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Infections of the External Ear

Anatomy and Physiology

The external ear is an area commonly subjected to acute and chronic inflammatory conditions. It consists of the auricle and external auditory meatus. The auricle is mostly composed of fibroelastic cartilage to which the skin and a small portion of subcutaneous tissue are closely attached, except in the lobule where there is fat and no cartilage. The external auditory meatus (EAM) is a skin-lined canal approximately 2.5 cm in length and ends medially at the tympanic membrane. The lateral cartilaginous portion comprises approximately 40% of its entire length, while the remaining 60% is osseous, formed primarily by the tympanic ring. The dehiscences in the anterior wall of the cartilaginous portion of the canal are known as the fissures of Santorini. They may allow spread of infection from the canal lumen into the preauricular soft tissues, parotid gland, and temporomandibular joint. Because of the oblique position of the tympanic membrane, the posterosuperior portion of the canal is 6 mm shorter than the anteroinferior portion. The canal is S-shaped, curving slightly superiorly and posteriorly from lateral to medial. The narrowest portion of the canal is at the junction of the cartilaginous and bony portions, termed the isthmus. Medial to the isthmus, the canal courses inferiorly and ends in the inferior tympanic recess.

The EAM is related to contiguous structures on all but its lateral surface. Medially, it is bound by the tympanic membrane, which when intact is a good barrier to the spread of infection. Superiorly, it is separated from the cranial fossa by a thick plate of bone, which usually prevents direct intracranial extension of infection. Posteriorly, the bony canal abuts the mastoid cavity. Several vessels penetrate the canal, which may be involved in the hematogenous extension of infection to the mastoid segment. Posterior to the cartilaginous canal, there is dense connective tissue overlying the mastoid portion of the temporal bone, which may become secondarily infected. Anteriorly, the canal is related to the glenoid fossa of the TMJ and the parotid gland. Inferiorly, the canal is related to the infratemporal fossa.

The external ear is innervated by contributions from the trigeminal, facial, glossopharyngeal, and vagal nerves as well as from the cervical plexus (greater auricular nerve). It receives its arterial blood supply from the superficial temporal and posterior auricular branches of the external carotid artery. The deep auricular branch of the internal maxillary artery serves the more medial canal and lateral TM. Venous drainage is via the superficial temporal and posterior auricular veins. The posterior auricular usually drains into the external jugular vein, but may also drain to the sigmoid sinus through the mastoid emissary vein. The lymphatic drainage of the canal is important with regard to the spread of infection and cancer. Inferiorly, the canal drains into the infra-auricular nodes posterior to the angle of the mandible. The anterosuperior canal empties into the preauricular nodes of the parotid and superior deep cervical nodes.

Posteriorly, the lymphatics drain to postauricular and superior deep cervical nodes. Finally, the lymphatics of the antihelix and concha empty into the nodes along the apex of the mastoid process, whereas those of the superior part of the auricle drain into postauricular nodes.

The entire EAM is lined with squamous epithelium, which is thicker in the cartilaginous portion (0.5 to 1mm) than the osseous portion (0.2mm). In the cartilaginous canal, the skin contains sebaceous and apocrine glands with many hair follicles. Together, the hair follicle, sebaceous gland, and apocrine gland are termed the apopilosebaceous unit. Invagination of the epidermis forms the outer wall of the hair follicle. The space between this outer wall and the hair shaft is termed the follicular canal. The excretory ducts of the sebaceous and apocrine alveoli drain into these follicular canals. In the normal ear, the secretions of these glands, combined with the desquamated keratin layer from the stratum corneum, form a water-repellant, acidic, waxy coat of cerumen that serves as a barrier against infection and injury to the skin. Motion of the ear canal provided by ordinary chewing movements together with the process of epithelial proliferation and lateral migration propel the cerumen outward in a self-cleansing manner.

