Chapter 5

EAR, NOSE, AND THROAT CONDITIONS

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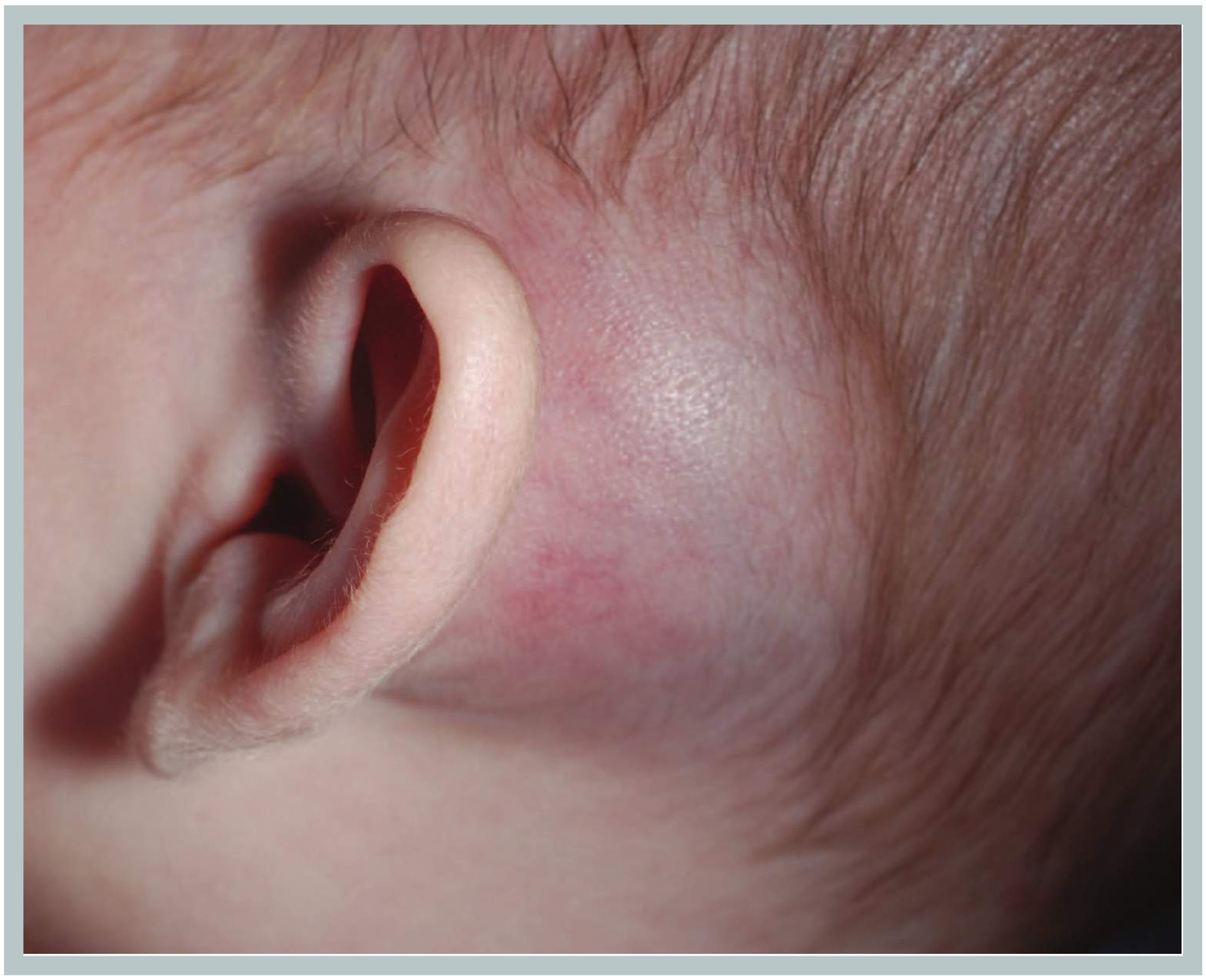

Mastoiditis. Postauricular redness, swelling, and proptosis in a young child with acute mastoiditis. (Photo contributor: Lawrence B. Stack, MD.)

The authors acknowledge Sean Barbabella and Francisco Fernandez for portions of this chapter written for the previous editions of this book.

Clinical Summary

Children between the ages of 6 months and 2 years are at highest risk of developing acute otitis media (AOM). Children at increased risk of recurrent AOM contract their first episode prior to 12 months, have a sibling with a history of recurrent AOM, are in day care, or have parents who smoke.

AOM is an acute inflammation and effusion of the middle ear. Viral, bacterial, and fungal pathogens may cause AOM. The pathogenesis of bacterial AOM is eustachian tube dysfunction, typically following a viral infection, allowing retention of secretions (serous otitis) and seeding of bacteria. The most common bacterial isolates are Streptococcus pneumoniae, Haemophilus influenzae, Moraxella catarrhalis, and Streptococcus pyogenes. There is an increased prevalence of antimicrobial resistance for S pneumoniae and β-lactamase-producing strains of H influenzae. Vaccination for strains of S pneumoniae has resulted in a modest decrease in incidence of AOM.

Patient presentations and complaints vary with age. Infants with AOM have vague, nonspecific symptoms (irritability, lethargy, and decreased oral intake). Young children can be irritable, often febrile, and frequently pull at their ears, but they may also be completely asymptomatic. Older children and adults note ear pain, impaired hearing, and occasionally otorrhea.

Otoscopy should focus on color, position, translucency, and mobility of the tympanic membrane (TM). Compared with the TM of a normal ear, AOM causes the TM to appear dull, erythematous or injected, bulging, and less mobile. The light reflex, normal TM landmarks, and malleus become obscured. Pneumatic otoscopy and tympanometry enhance accuracy in diagnosing AOM.

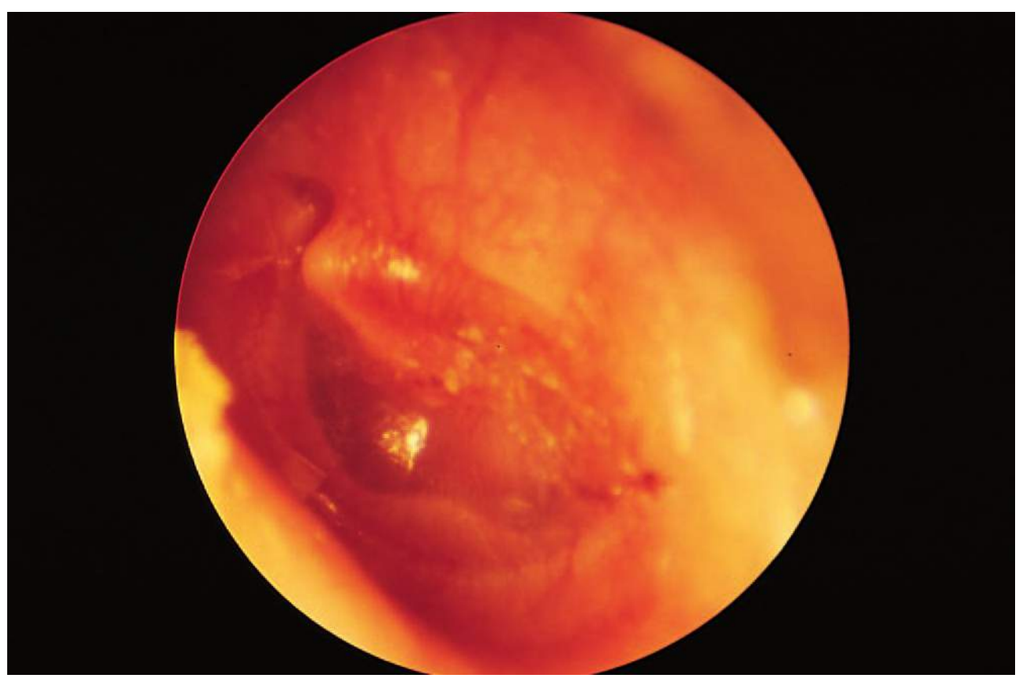

FIGURE 5.2 ■ **Early Acute Otitis Media.** A mildly erythematous tympanic membrane is seen with a small purulent effusion in the middle ear. (Photo contributor: C. Bruce MacDonald, MD.)

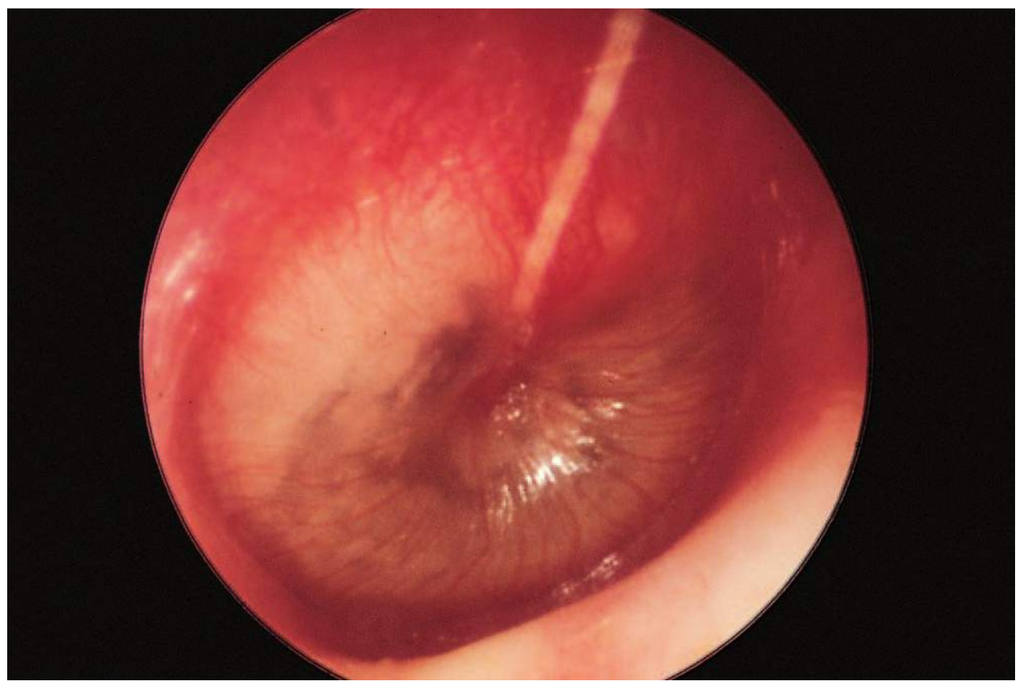

FIGURE 5.3 ■ **Acute Otitis Media.** The middle ear is filled with purulent material behind an erythematous, bulging tympanic membrane. (Photo contributor: Richard A. Chole, MD, PhD.)

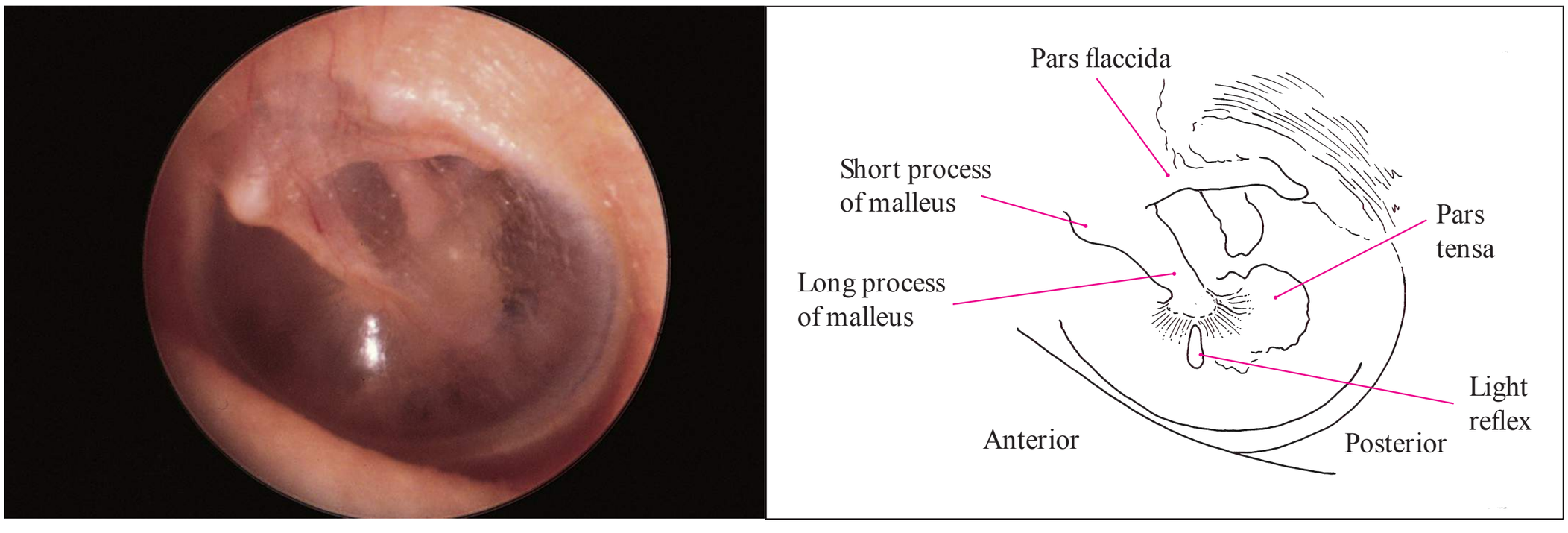

FIGURE 5.1 ■ **Normal Left Tympanic Membrane.** Normal tympanic membrane anatomy and landmarks. (Photo contributor: Richard A. Chole, MD, PhD.)

Management and Disposition

Although AOM generally resolves spontaneously, most patients are treated with antibiotics and analgesics. Steroids, decongestants, and antihistamines do not alter the course in AOM but may improve upper respiratory tract symptoms.

Follow-up in 10 to 14 days or return if symptoms persist or worsen after 48 hours is appropriate and patients should be referred to an otolaryngologist for further evaluation, an audiogram, and possible tympanostomy tubes if they have significant hearing loss, failure of two complete courses of outpatient antibiotics during a single event, chronic otitis media (OM) with or without acute exacerbations, or failure of prophylactic antibiotics.

Pearls

1. AOM is a clinical diagnosis based on the presence of distinct fullness or bulging of the TM.
2. In children, recurrent OM may be due to food allergies.
3. Only 4% of children less than 2 years with AOM develop temperatures greater than 104°F. Those with fever higher than 104°F or with signs of systemic toxicity should be closely evaluated for other causes before attributing the fever to AOM.
4. Most children over 2 years qualify for observation for 48 hours prior to initiation of antibiotics.
5. Management of AOM may require narcotic analgesia; topical Auralgan may decrease pain.

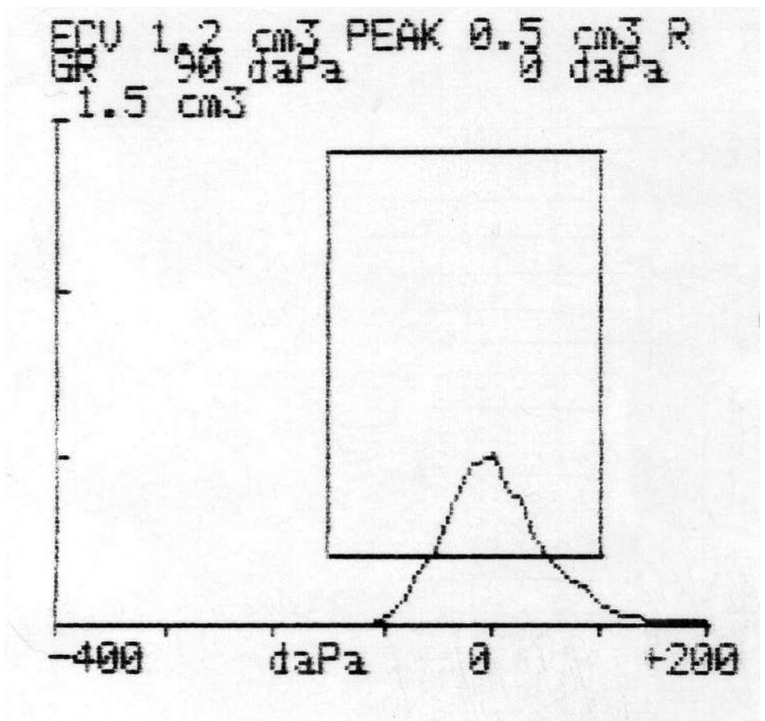

FIGURE 5.4 ■ **Tympanogram of Normal Ear.** The compliance tracing of a normal ear. (Photo contributor: Lawrence B. Stack, MD.)

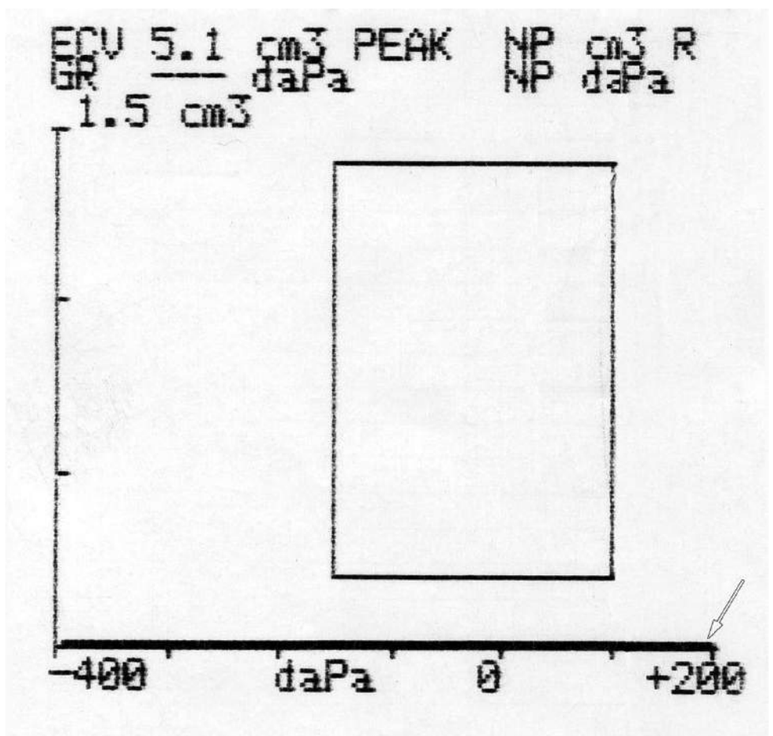

FIGURE 5.5 ■ **Tympanogram of Otitis Media.** The compliance tracing of an ear with otitis media. The line is flat (arrow). (Photo contributor: Lawrence B. Stack, MD.)

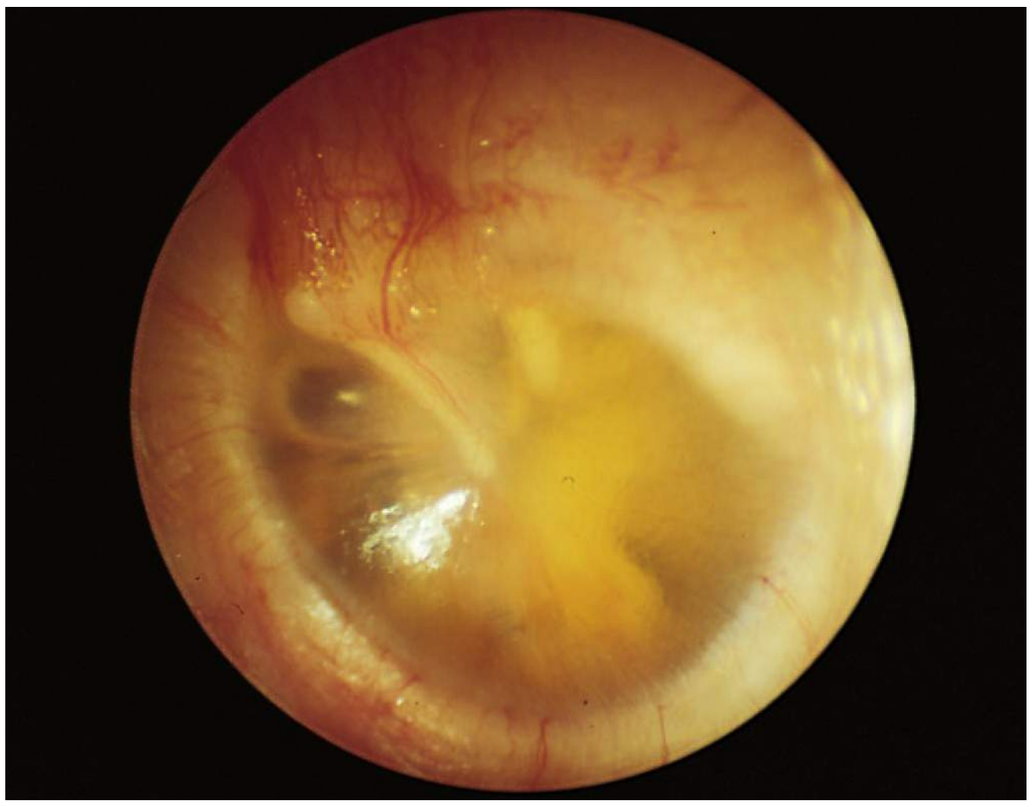

FIGURE 5.6 ■ **Serous Otitis Media with Effusion (OME).** Serous OME is commonly seen after AOM, but it is also common without this history. Any process that leads to obstruction of a eustachian tube may cause OME. A clear, amber-colored effusion with a single air-fluid level is seen in the middle ear behind a normal tympanic membrane. (Photo contributor: C. Bruce MacDonald, MD.)

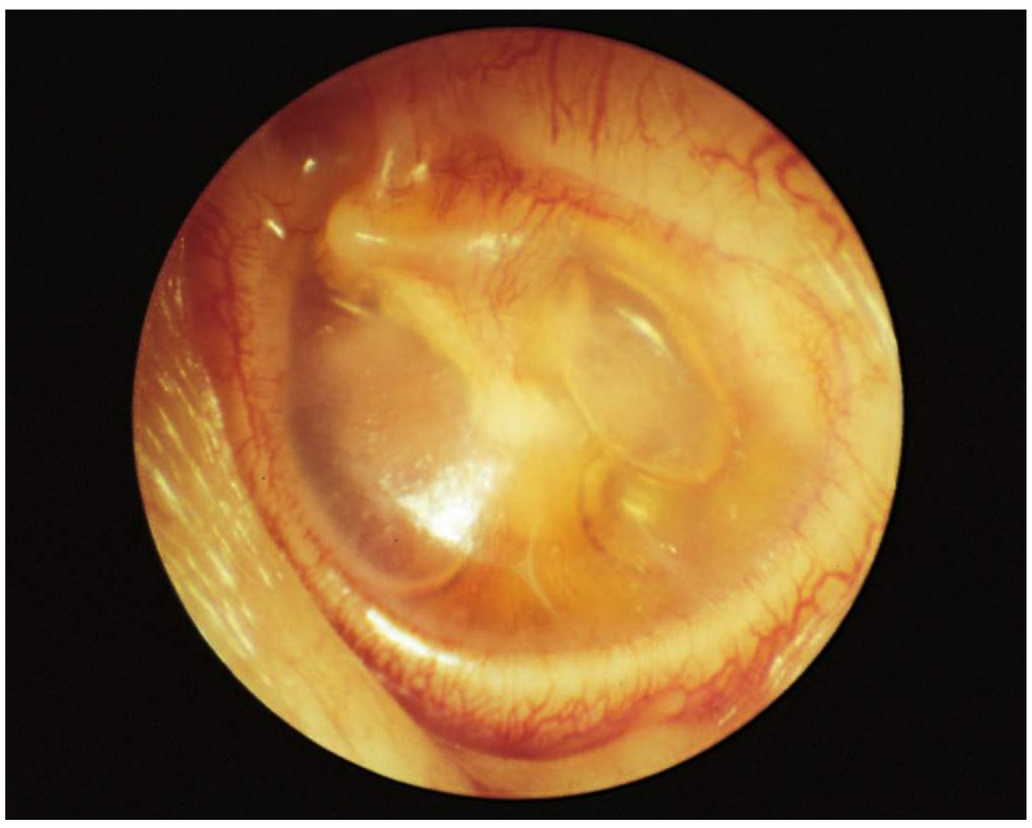

FIGURE 5.7 ■ **Serous OME.** A clear, amber-colored effusion with multiple air-fluid levels is seen in the middle ear behind a normal tympanic membrane. (Photo contributor: C. Bruce MacDonald, MD.)

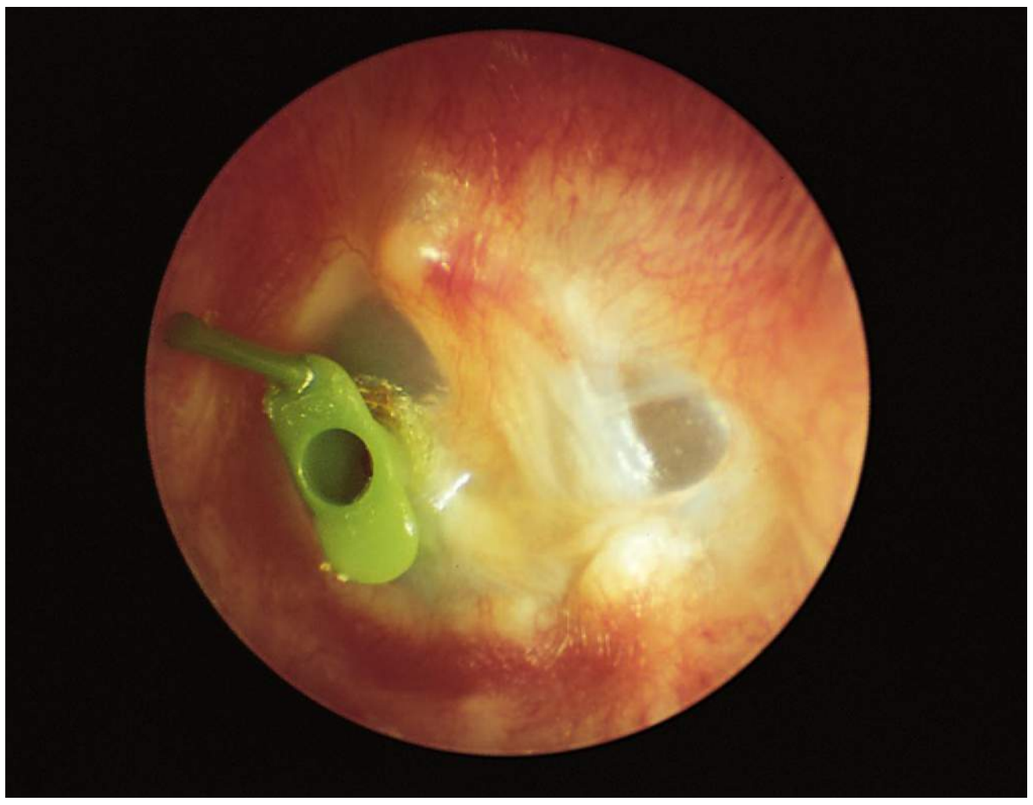

FIGURE 5.8 ■ **Tympanostomy Tube.** Typical appearance of a tympanostomy tube in the tympanic membrane. These tubes will migrate to the periphery and eventually drop out. Occasionally, they will be found in the external ear canal. (Photo contributor: C. Bruce MacDonald, MD.)

Clinical Summary

Bullous myringitis is a direct inflammation and infection of the TM secondary to a viral or bacterial agent. Vesicles or bullae filled with blood or serosanguinous fluid on an erythematous TM are the hallmarks. Frequently, a concomitant OM with effusion is noted. Typical pathogens are the same as seen in AOM.

The onset of bullous myringitis is preceded by an upper respiratory tract infection and is heralded by sudden onset of severe ear pain, scant serosanguinous drainage from the ear canal, and frequently some degree of hearing loss. Otoscopy reveals bullae on either the inner or outer surface of the TM. Patients presenting with fever, hearing loss, and purulent drainage are more likely to have concomitant infections, such as OM and otitis externa (OE).

Management and Disposition

Differentiation between viral and bacterial etiologies for TM bullae is not necessary. Although most episodes resolve spontaneously, many physicians prescribe antibiotics. Warm compresses, topical or strong analgesics, and oral decongestants provide symptomatic relief. Referral is not necessary in most cases unless rupture of the bullae is required for pain relief.

Pearls

1. Instruct parents that TM rupture may occur with sudden resolution of the pain and drainage from the ear canal.
2. Carefully differentiate TM bullae from cholesteatomas or herpetic vesicles.
3. Facial nerve paralysis associated with clear, fluid-filled TM vesicles is characteristic of herpes zoster oticus (HZO).

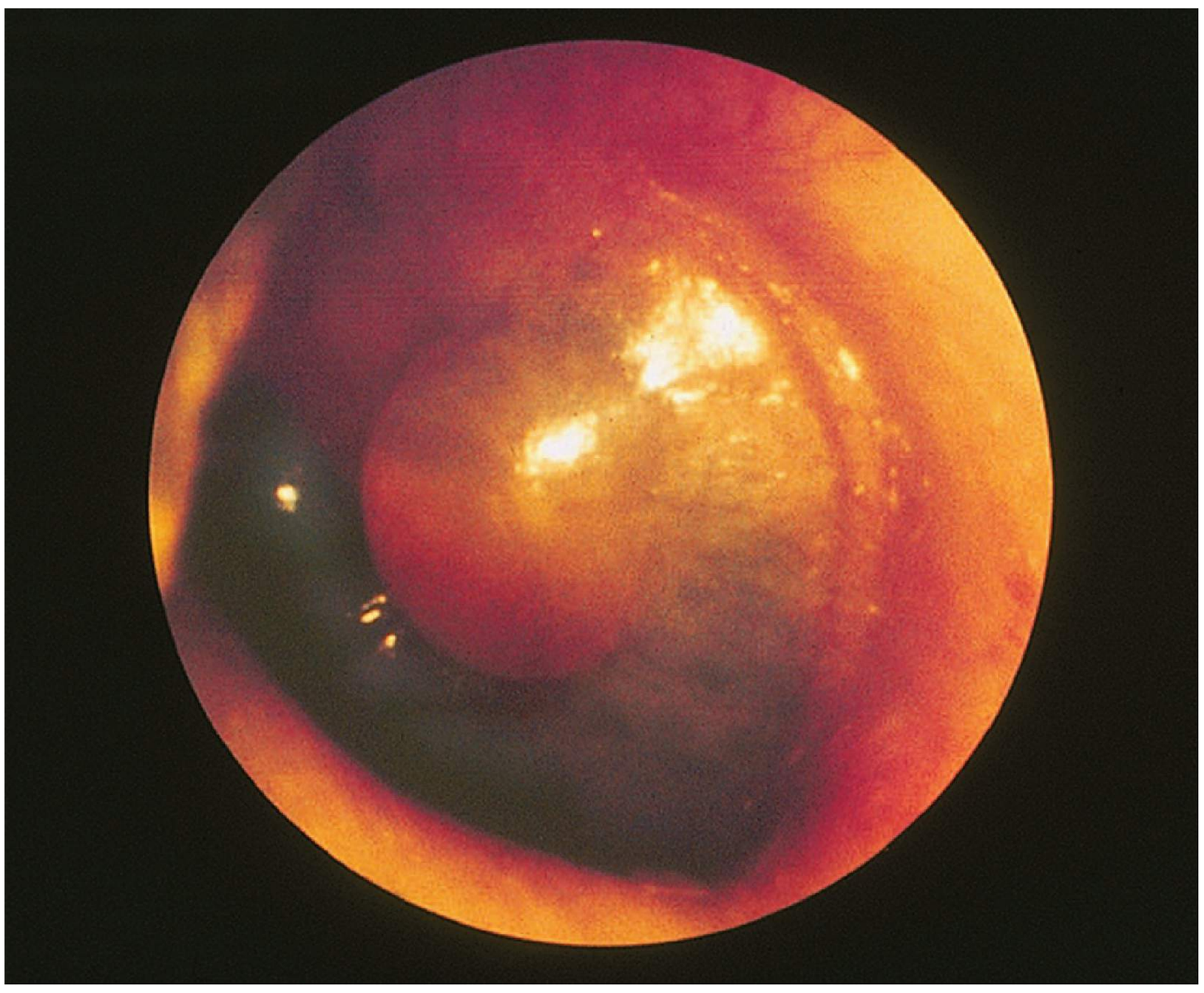

FIGURE 5.9 ■ **Bullous Myringitis.** A large fluid-filled bulla is seen distorting the surface of the tympanic membrane. (Photo contributor: Richard A. Chole, MD, PhD.)

Clinical Summary

Cholesteatomas are collections of desquamating stratified squamous epithelium found in the middle ear or mastoid air cells. Congenital cholesteatomas occur most frequently in children and young adults. Acquired cholesteatomas originate from perforation or retraction of the TM allowing migration of epithelium into the middle ear.

Injury may occur to middle ear ossicles through the production of collagenases, and may erode into the temporal bone, inner ear structures, mastoid sinus, or posterior fossa dura. Treatment delays can lead to permanent conductive hearing loss or infectious complications.

Many cholesteatomas have an insidious progression without associated pain or symptoms. Computed tomography (CT) scans may reveal bony destruction.

Management and Disposition

Refer initial diagnosis. No medical or surgical management is required unless they become symptomatic.

Pearls

1. Persistent pain associated with headache, facial motor weakness, nystagmus, or vertigo suggests inner ear or intracranial involvement.
2. Polyps found on the TM can indicate the presence of a cholesteatoma and require further evaluation to exclude its presence.

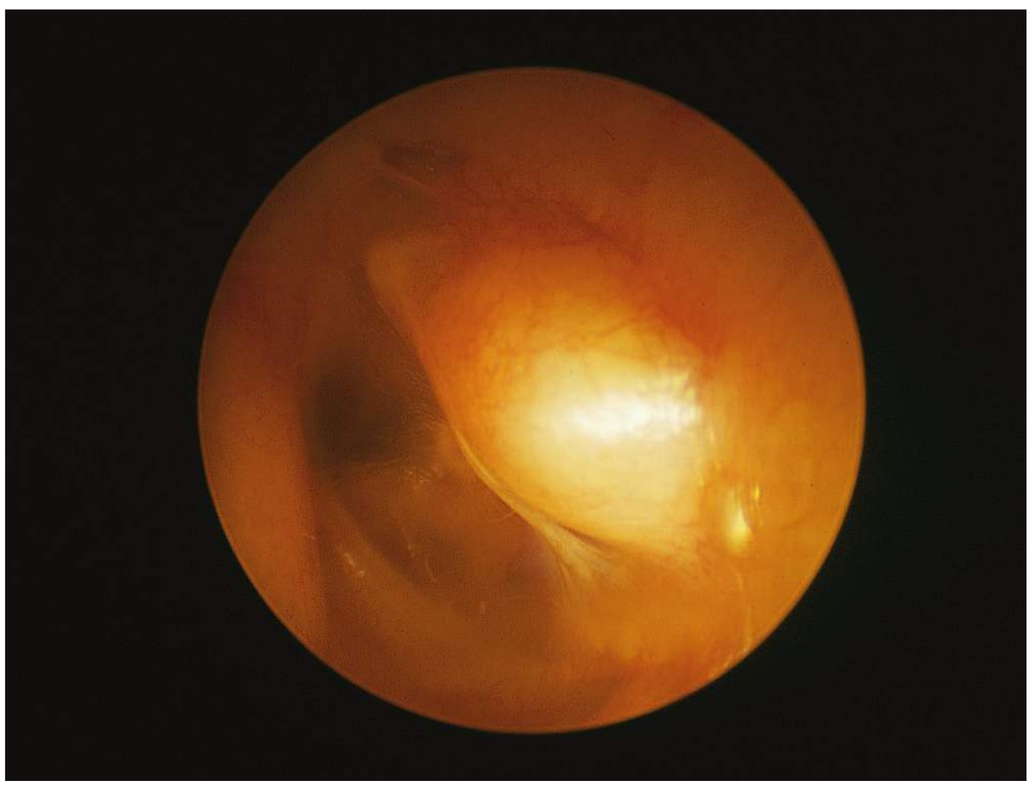

FIGURE 5.10 ■ **Congenital Cholesteatoma.** A congenital cholesteatoma is seen behind an intact tympanic membrane. (Photo contributor: C. Bruce MacDonald, MD.)

