 days), Rocky Mountain spotted fever begins initially as an undifferentiated febrile illness, with myalgias and headache, which may be severe. Associated signs and symptoms

TABLE 2. *Differential diagnosis of tickborne febrile illnesses*

Tickborne febrile illnesses	Other serious infections
Rocky Mountain spotted fever	Meningococcemia
Relapsing fever	Toxic shock syndrome
Tularemia	Leptospirosis
Lyme borreliosis	Influenza
Colorado tick fever	Murine typhus
Babesiosis	Q fever
Ehrlichiosis	Typhoid fever
	Bacterial sepsis
	Kawasaki disease
	Plague

are protean (Table 3), and gastrointestinal symptoms (nausea, vomiting, abdominal pain, and diarrhea) may be prominent. Rash is the major diagnostic sign. Typically it begins as a delicate macular eruption on extremities (particularly around the wrists and ankles) and spreads toward the trunk. As the disease progresses, the lesions involve the palms or the soles and become petechial and eventually purpuric. Of note, at least 10% of patients with RMSF have been reported to have no rash, and in approximately 50% of patients the rash does not occur within the first 3 days of illness (32). Most importantly, as many as 86% of patients with ultimately fatal outcomes do not have a rash when seen initially by a physician (33).

Development of hemorrhagic skin lesions is usually a sentinel event in the clinical course

TABLE 3. *Symptoms and signs of Rocky Mountain spotted fever*

Symptom or sign	Hazard (n = 13)	Sexton (n = 75)	Hattwick (n = 778)	Kaplowitz (n = 131)	Helmick (n = 262)	Kirk (n = 48)
Constitutional						
Fever (38°–39°C)	—[a]	—	—	100	99 (73)[b]	—
Fever (>39°C)	—	—	—	88	90 (63)	—
Fever (unspecified)	100	100	99	—	—	100
Shock	—	—	5	17	7 (<1)	—
Death	—	—	9	8	4 (<1)	2
Triad: fever/rash/tick exposure	—	—	—	—	67 (3)	
Cutaneous						
Any rash	100	100	—	90	88 (49)	96
Maculopapular	—	—	91	—	82 (46)	75
Palms/soles	—	80	—	82	74 (28)	—
Petechial, hemorrhagic	—	45	54	—	49 (13)	52
Gastrointestinal						
Nausea/vomiting	—	39	—	56	60 (38)	48
Abdominal pain	—	33	—	34	52 (30)	31
Splenomegaly	15	17	29	14	16 (3)	27
Hepatomegaly	15	—	—	15	12 (2)	27
Jaundice	—	—	—	8	9 (2)	—
Diarrhea	—	—	—	20	19 (9)	19
Neurologic						
Any headache	100	92	92	79	—	64
Headache, mild–moderate	—	—	—	—	91 (71)	—
Headache, severe	—	—	—	—	52 (40)	—
Ataxia	—	—	—	5	18 (7)	—
Myalgias	—	61	83	72	47 (25)	31
Meningismus	24	13	—	—	18 (5)	29
Stupor	77	—	37	21	26 (6)	40
Coma	—	—	13	10	9 (1)	17
Seizures	—	—	—	8	8 (2)	10
Decreased hearing	—	—	—	—	7(1)	—
Miscellaneous						
Conjunctivitis	60	15	—	30	30 (13)	—
Lymphadenopathy	0	—	—	—	27 (13)	25
Edema/periorbital edema	—	—	—	20	18 (3)	—
Pneumonitis	—	—	13	17	12 (2)	—
Arrhythmia	—	—	—	8	7 (1)	—
Myocarditis	—	—	—	26	5 (0)	—
Parotitis	—	—	—	—	4 (<1)	—
Arthralgias	—	17	—	—	—	10
Clinical bleeding	—	—	—	15	—	—
Gangrene	—	—	—	4	—	2

[a]— means data not recorded.

[b]( ) refers to frequency symptom present in first 72 hours of illness.

Source: Weber DJ, Walker DH. Rocky Mountain spotted fever. In: Weinberg AN, Weber DJ, eds. Animal-associated human infections. *Infect Dis Clin North Am* 5:19–35, 1991.

of RMSF, and it may be associated with significant neurologic, pulmonary, or renal complications. Severe neurologic syndromes (including encephalopathy, seizures, and paresis), which have been reported in about 40% of patients (34), may dominate the clinical presentation after the third day of illness. Pneumonitis is common and potentially life threatening (35). Renal insufficiency may be seen in 10% to 15% of cases (32).