Compromise of any the protective features of the canal can lead to colonization and invasion by pathogenic organisms. Obstruction of the drainage of the glands into the follicular canal can occur in response to increased temperature and humidity within the canal. Absorption of moisture by the stratum corneum leads to hyperhydration and maceration of tissue within the canal, which gives the patient an uncomfortable sense of fullness and itching. Any response that leads to trauma to the canal skin, such as instrumentation, excessive cleansing or scratching, allows for invasion of exogenous or endogenous organisms through breaks in the skin.

Otitis Externa

Otitis Externa is an infection of the external auditory canal (EAC) that can be divided according to the time course of the infection: acute, subacute, or chronic. Acute otitis externa (AOE) is a bacterial infection of the EAC, commonly referred to as "swimmer's ear" that can further be divided into preinflammatory and acute inflammatory stages. The acute inflammatory stage may be mild, moderate, or severe. The preinflammatory stage begins with itching, edema and a full sensation in the ear. As the infection progresses increased itching and pain ensues with mild erythema and edema on physical exam, however the canal lumen remains patent with cloudy secretions. During the moderate phase, the itching and pain intensify, and although the lumen remains patent, significant edema and debris decrease its size. Secretions are noted to be exudative and more profuse. Finally, in the severe stage of the disease, the pain is usually intolerable and is often intensified by manipulation of the skin and soft tissue around the ear. The lumen of the EAC may be obliterated by edema, debris and purulent otorrhea. The auricle and periauricular soft tissues are often involved, and regional lymph nodes may become palpable. In patients where the disease does not resolve after treatment, a subacute or chronic form may occur. This condition can be described as a spectrum of disease ranging from mild drying and scaling of the canal skin to complete obliteration of the lumen by the chronically infected and hypertrophic skin.

The most commonly isolated pathogens are *Pseudomonas aeruginosa* and *Staphylococcus aureus*. Other pathogens less commonly cultured include *Proteus mirabilis*, *Streptococci* species, coagulase negative *Staphylococci*, and various gram negative bacilli. The treatment of otitis externa involves a strategy intended to resolve the infection while promoting the restoration of the external auditory canal to its original healthy state. Four fundamental principles predominate and include: 1) frequent and thorough atraumatic cleansing of the canal through careful suctioning and debridement under microscopy (may need to be repeated frequently depending on severity), 2) use of the appropriate topical antibiotics (insertion of an otowick may be necessary to facilitate application of drops medially), 3) treatment of associated inflammation and pain, 4) and recommendations for prevention of future infections (i.e. dry ear precautions).

In most cases of uncomplicated AOE, topical antibiotics are the first-line treatment choice. There is no evidence that systemic antibiotics alone or combined with topical preparations improve treatment outcome over topical antibiotics alone. However, serious manifestations of the disease, such as periauricular cellulitis, necessitate the use of systemic antibiotics based on culture sensitivities. When the status of the TM is unknown, the ototoxic potential of topical antibiotics must be considered. The risk of ototoxicity by ototopical preparations has been debated for years. Estimates vary widely from 0.01% to 3%. Currently, the only ototopical drop approved by the FDA for use in an open middle ear is ofloxacin.

Chronic Otitis Externa

Chronic otitis externa (COE) is an inflammatory process of the ear canal due to bacterial, fungal, or other dermatologic disorders. COE can result from recurrent otitis externa, chronic purulent otitis media with perforation, or eczematoid dermatitis. The disease can be defined by having persistent symptoms for more than 2 months, which include unrelenting pruritus, mild discomfort, and dry flaky skin in the EAC. On exam, the EAC skin exhibits asteatosis (lack of cerumen), dryness and hypertrophy. Partial canal stenosis from the hypertrophied skin is common. Mucopurulent otorrhea is occasionally found. Culture reports vary widely and are often distorted because patients have been prescribed various antibiotics before referral to an otolaryngologist. One study reports *S. aureus*, *Pseudomonas*, and fungi as the most predominate pathogens. Management is similar to that of AOE. Multiagent topical treatments and frequent cleanings are often necessary. Topical steroid cream may help alleviate the chronic itching and resultant excoriations often present with this condition. Rarely is surgical intervention necessary. However, if medical management fails, surgical procedures to enlarge and resurface the EAC, such as conchal meatoplasty, are indicated.