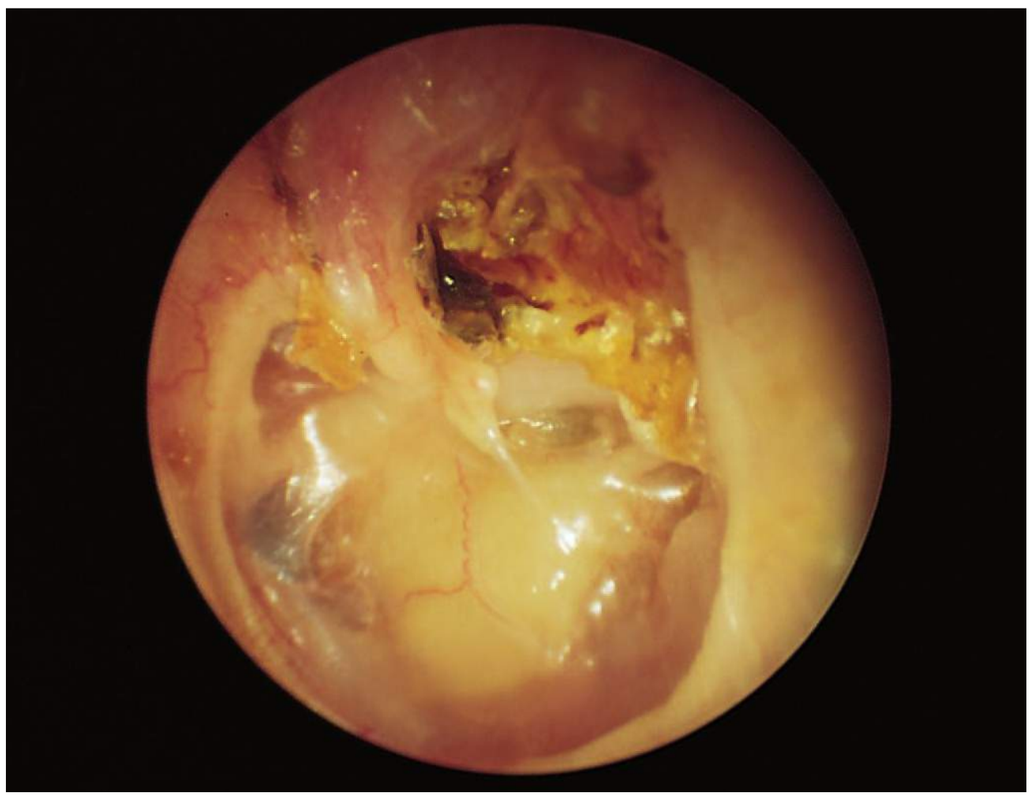

FIGURE 5.11 ■ **Acquired Cholesteatoma.** A cholesteatoma is seen. Note the yellow epithelial debris from the cholesteatoma in the area of the pars flaccida. Often there is an effusion and debris, which can distort the anatomy on otoscopy. (Photo contributor: C. Bruce MacDonald, MD.)

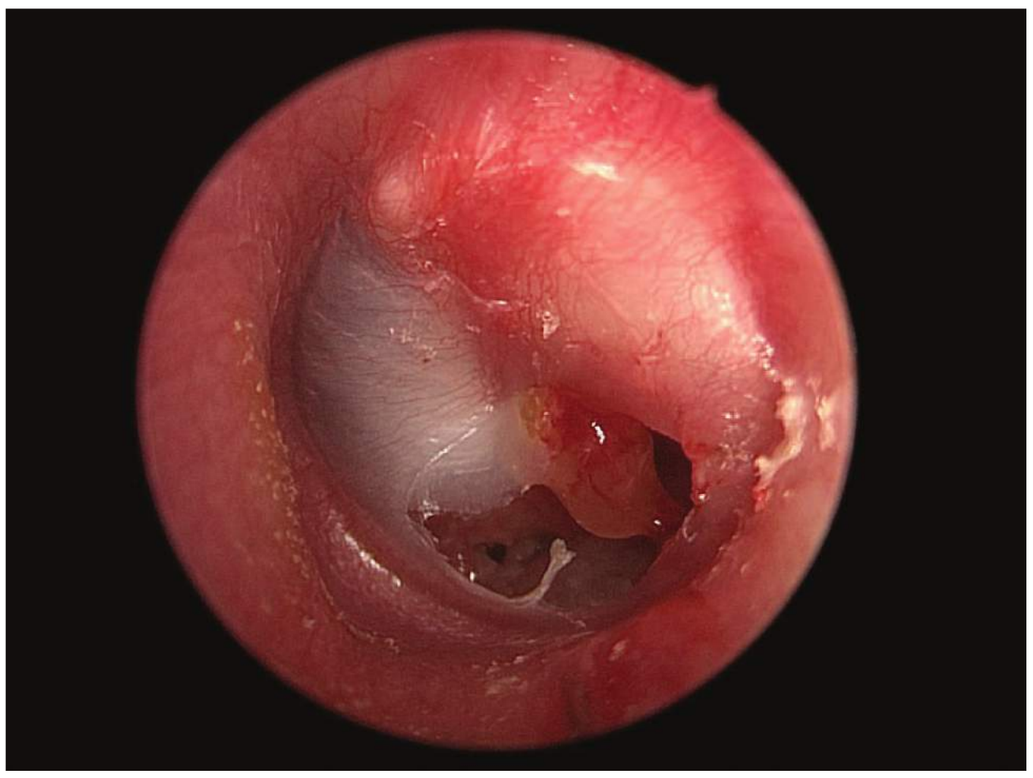

FIGURE 5.12 ■ **Acquired Cholesteatoma.** This is a left ear with an inferior perforation and granulation tissue present just below the tip of the malleus. A white mass is seen behind the eardrum in the posterosuperior quadrant, roughly 12-o'clock to 3-o'clock position. This patient had a history of PE tubes placed several years ago with a resultant perforation allowing squamous epithelium access to the middle ear. (Photo contributor: David R. White, MD)

Clinical Summary

Acute tympanic membrane (TM) perforations are caused by direct penetrating trauma, barotrauma, OM, corrosives, thermal injuries, and iatrogenic causes (foreign-body removal, tympanostomy tubes). TM perforations are occasionally accompanied by injuries to the ossicular chain and temporal bone.

Patients with an acute TM perforation complain of a sudden onset of ear pain, vertigo, tinnitus, and altered hearing after a specific event. Physical examination of the TM reveals a slit-shaped tear or a larger perforation with an irregular border, often associated with blood along the margins. Subacute or chronic perforations have smooth margins and a round or ovoid shape.

Management and Disposition

Treatment of acute TM perforations is tailored to the mechanism of injury. All easily removable foreign bodies should be extracted. Corrosive exposures require face, eye, and ear decontamination. Antibiotics and irrigation do not improve the rate or completeness of healing unless the injury is associated with OM. Systemic antibiotics should be reserved for perforations associated with OM, penetrating injury, and possibly water-sport injuries (see "Otitis Media" above). Topical steroids impede perforation healing.

Patients are instructed to avoid water in the ear while the perforation is healing and to return if symptoms of infection appear. While nearly 80% of all TM perforations heal spontaneously, all TM perforations require referral to an otolaryngologist for follow-up for possible myringoplasty.

Pearls

1. Corticosteroid eardrops of any formulation retard spontaneous healing and should be avoided.
2. Traumatic TM perforation associated with cranial nerve (CN) deficits or persistent vertigo requires immediate otolaryngology consultation due to possible temporal bone fractures or injury to the round or oval window.

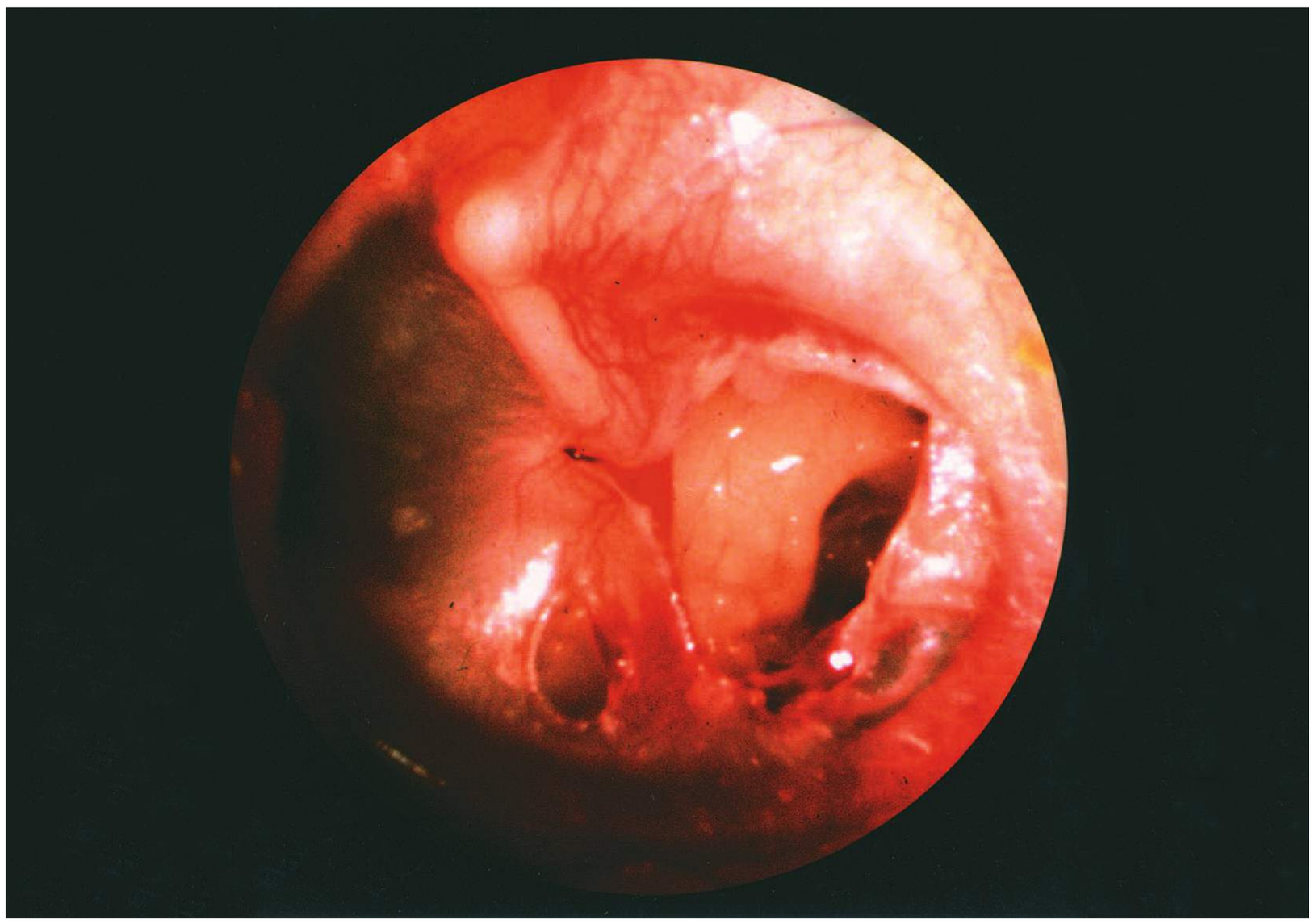

FIGURE 5.13 ■ **Acute Tympanic Membrane Perforation.** An acute tympanic membrane perforation is seen. Note the sharp edges of the ruptured tympanic membrane. (Photo contributor: Richard A. Chole, MD, PhD.)

OTITIS EXTERNA

Clinical Summary

Otitis externa (OE), or "swimmer's ear," is an inflammation and infection (bacterial or fungal) of the auricle and external auditory canal (EAC). Typical symptoms include otalgia, pruritus, otorrhea, and hearing loss. Physical examination reveals EAC hyperemia and edema, otorrhea, occlusion from debris and swelling, pain with manipulation of the tragus, and periauricular lymphadenopathy.

Several factors predispose the EAC to infection: increased humidity and heat, repeated water immersion, foreign bodies, trauma, hearing aids, and cerumen impaction. Bacterial OE is primarily an infection due to Pseudomonas species or Staphylococcus aureus. Diabetics are particularly prone to infections by Pseudomonas, Candida albicans, and, less commonly, Aspergillus niger.

Management and Disposition

EAC irrigation and suctioning is recommended to thoroughly evaluate the EAC. Topical antibiotic suspensions containing polymyxin, neomycin, and hydrocortisone or ciprofloxacin with ear wicks are effective. Topical solutions are not pH-balanced and thus are irritating and may cause inflammation in the middle ear if a perforation is present. Topical fluoroquinolones may be less irritating and are only given twice a day. Systemic antibiotics are not indicated unless extension into the periauricular tissues is noted. Patients should avoid swimming and prevent water from entering the ear while bathing. Dry heat aids in resolution, and analgesics provide symptomatic relief. Follow-up should be arranged in 10 days for routine cases.

Pearls

1. Resistant cases may have an allergic or eczematous component. These typically present with a dry, scaly, itchy EAC and are chronic in nature.
2. Drying the EAC after water exposure with a 50:50 mixture of isopropyl alcohol and water or with acetic acid (white vinegar) minimizes recurrence. If the TM is possibly perforated, isopropyl alcohol should be avoided.
3. If a TM perforation is suspected and antibiotic drops are indicated, a suspension is recommended.
4. Consider malignant OE, typically caused by Pseudomonas aeruginosa, in elderly, diabetics, or immunocompromised patients. Exposed bone, ulceration of the EAC, and facial nerve weakness are hallmarks.

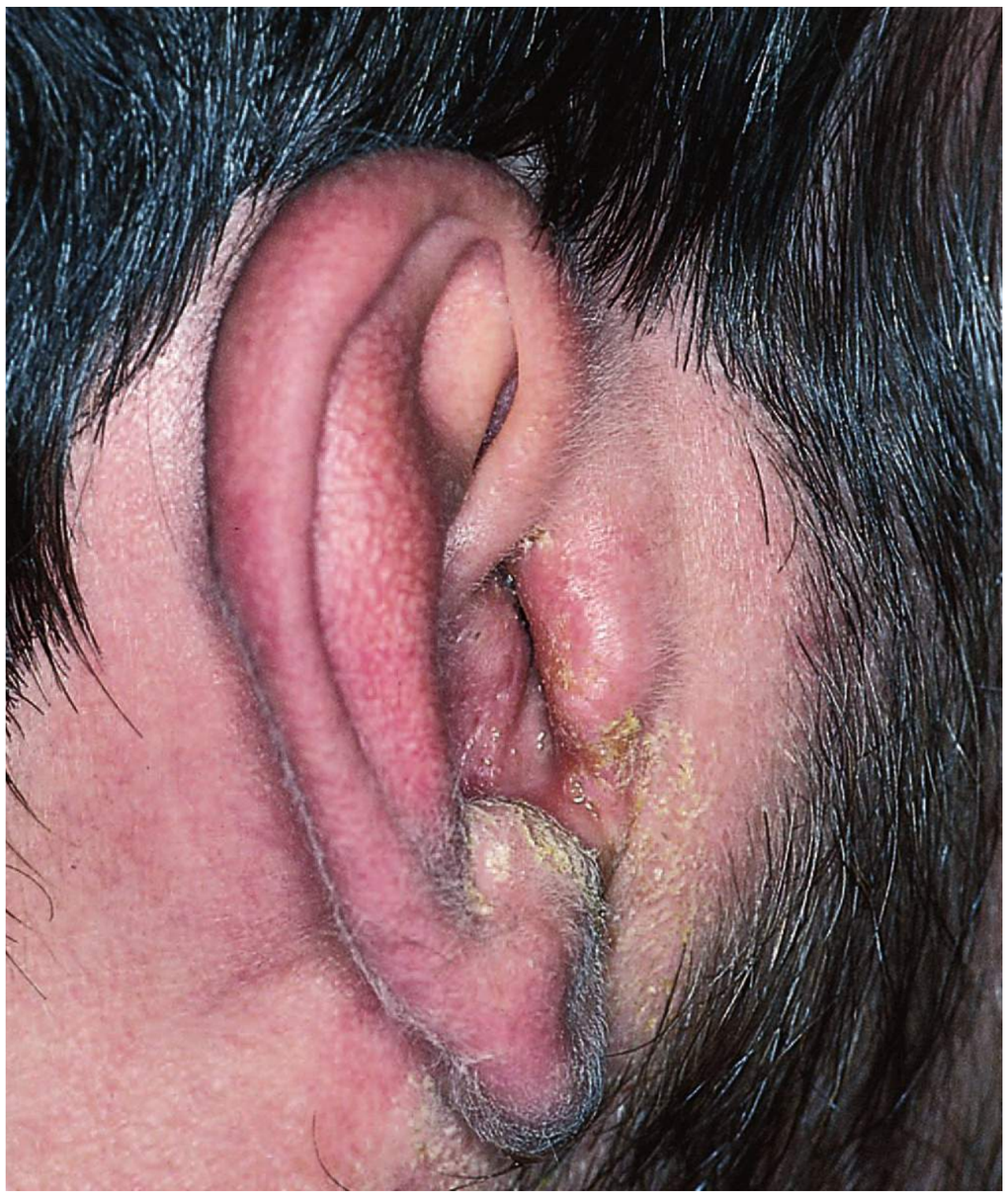

FIGURE 5.14 ■ **Otitis Externa.** A discharge is seen coming from the external auditory canal, which is swollen and almost completely occluded. An ear wick placed in the EAC facilitates delivery of topical antibiotic suspension and drainage of debris. (Photo contributor: Frank Birinyi, MD.)

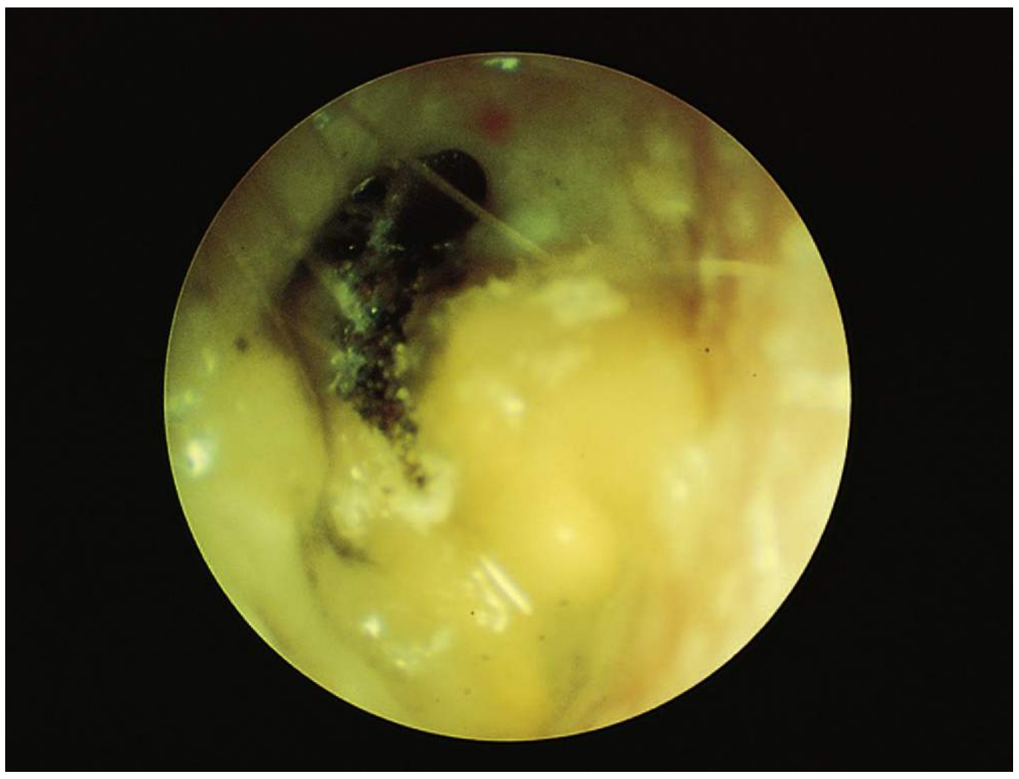

FIGURE 5.15 ■ **Aspergillus Otitis Externa.** Chronic otitis externa with copious debris, including black spores from A niger, cottony fungal elements, and wet debris. This patient had been treated with topical and systemic antibiotics. (Photo contributor: C. Bruce MacDonald, MD.)

PREAURICULAR SINUS ABSCESS

Clinical Summary

Preauricular sinus abscesses arise from pits or fissures originating from congenital malformations of the preauricular soft tissues. They are located near the front of the ear and mark the entrance to a sinus tract. These are lined with squamous epithelium and thus may produce epithelial-lined subcutaneous cysts, which may become infected, leading to cellulitis or abscess. Patients may have other congenital anomalies such as hearing loss or renal disease. Patients may present with recurrent ear discharge, pain, swelling, redness, itching, headache, and fever. The diagnosis is often overlooked in early stages.

Management and Disposition

Initiate oral antibiotics to cover for S aureus (most common). Incision and drainage should be avoided in the emergency department (ED). Needle aspiration may be attempted and may give some relief. Arrange follow-up with an otolaryngologist for complete excision of the sinus track.

Pearls

1. Preauricular sinus abscesses may be confused with first branchial cleft cysts.
2. Because of their close proximity to the facial nerve, referral to a specialist who is familiar with the underlying anatomy is recommended.

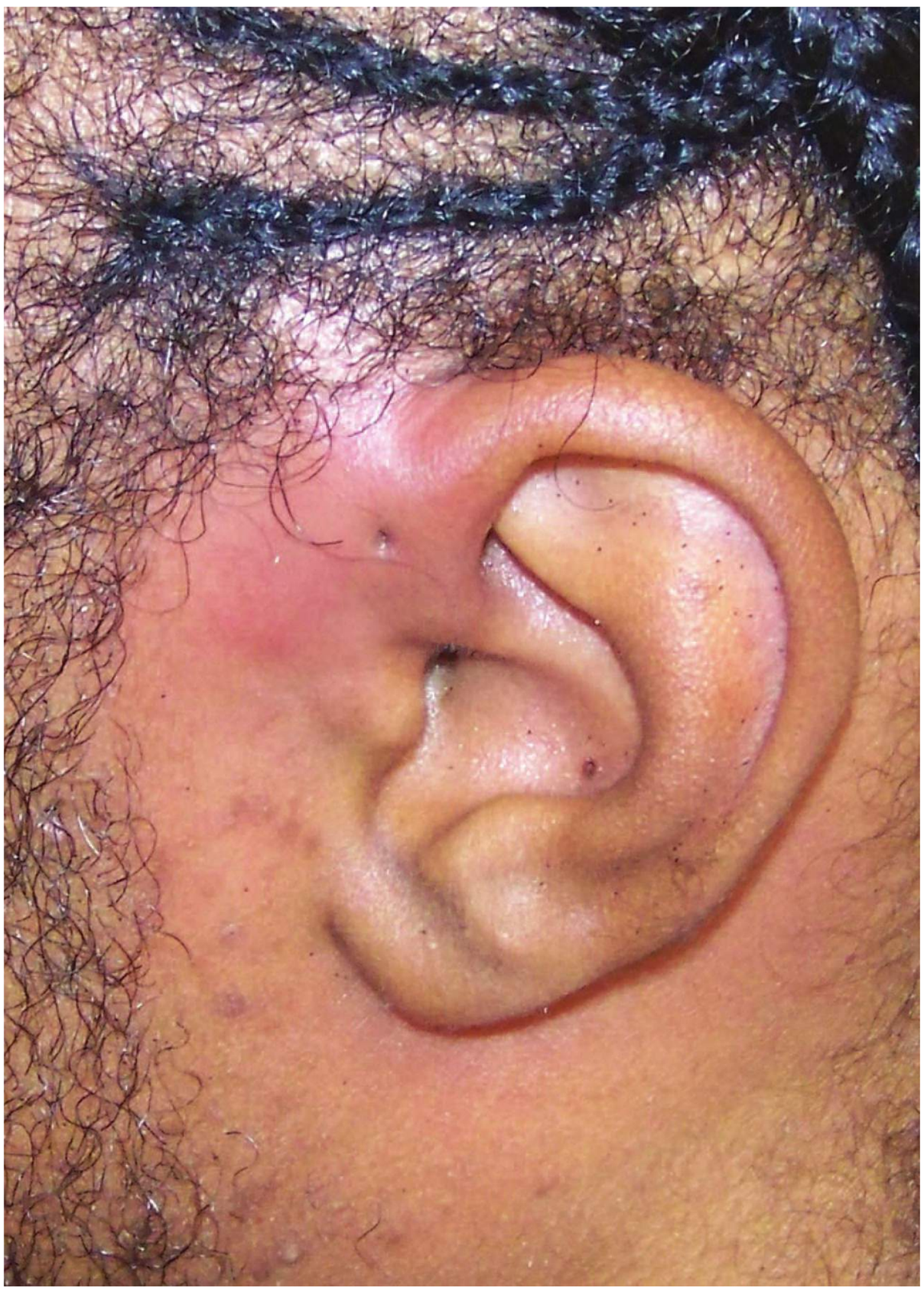

FIGURE 5.16 ■ **Preauricular Sinus Abscess.** Recurrent preauricular swelling and redness in the area of a "pit" suggests sinus abscess and was confirmed on contrast-enhanced CT scan. (Photo contributor: Lawrence B. Stack, MD.)

MASTOIDITIS

Clinical Summary

Mastoiditis is an infection or inflammation of the mastoid air cells often resulting from extension of purulent OM with progressive destruction and coalescence of air cells. Medial sinus wall erosion can cause cavernous sinus thrombosis, facial nerve palsy, meningitis, brain abscess, and sepsis. With the use of antibiotics for AOM, the incidence of mastoiditis has fallen sharply.

Patients present with fever, chills, postauricular ear pain, and otorrhea. Patients may have tenderness, erythema, swelling, and fluctuance over the mastoid process; proptosis of the pinna; erythema of the posterior-superior EAC wall; and otorrhea.

Management and Disposition

Initial evaluation includes a thorough head, neck, and CN examination. Contrasted CT of the head or mastoid sinus may reveal bone erosion and intracranial involvement.

While oral penicillinase-resistant penicillins, amoxicillin-clavulanic acid, third-generation cephalosporins, and the newer macrolides are effective in mild cases of mastoiditis, severe cases require parenteral antibiotics. Mastoiditis requires prompt consultation and close follow-up.

Pearls

1. Most patients require admission for parenteral antibiotics to cover H influenzae, M catarrhalis, streptococcal species, and S aureus.
2. Surgical incision and debridement, and possibly mastoidectomy, are reserved for refractory cases.
3. Chronic mastoiditis describes chronic otorrhea of at least 2-month duration. It is often associated with craniofacial anomalies.

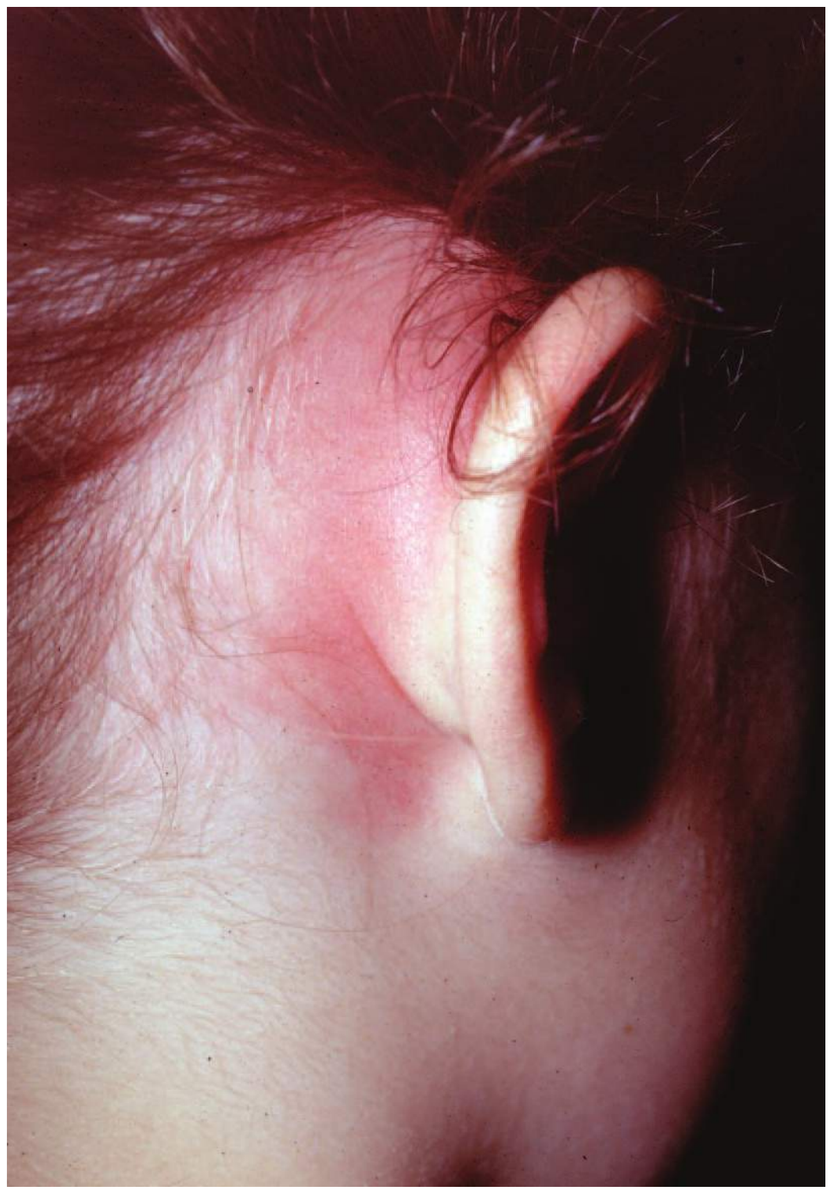

FIGURE 5.17 ■ **Acute Mastoiditis.** Postauricular swelling, redness, and proptosis in a young girl with acute mastoiditis and sinusitis. (Photo contributor: Lawrence B. Stack, MD.)

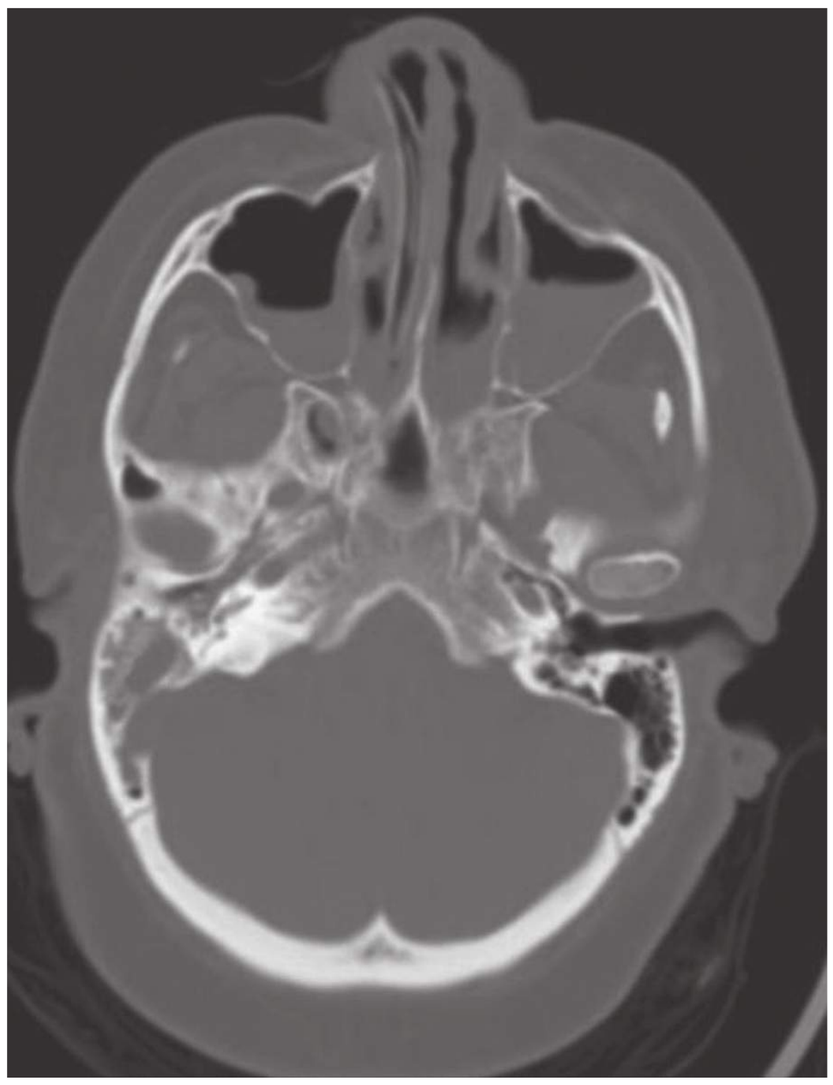

FIGURE 5.18 ■ **Acute Mastoiditis.** Bone window of a head CT scan which demonstrates fluid in the right mastoid air cells. Maxillary sinus disease is also present. (Photo contributor: Lawrence B. Stack, MD.)

AURICULAR PERICHONDRITIS

Clinical Summary

Auricular perichondritis is a bacterial infection of the overlying skin and perichondrium of the ear, by definition sparing the auricular cartilage. Causative organisms include P aeruginosa, S aureus, and S pyogenes. Predisposing factors for auricular perichondritis include surgery, ear piercing, burns, frostbite, insect bites, and contact sports. Auricular perichondritis may be an extension of OE. Clinical findings include a swollen, tender, erythematous, and warm auricle, which may involve the ear lobule, and often a fever; the TM is unaffected. Infectious perichondritis may be confused with relapsing polychondritis, an autoimmune condition involving the cartilage of the ears, nose, and trachea.