Diagnosis

As with other potentially lethal infections that may present as an undifferentiated febrile illness, the presumptive diagnosis of Rocky Mountain spotted fever is predicated on an index of suspicion. Clinical diagnosis remains essential for this life-threatening disease. Acutely ill febrile patients presenting in spring and summer months should have appropriate travel, recreational, and occupational histories reviewed, and they should be specifically queried about tick exposure. A careful skin survey may detect the early-onset rash. Serology is the usual method to confirm the diagnosis. The Weil-Felix test using Proteus OX-19 and OX-2 agglutination is not reliable owing to low sensitivity and specificity. Indirect hemagglutination and enzyme immunoassay are each valid methods for detecting antibodies to specific rickettsial antigens, but they are not immediately useful to the emergency physicians who must rely on a clinical diagnosis of this serious disease.

Immunohistologic demonstration of *R. rickettsii* by skin biopsy and PCR detection of rickettsial DNA in blood can yield a diagnosis of RMSF, but neither method is generally available.

Treatment

The morbidity and mortality of RMSF are significant. The rate of clinical complications is approximately 40%, and the mortality rate in patients treated with inappropriate antimicrobial agents may be as high as 20% (36). Both the morbidity and the mortality of RMSF are increased significantly by delay in specific antimicrobial therapy. A retrospective cohort study of patients with RMSF found a mortality rate of 6.5% in those receiving antirickettsial therapy within 5 days of the onset of symptoms compared to 22.9% for those who received treatment after the fifth day of illness (37).

Doxycycline at 100 mg q12h is the drug of choice. The antibiotic is administered for at least 7 days. RMSF also responds to oral tetracycline 25 to 50 mg/kg/day or chloramphenicol 50 to 75 mg/kg/day given in four divided doses. Severely ill patients require intensive care support and intravenous antibiotics.

For febrile patients with hemorrhagic skin lesions who clearly will be admitted to the hospital, RMSF should be considered in the design of an empiric antimicrobial regimen.

BARTONELLA INFECTIONS

Bartonella species are believed to be worldwide. In those with human immunodeficiency virus (HIV) in particular, these organisms are being recognized with increasing frequency.

Etiology

Nonbacilliformis *Bartonella* species are slow growing, small gram-negative, slightly curved bacilli. Since 1987 the number of *Bartonella* species recognized to cause human disease has increased from two to four (Table 4). Addition-

TABLE 4. *Diseases caused by Bartonella species*

Organism	Disease
Bartonella quintana	Trench fever
	Bacillary angiomatosis
	Bacteremia/endocarditis
Bartonella henselae	Cat-scratch disease
	Bacillary angiomatosis
	Bacillary peliosis
	Bacteremia/endocarditis
	Aseptic meningitis
Bartonella elizabethae	Endocarditis (one case)
Bartonella bacilliformis	Oroya fever
	Asymptomatic bacteremia
	Verruga peruana

ally, based on DNA hybridization *Rochalimaea quintana* was reclassified as *Bartonella quintana*. Special culture techniques accentuate cultivating the organisms.

Clinical Features

Nonbacilliformis *Bartonella* infection causes a broad spectrum of clinical syndromes including nonspecific febrile illness, cat-scratch disease, bacillary angiomatosis, bacillary peliosis, bacteremia, and endocarditis in HIV-infected and HIV-uninfected persons. In HIV seropositive individuals it is usually accompanied by weight loss, malaise, and fatigue, and it may be progressive. Aseptic meningitis and dementia also have been reported in HIV-infected patients (38). In Seattle, *B. quintana* bacteremia or endocarditis was reported in 10 homeless, inner-city persons with chronic alcoholism who were not infected with HIV (39).

Recent tick, flea, louse, or cat exposure may be the best clue to suggest the diagnosis.

Bacillary angiomatosis is a neovascular proliferative process involving the skin, lymph nodes, and a variety of internal organs (40,41). Although it was first described in HIV-infected persons with low CD4 counts (<100 cells per mm^3), it is seen in other immunocompromised, as well as immunocompetent hosts (42). Cutaneous bacillary angiomatosis may be confused with pyogenic granuloma and Kaposi's sarcoma. These nodular lesions frequently appear in crops, are skin-colored or red to purple, and may ulcerate. They range in size from a few millimeters to centimeters in diameter (43).

