is pruritus rather than pain; this condition must be differentiated from several others that produce a similar clinical picture, such as atopic dermatitis, seborrheic dermatitis, psoriasis, and dermatomycosis. Therapy consists of identifying and treating or removing the offending process, although successful resolution is frequently difficult.

Invasive otitis externa, also known as *malignant* or *necrotizing* otitis externa, is an aggressive and potentially life-threatening disease that occurs predominantly in elderly diabetic patients and other immunocompromised persons. The disease begins in the external canal as a soft tissue infection that progresses slowly over weeks to months and often is difficult to distinguish from a severe case of chronic otitis externa because of the presence of purulent otorrhea and an erythematous swollen ear and external canal. Severe, deep-seated otalgia, frequently out of proportion to findings on examination, is often noted and can help differentiate invasive from chronic otitis externa. The characteristic finding on examination is granulation tissue in the posteroinferior wall of the external canal, near the junction of bone and cartilage. If left unchecked, the infection can migrate to the base of the skull (resulting in skull-base osteomyelitis) and onward to the meninges and brain, with a high mortality rate. Cranial nerve involvement is seen occasionally, with the facial nerve usually affected first and most often. Thrombosis of the sigmoid sinus can occur if the infection extends to the area. CT, which can reveal osseous erosion of the temporal bone and skull base, can be used to help determine the extent of disease, as can gallium and technetium-99 scintigraphy studies. *P. aeruginosa* is by far the most common offender, although *S. aureus*, *Staphylococcus epidermidis*, *Aspergillus*, *Actinomyces*, and some gram-negative bacteria also have been associated with this disease. In all cases, the external ear canal should be cleansed and a biopsy specimen of the granulation tissue within the canal (or of deeper tissues) obtained for culture of the offending organism. IV antibiotic therapy should be given for a prolonged course (6–8 weeks) and directed specifically toward the recovered pathogen. For *P. aeruginosa*, the regimen typically includes an antipseudomonal penicillin or cephalosporin (e.g., piperacillin or cefepime), often with an aminoglycoside or a fluoroquinolone, the latter of which can even be administered orally given its excellent bioavailability. In addition, antibiotic drops containing an agent active against *Pseudomonas* (e.g., ciprofloxacin) are usually prescribed and are combined with glucocorticoids to reduce inflammation. Cases of invasive *Pseudomonas* otitis externa recognized in the early stages can sometimes be treated with oral and otic fluoroquinolones alone, albeit with close follow-up. Extensive surgical debridement, once an important component of the treatment approach, is now rarely indicated.

In *necrotizing otitis externa,* recurrence is documented up to 20% of the time. Aggressive glycemic control in diabetics is important not only for effective treatment but also for prevention of recurrence. The role of hyperbaric oxygen has not been clearly established.

INFECTIONS OF MIDDLE-EAR STRUCTURES

Otitis media is an inflammatory condition of the middle ear that results from dysfunction of the eustachian tube in association with a number of illnesses, including URIs and chronic rhinosinusitis. The inflammatory response in these conditions leads to the development of a sterile transudate within the middle ear and mastoid cavities. Infection may occur if bacteria or viruses from the nasopharynx contaminate this fluid, producing an acute (or sometimes chronic) illness.

Acute Otitis Media Acute otitis media results when pathogens from the nasopharynx are introduced into the inflammatory fluid collected in the middle ear (e.g., by nose blowing during a URI). Pathogenic proliferation in this space leads to the development of the typical signs and symptoms of acute middle-ear infection. The diagnosis of acute otitis media requires the demonstration of fluid in the middle ear (with tympanic membrane [TM] immobility) and the accompanying signs or symptoms of local or systemic illness **(Table 44-2)**.

ETIOLOGY Acute otitis media typically follows a viral URI. The causative viruses (most commonly RSV, influenza virus, rhinovirus, and enterovirus) can themselves cause subsequent acute otitis media; more often, they predispose the patient to bacterial otitis media. Studies using tympanocentesis have consistently found *S. pneumoniae* to be the most important bacterial cause, isolated in up to 35% of cases. *H. influenzae* (nontypable strains) and *M. catarrhalis* also are

TABLE 44-2 GUIDELINES FOR THE DIAGNOSIS AND TREATMENT OF ACUTE OTITIS MEDIA

Illness Severity	Diagnostic Criteria	Treatment Recommendations
Mild to moderate	>2 yrs *or* 6 mo to 2 yrs without middle-ear effusion	*Observation alone* (deferring antibiotic therapy for 48–72 h and limiting management to symptom relief)
	<6 mo; *or*	*Initial therapy*[a]
	6 mo to 2 yrs with middle-ear effusion (fluid in the middle ear, evidenced by decreased TM mobility, air/fluid level behind TM, bulging TM, purulent otorrhea) *and* acute onset of signs and symptoms of middle-ear inflammation, including fever, otalgia, decreased hearing, tinnitus, vertigo, erythematous TM; *or*	Amoxicillin, 80–90 mg/kg qd (up to 2 g) PO in divided doses (bid or tid); *or* Cefdinir, 14 mg/kg qd PO in 1 dose or divided doses (bid); *or* Cefuroxime, 30 mg/kg qd PO in divided doses (bid); *or* Azithromycin, 10 mg/kg qd PO on day 1 followed by 5 mg/kg qd PO for 4 d
	>2 yrs with bilateral disease, TM perforation, high fever, immunocompromise, emesis	*Exposure to antibiotics within 30 d or recent treatment failure*[a,b]*:* Amoxicillin, 90 mg/kg qd (up to 2 g) PO in divided doses (bid), plus clavulanate, 6.4 mg/kg qd PO in divided doses (bid); *or* Ceftriaxone, 50 mg/kg IV/IM qd for 3 d; *or* Clindamycin, 30–40 mg/kg qd PO in divided doses (tid)
Severe	As above, with temperature ≥39.0°C (102°F); *or* Moderate to severe otalgia	*Initial therapy*[a] Amoxicillin, 90 mg/kg qd (up to 2 g) PO in divided doses (bid), plus clavulanate, 6.4 mg/kg qd PO in divided doses (bid); *or* Ceftriaxone, 50 mg/kg IV/IM qd for 3 d *Exposure to antibiotics within 30 d or recent treatment failure*[a,b] Ceftriaxone, 50 mg/kg IV/IM qd for 3 d; *or* Clindamycin, 30–40 mg/kg qd PO in divided doses (tid); *or* Consider tympanocentesis with culture

[a]Duration (unless otherwise specified): 10 days for patients <6 years old and patients with severe disease; 5–7 days (with consideration of observation only in previously healthy individuals with mild disease) for patients ≥6 years old. [b]Failure to improve and/or clinical worsening after 48–72 h of observation or treatment.

Abbreviation: TM, tympanic membrane.

Source: American Academy of Pediatrics Subcommittee on Management of Acute Otitis Media, 2004.