Management and Disposition

Outpatient management of the healthy patient includes appropriate oral antibiotics and follow-up in 48 hours. Hospitalization for parenteral antibiotics in the immunocompromised or diabetic patient is advised. Topical antibiotic otic drops should be used if the external canal is involved. Relapsing polychondritis is treated as an outpatient with prednisone if involvement is confined to the ear.

Pearls

1. P aeruginosa is the most common bacteria causing auricular perichondritis.
2. Ear piercing is the most common activity resulting in auricular perichondritis.
3. Findings of fluctuance and auricular deformity suggest auricular chondritis, a complication of perichondritis.

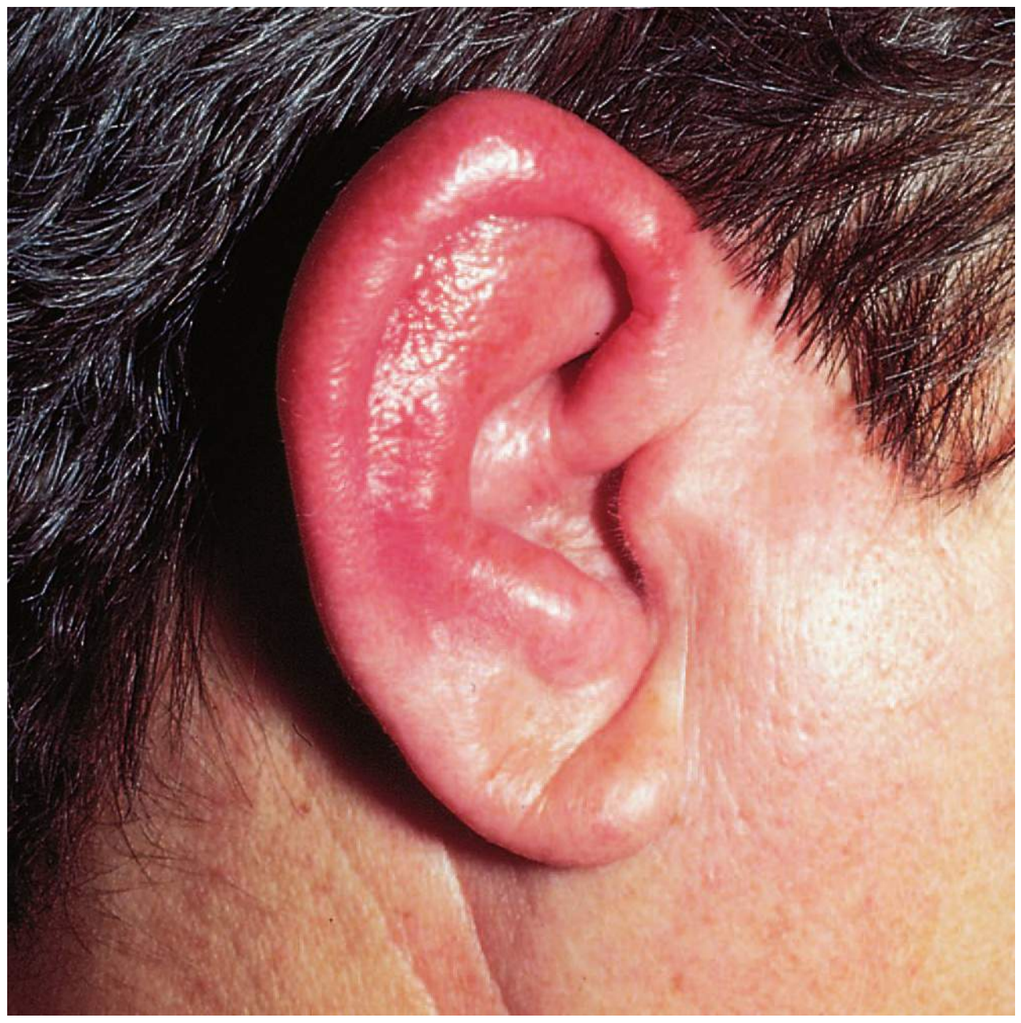

FIGURE 5.19 ■ **Perichondritis.** Erythema, swelling, and tenderness seen in the pinna with multiple ear piercings. (Photo contributor: Lawrence B. Stack, MD.)

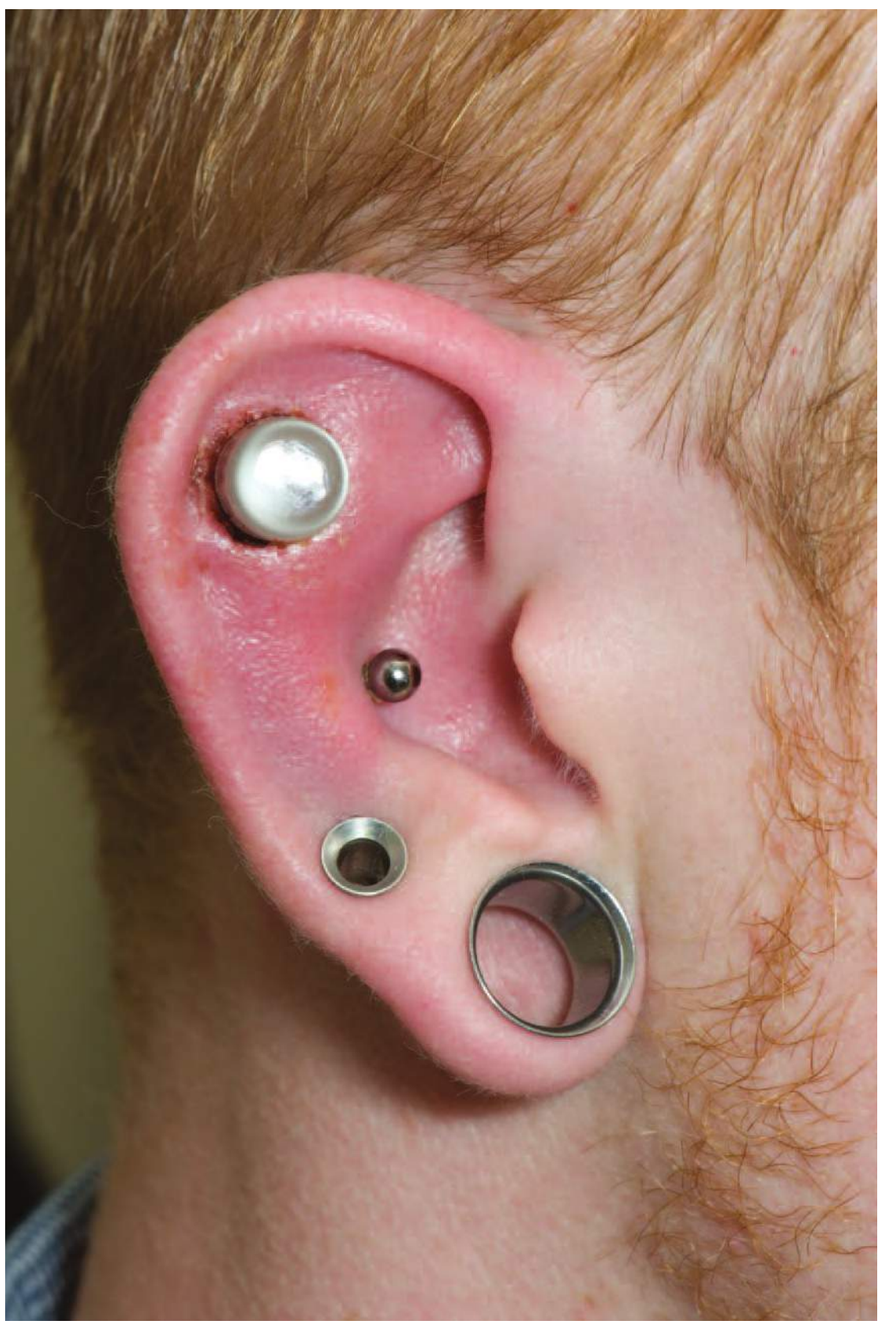

FIGURE 5.20 ■ **Perichondritis.** Erythema is seen surrounding the piercing sites of the ear of this patient in DKA. (Photo contributor: Lawrence B. Stack, MD.)

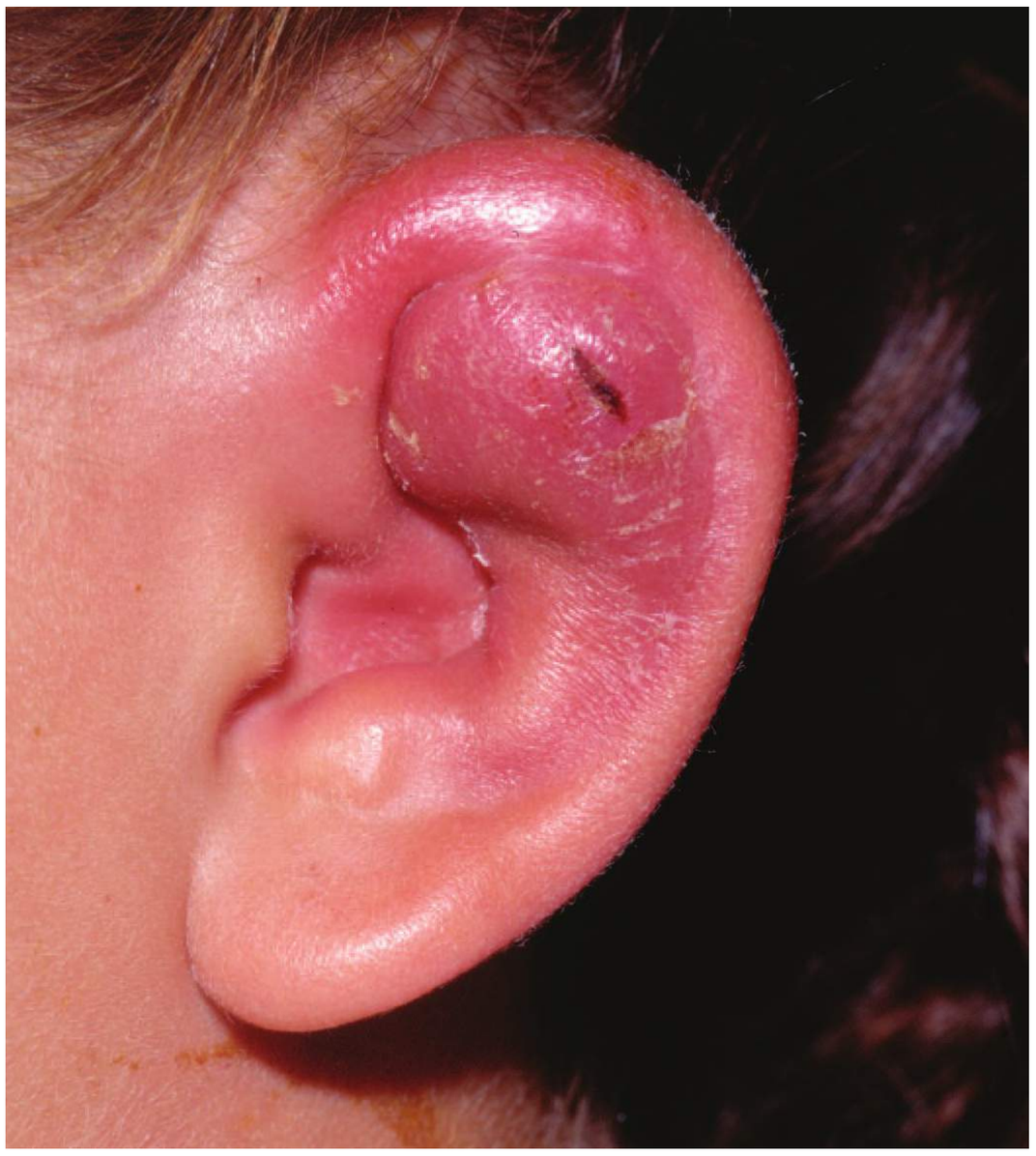

FIGURE 5.21 ■ **Auricular Chondritis.** Deformity of the auricular cartilage is seen in this patient with chondritis. Ear piercing caused the initial insult to this pinna. (Photo contributor: Lawrence B. Stack, MD.)

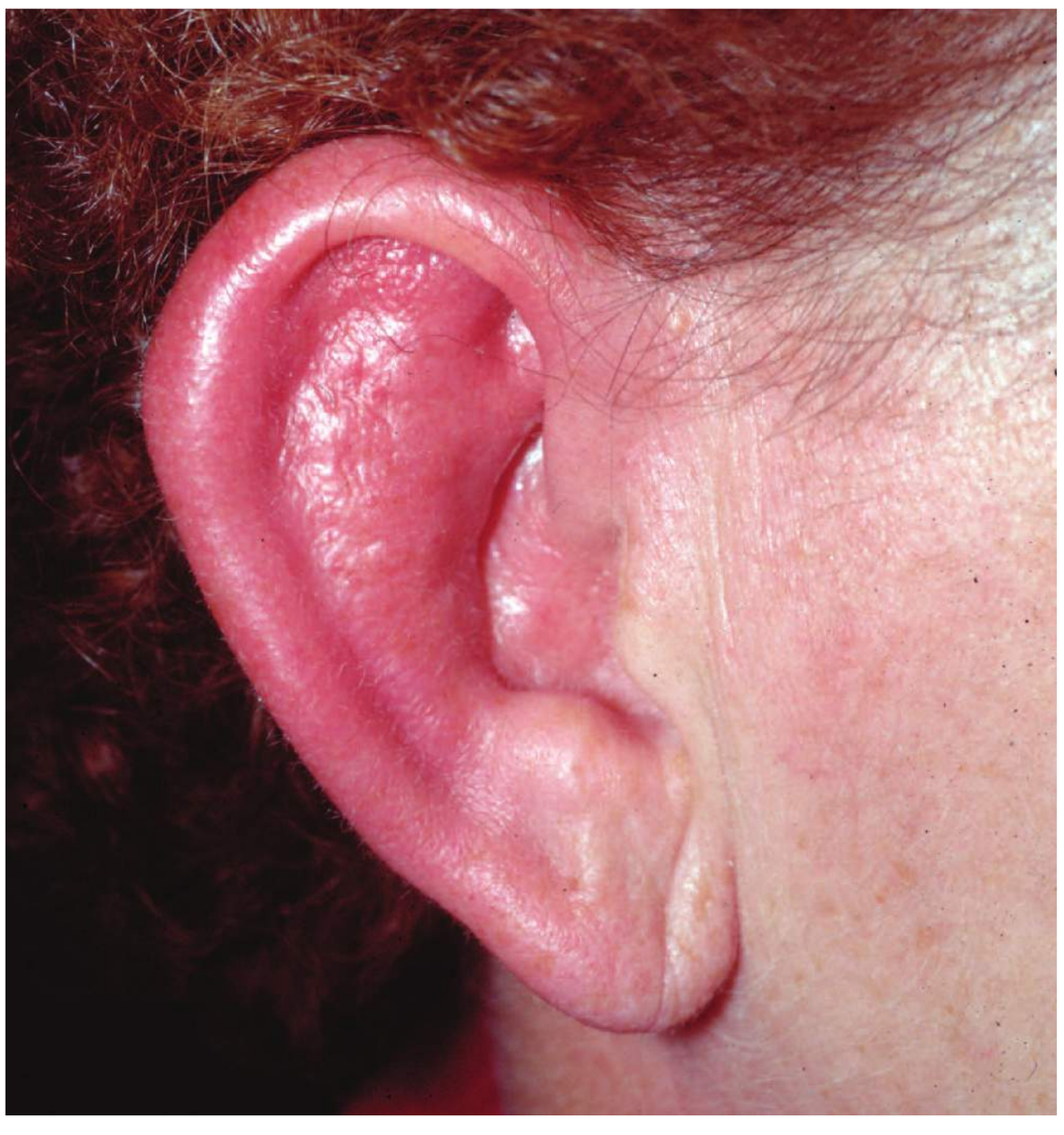

FIGURE 5.22 ■ **Relapsing Polychondritis.** Erythema of the right pinna that spares the lobe. A month prior, the left pinna was involved and was treated as infectious perichondritis. (Photo contributor: Lawrence B. Stack, MD.)

HERPES ZOSTER OTICUS (RAMSAY HUNT SYNDROME)

Clinical Summary

Herpes zoster oticus (HZO), or Ramsay Hunt syndrome, is the second most common cause of facial paralysis, representing 3% to 12% of cases. The syndrome consists of facial and neck pain, auditory symptoms, and facial palsy associated with the reactivation of latent varicella zoster virus in the facial nerve and geniculate ganglion. Patients first note pruritus, followed by pain out of proportion to the physical examination over the face and ear. Patients may also experience vertigo, hearing loss from involvement of the eighth CN, tinnitus, rapid onset of facial paralysis, decrease in salivation, loss of taste sensation over the posterolateral tongue, and vesicles on the ear, EAC, and face.

Management and Disposition

The diagnosis of HZO is based largely on history and physical examination. Tzanck preparations may be difficult because of the vesicles' location. Magnetic resonance imaging (MRI) with contrast, if performed, may show enhancement of the geniculate ganglion and facial nerve.

Oral antivirals and steroids are mainstays of treatment. It is important to protect the involved eye from corneal abrasions and ulcerations by using lubricating drops. Referral to a specialist should be made for follow-up care.

Pearls

1. The prognosis for facial paralysis due to HZO is worse than that for Bell palsy. Approximately 10% and 66% of patients with full and partial facial paralysis, respectively, recover fully. The prognosis improves if the symptoms of HZO are preceded by the vesicular eruption.
2. The combination of antivirals with steroids produces better outcomes than with either agent alone.

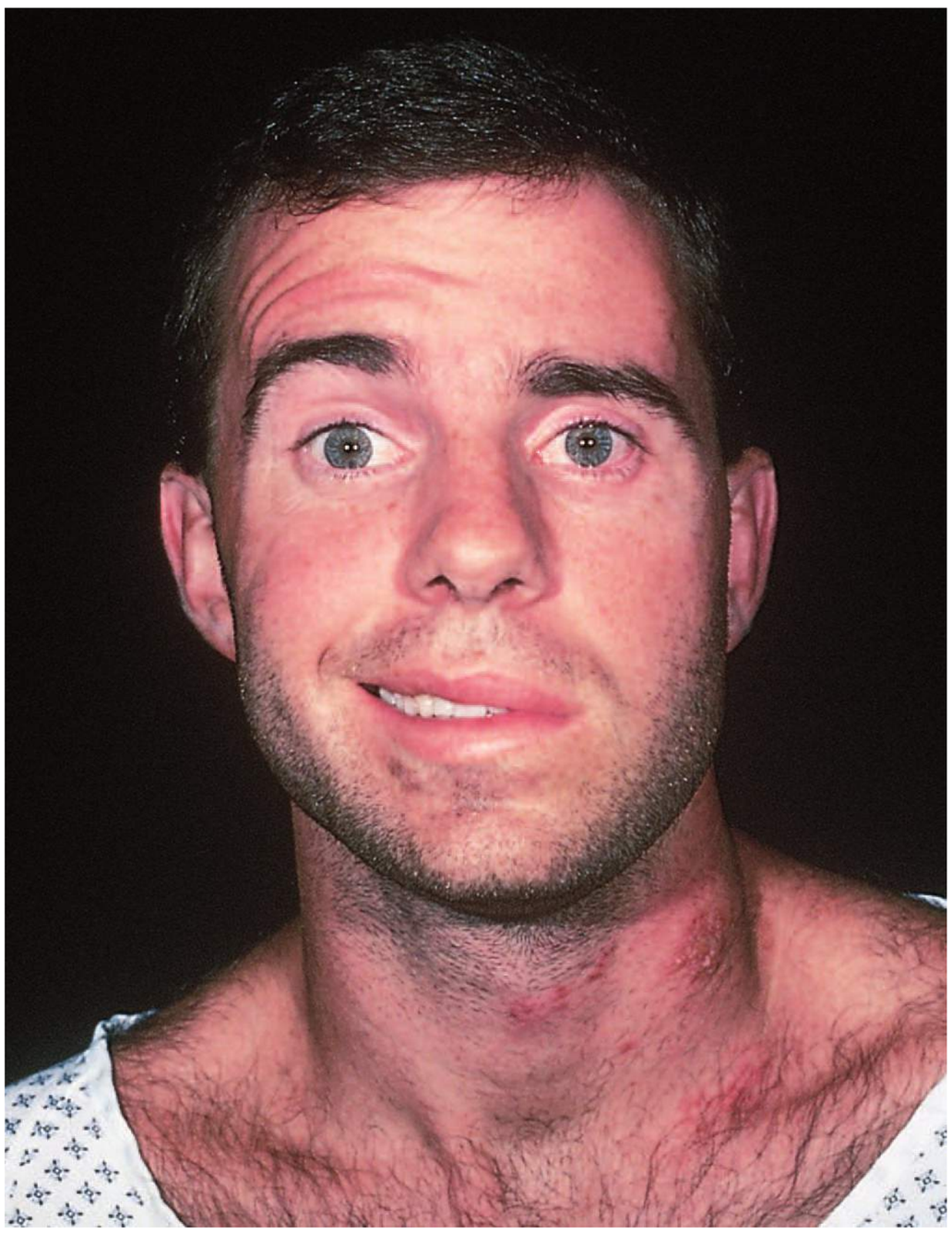

FIGURE 5.23 ■ **Herpes Zoster Oticus.** Facial palsy in a young adult. Note the vesicular eruptions on the neck. (Photo contributor: Frank Birinyi, MD.)

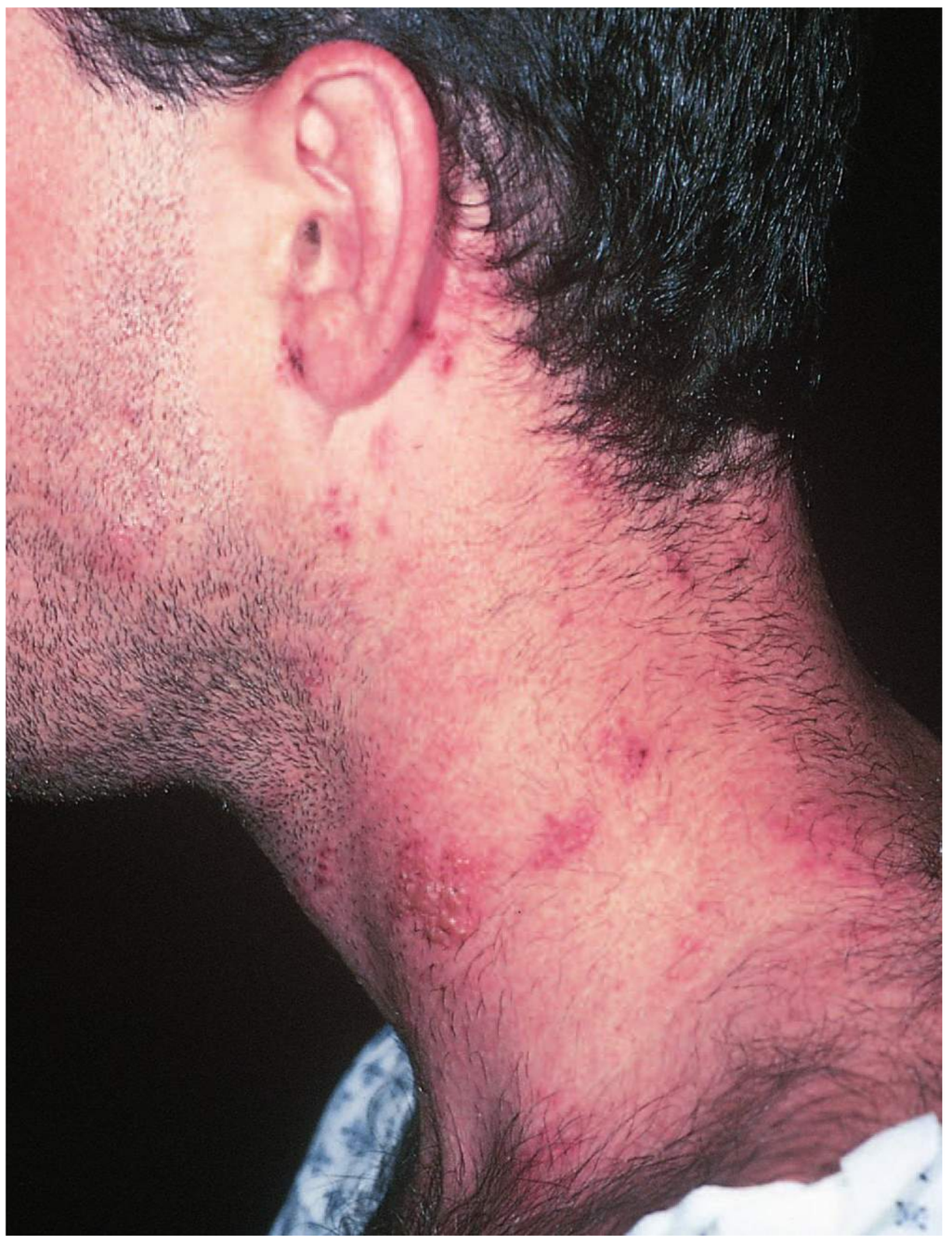

FIGURE 5.24 ■ **Herpes Zoster Oticus.** On closer examination, the vesicles extend up the neck to the external auditory canal. (Photo contributor: Frank Birinyi, MD.)

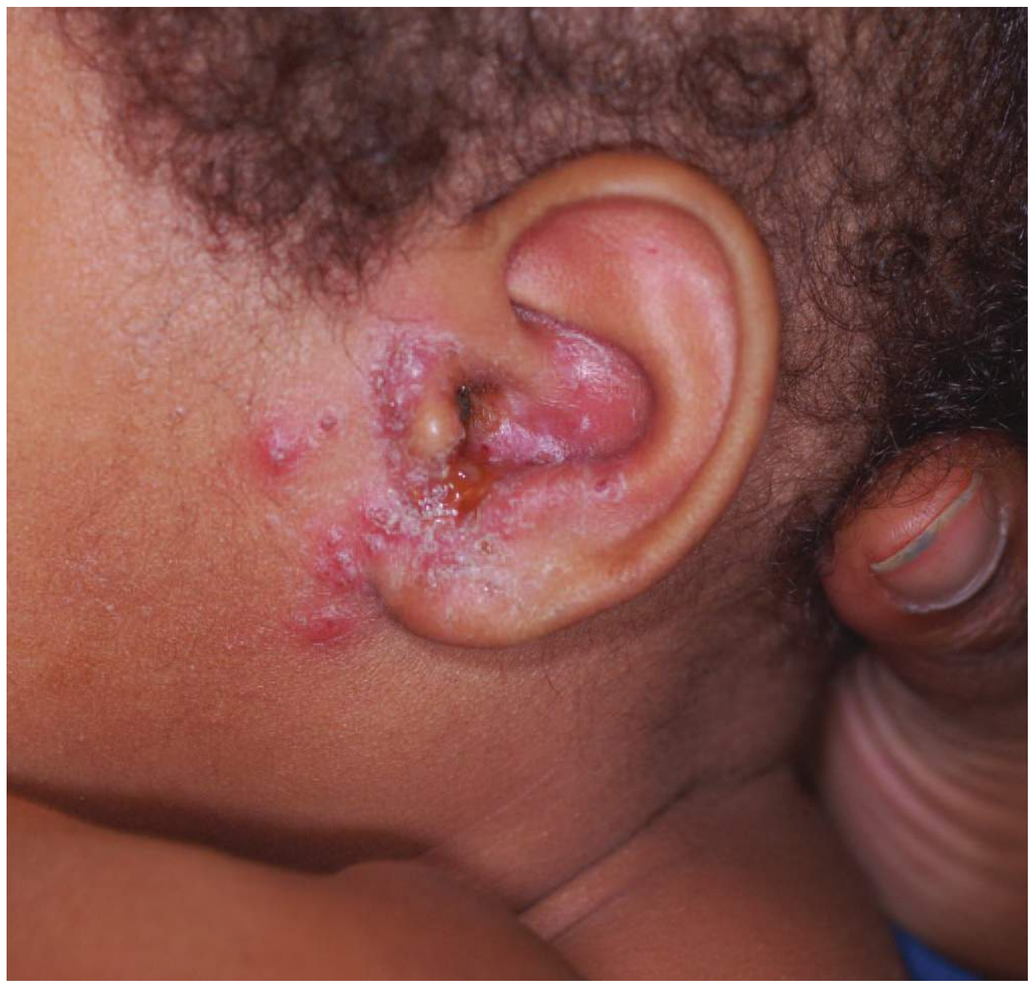

FIGURE 5.25 ■ **Herpes Zoster Oticus.** Erythema and drainage coming from the EAC is seen in this patient with herpes zoster oticus. Otitis externa can have a similar appearance but does not have vesicles, as seen in this patient. (Photo contributor: David Effron, MD.)

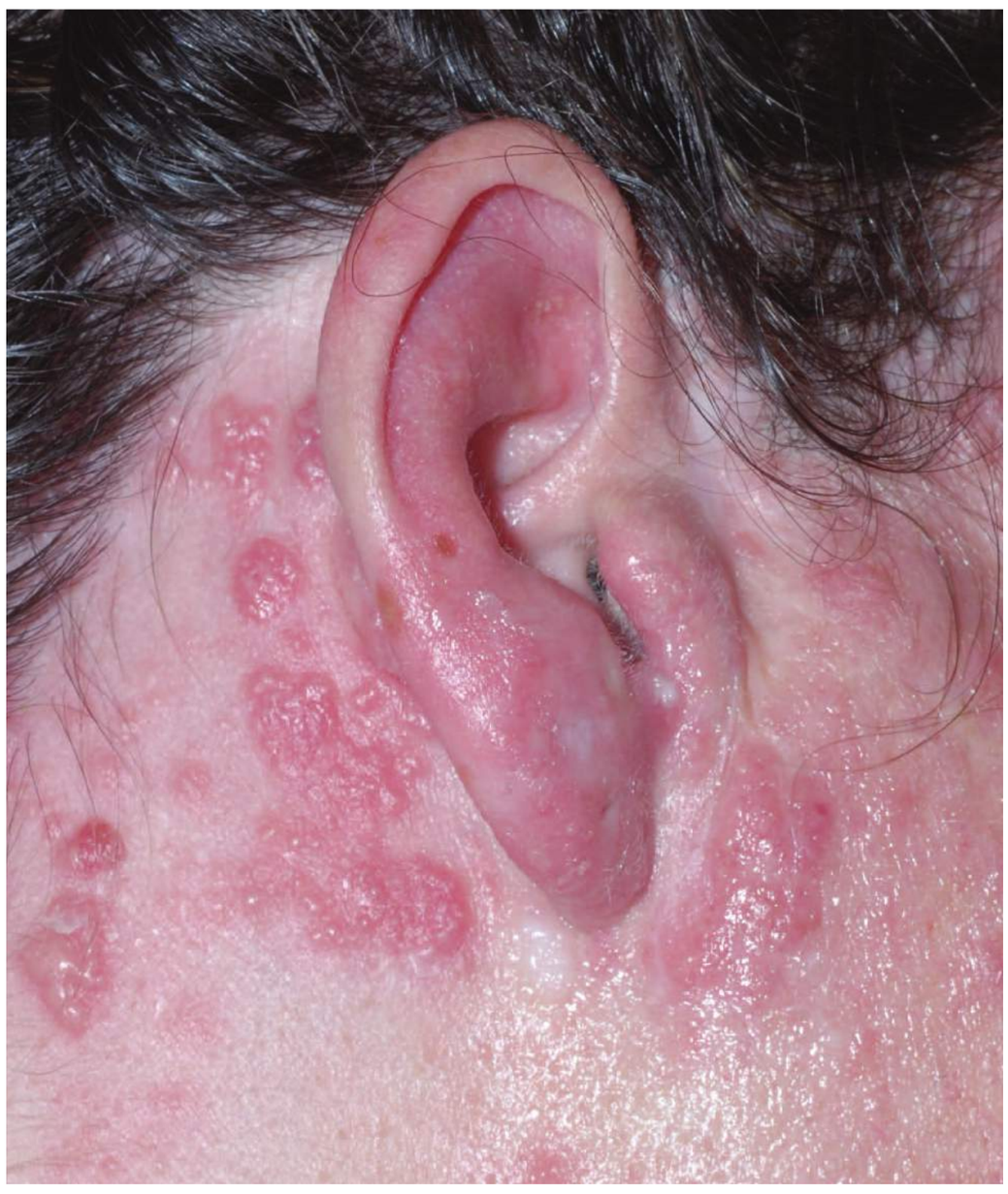

FIGURE 5.26 ■ **Herpes Zoster Oticus.** Vesicles of zoster are clearly seen in this patient. (Photo contributor: Lawrence B. Stack, MD.)

Clinical Summary

CN VII innervates the facial muscles via the five branches of the motor root, the submandibular, sublingual, and lacrimal glands, and the taste organs on the anterior two-thirds of the tongue; and it provides sensation to the pinna of the ear. Facial palsies are either central or peripheral. Central lesions occur proximal to the CN VII nucleus in the pons. Lesions distal to the nucleus are classified as peripheral lesions. The ipsilateral frontalis muscle is functional or "spared" in central lesions, since it receives innervation in the nucleus from both ipsilateral and contralateral motor cortices. Peripheral injuries involve the entire side of the face, including the forehead; thus, the forehead is not "spared."

Most commonly, seventh-nerve dysfunction is idiopathic (Bell palsy). One percent of patients have bilateral involvement; 60% have a viral prodrome. There is no age, sex, or racial predilection. Patients note an acute onset of facial weakness and may have numbness or pain on the ipsilateral face, ear, tongue, and neck as well as a decrease or loss of ipsilateral tearing and saliva flow. Hearing is preserved.

Prognosis is variable. Facial weakness has a better prognosis for full recovery than complete paralysis. Palsies due to herpes zoster have a protracted course, and many do not fully resolve. In comparison, 80% of patients with Bell palsy completely recover within 3 months.

Management and Disposition

Initial examination should include a thorough examination of the ear (including sensorineural or conductive hearing loss), the eye (including lacrimation), and the CNs—especially extraocular muscles (EOMs). Motor function of the seventh CN is evaluated by having the patient raise their eyebrows, smile, pucker, and frown. No single laboratory test is diagnostic. Screening CT or MRI of the head is of little value in the absence of additional findings on physical examination.

Initiate steroids within 72 hours of symptom onset for age >16 years and antivirals with steroids if viral etiology suspected. Eye lubricants and taping the eye shut at night help prevent keratitis and ulceration. Referral to a neurologist should be made for follow-up care.