Bacillary peliosis involves the visceral organs of HIV-infected patients and other severely immunocompromised hosts, some who have concurrent bacillary angiomatosis (44, 45). These inflammatory lesions consist of collections of lymphocytes, histiocytes, and neurophils microscopically resembling nongranulomatous abscesses (46).

Cat-scratch disease (CSD) has now been associated with two organisms: *Afipia felis* and *B. henselae*; the latter seems to be the most common possibly owing to a reported 30% seroprevalance in cats (47–49). CSD is a disease primarily of children and adolescence. The median age of patients with CSD in one large epidemiologic study was 14 years (range 1–64) with the age-specific attack rate highest among persons less than 10 years of age and decreased with increasing age (50). For clinical manifestations, see Chapter 9.

Diagnosis

Until recently we relied on the finding of noncasseating granulomas on lymph node biopsies to make the diagnosis. Diagnosis by culture requires special laboratory technique and expertise. The clinician suspecting *Bartonella* infection should inform the laboratory for alternative approaches to routine blood and tissue cultures. Nonculture diagnostic assays are being vigorously pursued. Several assays have been tested (e.g., enzyme immunoassays, immunofluorescence assay, monoclonal antibodies, and PCR amplification), which are available at selected medical centers or reference laboratories.

Treatment

Recommended therapy for nonbacilliformis *Bartonella* infections is oral doxycycline 100 mg bid or erythromycin 500 to 1000 mg qid for 2–4 weeks. Minocycline, chloramphenicol azithromycin, and clarithromycin are also active against *Bartonella* species. For bacteremia, at least 4 weeks of therapy is indicated (54). Two to 3 months of therapy may be required in HIV-infected patients. For cutaneous bacillary angiomatosis, 2 to 3 months of oral therapy is generally recommended. Parenteral therapy initially is appropriate for more serious forms of disease involving bone or viscera. Relapse is problematic in any patient if the duration of therapy is abbreviated. Relapsing fever and illness is common in untreated immunocompetent patients. In immunocompromised hosts without appropriate treatment, these infections cause progressive illness and death.

BRUCELLOSIS

Brucellosis is a zoonotic disease that exists worldwide but is becoming much less common in the United States. It along with tuberculosis and syphilis are considered to be the most protean in clinical manifestations (51). In the United States, the organism (*Brucella* spp.) is found primarily in domestic animals, especially cattle, goats and sheep, swine, and dogs. Most cases are reported from Texas, California, Virginia, and Florida. Humans become infected by direct contact with infected animals or their carcasses, inhalation from these sources, or by ingestion of unpasteurized dairy products (52). Brucellosis is an important occupational hazard for medical laboratory personnel because the pathogen is highly contagious (53).

Etiology

Brucellae are small, facultative intracellular, gram-negative coccobacilli. *Brucella abortus* is found principally in cattle. *B. melitensis* in goats and sheep, *B. suis* in swine, and *B. canis* in kennel-raised dogs.

Pathogenesis

Incubation period is 2 to 3 weeks. Once inside the host, hematogenous dissemination occurs with localization in multiple organs, especially the liver, spleen, lymph nodes, bone marrow, and kidneys. As intracellular pathogens *Brucellae* are able to survive and multiply. Humoral antibodies and cell-mediated immunity appear to play key roles in recovery and protection against reinfection (52).

Clinical Features

Because of such a myriad of clinical manifestations diagnosis is difficult. The onset may be acute or insidious 2 to 8 weeks after inoculation. Patients complain of multiple nonspecific symptoms (fever, sweats, malaise, headache, myalgia), but with few abnormal physical findings. This subjective-objective discrepancy is an important clue to the diagnosis. Chronic forms of the disease resemble the "chronic fatigue syndrome." Relapse of brucellosis can be insidious and difficult to diagnose as well (53).

Complications, which are common, involve the gastrointestinal tract (70%) (granulomatous hepatitis, cholecystitis, pancreatitis, spontaneous bacterial peritonitis, splenic abscesses); skeleton (20% to 60%) (54); pulmonary system (25%); central nervous system (5%), usually as acute or chronic meningitis with lymphocytic pleocytosis (55), infective endocarditis (<2%), and others. Anemia and thrombocytopenia are common. A rash is infrequent and is reported in about 5% of cases.