Furunculosis (Acute localized otitis externa)

Furunculosis is a localized infection, usually found in the lateral one third of the posterosuperior aspect of the EAM that results from obstructed apopilosebaceous units. The medial canal is often normal in appearance and to palpation. The most common pathogen is *Staphylococcus aureus*. The primary lesion is often a small pustule that may enlarge to become a furuncle. The symptoms include localized pain and itching, and may include pain with mastication if the lesion involves the anterior wall. If the lesion occludes the canal, hearing loss may be present. Upon examination, edema, erythema, tenderness and occasionally fluctuance are present. Limited lesions that have not progressed to form an abscess are treated with local heat, analgesics and oral anti-staphylococcal antibiotics. If spontaneous drainage does not occur, and the lesion progresses into an abscess with cellulitis, incision and drainage after local anesthesia is indicated. Extension of the infection to the pinna and periauricular soft tissues may warrant parenteral antibiotic therapy.

Otomycosis

Otomycosis is a fungal infection of the skin of the EAC. Fungi can be either the primary pathogen or superimposed on bacterial infections. Many fungi have been implicated in the disease process, however the most common organisms isolated are *Aspergillus* and *Candida*. The initial symptoms of fungal otitis externa are often indistinguishable from bacterial OE. The most common symptoms of otomycosis are pruritus deep within the ear and an irresistible urge to scratch. The itching generally progresses to dull pain with or without drainage. The accumulation of fungal debris in the inflamed, narrowed canal often leads to a complaint of hearing loss. Tinnitus is also a common presenting complaint. Physical examination generally demonstrates canal erythema, mild edema and the presence of white, gray, or black fungal debris within the canal. Treatment is directed at thorough cleaning and drying of the canal followed by the application of topical antifungal medication. There are currently over 20 topical agents recommended for the treatment of otomycosis, however, controversy exists as to the first-line agent of choice. Lucente describes an effective regimen as: 1) clean the canal thoroughly and dry completely, 2) apply topical Cresylate for 5 minutes, 3) flush the canal gently with Domeboro solution and dry again, 3) apply a thin coating of nystatin-triamcinolone ointment (Mycolog II) throughout the length of the canal under microscopy, 5) give the patient a prescription for Mycolog II to use at home once daily. Occasionally, Lucente describes having to use systemic antifungal agents in conjunction with intensive topical therapy in patients with refractory diseases. Should the ear become macerated and wet topical powders are preferable. In patients with previous mastoid surgery, Gentian violet is well tolerated for fungal infections involving the mastoid cavity.

Granular Myringitis

Granular Myringitis is the result of localized chronic inflammation of the lateral surface of the pars tensa of the tympanic membrane and is characterized by persistent, incompletely epithelialized granulation tissue over the involved area. It is a poorly understood entity with few cases documented in the literature, which is why its incidence is difficult to estimate. Toynebee was the first to record a description of granular myringitis in 1860, when he noted a case of “catarrhal inflammation of the dermoid layer after measles” with a “polypoid growth from the surface, ...especially posteriorly”. It has been reported to occur as a result of primary acute myringitis, a sequela of a previous OE, or a perforation of the TM. Gram – negative bacilli are the most commonly cultured organisms, especially *Pseudomonas* and *Proteus* species, however, there is no evidence that any one type of bacteria or fungi is associated with this disease.

The course is generally chronic with inflammation confined to the outer epithelial and underlying fibrous middle layers of the TM. The layers become replaced by granulation tissue, which may extend over the entire surface of the eardrum, if neglected. The usual presenting complaint is a foul smelling discharge from one ear, although many patients remain asymptomatic. Other common complaints are slight irritation and fullness in the ear without significant pain or hearing impairment. On physical examination, the TM is usually obscured by a mucopurulent discharge with “peeping” granulations. There is no perforation of the TM to be found with granular myringitis, which distinguishes it from chronic suppurative otitis media with perforation.