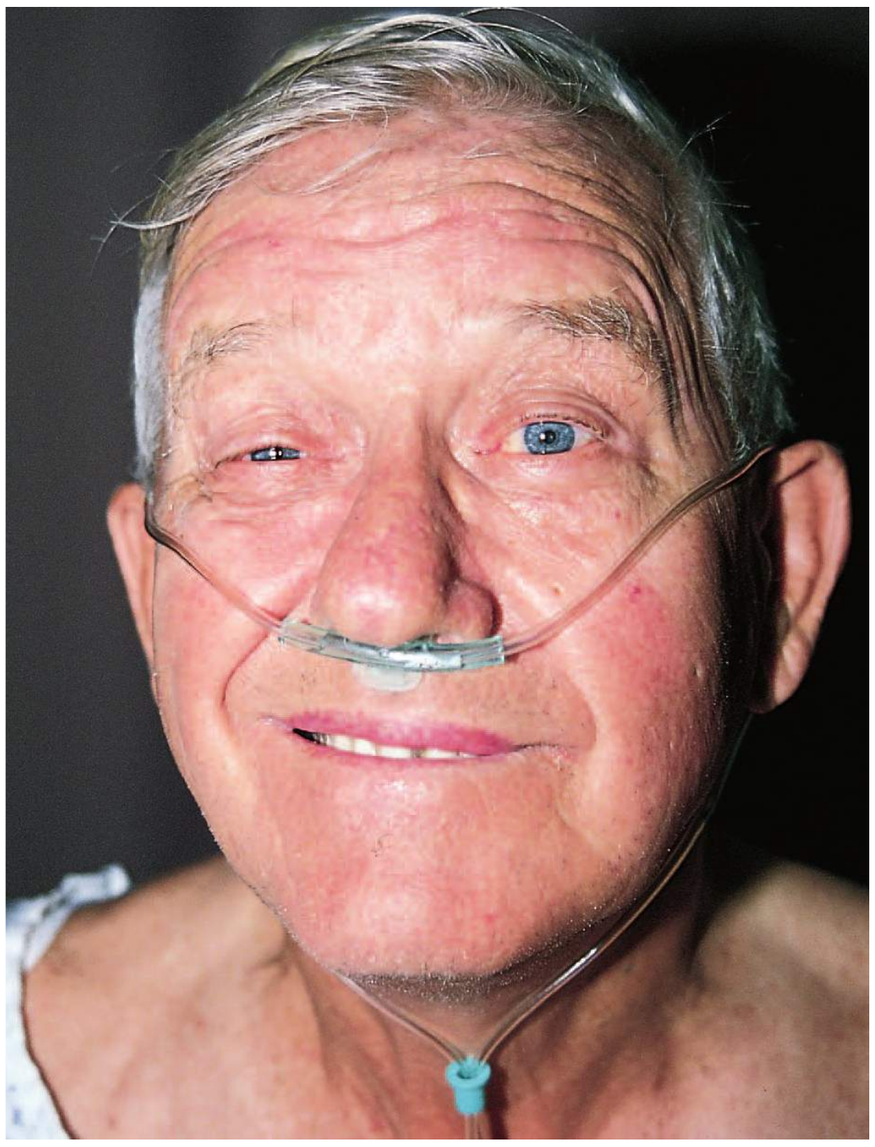

FIGURE 5.27 ■ **Central Seventh-Nerve Palsy.** Central facial nerve paralysis with forehead sparing. (Photo contributor: Frank Birinyi, MD.)

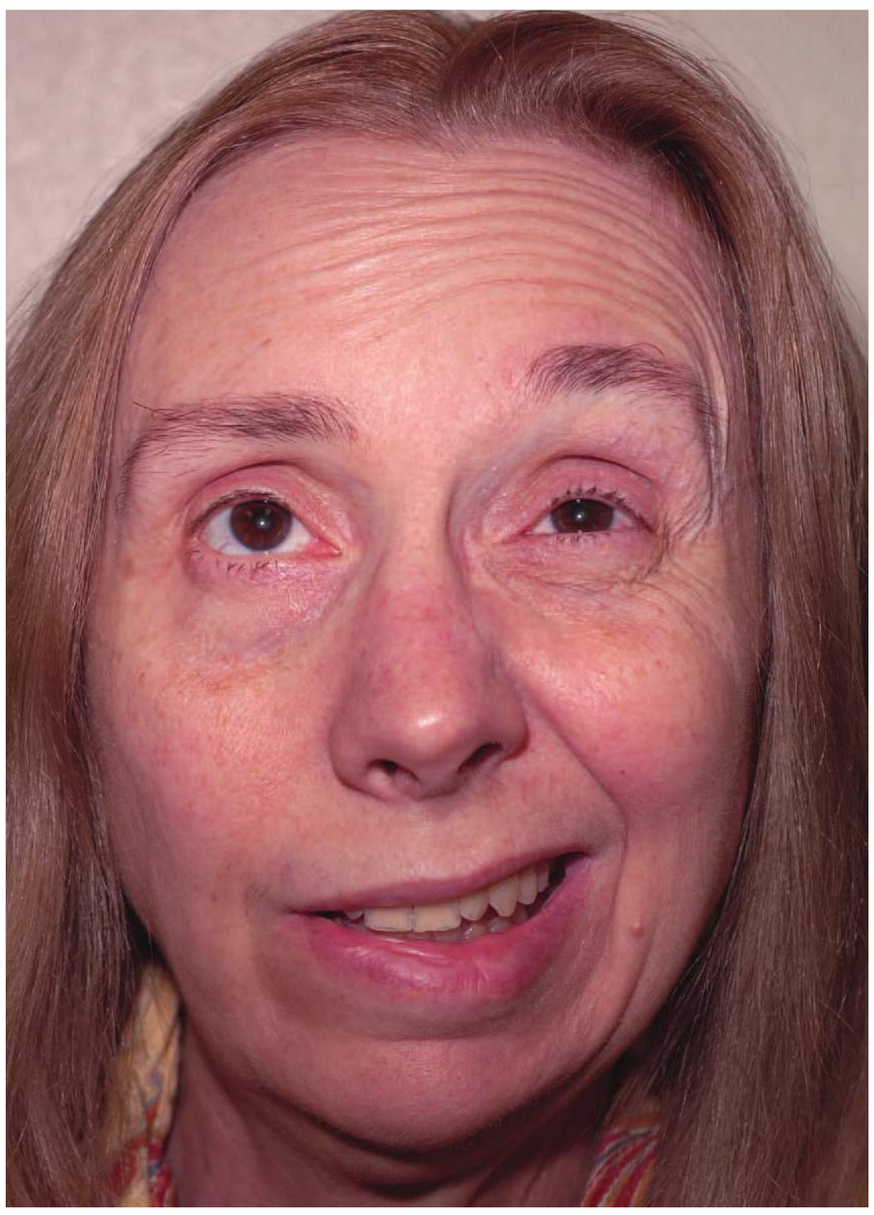

FIGURE 5.28 ■ **Peripheral Seventh-Nerve Palsy.** A peripheral nerve paralysis involving the entire ipsilateral face, including the forehead, is seen in this patient with Bell palsy. (Photo contributor: Lawrence B. Stack, MD.)

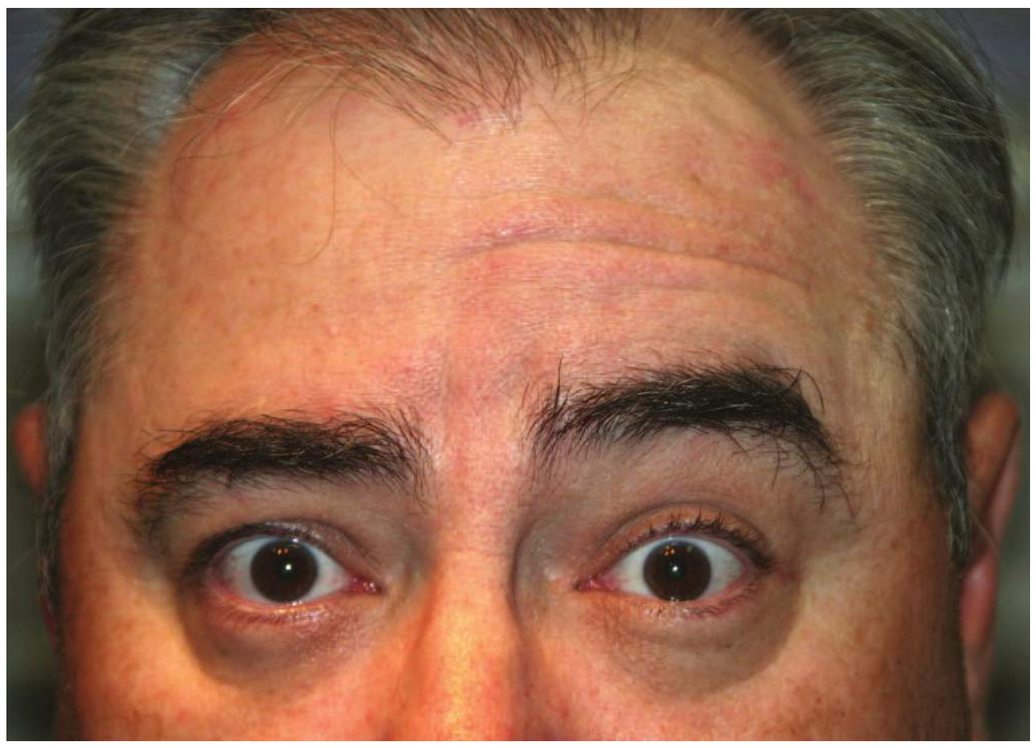

FIGURE 5.29 ■ **Peripheral Seventh-Nerve Palsy.** Forehead muscle paralysis is clearly apparent in this patient with Bell palsy. (Photo contributor: Suzanne Dooley-Hash, MD.)

Pearls

1. Facial nerve paralysis is a symptom, not a diagnosis.
2. If a provisional diagnosis of Bell palsy is made and no resolution of symptoms occurs, the diagnosis must be reconsidered. In patients misdiagnosed with Bell palsy, tumors are the most common missed etiology. MRI of the brainstem and internal auditory canal is indicated in these circumstances.
3. The finding of CN VI (lateral rectus) palsy along with CN VII palsy is diagnostic of a CN VI stroke, which involves the ipsilateral CN VII as it partially surrounds the CN VI nucleus. Hence, always evaluate for CN VI palsy when evaluating CN VII palsy.
4. Further ED evaluation is required for facial palsy presenting with sparing of the ipsilateral frontalis muscle as this represents a central lesion that may be caused by neoplasm or stroke.

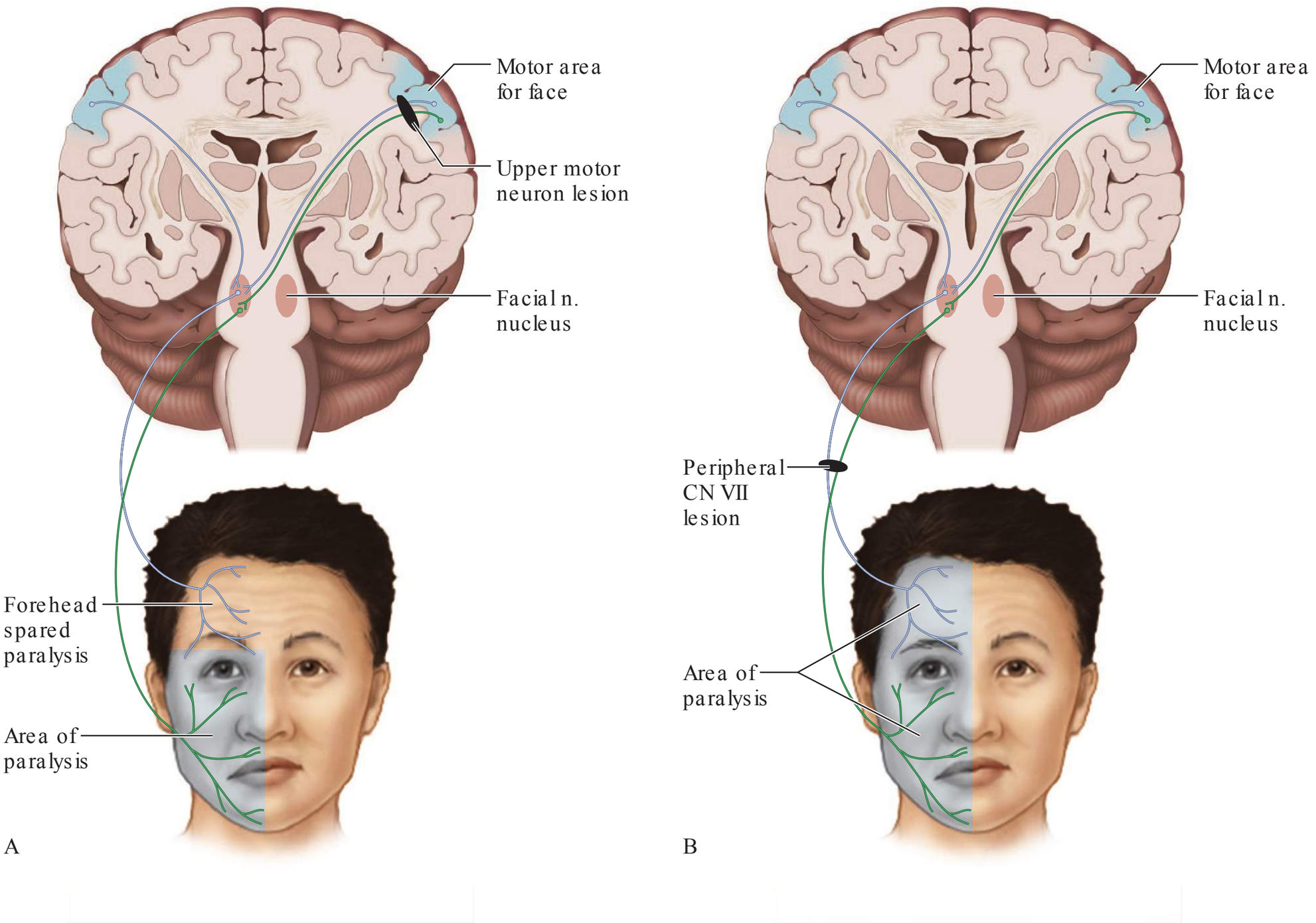

FIGURE 5.30 ■ **Cranial Nerve VII Neuropathways.** (**A**) Central CN VII Lesion. Crossed fibers allow forehead sparing in an upper motor neuron lesion identifying this presentation as a CVA vs a Bell palsy. (**B**) Peripheral CN VII nerve lesion causes paresis of both forehead and lower facial muscles.

Clinical Summary

Angioedema is clinically characterized by acute onset of well-demarcated cutaneous swelling of the face, lips, and tongue; edema of the mucous membranes of the mouth, throat, or abdominal viscera; or nonpitting edema of the hands and feet (often asymmetric). It is hereditary, allergic, or idiopathic. Hereditary angioedema is an autosomal dominant trait associated with a deficiency of serum inhibitor of the activated first component of complement (C1). Allergic angioedema can result from medications or contrast agents, environmental antigens, or local trauma. Complications range from dysphagia and dysphonia to respiratory distress, airway obstruction, and death. Angiotensin-converting enzyme (ACE) inhibitor–induced angioedema has a predilection for involvement of the lips, face, tongue, and glottis, and like hereditary angioedema, is often refractory to medical therapy.

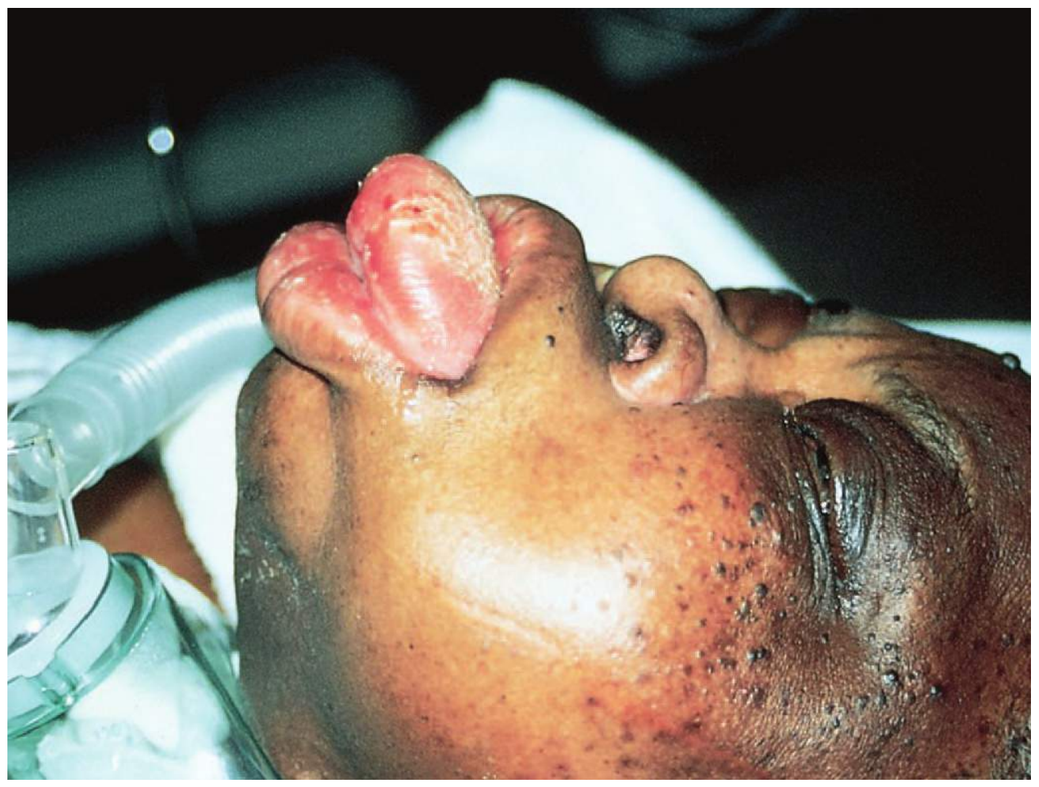

FIGURE 5.31 ■ **Angioedema.** Severe angioedema of the face and tongue requiring emergent cricothyroidotomy. (Photo contributor: W. Brian Gibler, MD.)

Management and Disposition

Airway protection remains the primary focus of emergency treatment. Frequent reassessment and early airway management is mandatory as deterioration due to edema formation can be rapid.

Medical therapy includes steroids, H_1 and H_2 histamine blockers, and subcutaneous or intramuscular epinephrine. Chronic angioedema responds better to corticosteroids and H_2 blockers. Hereditary angioedema may be treated with C-1 esterase inhibitors (eg, Cinryze).

Disposition depends on the severity and resolution of symptoms. Patients with symptomatic improvement or showing no worsening after 4 hours of observation may be discharged home. Discontinue suspect medications. Airway involvement requires admission to a monitored environment with surgical airway equipment at the bedside.

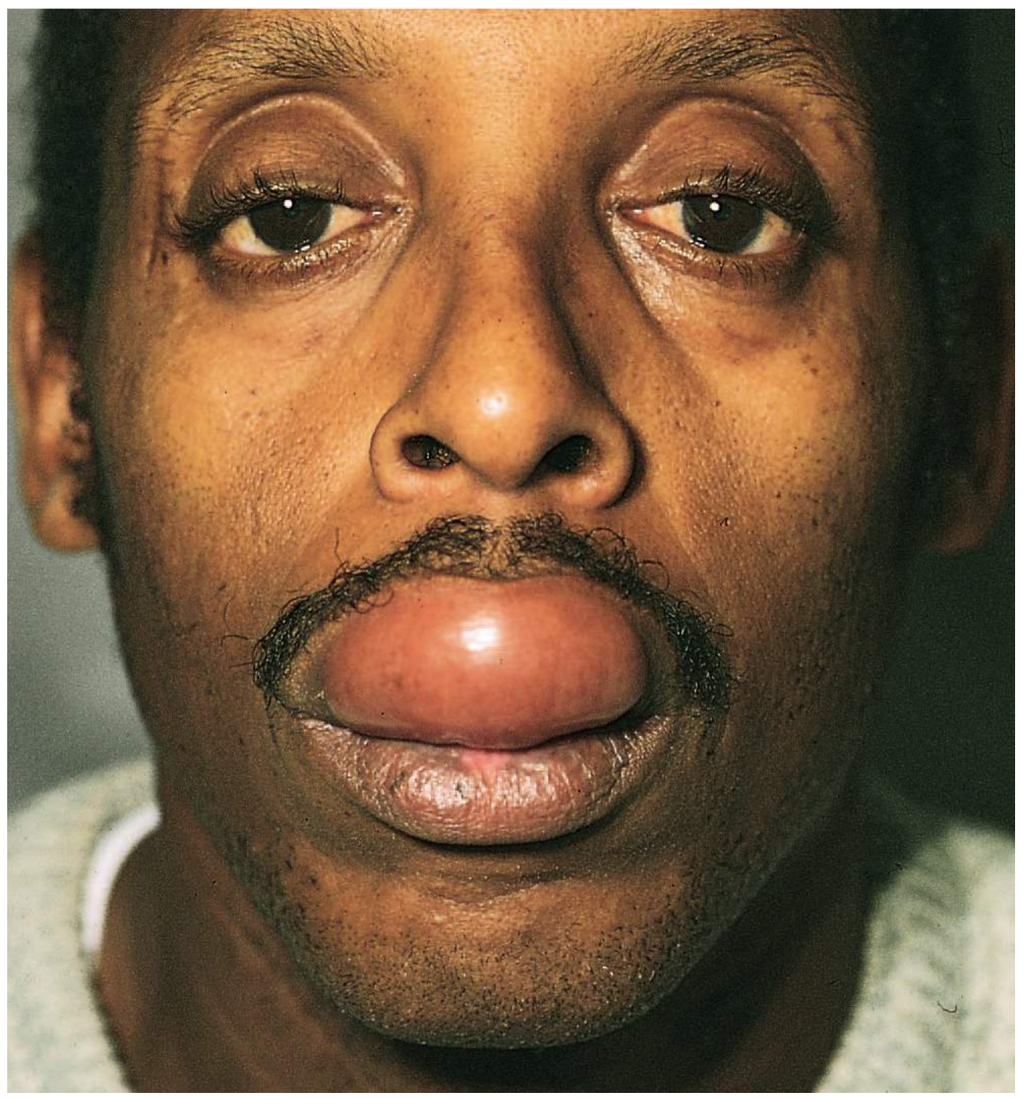

FIGURE 5.32 ■ **ACE Inhibitor–Induced Angioedema.** Angioedema of the upper lip in a man who had been taking an ACE inhibitor for 2 years. The patient had no previous episodes. (Photo contributor: Kevin J. Knoop, MD, MS.)

Pearls

1. Do not underestimate the degree of airway involvement; act early to preserve airway patency.
2. Angioedema can also cause gastrointestinal and neurologic involvement.
3. Early response to medical intervention does not preclude rebound of symptoms to a greater extent than at presentation.
4. Patients who have been using ACE inhibitors for months or years can still develop angioedema from these agents.

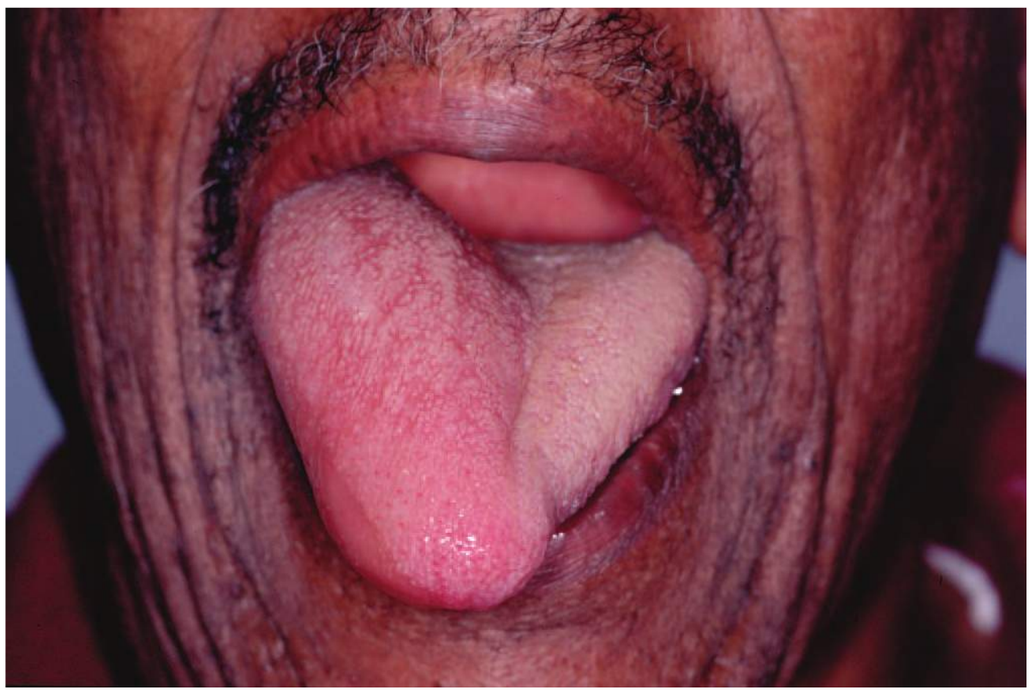

FIGURE 5.33 ■ **ACE Inhibitor–Induced Angioedema.** Unilateral tongue swelling in a man who had been taking ACE inhibitors for 5 years. (Photo contributor: Lawrence B. Stack, MD.)

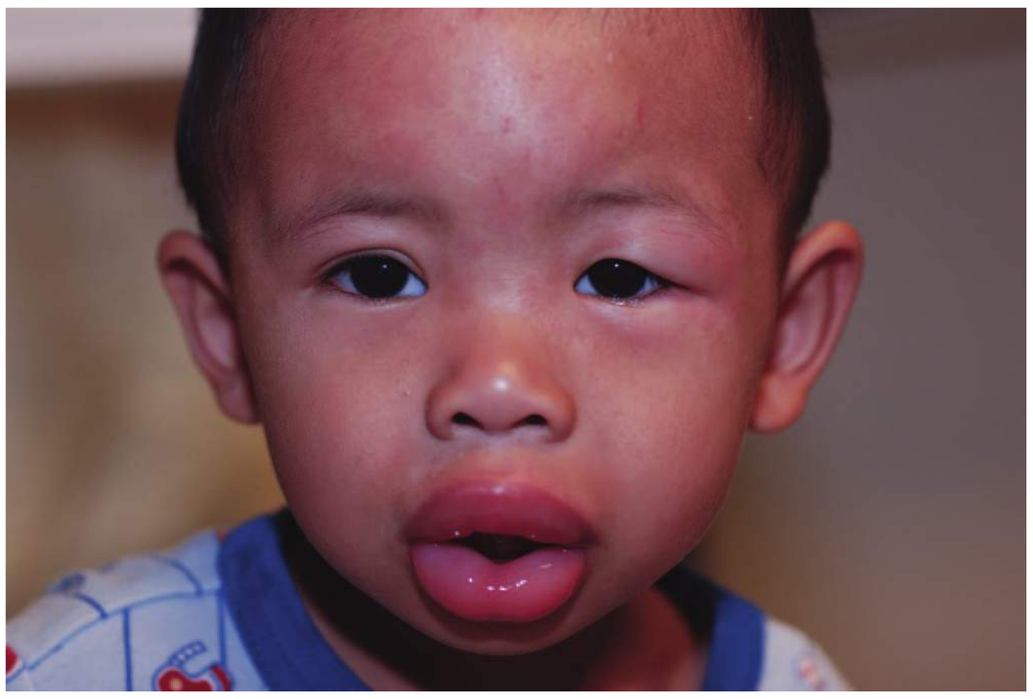

FIGURE 5.34 ■ **Angioedema.** Severe lip swelling in a patient with hereditary angioedema. (Photo contributor: Clay B. Smith, MD.)

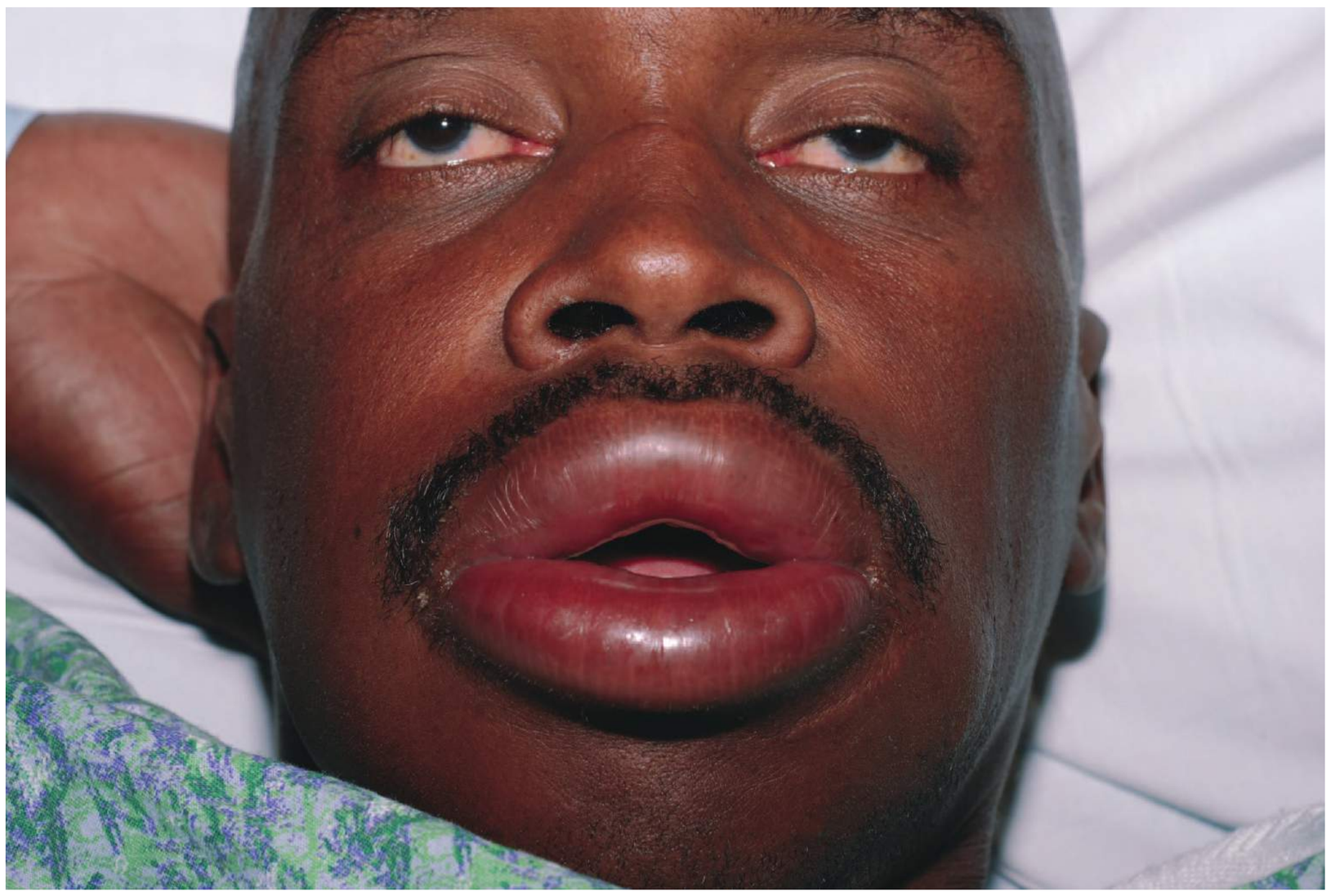

FIGURE 5.35 ■ **Angioedema.** Severe lip swelling from an unknown cause. (Photo contributor: Clay B. Smith, MD.)

Clinical Summary

Pharyngitis is an inflammation and commonly an infection of the pharynx and its lymphoid tissues. Viral causes account for 90% of all cases. Group A β-hemolytic streptococci (GABHS) is responsible for up to 50% of bacterial infections. Other bacterial causes include other streptococci, Mycoplasma pneumoniae, Neisseria gonorrhea, and Corynebacterium diphtheriae. In immunocompromised patients and patients on antibiotics, Candida species can cause thrush. Sore throats that last longer than 2 weeks should increase suspicion for either a deep-space neck infection or a neoplastic cause.

Patients with bacterial (especially GABHS) pharyngitis present with an acute onset of sore throat and fever, frequently accompanied by nausea, vomiting, headache, and abdominal cramping. They may have a mild to moderate fever, an erythematous posterior pharynx and palatine tonsils, tender cervical lymphadenopathy, and palatal petechiae. Classically, the tonsils have a white or yellow exudate with debris in the crypts; however, many patients do not have exudate on examination. Viral pharyngitis is typically more benign, with a gradual onset, less fever, and less impressive erythema and swelling of the pharynx. Except for infectious mononucleosis, which can take weeks to resolve, most cases of viral pharyngitis are self-limited, with spontaneous resolution in a matter of days. Lingual and adenoid tonsillitis may also be present.

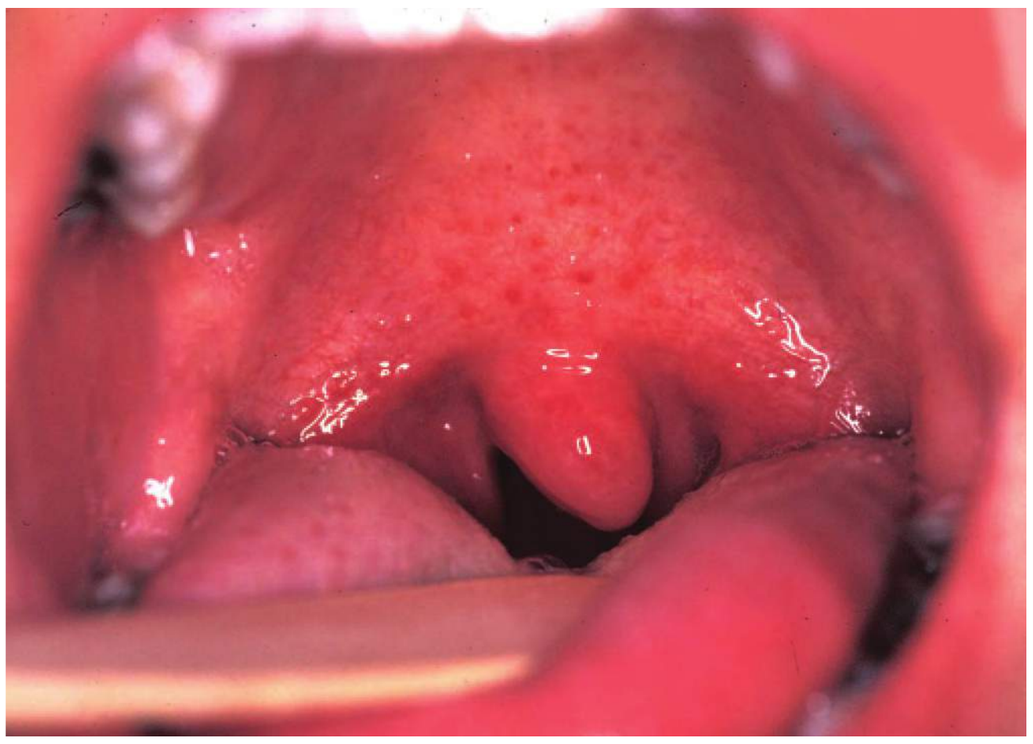

FIGURE 5.36 ■ **Palatal Petechiae.** Palatal petechiae and erythema of the tonsillar pillars in a patient with streptococcal pharyngitis. (Photo contributor: Kevin J. Knoop, MD, MS.)