Diagnosis

Because of the myriad of nonspecific symptoms and the lack of consistent physical findings for brucella infection, the diagnosis is seldom made in the emergency department setting. Only after time for the clinician to obtain a detailed history will the disease seem plausible. Diagnosis is by bacteriologic confirmation or serologic testing. *Brucellae* spp. may be recovered from the blood in 15% to 70% of cases. The key to obtaining a positive culture is incubating the cultures for at least 30 days because *Brucellae* organisms grow slowly in vitro. Automated culture systems have shortened the isolation from weeks to days (56). False-positive serologic reactions can occur in patients with cholera, tularemia, and plague.

Treatment

Brucella species is an intracellular organism; therefore, clinical efficacy correlates with prolonged treatment with agents that have good penetration into cells. Doxycycline 100 mg/day (or tetracycline) orally for 6 weeks in combination with an aminoglycoside (streptomycin or gentamicin) for 2 to 3 weeks is the regimen preferred by many experts. This combination therapy, which is recommended to prevent relapses, does have a relapse rate of less than

5%. In 1986, the Joint Food and Agriculture Organization/World Health Organization Expert Committee on Brucellosis recommended therapy with the combination of doxycycline (2 g/day) plus rifampin (600–900 mg/day) for 6 weeks. This regimen compares favorably with doxycycline/streptomycin in comparative studies (57). Ofloxacin 400 mg/day when used in combination with rifampin 600 mg/day for 6 weeks has yielded comparable results to doxycycline plus rifampin (58). Finally, note that treatment of life-threatening complications such as endocarditis or CNS infections require a much longer course (i.e., 6–9 months).

LISTERIOSIS

Listeriosis is an uncommon infection but with a high mortality rate. Although generally considered a zoonosis, most cases in the United States occur in urban areas and are foodborne. In these cases, the sources implicated are vegetables, dairy products, and meats (59–62). Hospital outbreaks have also been reported. The annual incidence of listeriosis is less than 1/100,000 cases (63). An estimated 1965 cases and 248 deaths occurred in 1993. Most listeria infections occur in immunocompromised hosts, particularly patients with cancer, collagen vascular diseases, diabetes mellitus, and HIV infections; patients receiving corticosteroid therapy; and pregnant women, newborns, and others with serious illnesses (64,65). Only approximately 15% of listeria infections occur in previously normal and healthy adults (66).

Etiology

Listeria monocytogenes is a gram-positive, nonspore-forming aerobic bacillus. It is ubiquitous in nature having been isolated from mammals, birds, fish, crustaceans, ticks, food, soil, water, and so forth.

Clinical Features

Listeriosis is well known for its varied clinical syndrome, but usually it presents as bacteremia or meningitis, with pregnant women accounting for up to a third of reported cases. Listeriosis in pregnancy presents with fever, chills, and often back pain with no other clues at time of presentation; only a positive blood culture reveals the diagnosis. The pregnant woman's fever may subside without treatment. Premature labor with birth of a dead or infected baby may occur. Babies infected in utero and alive at birth are seriously ill with granulomatosis infantiseptica. The mortality is nearly 100%.

A third clinical syndrome is sepsis in adults, usually immunocompromised, or in neonates. The features are similar to those of gram-negative sepsis and a blood culture is required for diagnosis. Acute meningoencephalitis, a fourth clinical syndrome, may complicate sepsis or it may occur in a more subtle form, often subacute in adults. Listeria is a common cause of meningitis in organ transplant and cancer patients. It is the fourth most common cause (10%) of meningitis (67,68). In addition, cirrhotic patients have increased incidence of listerial infection. Acute cerebritis with focal neurologic findings is being increasingly reported (69,70).

Finally, focal infections involving any tissue or organ may occur (71). Unfortunately, there is nothing distinctive about the clinical lesions.

Diagnosis

Listeria can be identified in the microbiology laboratory from appropriately submitted clinical specimens. It grows well on blood agar with the colonies resembling group B β-hemolytic streptocci. On Gram's stain the organisms may be interpreted as a diphtheroid and therefore considered a contaminate.