Treatment includes careful and frequent debridement of the ear with the application of anti-Pseudomonal antibiotics, occasionally combined with steroids for at least two weeks. If no resolution of the granulation tissue occurs after topical treatment, some form of topical chemical destruction of the granulation tissue should be used without destroying the underlying fibrous middle layer of the TM. Any chemical agent should be left in contact for less than 2 minutes and only applied once a week in order to avoid necrosis and perforation of the fibrous layer. One example, described by Yinglin, is a 0.5% solution of formalin. Other agents include 50% chromic acid, ferric perchloride, solid silver nitrate, trichloroacetic acid, and pure carbolic acid.

Bullous Myringitis

This is a form of viral involvement often confined to the tympanic membrane and primarily involves younger children. The presenting symptom is one of severe pain without fever and hearing loss. Upon examination of the ear, the inflammation is limited to the TM and adjacent canal wall and appears as multiple blebs that are reddened and inflamed. The vesicles are usually hemorrhagic and when ruptured produce a significant amount of bloody otorrhea. Unless there is a secondary bacterial invasion, the middle ear is not involved. The condition is self-limiting with resolution in 2 to 3 days. Treatment is aimed at pain relief and systemic and topical antibiotics to prevent secondary bacterial infection. Incision of the blebs is not recommended, due to the possibility of secondary infection, as this does not appear to change the rate of recovery.

Necrotizing External Otitis (Malignant Otitis Externa)

Necrotizing External Otitis (NEO) is a potentially lethal infection of the EAC and adjacent structures typically seen in elderly diabetic or immunocompromised patients. *Pseudomonas aeruginosa* is the bacteria most commonly responsible for this infection, which begins as an acute otitis externa and frequently progresses to a skull base osteomyelitis with resultant cranial neuropathies. Meltzer and Kelemen first described the disease process in 1959, but the name is credited to Chandler with his precise description of the clinical entity in 1968. The diagnosis of NEO is based on clinical and laboratory evidence along with the suspicions of the treating physician.

The typical patient is an elderly diabetic with poor metabolic control and evidence of otitis externa not responding to the usual local therapy. The typical complaints are deep-seated aural pain, discharge, and fullness. A history of diabetes or an immunocompromised state (neoplasm, immunosuppressive therapy, HIV, etc.) should be elicited. Examination of the involved ear canal reveals inflammation and granulation tissue at the bony cartilaginous junction. Purulent secretions are common, and excessive inflammation may occlude the canal and obscure the TM. Disease beyond the EAC may extend anteriorly into the parotid through the fissures of Santorini or inferiorly into the soft tissue below the tympanic ring. Cranial nerve involvement may appear as early as one week after the onset of symptoms, with the facial nerve most commonly involved, followed by X and XI. Various imaging techniques have been employed to help is the

diagnosis of NEO, including plain films, computerized tomography (CT), technetium-99(Tc99) bone scan, gallium scan, and magnetic resonance imaging. Computerized tomography scanning is particularly useful for following soft tissue extension of infection and subtle bony changes, and is the radiological test of choice today. Tc99 scanning and gallium scans are reliable in identifying osteomyelitis of the temporal bone and skull base. The gallium scan reverts to normal with successful treatment, and is therefore useful for evaluating effectiveness of therapy.

Cohen and Friedman established diagnostic criteria to distinguish NEO from AEO, based on obligatory and occasional signs. The signs were determined from a review of the current literature and were divided into major signs (appeared in 100% of cases) and minor signs (appeared only in some of cases). Major signs included: pain, exudates, edema, granulations, microabscess, positive Tc99 scan, and failure of local treatment after more than 1 week. Minor signs included: *Pseudomonas*, positive radiograph, diabetes, cranial nerve involvement, debilitating condition, and old age. It was noted, however, that *Pseudomonas* was found in 98% of cases reported, but did not technically meet the requirement for being a major sign of NEO.