Management and Disposition

Treatment is largely supportive except for antibiotics and rehydration. Analgesics, antipyretics, and throat sprays or gargles can provide symptomatic relief. Patients with known or suspected GABHS require antibiotics primarily to prevent rheumatic fever and suppurative complications. The Centor criteria are clinical decision rules developed to guide physicians in testing and prescribing of antibiotics. Criteria include: (1) tonsillar exudates, (2) tender anterior cervical adenopathy, (3) fever by history, and (4) absence of cough. Patients with fewer than two criteria should not receive either antibiotic treatment or diagnostic testing. Most authorities now favor evaluation using a sensitive rapid streptococcal antigen test (RSAT) for identification of GABHS, without throat culture for negative results, in adult patients with two or more Centor criteria. However, in children it is recommended that all negative RSAT be followed up with a throat culture. Current first-line antibiotic therapies remain a single dose of intramuscular benzathine penicillin or oral penicillin for 10 days.

Pearls

1. Other manifestations of pharyngitis should be sought in patients with sore throat. Sandpaper rash, Pastia lines, and desquamation are suggestive of scarlet fever. Hepatomegaly and/or splenomegaly should raise suspicion for infectious mononucleosis.
2. Sore throat or pharyngitis that lasts more than 2 weeks must be referred for further evaluation to rule out possible neoplastic or neurologic causes, especially in patients over 50 years old who have a smoking or chewing tobacco history.
3. Amoxicillin should be avoided if infectious mononucleosis is a possibility, as a morbilliform eruption will occur in up to 80% of patients.
4. Pharyngitis itself may be a prodrome for other pathologic conditions, such as measles, scarlet fever, and influenza.

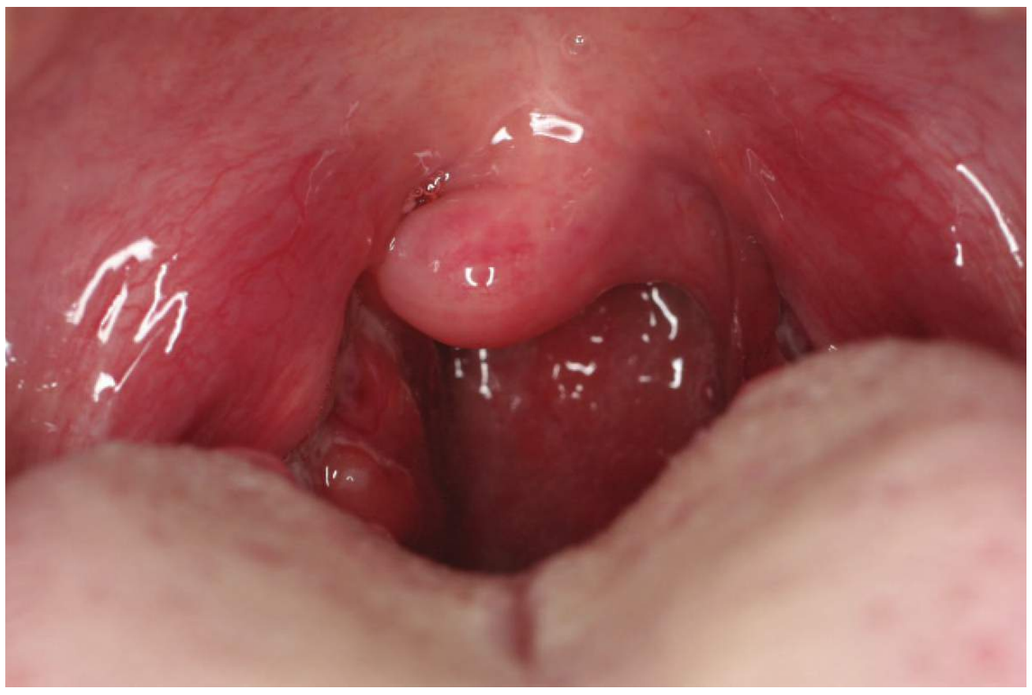

FIGURE 5.37 ■ **Exudative Pharyngitis.** Intense erythema with scant exudates is seen in this early (<24 hours) case of GABH pharyngitis. (Photo contributor: Kevin J. Knoop, MD, MS.)

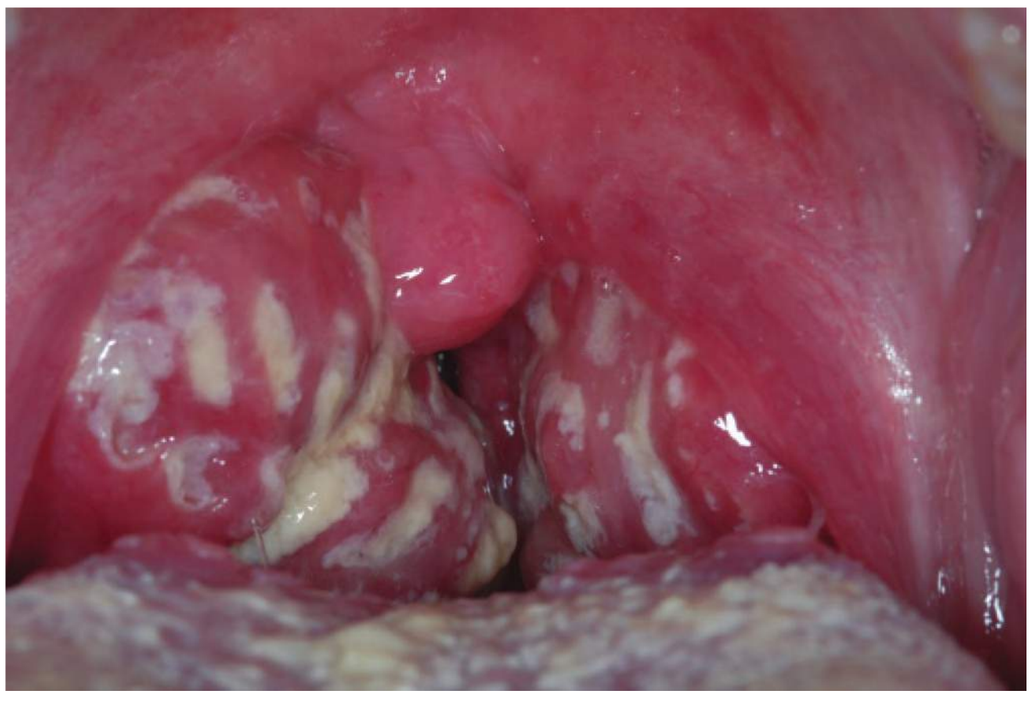

FIGURE 5.38 ■ **Tonsillar Exudate.** White and yellow cryptic exudates are seen in this patient with rapid strep test proven streptococcal pharyngitis. (Photo contributor: Lawrence B. Stack, MD.)

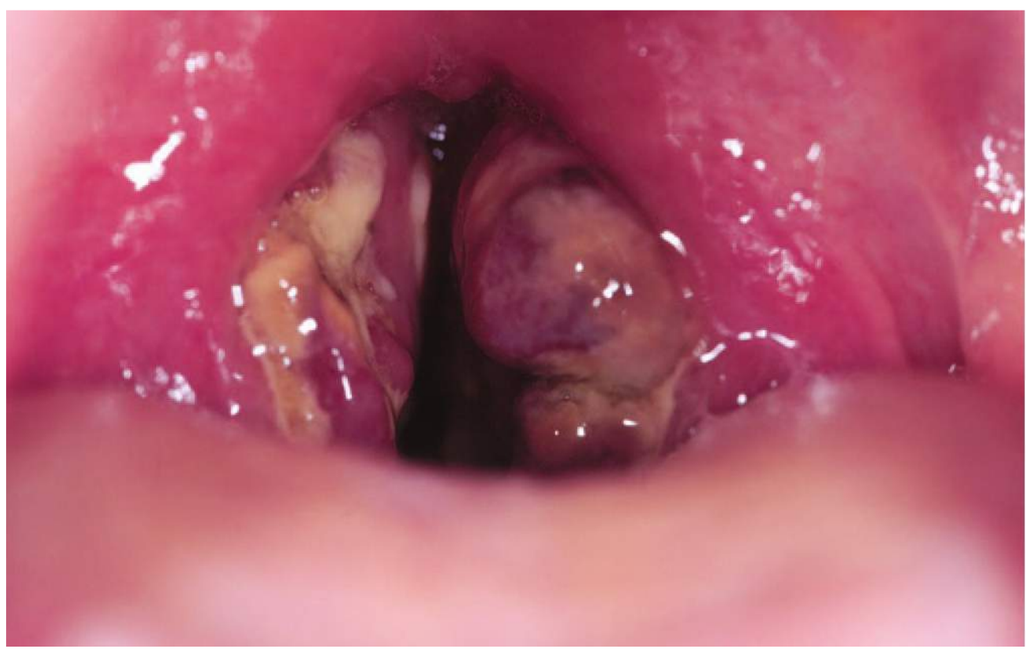

FIGURE 5.39 ■ **Infectious Mononucleosis.** Nearly touching exudative tonsils in a patient with elevated liver transaminases and hepatosplenomegaly. (Photo contributor: Lawrence B. Stack, MD.)

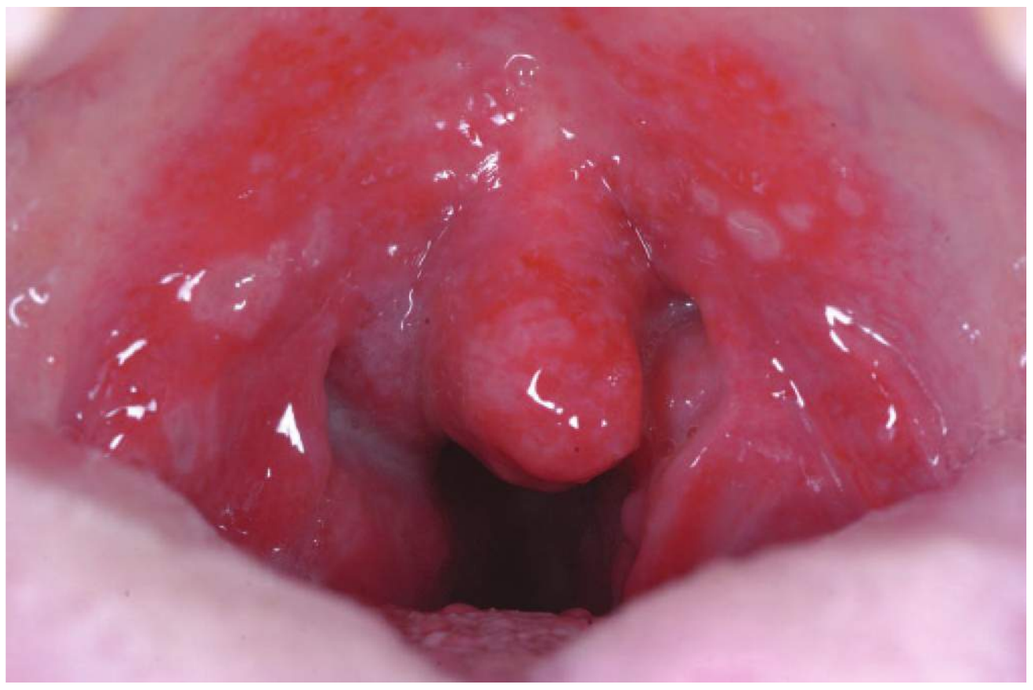

FIGURE 5.40 ■ **Viral Pharyngitis.** Intense erythema of the uvula and tonsillar pillars and ulcerations are typical of viral pharyngitis. (Photo contributor: Lawrence B. Stack, MD.)

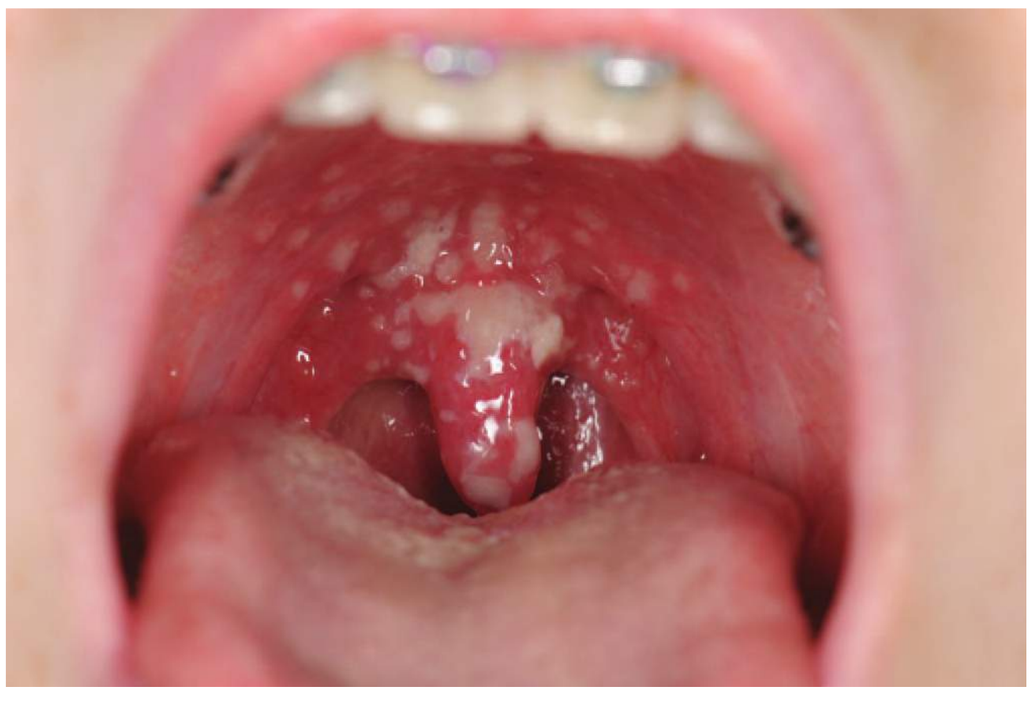

FIGURE 5.41 ■ **Inflammatory Pharyngitis.** Inflammation and ulcerations of the uvula and soft palate seen in this adolescent patient with a Crohn disease flare-up. (Photo contributor: Lawrence B. Stack, MD.)

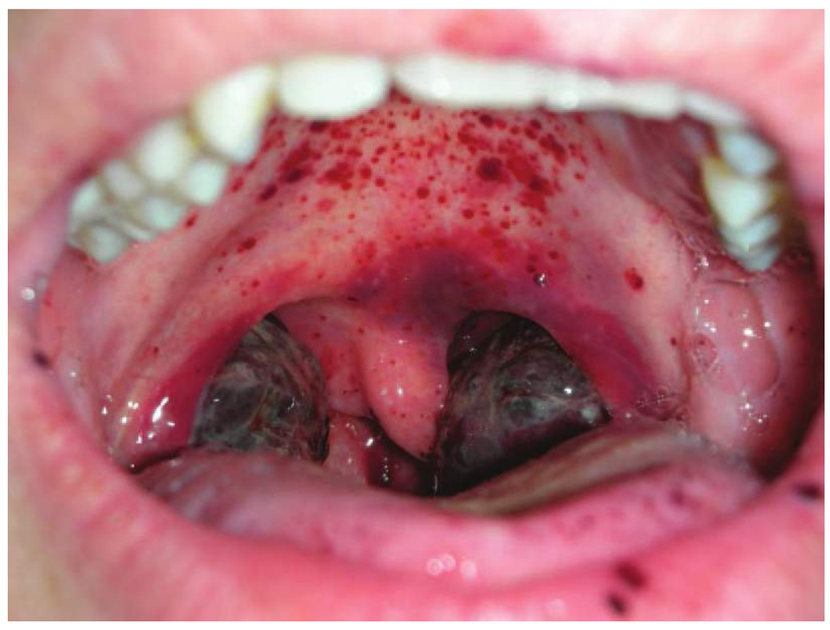

FIGURE 5.42 ■ **Hemorrhagic Pharyngitis.** Hemorrhagic tonsils, palatal petechiae, and pinch purpura of the buccal mucosa are seen in this patient with infectious mononucleosis-induced immunologic thrombocytopenic purpura. (Photo contributor: Sheila McMorrow-Jones, MD.)

Clinical Summary

Septal hematoma, with or without abscess formation, is an uncommon complication of septal surgery or direct nasal trauma. Bleeding from submucosal blood vessels leads to an accumulation of blood between the mucoperichondrium and the septal cartilage, which may lead to ischemic avascular necrosis of the underlying cartilage, destruction of the cartilage, and saddle nose deformity (see Fig. 1.13). The hematoma and any necrotic cartilage may then serve as a nidus for infection, resulting in a septal abscess. Chronic cocaine use results in vasoconstriction and eventual necrosis of the septal cartilage leading to perforation.

Septal injuries may lead to cosmetic nasal deformity, chronic sinus infections, recurrent epistaxis, and sleep disturbances. Rarely, it can result in more serious complications such as cavernous sinus thrombosis and meningitis. Since the original trauma is often minor, patients may present days to weeks after the injury. Young children and infants may present with poor feeding, fever, and rhinorrhea. Older children and adults may note bleeding, headache, and more focal pain.

Examination reveals a large, red, round swelling originating off the septum and occluding most of the nasal cavity. The mass is tender to palpation and may cause the outer aspects of the nose to be tender as well. Septal abscesses tend to be more painful and larger than uncomplicated hematomas. Fever is frequently present. S aureus, group A β-hemolytic streptococcus, H influenzae, and S pneumoniae are the organisms most commonly isolated in septal abscesses. A septal perforation is easily seen with penlight examination.

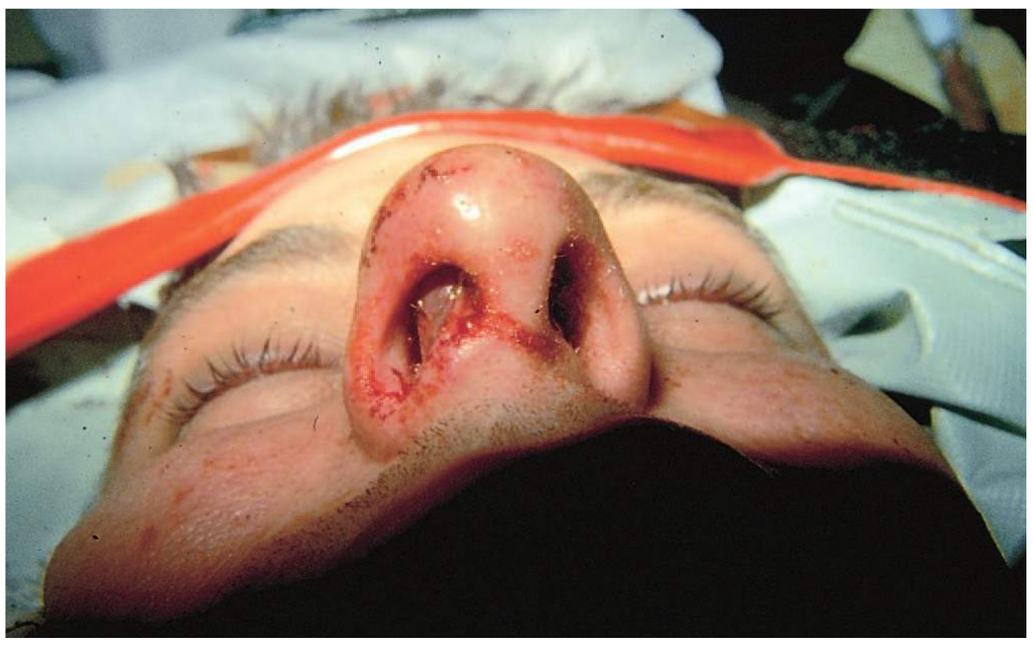

FIGURE 5.43 ■ **Septal Hematoma.** Fluctuant grapelike structure in the right naris after blunt facial trauma consistent with a septal hematoma. (Photo contributor: Lawrence B. Stack, MD.)

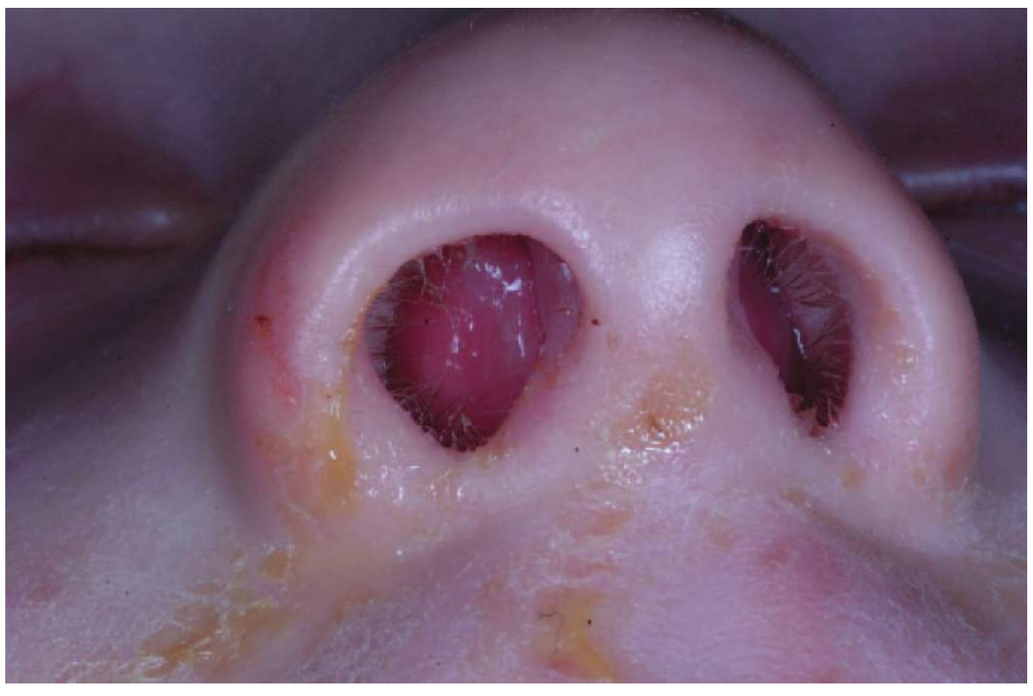

FIGURE 5.44 ■ **Septal Abscess.** A septal abscess is seen in both nares 1 week after blunt nasal trauma. (Photo contributor: Lawrence B. Stack, MD.)

Management and Disposition

Suspicion and recognition are essential in diagnosing septal injuries. Prompt referral to an otolaryngologist is mandatory for incision and drainage of the hematoma or abscess.

Pearls

1. Intranasal examination for septal hematoma in all patients with a history of nasal trauma regardless of severity is crucial.
2. Antibiotics are required in septal hematomas with a clinical suspicion for a secondary infection or abscess.
3. The physician must explore the possibility of child abuse in young children and infants with a septal hematoma or abscess.

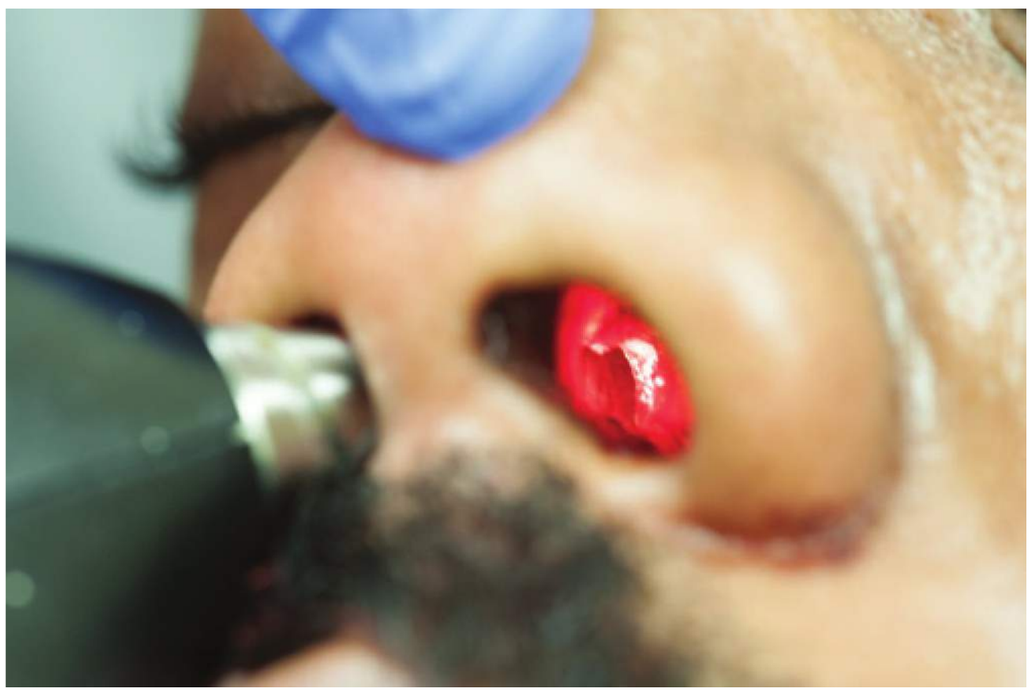

FIGURE 5.45 ■ **Septal Perforation**. A septal perforation is illuminated with a light in a chronic cocaine user. (Photo contributor: Lawrence B. Stack, MD.)

NASAL CELLULITIS

Clinical Summary

Nasal cellulitis is an infection of the skin and subcutaneous tissues, and does not include nasal cartilage. It is most common at the extremes of age. Bacterial invasion due to disruption of the skin is the usual cause. S pyogenes and S aureus cause most infections. Risk factors include nasal surgery, instrumentation, diabetes, immunodeficiency, and nasal piercing. Clinical features include pain, redness, and swelling of the nasal tissues. Headache, fever, and malaise suggest complicated disease. Complications of nasal cellulitis include abscess, cavernous sinus thrombosis, chondritis of the nasal cartilage, bacteremia, and sepsis.

Management and Disposition

The diagnosis of nasal cellulitis is clinical. However, evaluation may include complete blood count, blood and tissue cultures, contrasted CT if abscess is suspected, and CT-V if a cavernous sinus thrombosis is suspected. Remove any foreign body that might be a nidus of infection. Amoxicillin-clavulanate or amoxicillin-sulbactam is first-line antibiotics. Clindamycin, vancomycin, trimethoprim-sulfamethoxazole, and first-generation cephalosporins are other options. Patients should be hospitalized if they have systemic symptoms, are immunocompromised, have diabetes or a suspected abscess, or have retained foreign bodies. Patients at extremes of age should be strongly considered for admission.

Pearls

1. Threshold for hospital admission for patients with nasal cellulitis should be low.
2. In children, consider H influenza and S pneumoniae as causative organisms of facial cellulitis.
3. Patients with pain at the nasal entrance but no overt physical findings of cellulitis may have vestibulitis, responsive to topical antibiotics (mupirocin).

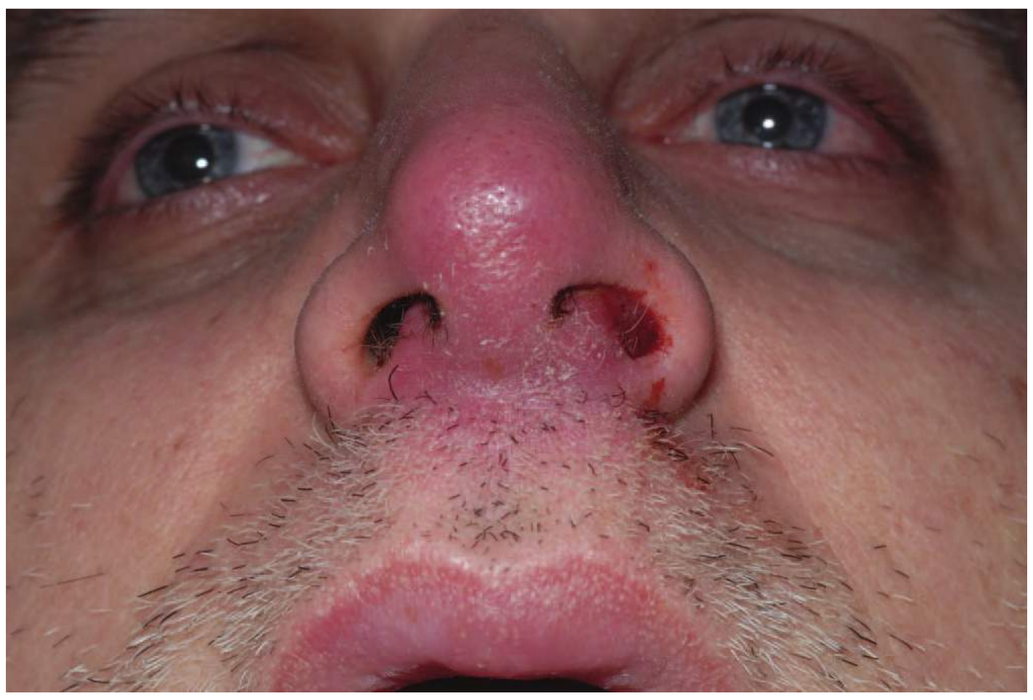

FIGURE 5.46 ■ **Nasal Cellulitis.** Swelling and erythema of the nose in a middle-aged man. A contrasted CT was done to exclude abscess because of the marked swelling of the nasal septum. (Photo contributor: Lawrence B. Stack, MD.)

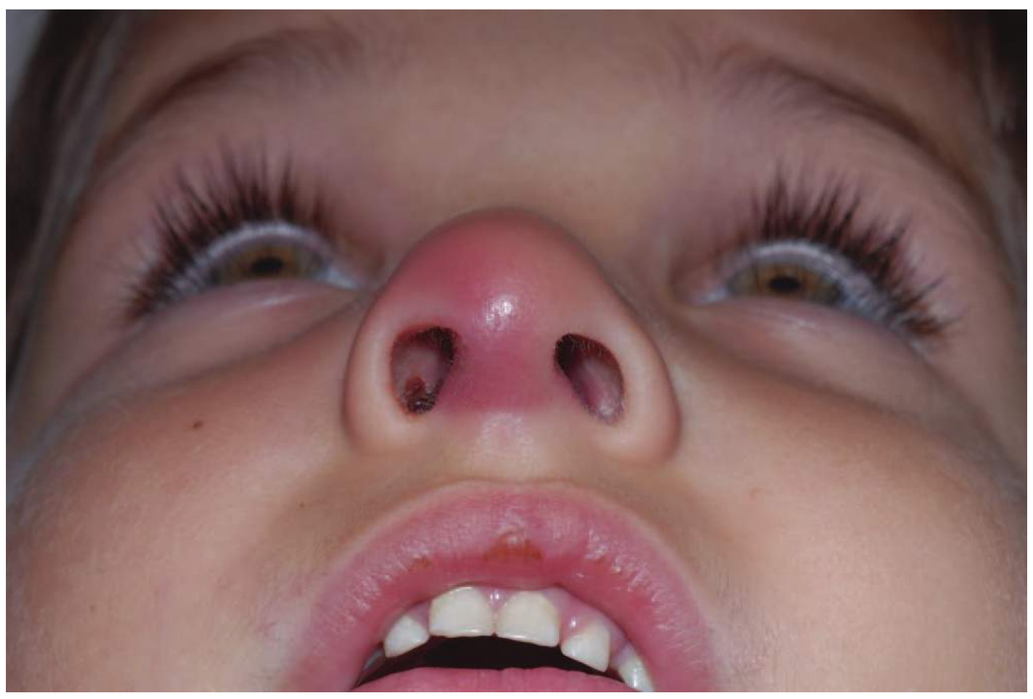

FIGURE 5.47 ■ **Nasal Cellulitis.** Swelling and erythema of the nose of a 2-year-old child, suggesting nasal cellulitis. (Photo contributor: Lawrence B. Stack, MD.)

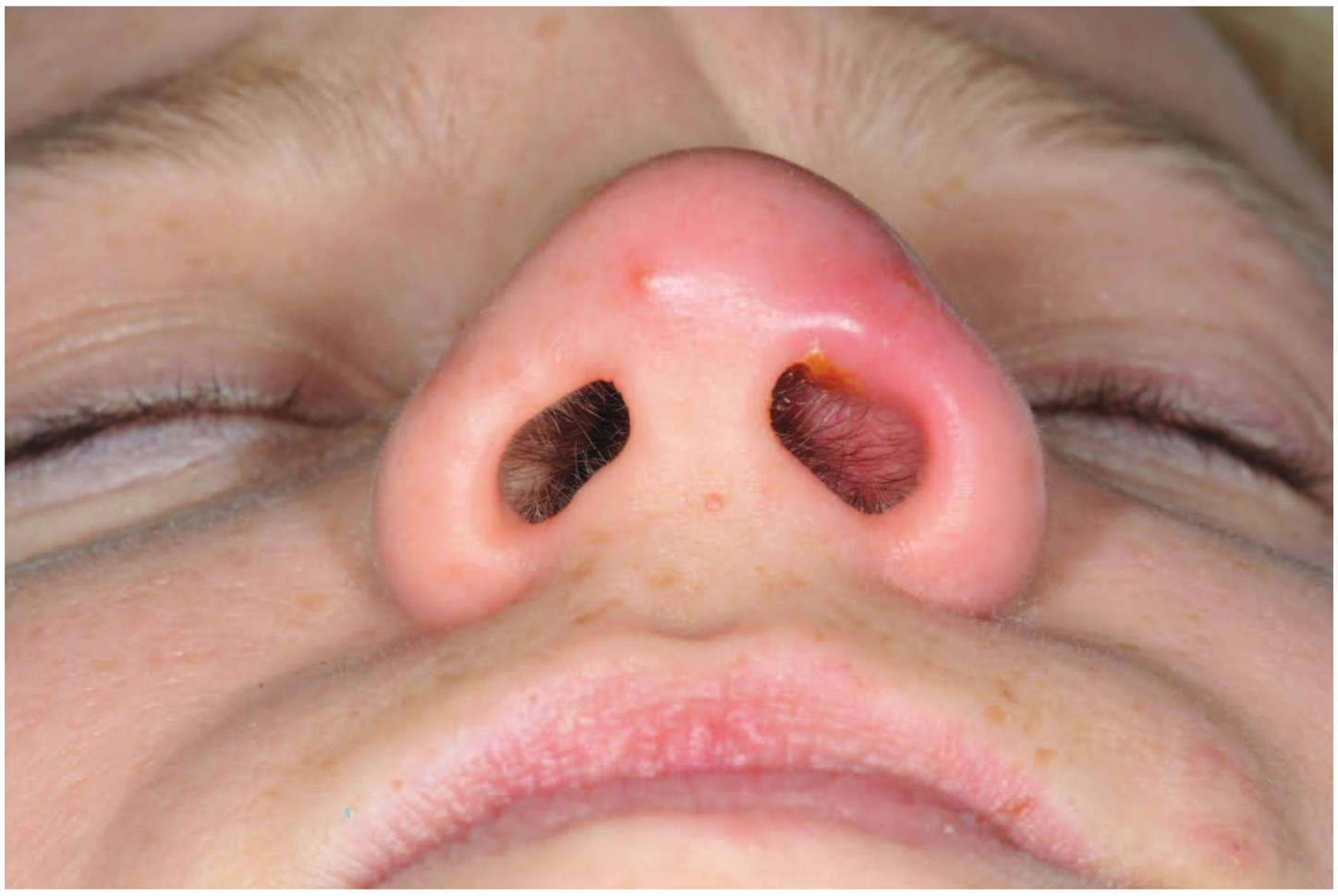

FIGURE 5.48 ■ **Nasal Cellulitis.** Swelling and erythema of soft tissue surrounding the internal and external sides of nasal cartilage seen in early nasal cellulitis. (Photo contributor: Lawrence B. Stack, MD.)