Treatment

Ampicillin or penicillin plus gentamicin in combination with either drug is the preferred antibiotic regimen for most clinical syndromes (72,73). Trimethoprim-sulfamethoxazole or vancomycin can be used in penicillin-allergic patients. Therapy duration is from 2 to 6 weeks

depending on the syndrome, state of the host, and illness severity.

LEPTOSPIROSIS

Leptospirosis is an acute multisystem zoonosis of worldwide distribution. As a zoonosis humans are infected by direct or indirect contact (water or soil contaminated with infected urine) with infected animals, usually mammals. In the United States the most common sources are dogs, livestock, rodents, wild mammals, and cats (74).

Etiology

Leptospiras are spirochetes that are different than other spirochetes in that they can be grown on artificial media. Humans are infected by a single species, *L. interrogans*, but there are over 200 serovars (serotypes) of this species (75).

Pathogenesis

Following accidental inoculation of *Leptospira* through the skin or mucous membranes, the organism enters the bloodstream and spreads to all organs including the eye and CNS. Vasculitis is the principal pathologic disorder responsible for the clinical manifestations.

Clinical Features

Two clinical syndromes are associated with Leptospira infections: a milder anicteric form and a severe icteric form (Weil's disease). In the milder form, leptospirosis is characterized by sudden onset of fever, severe headache, severe myalgias, nausea, vomiting, and abdominal pain. No specific physical findings are present in this early stage. By day 7, the illness subsides and a second phase may not occur. If the second stage does appear, the predominant findings are intense headache and severe myalgias. Nausea and vomiting are present in 95% of the patients. In this phase the clinician will find muscle tenderness, lymphadenopathy, hepatosplenomegaly, conjunctival suffusion, and rashes. Pulmonary infiltrates are reported in 20% to 70% of patients, and they are primarily hemorrhagic (75) and 50% of patients will have clinical signs of aseptic meningitis (75).

Icteric leptospirosis is the severe form of the infection with a mortality of 5% to 10%; it is characterized by jaundice due to impaired hepatic function and acute renal failure from acute tubular necrosis. Finding elevated creatine phosphokinase and liver function tests in an acutely febrile jaundiced patient suggests leptospirosis.

Diagnosis

The definitive diagnosis is by cultivation of leptospiras from body fluids or tissues. Isolation of leptospiras requires special laboratory techniques and media.

The diagnosis of leptospirosis is frequently missed. Leptospirosis is endemic to Hawaii, but during an active surveillance study on the islands, physicians suspected leptospirosis at the time of initial clinical presentations for only 30% of the patients in whom the diagnosis was later confirmed (76). The definitive diagnosis is by cultivation of leptospires from body fluids, including urine, beginning in the second week of the illness, or from tissues. Isolation of leptospires requires special laboratory techniques and media.

The most common method of diagnosis is through serologic tests. Several tests are available, such as the microscopic agglutination test, ELISA, and dot-ELISA procedures, and PCR assays.

Treatment

For severely ill patients, treatment with either penicillin G (1.5 million units q6h) or ampicillin (500–1000 mg q6h), should be initiated intravenously. In mild-to-moderate illness, doxycycline (100 mg bid), ampicillin (500–750 mg q6h), or amoxicillin (500 mg q6h) orally administered is recommended. Treatment should be given for 5 to 7 days (77).

TULAREMIA

Tularemia is a zoonotic infection, and it is present in most parts of the world. In the United States it has also been called "rabbit fever, deerfly fever, and market men's disease." Significant morbidity and mortality are associated with this disease.

Etiology

Francisella tularensis, a small, aerobic pleomorphic, gram-negative coccobacilli, is the agent of tularemia. There are several biogroups, some more virulent than others. Transmission of *F. tularensis* to humans occurs most often through an insect bite or contact with contaminated animal products. Ticks and flies are the main vectors in the United States. At least 13 species of ticks, including the dog tick, wood tick, and Lone Star tick, are naturally infected with *F. tularensis*. Direct inoculation through inapparent skin breaks, airborne transmission, and ingestion associated with animal contact is another important mode of *F. tularensis* transmission to humans.

Tularemia is primarily a disease of the northern hemisphere. Since the 1950s, a steady decline in cases in the United States has been recognized with over 50% reported from just three states, Missouri, Arkansas, and Oklahoma (78). The bimodal peak incidence in human infections correlate with a summer peak associated with tick bites and a winter peak associated with direct animal contact during hunting and trapping activities.