Treatment with parenteral anti-Pseudomonal antibiotics should be continued for a minimum of 4 weeks. Local canal debridement is an essential part of therapy and should be started immediately and continued until granulation tissue resolves and healing ensues. Pain control is usually necessary and, underlying disease states must be controlled. The use of topical antimicrobial agents is controversial, because they are insufficient for invasive infection and tend to hinder culture isolation of the offending pathogen. Hyperbaric oxygen has been used with varying success in some reports. Resolution of otalgia, decreased drainage and a falling ESR indicate a response to therapy. The duration of antimicrobial therapy depends on serial gallium scans performed at 4-week intervals. Surgical debridement of tissue and infected bone is usually reserved for those patients not responding to medical management. There is no universal agreement on the need for prophylactic or therapeutic facial nerve decompression.

Mortality remains significant with the death rate essentially unchanged in 20 years despite the introduction of newer antibiotics. Increased mortality is associated with mental status deterioration and cranial nerve involvement, with the highest mortality seen in cranial polyneuropathies. Recurrence is not uncommon with rates ranging from 9% to 27%. Infection can recur as long as four to 12 months after cessation of antibiotic therapy, so periodic follow-up and re-evaluation of ESR is essential to proper management of this disease.

Perichondritis and Chondritis

Bacterial infection of the perichondrium or cartilage is usually a result of accidental or surgical trauma to the auricle. In cases where it appears spontaneously, a high index of suspicion for overt or latent diabetes mellitus should be raised. The presenting complaint is pain and severe itching deep in the canal. The skin over the pinna is tender, indurated and edematous. In more advanced cases, the affected area may become crusted and weep purulent exudates with involvement of the surrounding soft tissues of the face and neck. Mild cases can be treated with debridement and topical and oral antibiotics. Antibiotics should be directed toward the offending organism from culture sensitivities. If the infection spreads to involve regional soft tissues and lymphatics, hospitalization and parenteral antibiotics are necessary. If subacute or chronic infection becomes established in the perichondrium or cartilage and continues despite treatment, surgical intervention under a controlled setting is indicated. Surgery involves excision of necrotic tissue with coverage by local skin flaps. Small irrigation drains should be placed beneath the flaps and irrigated with an antibiotic solution three times per day. The drains can be advanced as the condition resolves.

Relapsing Polychondritis

Relapsing polychondritis is an episodic and generally progressive inflammation of the cartilaginous structures of the body. An autoimmune etiology is suspected since many of these patients have circulating antibodies to type II collagen. Cartilage of the external ear, larynx, trachea, bronchi, and nose may be involved. During exacerbations, findings include fever, erythema, swelling, pain, anemia and an elevated ESR. With progression of disease, involvement of the larynx and trachea causes increased respiratory obstruction. Treatment is with oral corticosteroids.

Herpes Zoster Oticus (Ramsay Hunt syndrome)

Herpes zoster oticus is a viral infection of the ear caused by the varicella zoster virus. The virus causes infection along the dermatomes of one or more cranial nerves, thus describing shingles. In 1907, J.Ramsay Hunt, a neurologist described the cutaneous areas that can be involved in this infection. He noted that the trigeminal, geniculate, and upper cervical root ganglia might be involved, individually or combined. Ramsay Hunt syndrome has evolved to describe herpes zoster of the pinna with otalgia and facial paralysis. The earliest symptom is a burning pain in one ear, which may be accompanied by headache, malaise and fever for a couple of days. Vesicles usually appear 3 to 7 days after the onset of pain, and usually erupt on the antihelix, conchal bowl, and posterior lateral EAC. Infection of the geniculate ganglion may also present with facial paresis, or complete paralysis. In the case of complete paralysis, corneal protection with ophthalmic drops and lubrication at night is indicated. Oral steroids are also commonly prescribed and tapered over 10 to 14 days. Treatment with acyclovir, famcyclovir and valacyclovir have been shown to be effective in shortening the phase of viral shedding and reducing otalgia.

Erysipelas

Erysipelas is an acute, localized but spreading superficial cellulitis that may involve the auricle. It is caused by group A beta hemolytic streptococci and characterized by involvement of the lymphatics. The affected skin is bright red, well demarcated and tender, with a distinctly advancing margin. Treatment with oral antibiotics should be started promptly, however, hospitalization and intravenous antibiotics may be necessary, if a rapid response is not evoked.

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