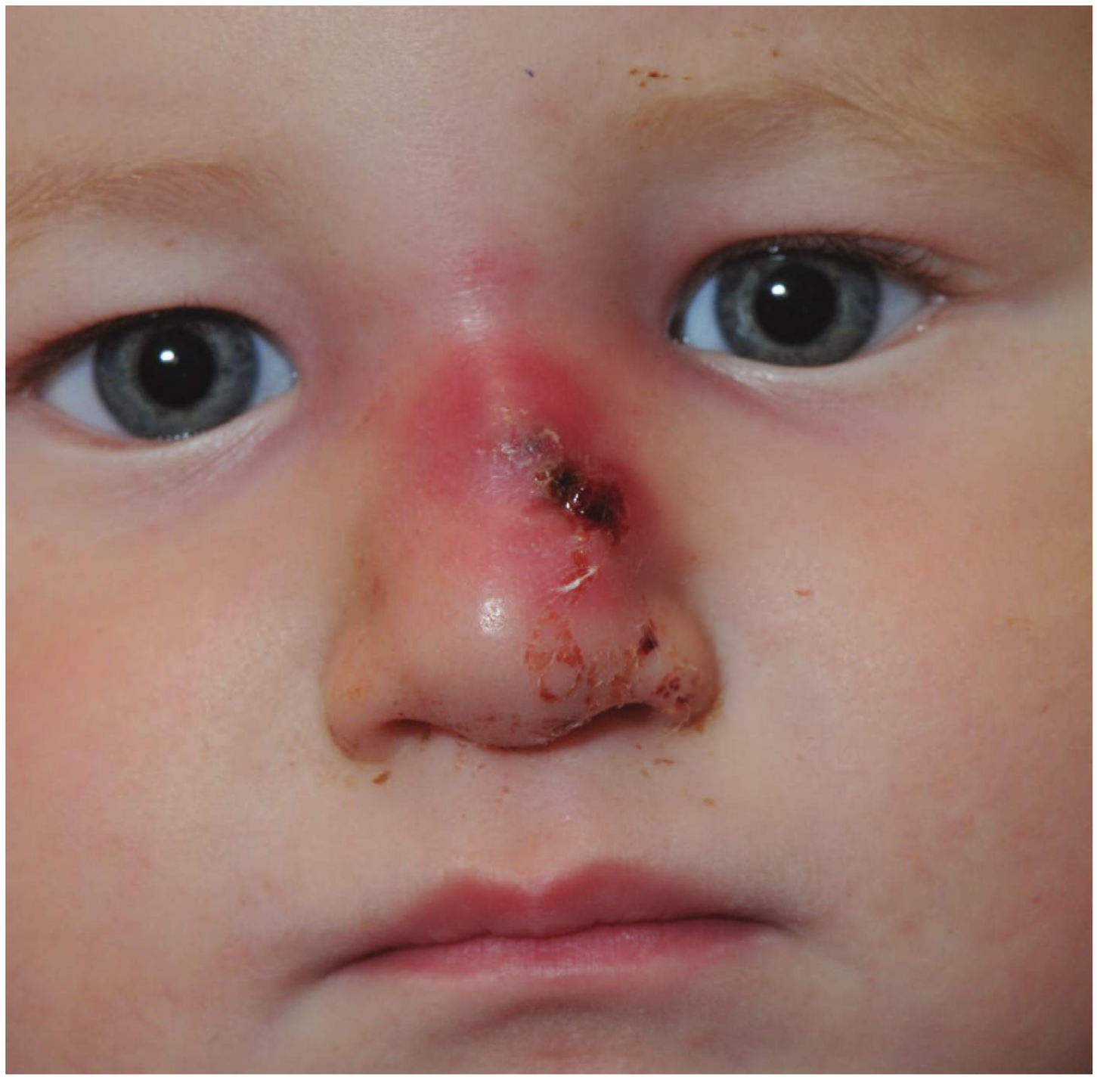

FIGURE 5.49 ■ **Nasal Cellulitis.** Intense erythema, swelling, and drainage consistent with MRSA abscess and cellulitis of the nose. (Photo contributor: Lawrence B. Stack, MD.)

DIPHTHERIA

Clinical Summary

Diphtheria is a highly contagious disease caused by the exotoxin-producing bacterium C diphtheriae. It is transmitted either by direct contact or through respiratory aerosolization. Many adults are now susceptible to diphtheria because their vaccine-induced immunity decreased over time or owing to decreased opportunity for naturally acquired immunity.

Diphtheria may involve any mucous membrane; but most commonly, it affects the mucosa of the upper respiratory tract. It typically produces an ulcerated pharyngeal mucosa with a white-to-gray inflammatory pseudomembrane, classically with a "wet mouse" odor. Patients present with symptoms, in order of frequency, of fever, sore throat, weakness, pain with swallowing, change in voice, loss of appetite, neck swelling, difficulty breathing, and nasal discharge.

While the organism remains localized to the mucosa, hematogenous spread of the exotoxin typically produces myocarditis or peripheral neuropathies. Deaths from diphtheria occur from either tracheobronchial obstruction by the pseudomembrane acutely or cardiac complications several weeks after the primary infection.

Management and Disposition

The diagnosis is initially made clinically and confirmed by successful isolation and toxigenicity testing of C diphtheriae.

Antitoxin, available from the Centers for Disease Control and Prevention (CDC), is the mainstay of therapy and must be given even before laboratory confirmation. Erythromycin or penicillin given promptly when diphtheria is suspected has been shown to decrease both exotoxin production and spread of the bacterium.

Patients require hospital admission for observation of airway obstruction, pulmonary support, and intravenous hydration and antibiotics. Strict isolation is essential.

Pearls

1. Outcome is improved with early treatment; thus, the diagnosis of diphtheria must be made clinically and treatment begun empirically before bacteriologic confirmation.
2. Patients with a membranous pharyngitis need to be questioned regarding immunization, exposures, and recent travel.
3. All contacts should have a booster dose of vaccine (TD or Td, depending on age), while nonimmune contacts should also be given prophylactic antibiotics after a throat swab.

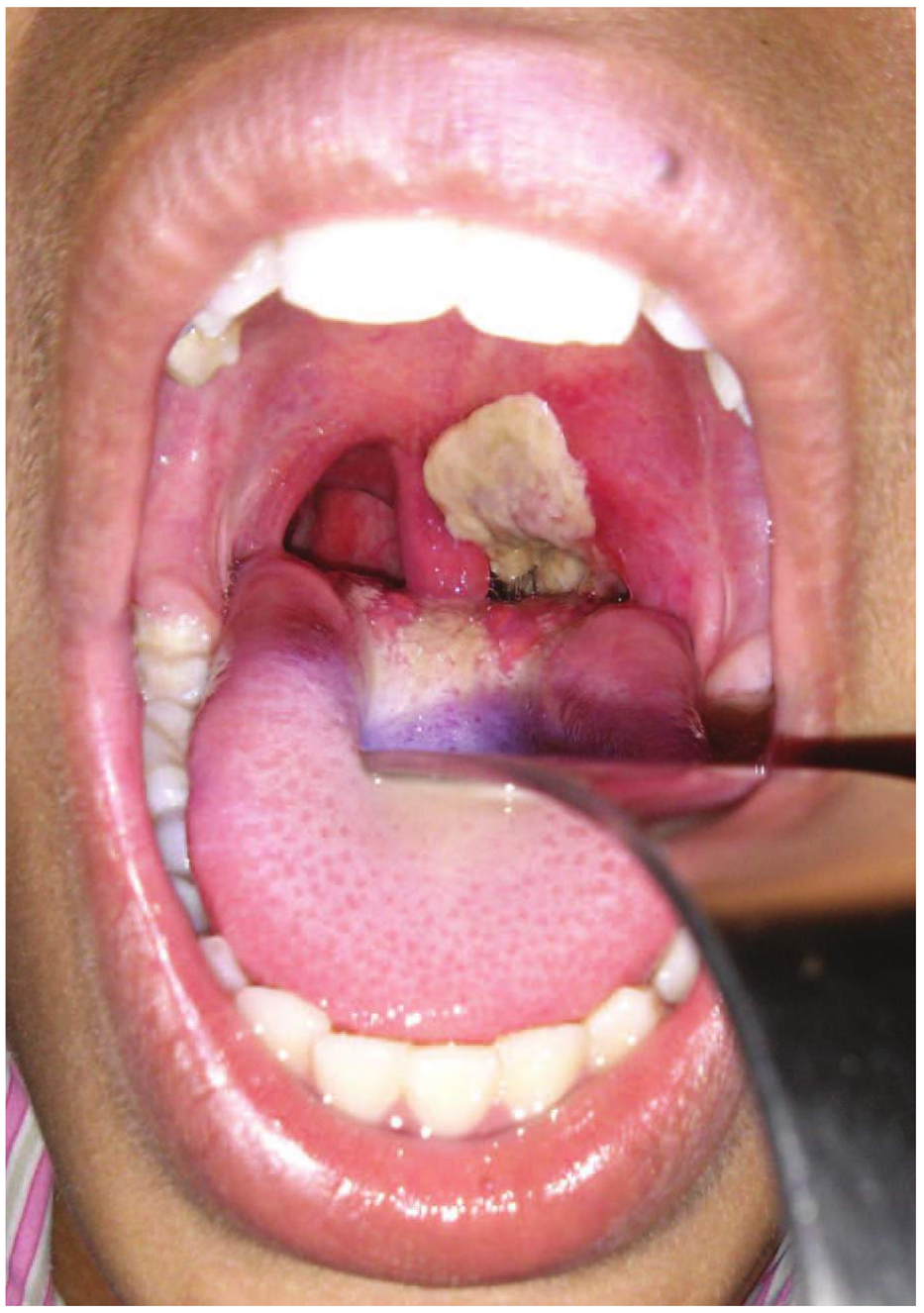

FIGURE 5.50 ■ **Diphtheria Pharyngitis.** An exudative pharyngitis with a gray pseudomembrane is seen in this patient with diphtheria. (Photo contributors: Dileep C. Unnikrishnan, MD, and Nadeem Kocheri, MD.)

Clinical Summary

Peritonsillar abscess (PTA), or quinsy, is the most common deep neck infection. Although most occur in young adults, immune compromised and diabetic patients are at increased risk. Most abscesses develop as a complication of tonsillitis or pharyngitis, but may also result from odontogenic spread, recent dental procedures, and local mucosal trauma.

The pathogens involved are similar to those causing tonsillitis, especially streptococcal species, but many infections are polymicrobial and involve anaerobic bacteria. Patients present with a fever, severe sore throat that is often out of proportion to physical findings, localization of symptoms to one side of the throat, trismus, drooling, dysphagia, dysphonia, fetid breath, and ipsilateral ear pain.

During the early stages, the tonsil and anterior pillar are erythematous, appear full, and may be shifted medially. Later, the uvula and soft palate are shifted to the contralateral side. The tonsillar pillar may feel fluctuant and tender.

Management and Disposition

Most patients can have needle aspiration performed as the sole surgical drainage procedure and expect a satisfactory outcome. Alternative surgical drainage procedures—including incision and drainage and abscess tonsillectomy—can be performed by an otolaryngologist or oral surgeon. Most are managed as outpatients on oral antibiotics following drainage. Patients who are immunocompromised, have airway involvement, appear toxic, or cannot tolerate oral intake require admission for rehydration, parenteral antibiotics, and specialty consultation.

Pearls

1. The value of culturing aspirates is questionable, with a review of several studies showing no clinical benefit from the cultures unless the patient is immunocompromised.
2. ED ultrasound along with palpation of the mass is a valuable adjunct in confirming diagnosis and verifying landmarks prior to aspiration.
3. A contrasted CT scan of the neck will confirm the presence of a PTA if the diagnosis is uncertain.

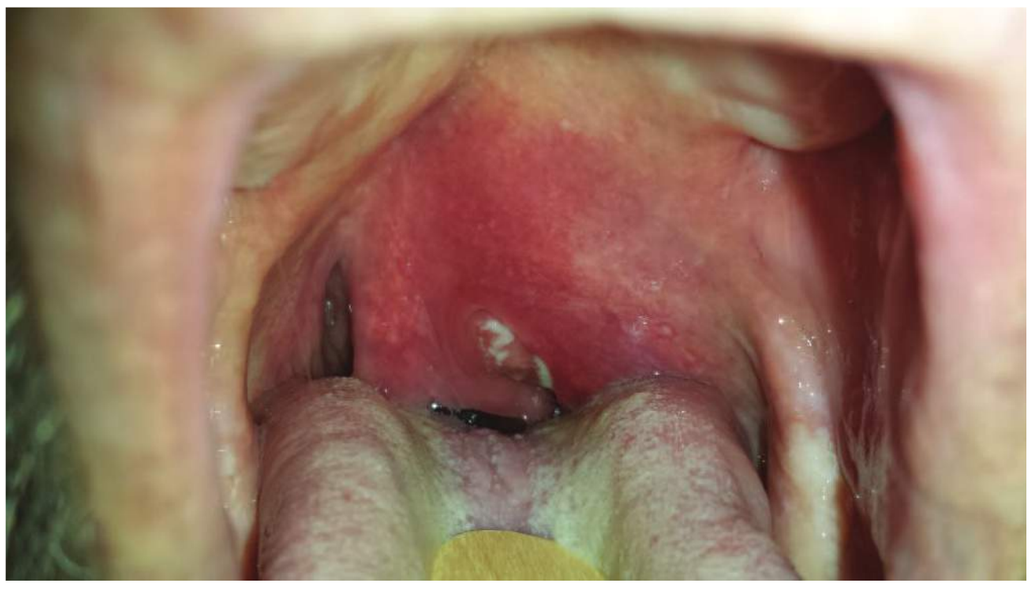

FIGURE 5.52 ■ **Peritonsillar Abscess.** Acute peritonsillar abscess showing medial displacement of the uvula, palatine tonsil, and anterior pillar. (Photo contributor: Lawrence B. Stack, MD)

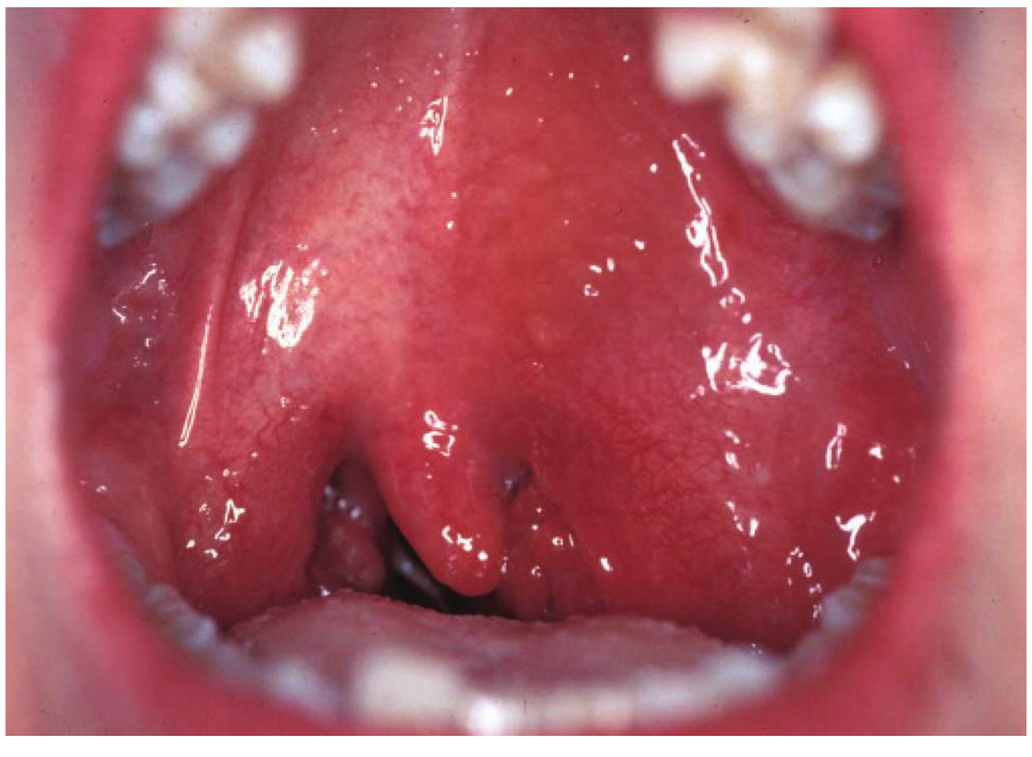

FIGURE 5.51 ■ **Early Peritonsillar Abscess.** Edema and marked erythema of the left tonsillar pillar in early peritonsillar abscess. (Photo contributor: Kevin J. Knoop, MD, MS.)

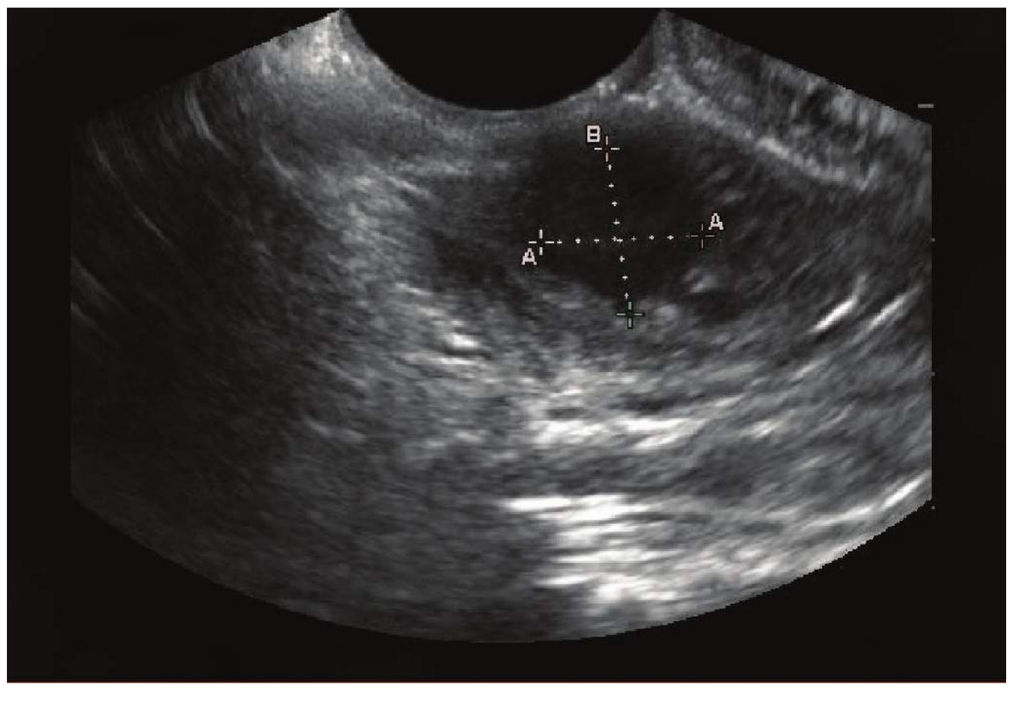

FIGURE 5.53 ■ **Peritonsillar Abscess—Ultrasound.** Ultrasound demonstrates size and location of abscess, in addition to confirming relationship to vascular structures. Over 2 cc of pus was aspirated from this abscess. (Photo contributor: Kevin J. Knoop, MD, MS.)

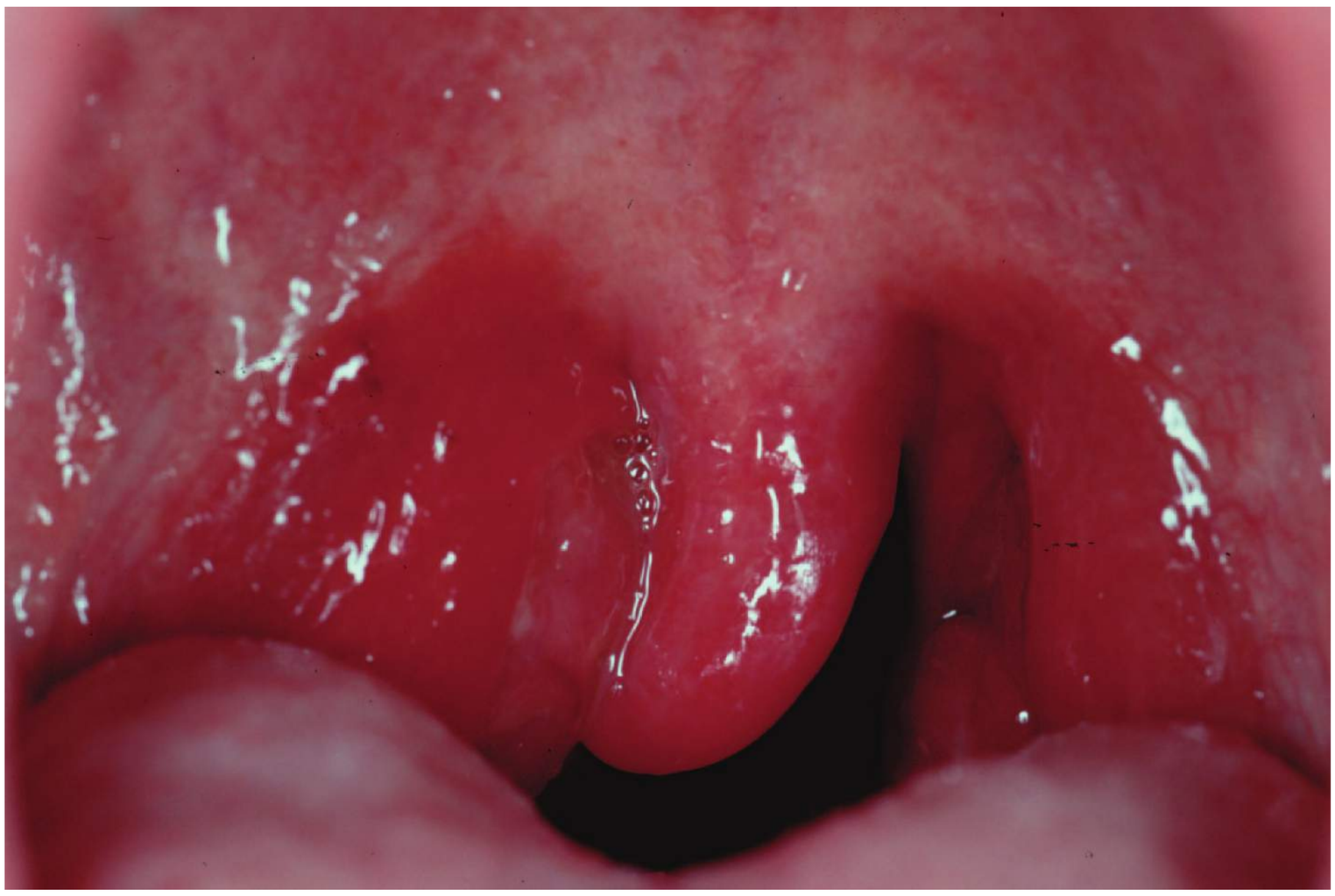

FIGURE 5.54 ■ **Peritonsillar Phlegmon.** Marked erythema of the tonsillar pillars is seen in this patient currently on oral penicillin. No swelling or fluctuance is present. (Photo contributor: Lawrence B. Stack, MD.)

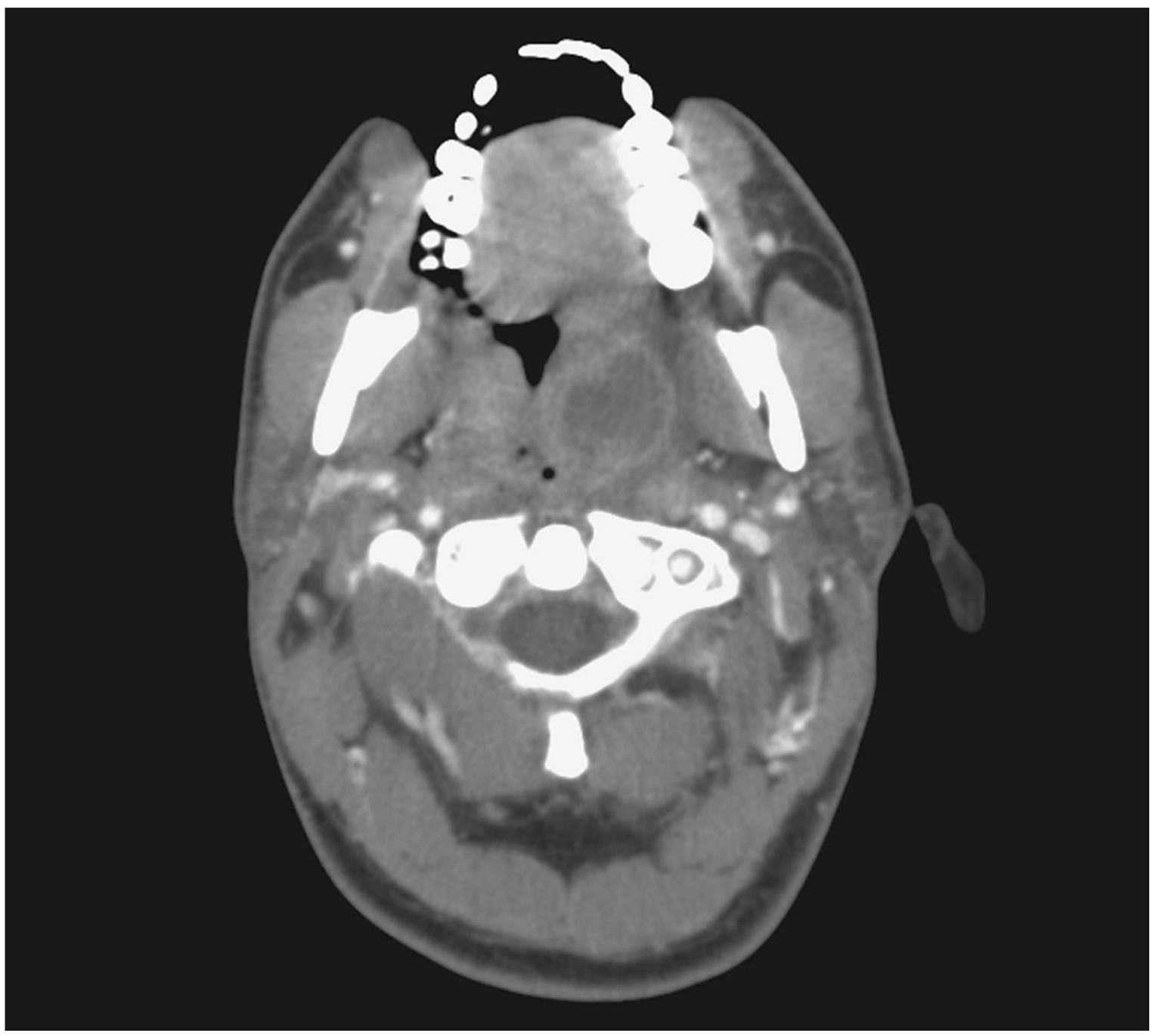

FIGURE 5.55 ■ **Peritonsillar Abscess—CT.** Ring-enhancing lesion with a hypodense core consistent with a left peritonsillar abscess. (Photo contributor: Lawrence B. Stack, MD.)

Clinical Summary

Epiglottitis or supraglottitis is an infection of the epiglottis and adjacent tissues. Bacterial epiglottitis, a rare but potentially fatal infection, is caused primarily by H influenzae, but S pneumoniae, S aureus, and β-hemolytic streptococcus have also been isolated. The advent of the H influenzae B vaccination for infants has changed what used to be a disease primarily of children, with a peak age range from 2 to 6 years, to one occurring predominantly in adults. Bacterial epiglottitis occurs most commonly in the winter and spring.

Patients, especially children, with acute epiglottitis appear quite ill. They present with sore throat, fever, drooling, severe dysphagia, dyspnea, muffled or hoarse voice, and occasionally inspiratory stridor. Patients with severe respiratory distress assume the "tripod" position: sitting upright with the neck extended, arms supporting the trunk, and the jaw thrust forward. This position maximizes airway patency and caliber. Adults typically have an indolent course with a prodromal viral illness, but many children have a sudden onset and rapid progression to respiratory distress.

Management and Disposition

Airway management is paramount. Even prior to diagnosis, children should be calmed, comforted by a parent, and allowed to assume whatever position they feel is most comfortable. Anesthesiology and ENT should be consulted immediately. Indications for intubation are clinical, but severe stridor and respiratory distress are clear reasons to intervene. Nasotracheal intubation over a flexible endoscope is preferred. Needle cricothyrotomy can provide temporary oxygenation until a surgical airway is provided.

Radiographs of the neck may reveal the classic "thumb" sign, a thickened epiglottis on the lateral soft-tissue neck radiograph. Visualization of the epiglottis is possible in the stable adult patient via direct and indirect laryngoscopy and fiberoptic nasopharyngoscopy. In children, the top of the swollen epiglottis may be visualized on careful oral examination, whereas pharyngoscopy is typically reserved for an experienced anesthesiologist or otolaryngologist in a controlled setting.

The mainstay of epiglottitis treatment is antibiotic therapy. Third-generation parenteral cephalosporins, ampicillin with sulbactam or trimethoprim-sulfamethoxazole, have proven efficacy in treating epiglottitis. Steroids or epinephrine, either nebulized or subcutaneous, may provide some improvement in edema.

In addition to airway compromise, complications of epiglottitis include epiglottic abscess, meningitis, pulmonary edema, pneumonia, and empyema (associated with H influenzae).

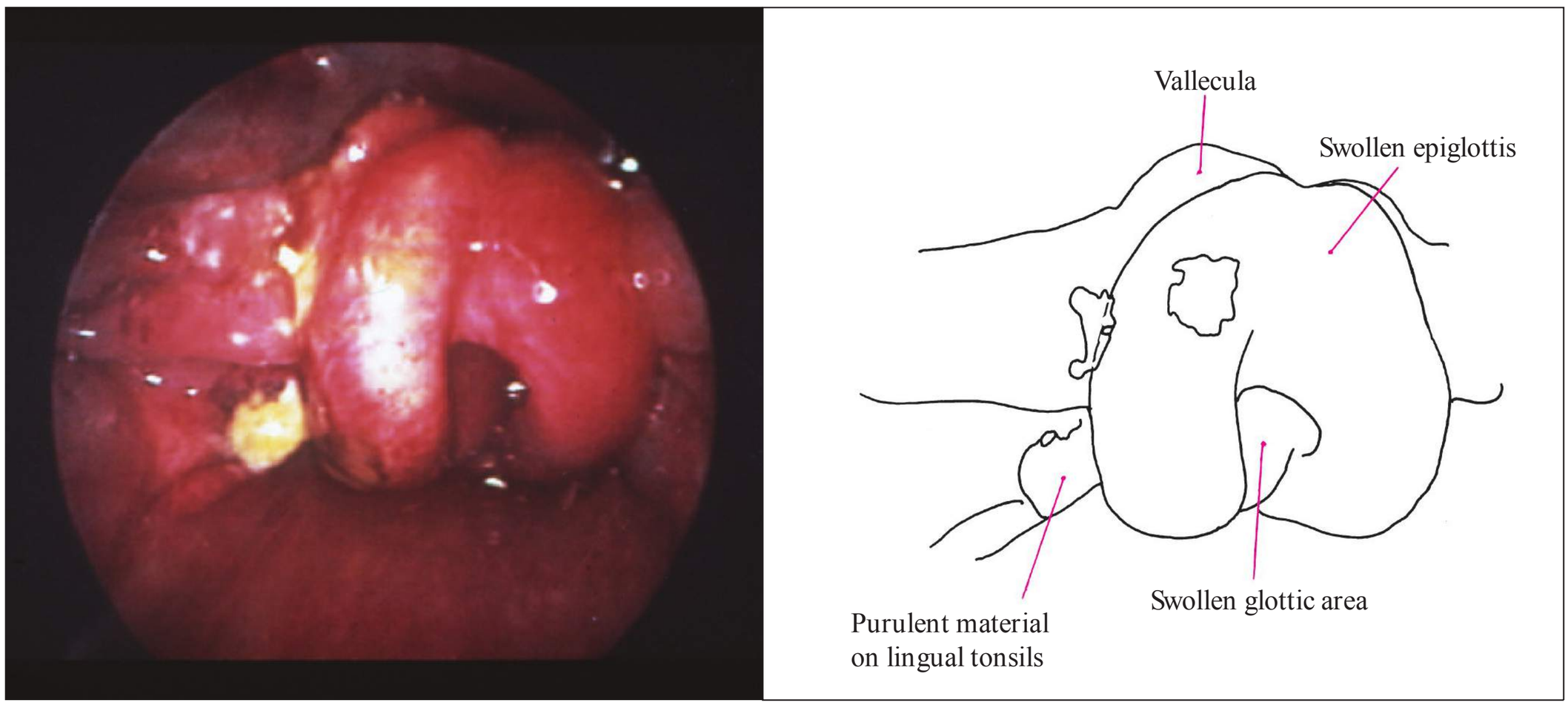

FIGURE 5.56 ■ **Adult Epiglottitis.** Fiberoptic laryngoscopy showing an edematous epiglottis and glottic area with marked airway compromise in an adult with epiglottitis. (Photo contributor: Timothy L. Smith, MD.)

Pearls

1. Transport of patients with suspected epiglottitis must be done by an experienced transport team. The airway must be secured before transport of all but the most stable patients.
2. During intubation, pushing on the patient's chest may cause a bubble to form at the airway orifice, guiding placement of the tube.
3. In areas with a significant prevalence of infection with community-associated methicillin-resistant S aureus (MRSA), clindamycin should be considered as part of the empiric choice for gram-positive coverage.
4. Failure to intervene prior to loss of the airway carries a six-fold increase in mortality.