Clinical Features

Patients will present to the emergency department with one of six predominant features: glandular, ulceroglandular, oculoglandular, pharyngeal, typhoidal, or pneumonic. Following an average incubation period of 3 to 5 days, patients develop an acute undifferentiated febrile illness with associated chills, headache, malaise, and fatigue. In the glandular form, fever up to 101°F and tender regional lymphadenopathy persist for weeks or months without treatment. Ulceroglandular is the most common form, and it is most quickly recognized. The skin lesion often starts as a small painful erythematous papule that then undergoes necrosis, leaving an ulcer with a very tender, enlarged regional lymph node. Notably, lymphangitis is rare. Inguinal adenopathy is most common in adults; cervical and occipital in children (79). Oculoglandular tularemia, which is uncommon, results from contaminated fingers or aerosols coming into contact with the eye. Unilateral painful conjunctivitis with either preauricular, submandibular, or cervical lymphadenopathy is typical of this form. The differential diagnosis of fever and lymphadenopathy is covered in Chapter 9. Pharyngeal tularemia is also uncommon and results from infection of the oropharynx by infected aerosols or food. Pharyngeal exudate with cervical adenopathy resembles streptococcal pharyngitis (79). Chapter 10 discusses other zoonotic causes of pharyngitis, and the differential diagnosis of oropharyngitis is reviewed in Chapter 19.

The most difficult form to diagnose is the typhoidal tularemia. Rapid deterioration and death is characteristic from the multisystem infection by *F. tularensis* (79). Prominent symptoms and signs include high fever and chills, abdominal pain and diarrhea, hypotension, acute renal failure, disseminated intravascular coagulopathy, hepatitis, meningitis, and (in nearly 50% of the patients) pneumonia and/or pleural effusions. This septic presentation always demands hospital admission and differentiation from other causes of sepsis, acute endocarditis, and other difficult diseases to diagnose such as typhoid fever, malaria, rickettsioses, ehrlichiosis, and babesiosis (80). See Chapter 10 for a more extensive differential of zoonotic infections.

Pneumonic tularemia is one of the more obscure atypical pneumonias (see Chap. 21). Patchy infiltrates or lobar consolidation with effusion in upper or lower lobes is recognized and, as in other forms of pneumonia, is not

characteristic of the causative agent. Acute respiratory distress syndrome may complicate the pneumonic form of disease. Clues to the diagnoses is history of possible exposure to *F. tularensis* or failure to respond to routine pneumonia therapy (79).

Finally, unlike plague in which skin rashes are uncommon, rashes including diffuse maculopapular and vesiculopapular rashes, as well as erythema nodosum, erythema multiforme and urticaria are common in tularemia (79).

Diagnosis

Routine gram-stained smears and cultures of fluid or tissue samples do not ordinarily reveal *F. tularensis*. Special media is required to support the growth of this organism, which means timely communication with laboratory personnel is paramount for diagnosis. In addition, tularemia is a significant occupational infectious disease risk for unsuspecting laboratory workers. Serologic methods are most commonly relied on for diagnosis. ELISA, tube agglutination, hemagglutination, and microagglutination are all available. ELISA is most preferred (79). PCR amplification appears promising.

Treatment

The mortality for untreated or inappropriately treated patients with tularemia is as high as 33% (81). Appropriate treatment lowers the death rate to less than 4%. The therapy of choice for all forms except meningitis is streptomycin for 7 to 14 days. The adult dose is 7.5 to 10 mg/kg IM q12h; pediatric dose is 30 to 40 mg/kg IM in two divided doses. Some variations of these doses exist depending on age, complications, and illness severity. Gentamicin has also proved to be efficacious. Tetracycline and chloramphenicol are not preferred because they are bacteriostatic for *F. tularensis* (78). Although, some advocate the use of chloramphenicol with streptomycin to treat meningitis; others recommend the third-generation cephalosporins. Many other agents, such as the fluorquinolones and imipenem, show promise.

A note about prevention is appropriate. Although *F. tularensis* is a biohazard for laboratory personnel, emergency department or hospitalized patients with tularemia do not need special isolation because person-to-person spread does not occur.

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