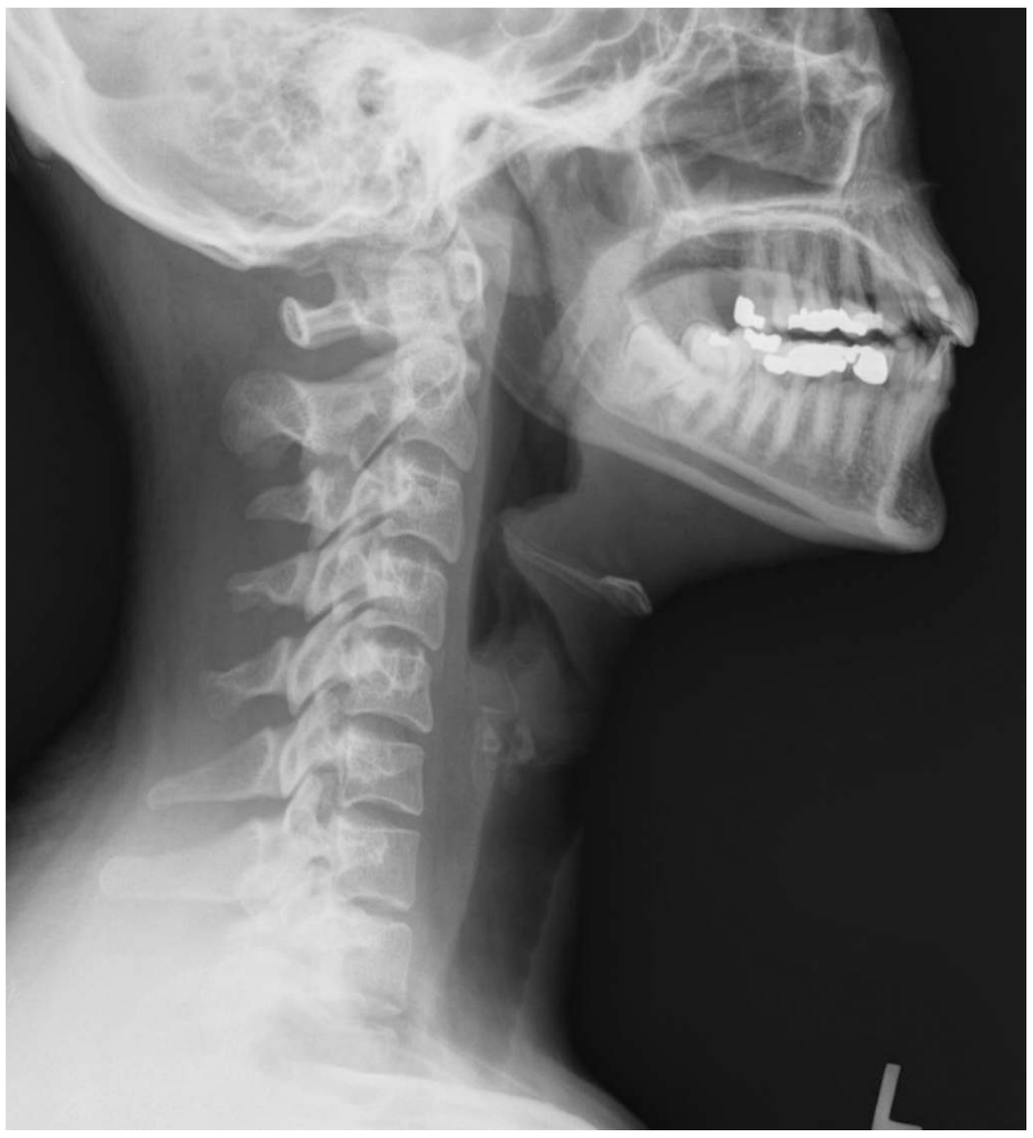

FIGURE 5.57 ■ **Adult Epiglottitis.** Soft-tissue lateral neck radiograph of an adult with epiglottitis demonstrating the classic "thumb" sign of a swollen epiglottis. (Photo contributor: Kevin J. Knoop, MD, MS.)

Clinical Summary

Except for idiopathic, the two most common causes of uvular enlargement are infections and angioedema. Most patients complain of a sore throat, a gagging sensation, or a foreign-body sensation in the back of the mouth.

The infectious etiologies of uvulitis are bacterial, including H influenzae and streptococci; fungal, such as C albicans; and viral. Infections are typically extensions from adjacent infections, such as epiglottitis, tonsillitis, PTAs, and pharyngitis. Patients note fever, odynophagia, trismus, facial pain, hoarseness, neck pain, and headache. On examination, the uvula is red, firm, swollen, and very tender to palpation.

Angioedema of the uvula, known as the Quincke disease, can be hereditary, acquired, or idiopathic. Medications, allergens, thermal stimuli, pressure, and iatrogenic or accidental trauma can initiate angioedema. In addition to the swollen uvula, patients may note pruritus, urticaria, and wheezing. With uvular edema, the angioedema may also involve the face, tongue, and oropharynx. Airway compromise is more common in angioedema of the uvula which appears pale, boggy, and edematous, resembling a large white grape (uvular hydrops).

Management and Disposition

Most cases of uvulitis are benign and self-limited. Angioedematous uvulitis is treated with steroids, antihistamines, and epinephrine in severe cases, either subcutaneously or nebulized. For infectious uvulitis, antibiotic coverage is dictated by the primary source of infection. Admission is based on severity of airway compromise and accompanying infections.

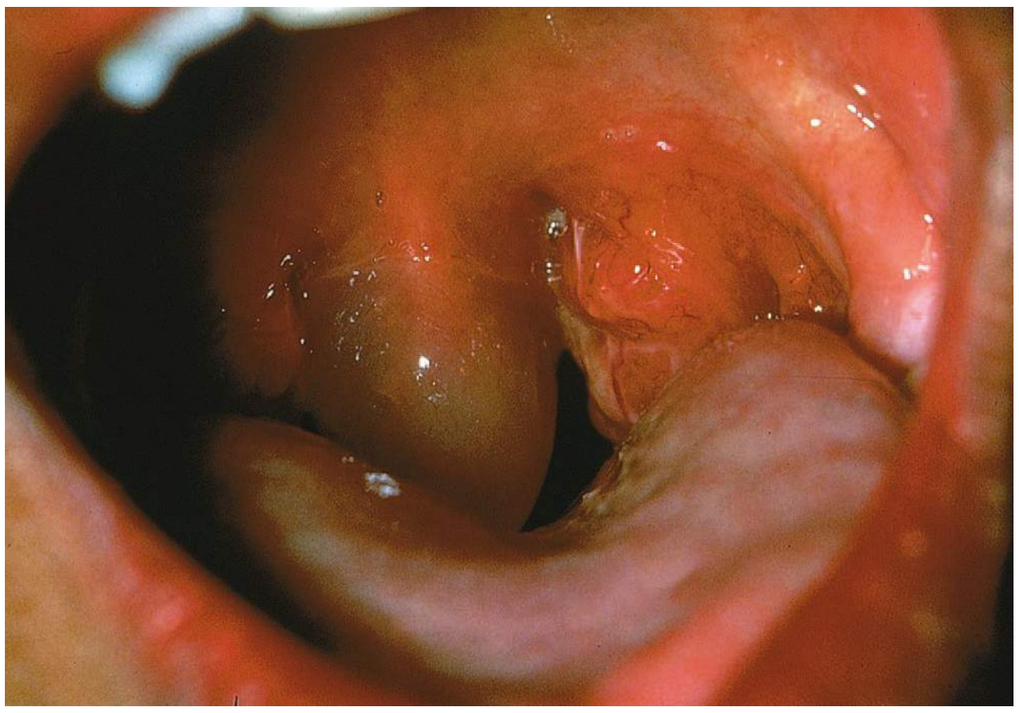

FIGURE 5.59 ■ **Uvulitis.** Angioedema of the uvula, known as the Quincke disease. (Photo contributor: Robin T. Cotton, MD.)

Pearls

1. Any airway symptom prompts an evaluation of the hypopharynx, by either radiographic imaging, or fiber-optic nasopharyngoscope, or direct laryngoscopy.

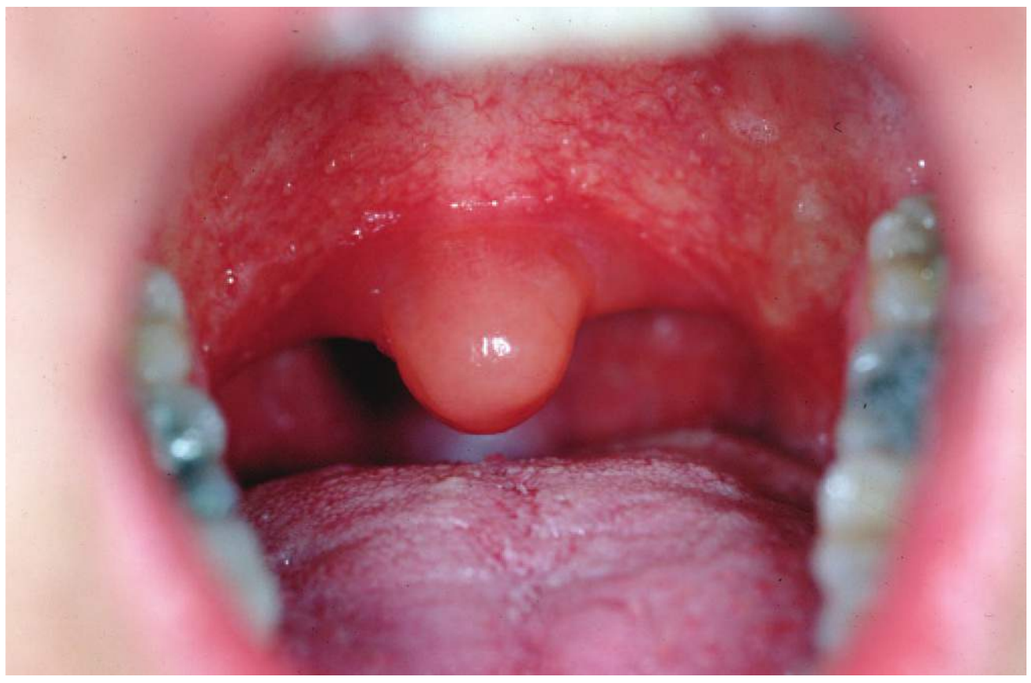

FIGURE 5.58 ■ **Uvulitis.** Isolated edema of the uvula in a patient who presents with a foreign-body sensation of the throat. (Photo contributor: Lawrence B. Stack, MD.)

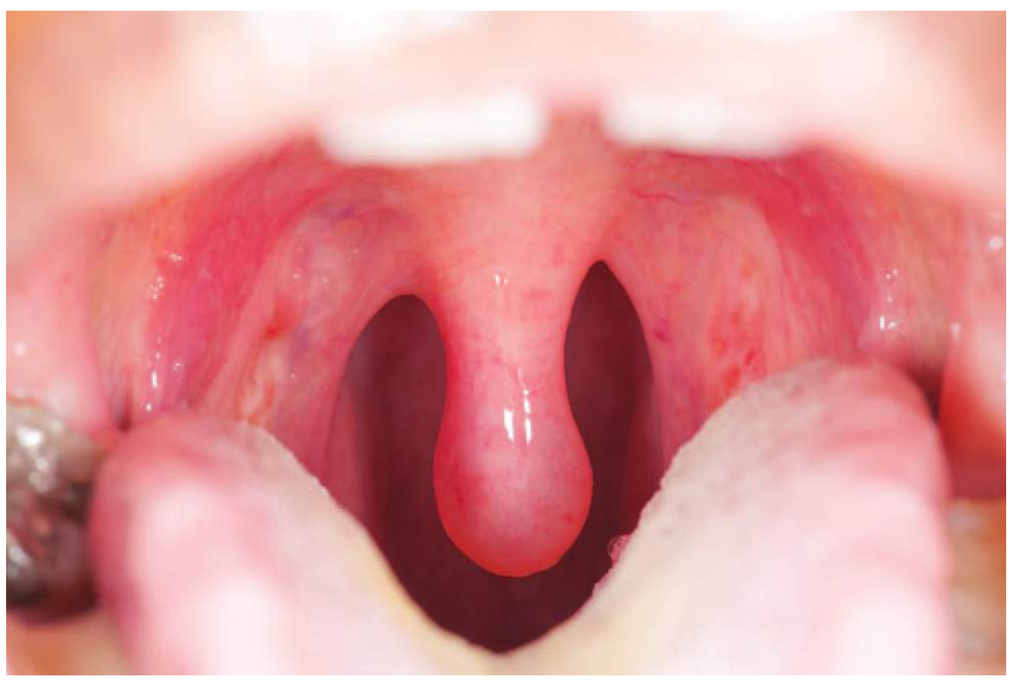

FIGURE 5.60 ■ **Uvulitis.** Isolated edema of the uvula showing a globular structure that presents with foreign-body sensation. (Photo contributor: Lawrence B. Stack, MD.)

MUCOCELE (RANULA)

Clinical Summary

Ranulas are mucoceles (mucous retention cysts) that develop in the floor of the mouth, arising from obstructed sublingual or submandibular ducts or smaller minor salivary glands. Initially, the cysts are small and barely noticeable, but over time they can expand outward or deeper into the neck (plunging ranula). Large cysts can displace the tongue forward and upward, making the patient uncomfortable. Unlike those with sialolithiasis, patients with ranulas may not always notice an increase in swelling associated with eating. Physical examination reveals a soft, minimally tender, translucent cyst with dilated veins running over its surface. Unlike carcinomas, no ulceration is noted with ranulas, and they are generally softer.

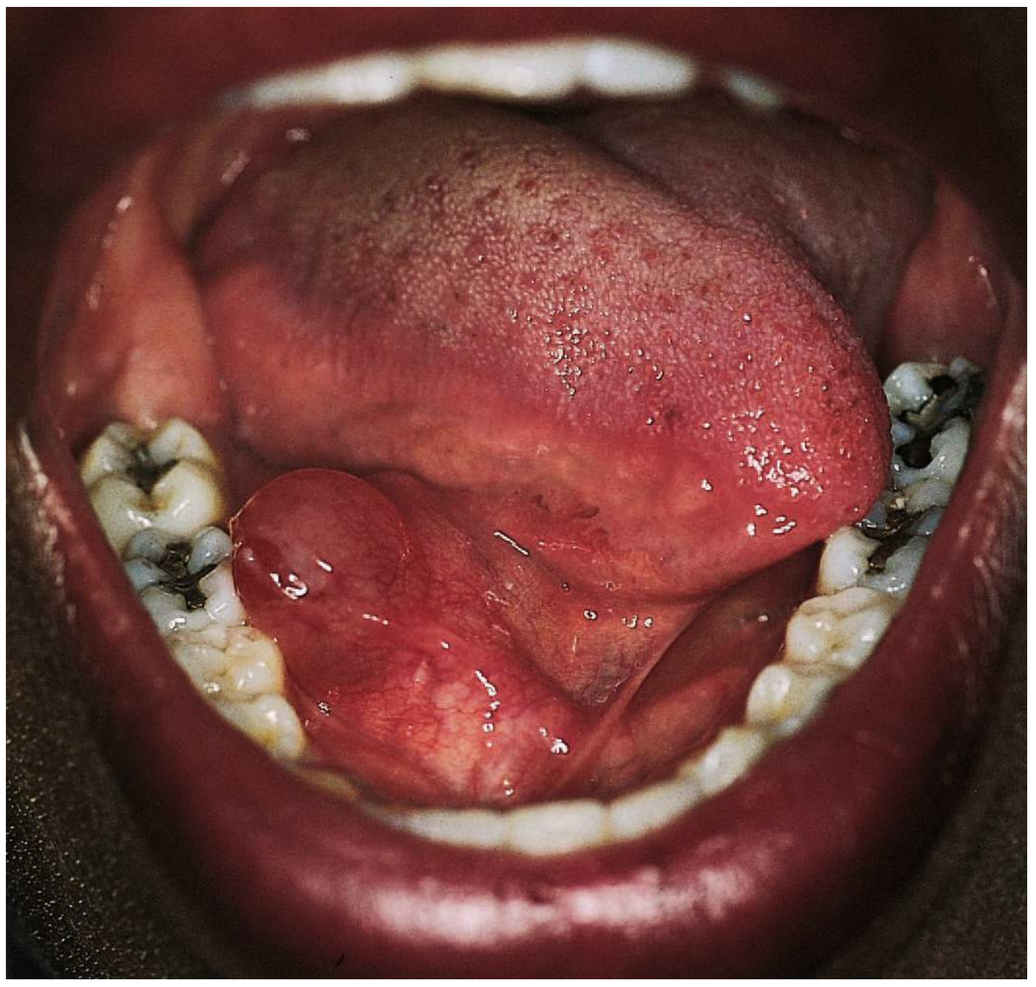

FIGURE 5.61 ■ **Ranula.** Sublingual ranula, or mucocele, lateral to Wharton duct. The patient was asymptomatic except for being aware of the lesion. (Photo contributor: Kevin J. Knoop, MD, MS.)

Management and Disposition

Recognition by the physician is essential for proper referral. Definitive treatment is excision or marsupialization, although needle aspiration of the cyst can provide temporary relief. Unless there is a secondary infection, no antibiotic coverage is required.

Pearls

1. Most ranulas are painless and are incidental findings on routine examinations.
2. Ranulas often recur, requiring total excision of the offending salivary gland.

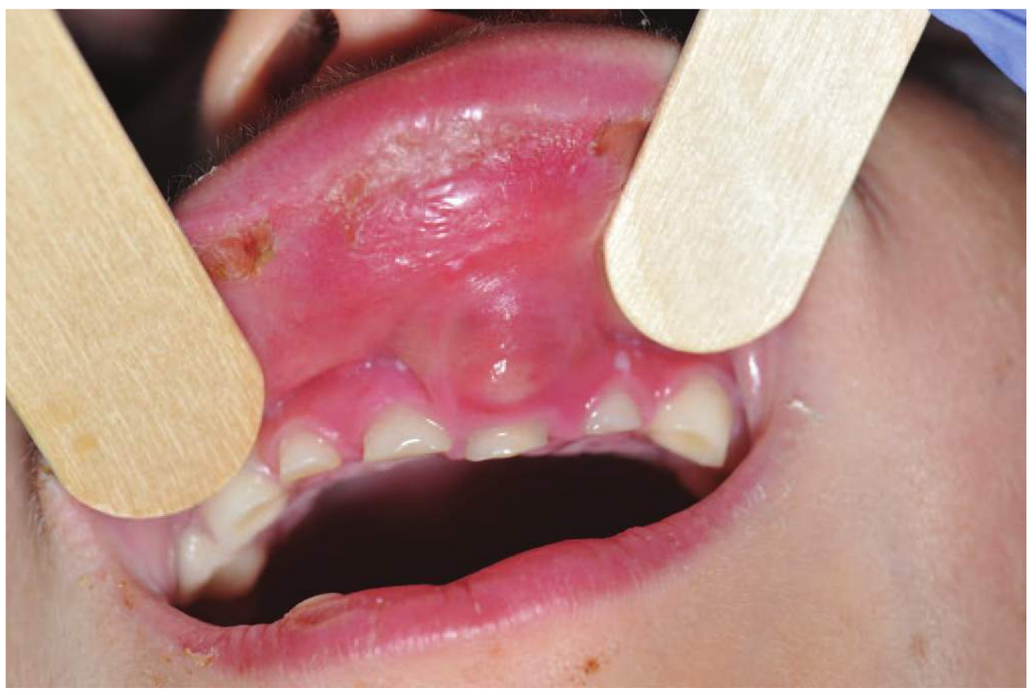

FIGURE 5.62 ■ **Mucocele.** Focal swelling of the upper lip that developed months after local trauma consistent with a mucocele. (Photo contributor: Lawrence B. Stack, MD.)

Clinical Summary

Sialoadenitis is a general term describing inflammation of any salivary gland. The three major salivary gland pairs are the parotid, submandibular, and sublingual. There are also numerous smaller salivary glands that empty into the oral cavity and all are capable of becoming inflamed. Salivary gland disorders have a broad spectrum of causes, including acute and chronic infections; metabolic, systemic, and endocrine disorders; infiltrative processes; obstructions; allergic inflammation; and neoplastic diseases. Key features in the history are the duration and course of the symptoms, complaints of pain, and unilateral or bilateral location.

Both viral and bacterial infections of the salivary gland can lead to enlarged, swollen, painful masses. Suppurative sialoadenitis is most commonly caused by S aureus and is found in patients who are elderly, diabetic, or have poor oral hygiene. It may also follow episodes of dehydration, such as those due to surgery or debilitation. Viral sialoadenitis, such as mumps parotitis, is the most common cause. It occurs with a concomitant viral illness and is usually bilateral, whereas bacterial infections are primarily unilateral.

Obstructive sialoadenitis occurs from a stone or calculus in the salivary gland or duct, most commonly in the submandibular gland. The flow of saliva is obstructed, causing swelling, pain, and firmness. Patients with sialolithiasis note general xerostomia and recurrent worsening of swelling and pain during eating.

A thorough head and neck examination is essential, especially a bimanual examination of the major salivary glands. In suppurative sialoadenitis, purulent drainage may be expressed from the submandibular duct (Wharton) or parotid duct (Stensen) and the glands are very tender and painful to examination. Sialolithiasis can manifest as enlargement of the ducts with minimal saliva expressed on stripping and, rarely, a palpable or visible stone or duct thickening. Facial radiographs are of limited utility. Ultrasound or CT may be useful to detect abscesses.

Management and Disposition

Treatment of suppurative sialoadenitis requires antibiotics with coverage of Staphylococcus and oral flora, rehydration, proper oral hygiene, sialogogues, local heat, and occasionally surgical irrigation and drainage of abscesses. Obstructive sialoadenitis is rarely an emergency. Most salivary stones pass spontaneously without complication, and patients can be discharged home on lozenges to stimulate salivary secretions. Prompt follow-up of sialoadenitis is essential to prevent possible morbidity and mortality associated with infections or neoplasms.

Pearls

1. Examine secretions of both mouth and eyes, and elicit any history of dry eyes, keratoconjunctivitis, cutaneous lesions, or rheumatoid arthritis to establish the diagnosis of a systemic disorder.
2. Medications such as antihistamines, psychotropic drugs, and those possessing atropine-like side effects can cause xerostomia, exacerbating or predisposing to sialoadenitis.
3. Lack of improvement on antibiotics suggests an abscess or multiple loculated abscesses that require drainage.

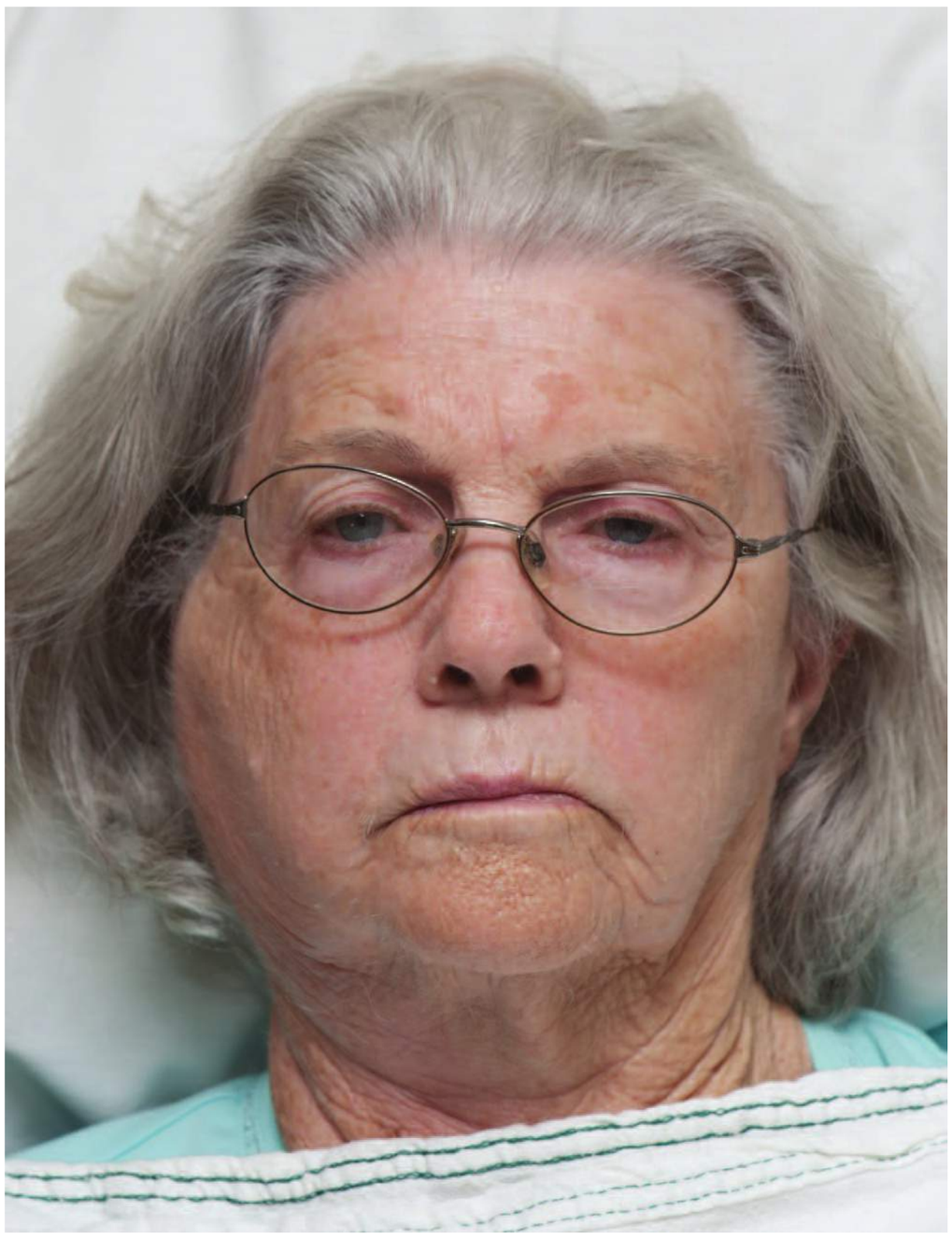

FIGURE 5.63 ■ **Suppurative Parotid Sialoadenitis**. Fever, swelling, and tenderness over the parotid gland along with purulent discharge expressed from Stensen duct suggest suppurative parotid sialoadenitis. (Photo contributor: Lawrence B. Stack, MD.)

FIGURE 5.64 ■ **Suppurative Parotid Sialoadenitis.** Pus from Stensen duct confirms suppurative parotid sialoadenitis. (Photo contributor: Lawrence B. Stack, MD.)

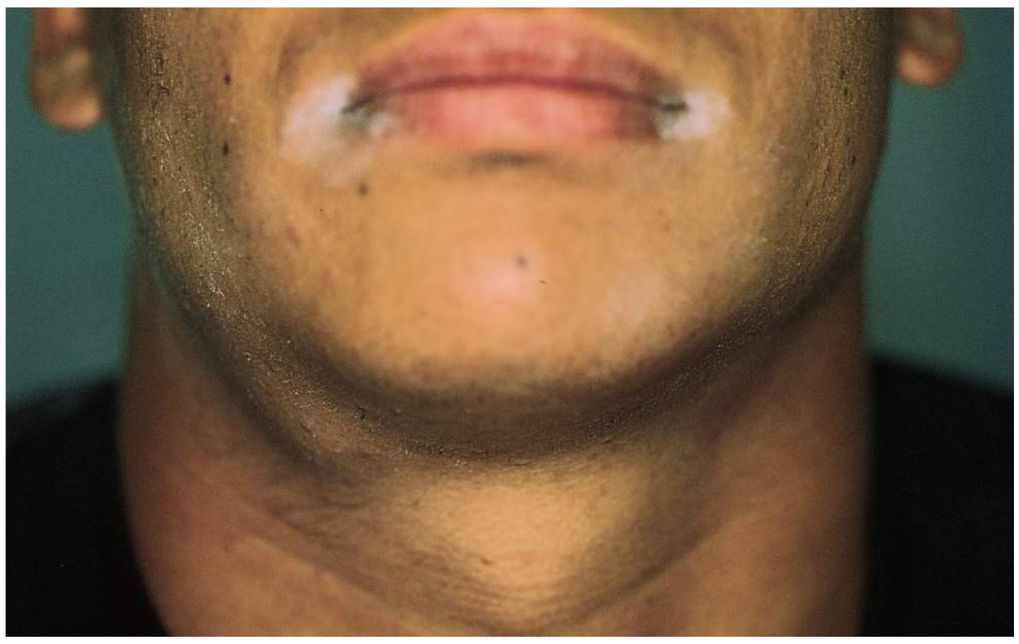

FIGURE 5.65 ■ **Suppurative Submandibular Sialoadenitis**. Unilateral submandibular swelling. (Photo contributor: Jeffery D. Bondesson, MD.)

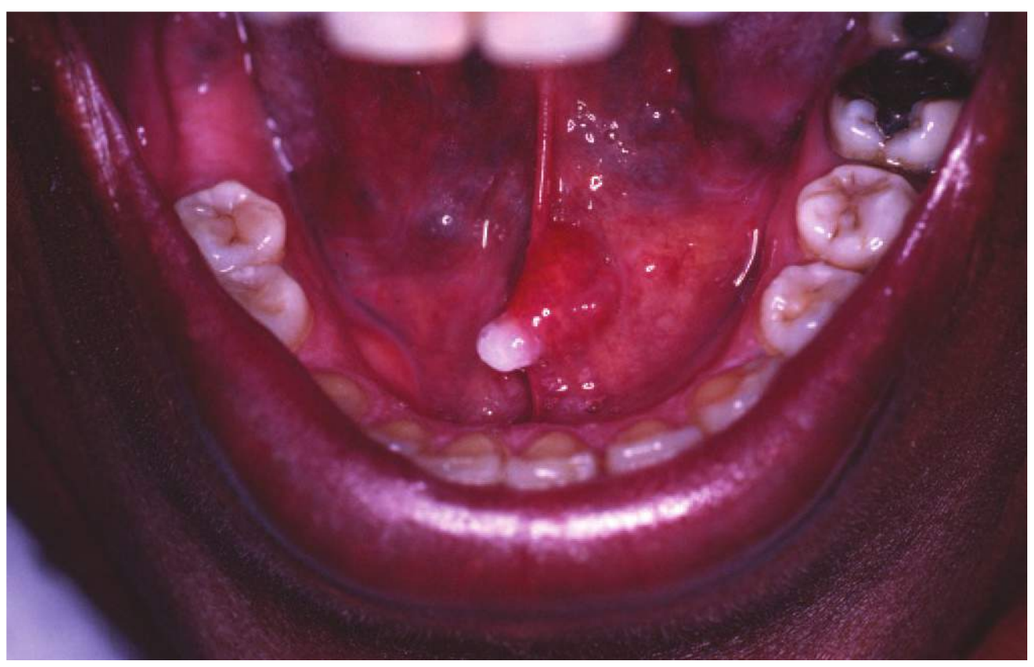

FIGURE 5.66 ■ **Sialolithiasis.** A stone is seen at the orifice of Wharton duct. Small ranula are also seen at the tongue base. (Photo contributor: David Effron, MD.)

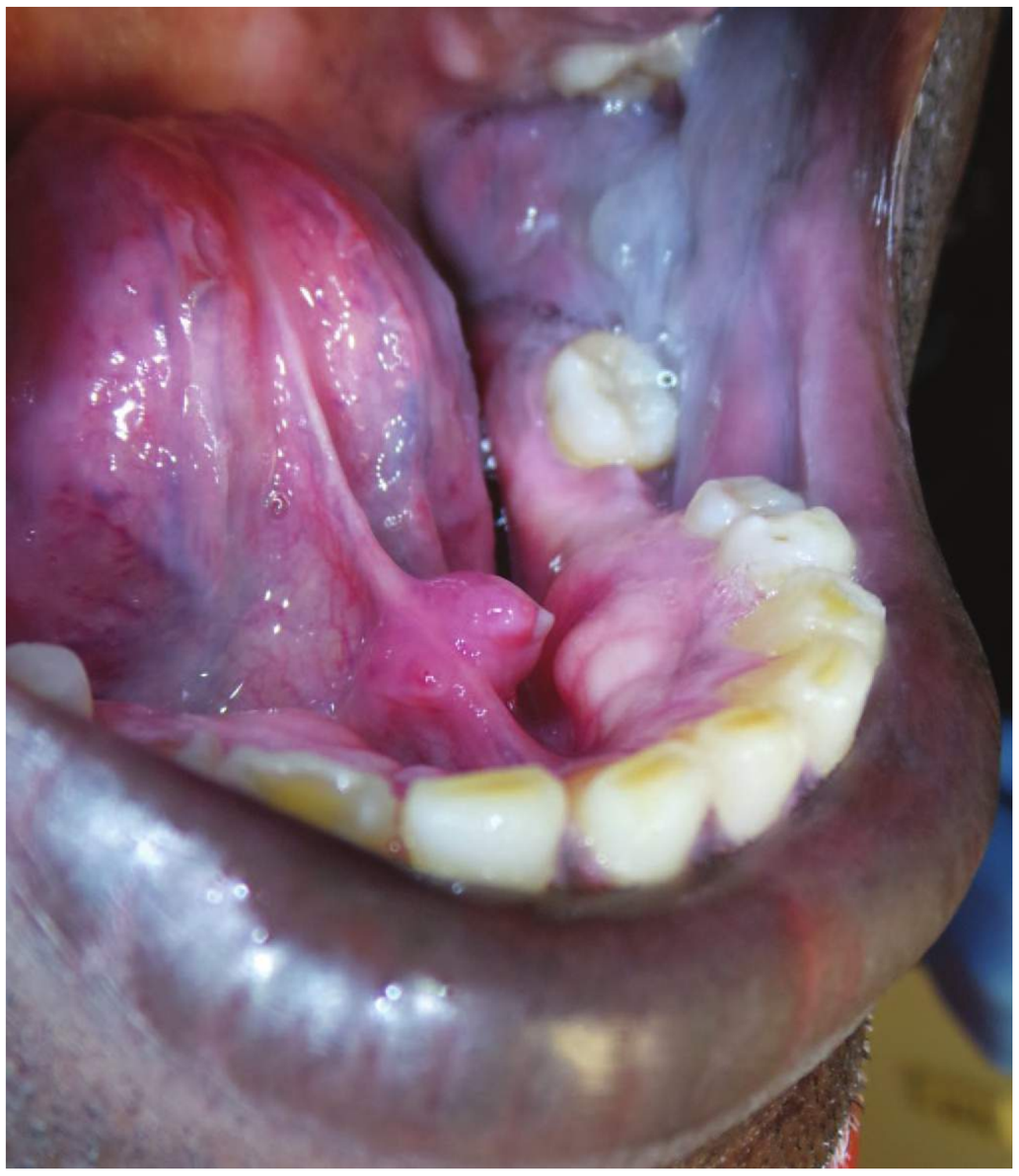

FIGURE 5.67 ■ **Sialolithiasis.** A stone is seen at the orifice of Wharton duct. (Photo contributor: Larry B. Mellick, MD

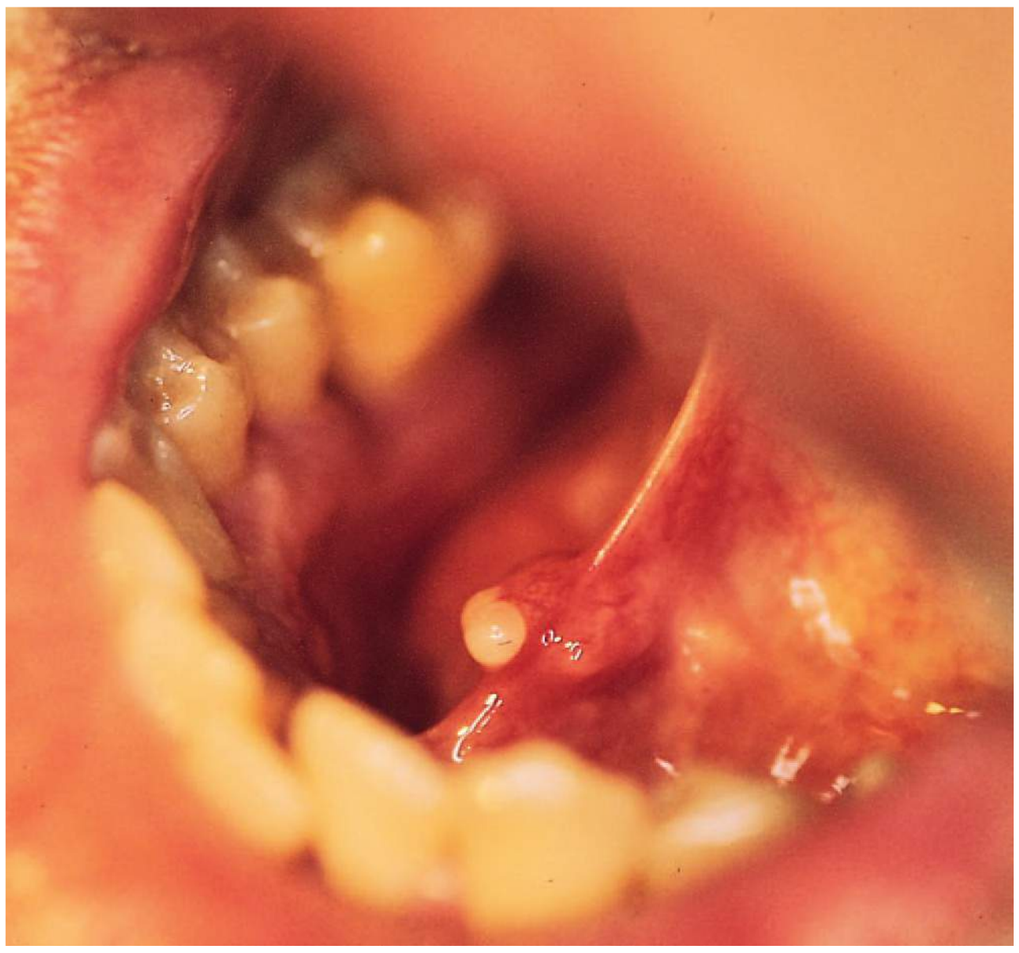

FIGURE 5.68 ■ **Suppurative Submandibular Sialoadenitis.** After applying firm pressure, purulent discharge is seen coming from Wharton duct. (Photo contributor: Jeffery D. Bondesson, MD.)

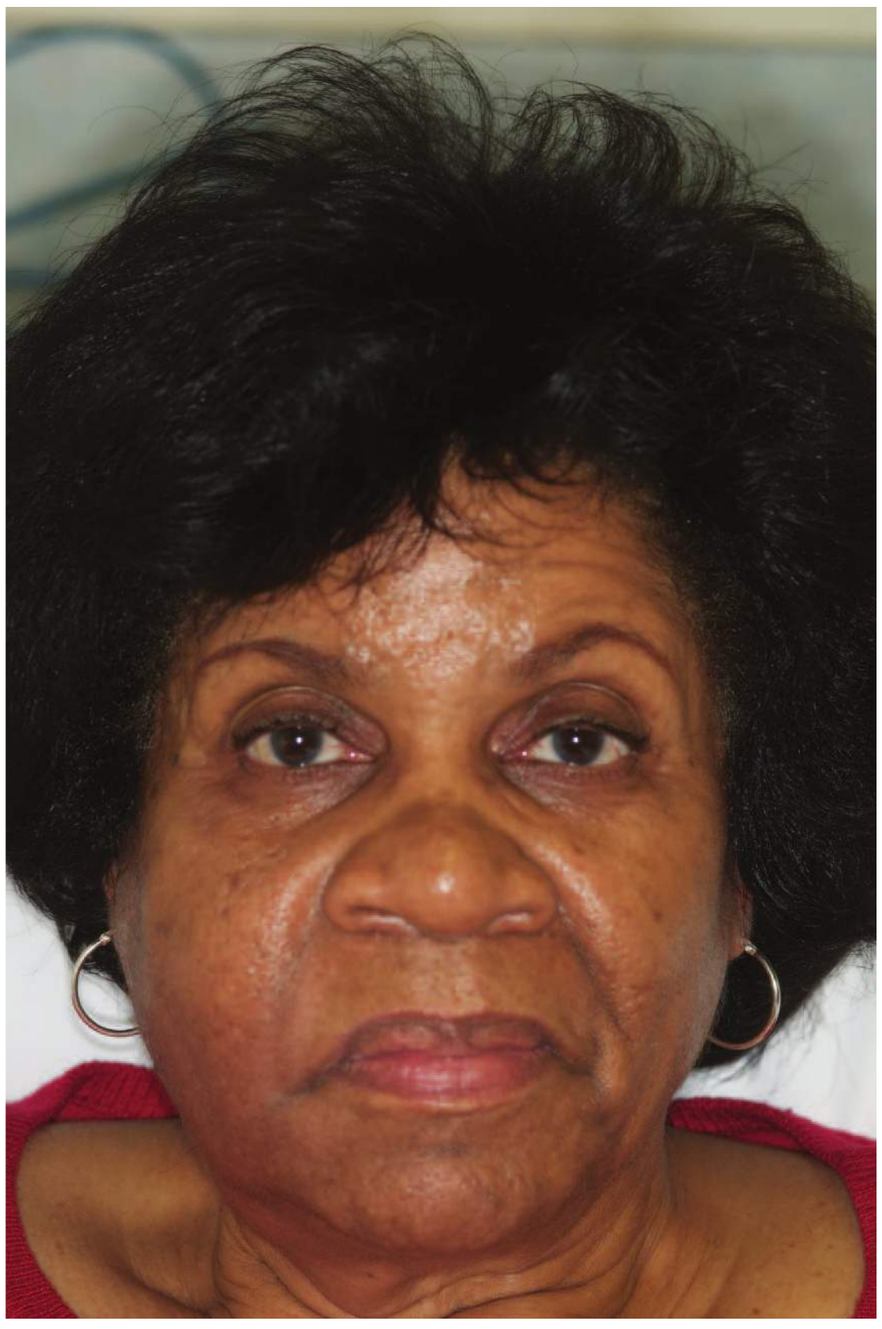

FIGURE 5.69 ■ **Sialolithiasis.** Rapid swelling and pain of the right parotid gland suggest an obstructed parotid duct. (Photo contributor: Lawrence B. Stack, MD.)

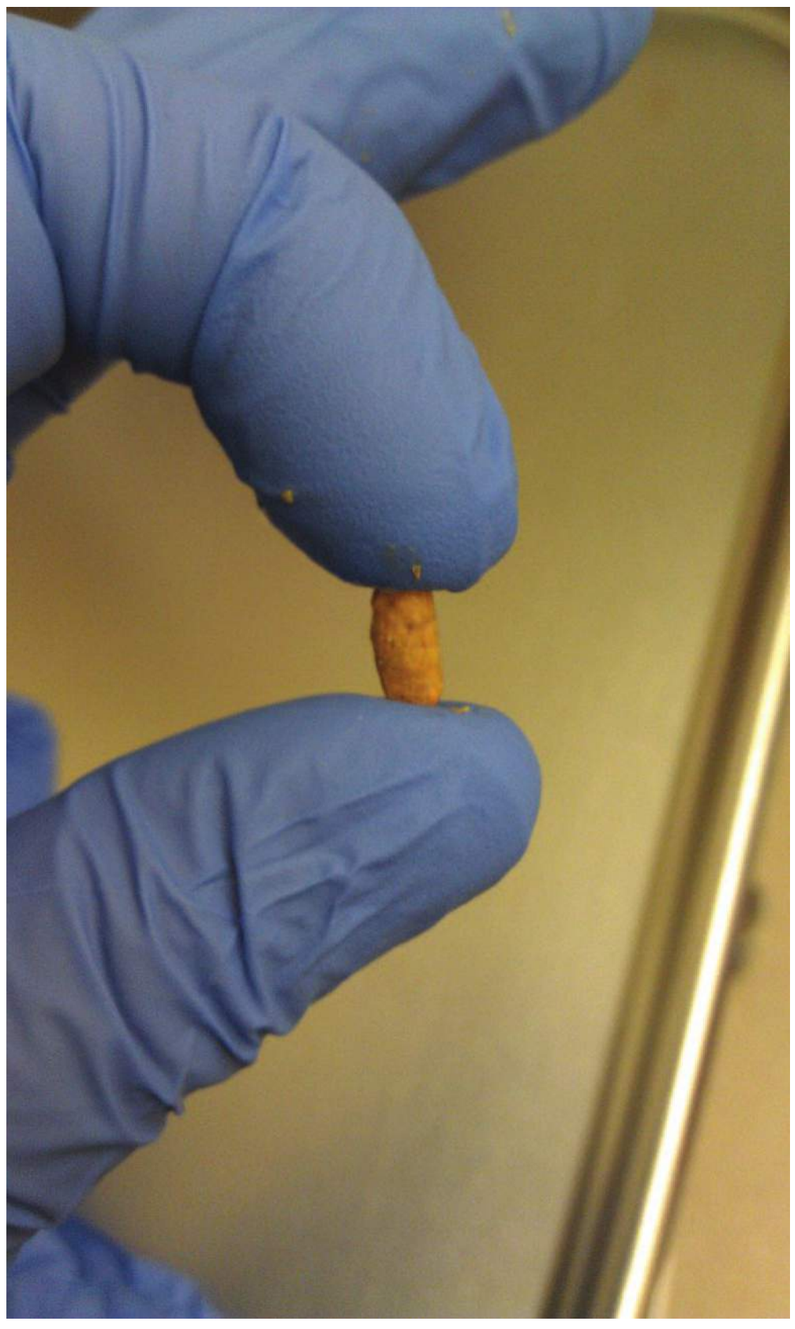

FIGURE 5.70 ■ **Sialolithiasis.** This stone was subsequently delivered through Stensen duct. (Photo contributor: Lawrence B. Stack, MD.)

SINUSITIS

Clinical Summary

Sinusitis results from an impairment of mucociliary clearance and subsequent inflammation of the paranasal sinuses. Most cases of bacterial sinusitis are associated with antecedent viral upper respiratory tract infection.

Maxillary sinusitis (most common) is associated with paranasal facial pain, maxillary dental pain, purulent rhinorrhea, retroocular pain, and conjunctivitis. Ethmoid sinusitis, more common in children, produces a low-grade fever and periorbital pain. Frontal sinusitis can cause a severe supraorbital headache, which is exacerbated by leaning forward; a low-grade fever; upper lid edema; and rhinorrhea. Sphenoid sinusitis is rare, and patients typically complain of a vertex headache and retroocular pain. It can involve CNs, the pituitary gland, and the cavernous sinus. Involvement of all sinus cavities is referred to as pansinusitis. Important complications of sinusitis include periorbital and orbital cellulitis, cavernous sinus thrombosis, and intracranial abscess.

Pott's puffy tumor (a rare osteomyelitis of the cranium from direct extension of a frontal sinusitis) presents as a boggy, tender swelling overlying the frontal sinus.

H influenzae and S pneumoniae together represent 60% to 70% of all bacterial causes. Others include S pyogenes, S aureus, and M catarrhalis. Immunocompromised patients are susceptible to fungal infections, including Aspergillus and Mucor species.

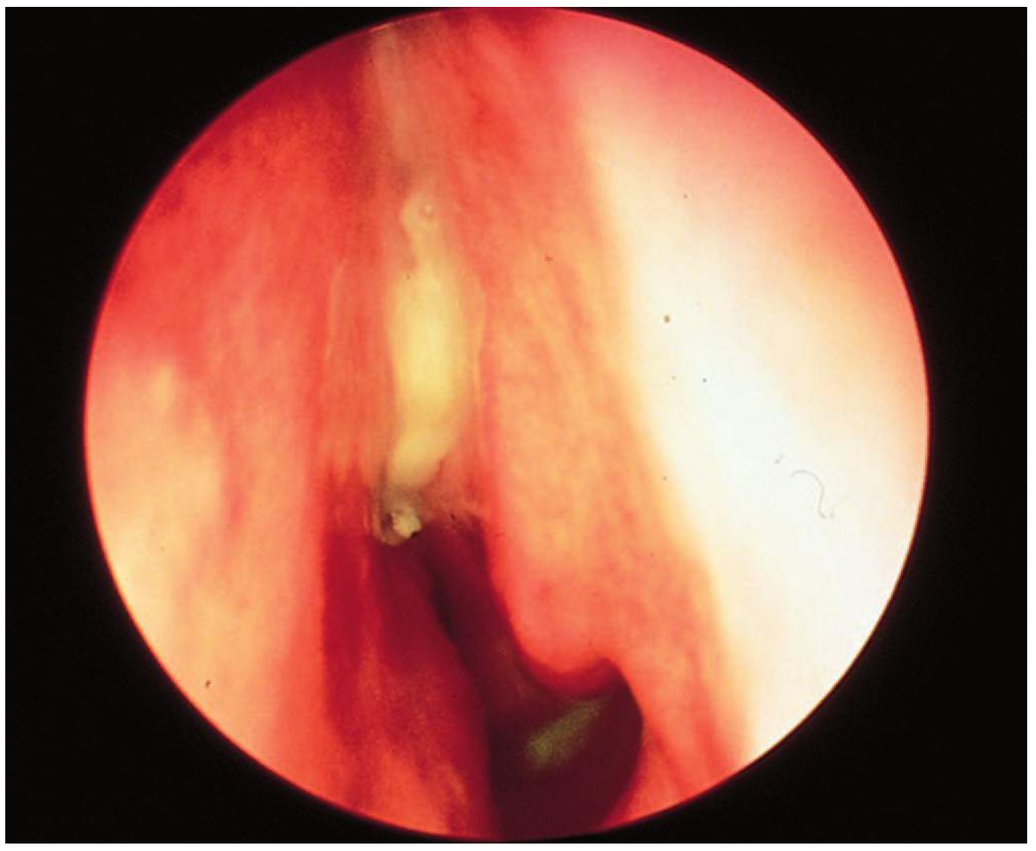

FIGURE 5.71 ■ **Sinusitis.** Purulent drainage from the maxillary sinus ostium in a patient with maxillary sinusitis. Drainage may not always be apparent, since the ostium may be occluded from swelling and inflammation. (Photo contributor: Robin T. Cotton, MD.)

Management and Disposition

Treat most patients with decongestants and analgesics. Decongestants reduce local edema, increase air movement within the sinuses, and decrease local secretions. Humidified air, steam, or saline nasal sprays also facilitate drainage. Patients should be strongly encouraged to stop smoking. If symptoms persist beyond 7 days, consider antibiotics.

Parenteral steroids are not used in acute or recurrent sinusitis. Intranasal steroids may have a role in allergic and chronic sinusitis. CT is the most sensitive and specific imaging modality and allows for better delineation of the sphenoid and ethmoid sinuses.

Otolaryngologist or primary care referral or follow-up should be made for all patients within 3 weeks for routine cases. Patients with comorbid illnesses or more complicated sinusitis should be admitted for parenteral antibiotic therapy and supportive care.

Pearls

1. Chronic sinusitis may be due to mucoid retention cysts, deviated septum, or polyps, which are often visible on plain radiographs. Refer these patients for possible surgery.
2. Physicians must consider fungal etiologies in immunocompromised patients or those with comorbid illnesses.

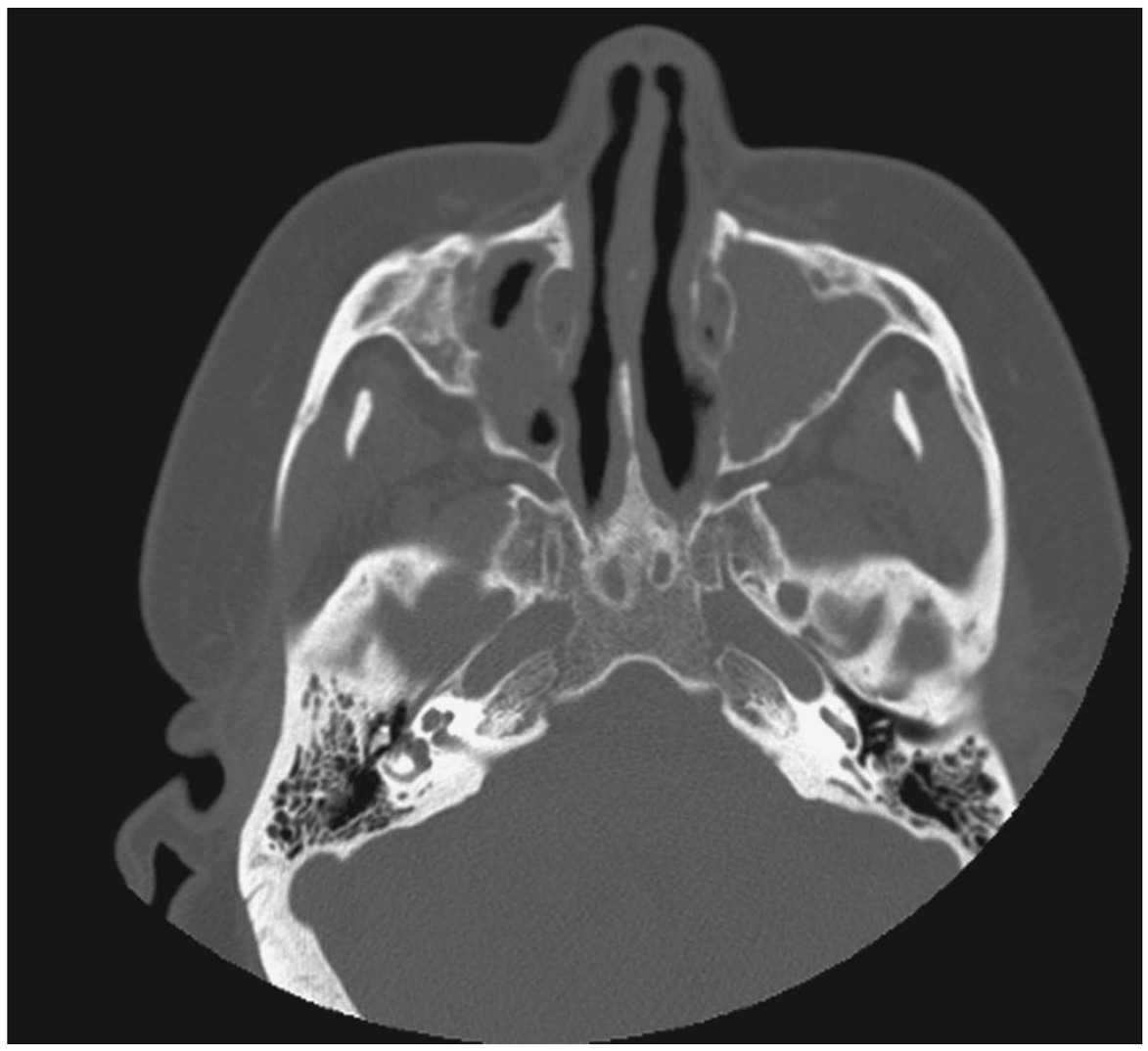

FIGURE 5.72 ■ **Sinusitis.** Bone windows of a sinus CT demonstrating bilateral maxillary sinus disease. (Photo contributor: Lawrence B. Stack, MD.)

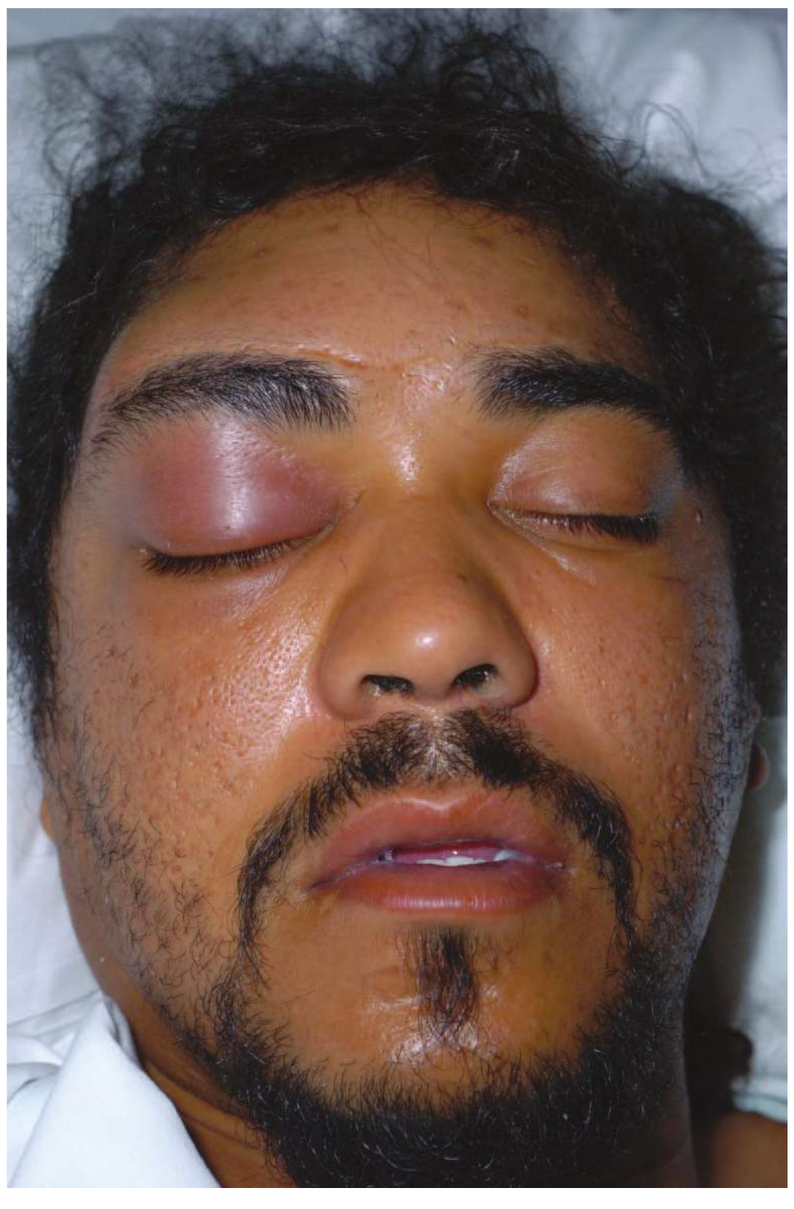

FIGURE 5.73 ■ **Sinusitis—Pott's Puffy Tumor.** Forehead and right periorbital edema in a patient with pansinusitis suggests osteomyelitis of the anterior table of the frontal sinus. (Photo contributor: Lawrence B. Stack, MD.)

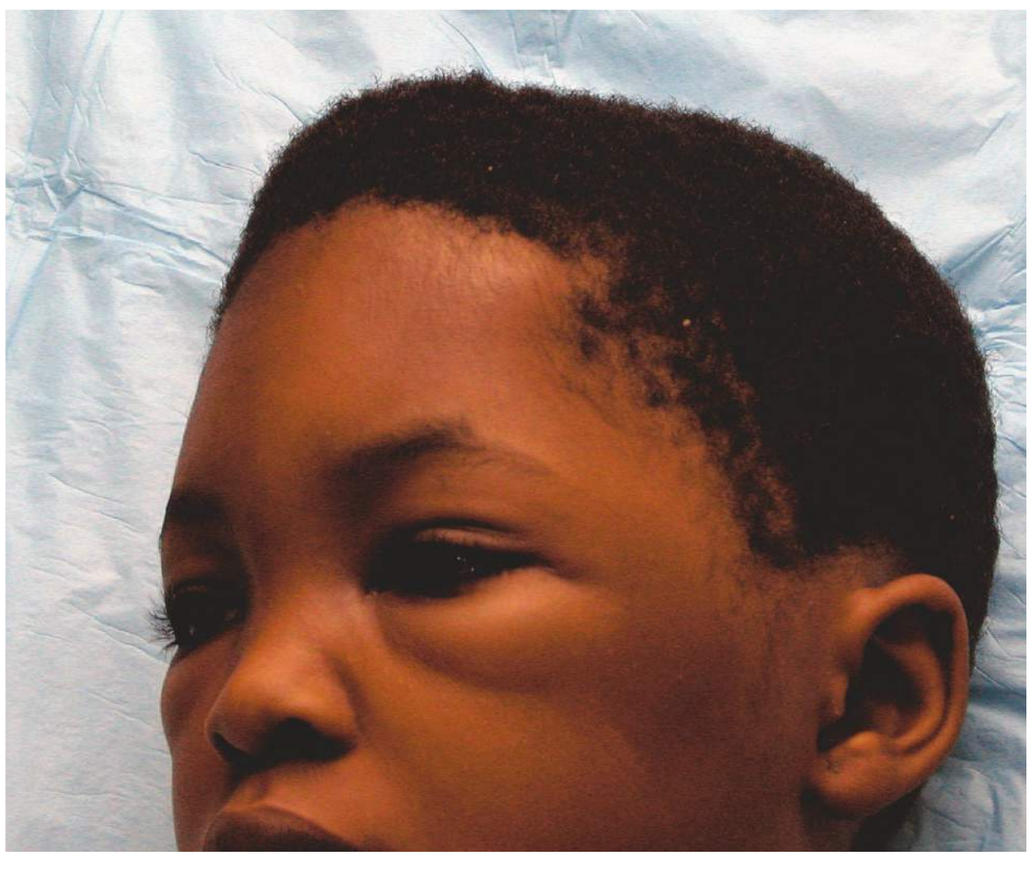

FIGURE 5.74 ■ **Sinusitis—Pott's Puffy Tumor.** Forehead and periorbital edema in a child with pansinusitis suggests osteomyelitis of the anterior table of the frontal sinus. (Photo contributor: David Effron, MD.)

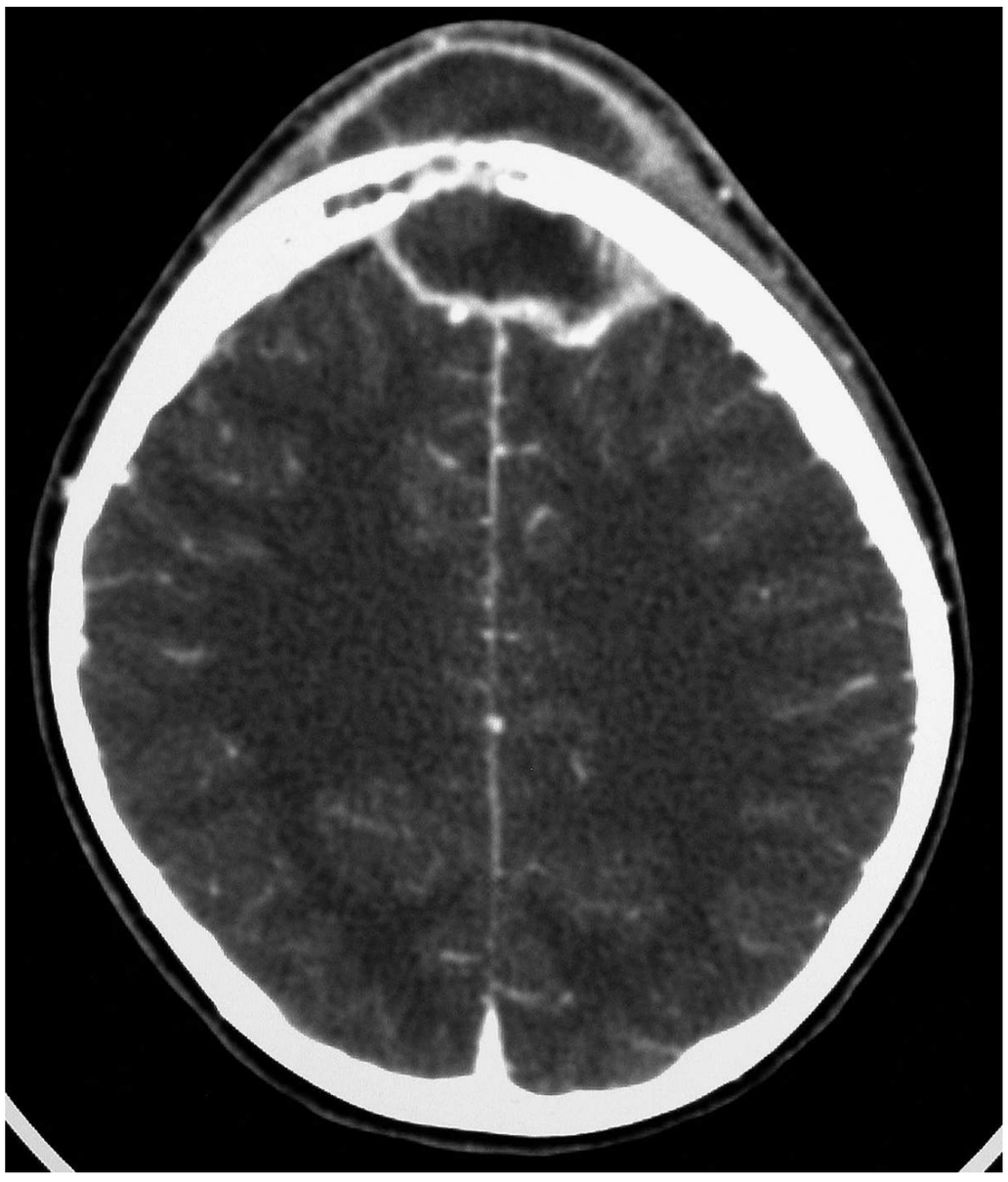

FIGURE 5.75 ■ **Sinusitis—Pott's Puffy Tumor.** CT (parenchymal view) demonstrating intracranial abscess formation and bony destruction of frontal bone from sinusitis. (Photo contributor: David Effron, MD.)

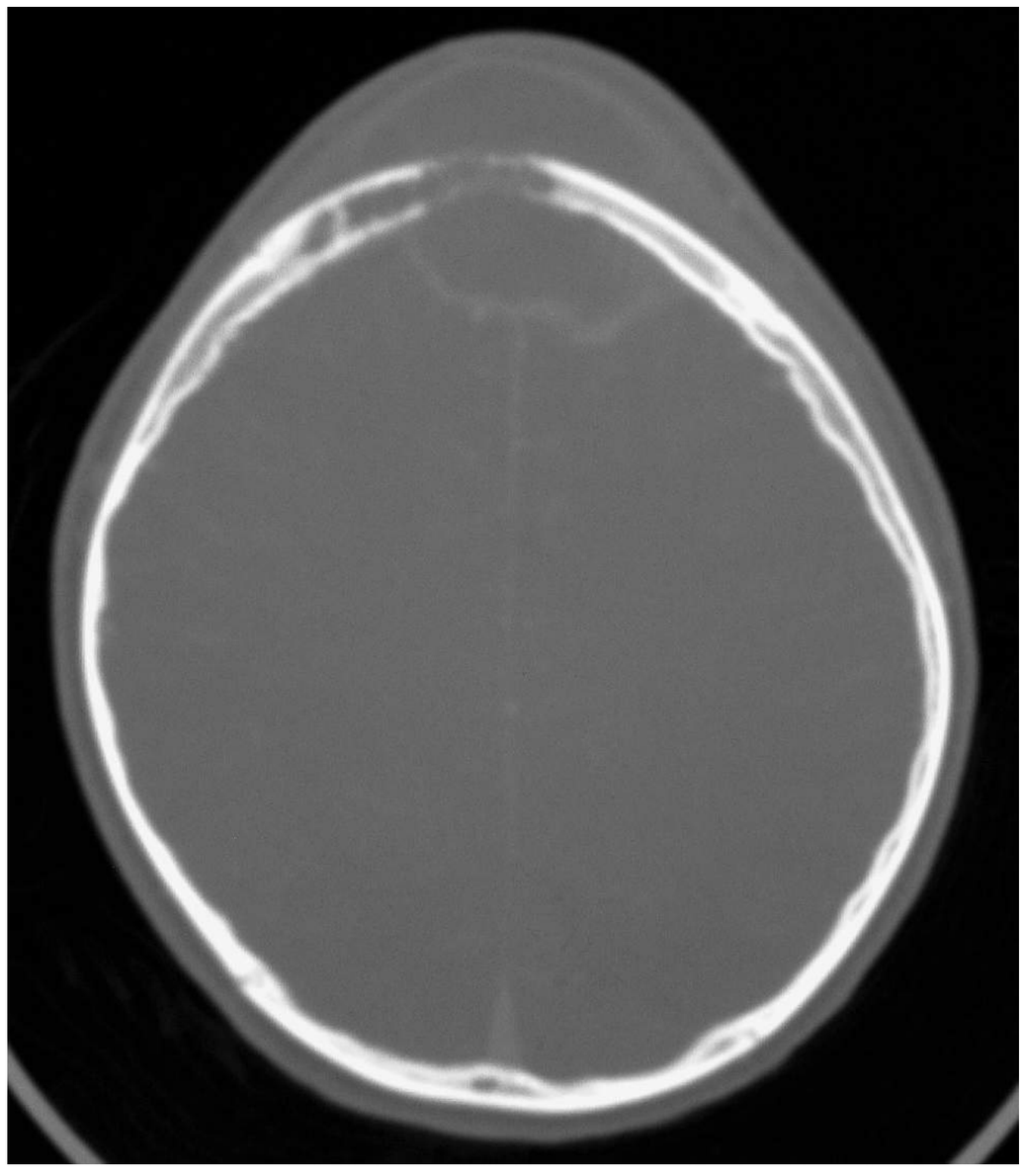

FIGURE 5.76 ■ **Sinusitis—Pott's Puffy Tumor.** CT (bone window) highlighting bony destruction of inner and outer table of frontal bone from sinusitis. (Photo contributor: David Effron, MD.)