Review Article

Infections of the External Ear

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Abstract

Otitis externa is one of the most common conditions seen in the otolaryngology practice. It encompasses a wide range of conditions, from those that cause mild inflammation and discomfort to those that are life-threatening. The management of these conditions requires a clear understanding of the anatomy and physiology of the ear canal, the microbiology of pathogens and familiarity with the clinical presentation.

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Introduction

Otitis externa refers to a spectrum of infections of the external auditory canal and auricle. It is a common condition and affects between 5% to 20% of the patients attending ENT clinics.[1] It may be classified on the basis of aetiology, location, and time course of the illness. These include acute diffuse otitis externa, acute localised otitis externa, chronic otitis externa, otomycosis, herpes oticus, dermatoses and malignant otitis externa.

Anatomy and Physiology

The external ear is composed of the auricle (pinna) and external auditory canal. Both contain elastic cartilage (except the earlobe) and a small amount of subcutaneous fat, covered by skin and its adnexal appendages including the sebaceous glands, sweat glands and hair.

The external auditory canal serves as a conduit for sound transmission to the middle ear and protects the middle and inner ear. Its average length is 2.5 cm. The lateral one-third is cartilaginous while the medial two-thirds is osseous. The skin of the cartilaginous canal contains hair cells, sebaceous (lipid-producing) and apocrine (ceruminous) glands, in contrast to the osseous canal, which contains neither glands nor hair follicles. Together, these 3 adnexal structures are termed the apopilosebaceous unit and serve a protective function. Cerumen (ear wax) is a result of secretions produced by the sebaceous and apocrine glands admixed with desquamated epithelial cells. Apart from physically

protecting the canal skin, its acidic coat creates an inhospitable environment for pathogens and it also produces antimicrobial compounds such as lysozymes.[2]

The canal is a self-cleansing structure as the cerumen coat migrates laterally and sloughs externally. Instrumentation and excessive cleansing of the canal predisposes one to infection in 2 ways. First, the act of removing cerumen, even using one's own fingernail, may be traumatic, as it can abrade the canal skin and allow the introduction of bacteria. Secondly, the removal of cerumen leads to the disruption of this protective barrier.

The 2 most common organisms isolated in the external auditory canal of normal individuals are the *Staphylococcus* species (*S. auricularis, S. epidermidis, S. capitis*) and the *Corynebacterium* species (*Turicella otitidis, C. auris*). The third most frequently recovered bacteria are the *Streptococci* and *Enterococci* group (*Alloiococcus otitis*). Together, they account for more than 90% of the normal flora in the external auditory canal.[3]

Acute Diffuse Otitis Externa

Acute diffuse otitis externa is a bacterial infection of the canal caused by a break in the normal skin/cerumen protective barrier in the presence of elevated humidity and temperature.[4] It is commonly known as "swimmer's ear", though anything that disrupts this protective lipid layer can lead to the introduction and proliferation of bacteria.[5,6] Trauma from cleaning the ears with fingernails or cotton

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buds has been identified as the most common predisposing factor locally.[7] Other predisposing factors include inherited narrow ear canals and non-atopic eczema. Pain (70%), itch (60%), deafness (32%) and fullness (22%) are the main cardinal symptoms.[8] Signs on examination include erythema, oedema, purulent otorrhoea and crusting of the canal wall skin. A gentle tug of the auricle upward and backward usually causes pain, and this distinguishes it from patients with otitis media.[4]

Senturia et al[8] divided the clinical course of otitis externa into 3 stages: preinflammatory, acute inflammatory and chronic inflammatory. Acute inflammatory stage is further divided into mild, moderate or severe categories. The preinflammatory state is characterised by itch, oedema and a sensation of fullness. The acute stage is accompanied by pain and auricular tenderness. As the infections progress from mild to severe, the itch, pain and auricular tenderness are all intensified. The canal becomes more oedematous and erythematous. The secretion, initially clear and odourless, will turn into a thick, profuse and seropurulent exudate. In the severe stage, the lumen becomes obliterated due to the increasing oedema and seropurulent material. The patient complains of intense pain, especially on chewing or tragal manipulation. Fever, periauricular oedema and cervical lymphadenopathy may also be present.

Pseudomonas aeruginosa, S. epidermidis and *S. aureus* are the 3 most common pathogens isolated in acute diffuse otitis externa locally.[7] This is similar to a recent study involving 2039 subjects with acute otitis externa, which identified the above 3 organisms as the most common bacteria isolated.[9] The same study also identified the *Microbacterium* species as important pathogens in acute otitis externa. They are members of the *Corynebacterium* group and include *M. otitidis* and *M. alconae*. Culture of the canal is usually only required in recalcitrant cases, where it may assist in the choice of antibiotic therapy.

The key to success in managing external ear infections is regular and meticulous aural toilet. In the general practice setting, commonly used techniques include syringing or swabbing with cotton-tipped applicators. Syringing should only be done judiciously and in the presence of an intact tympanic membrane. It can be followed by drying the ear with a hair drier set on low heat and aimed from 1 foot away for 60 seconds (care must be taken when using an electric appliance near water).[10] Another method is that of mopping using a cotton wool. Cotton wool is twisted or folded onto the wool carrier end of a Jobson Horne probe or any wool carrier in order to create a tool like a small paintbrush. This is a gentle method but may not be able to clean the ear thoroughly, especially at the medial end. Mounting the cotton wool on the applicator can also be technically difficult and requires patience and experience. The best

method is probably microsuction as it allows direct visualisation of the ear canal and complete clearance of any debris. The only limitation is the need for a microscope. If the canal is obliterated by oedema and debris, a pope wick is inserted to stent the canal and allows the application of antibiotic/steroid drops. The wick is generally removed 24 to 72 hours later, although it can be left in the canal for up to 1 week. Once the ear is cleaned, an antibiotic/steroid eardrop is administered for 1 week. Topical antibiotics and steroids are based in an acidic solution to inhibit bacteria growth and many contain glycerol, which acts as a desiccant.[10] The major adverse reaction to the use of an acidic agent is burning on application, which may affect the patient's compliance.[2] Ophthalmic preparations, on the other hand, are less acidic than otic preparations and may be better tolerated by these patients. It also has a low viscosity, allowing improved penetration in narrow lumens.[2]

Eardrops containing gentamyicin or polymyxin appear to be most effective against the 3 most common bacteria locally.[7] Ciprofloxacin/ofloxacin otic solution is a new topical formulation that has a wide spectrum of activity against most common ear pathogens.[11] Even though some organisms may exhibit resistance to antibiotics in vitro, the high concentration of antibiotics in topical antibiotic preparations will be lethal to those with resistance, provided the topical solution can penetrate to infected tissues.[9] In severe cases where cellulitis is present, systemic antibiotics are indicated. Analgesia is also required for pain control. Patients are advised to resist digital manipulation, avoid swimming during the treatment period and to prevent water from entering the ear canal while taking a shower or a bath.

Education is also important in preventing future episodes. This is aimed at minimising ear canal trauma and the avoidance of exposure to water. Earplugs may be used during swimming or bathing to decrease the amount of moisture entering the ear. Applications of topical acidifying agents (acetic acid) or 70% alcohol, which recreate an acidic environment, are also advocated.

A few of the common differential diagnoses include malignant otitis externa, bullous external otitis, granular external otitis, furunculosis, dermatoses, such as seborrheic dermatitis. Another important differential diagnosis is carcinoma of the external auditory canal, which may present as infection, and in its earliest stages are often mistaken for infection and inappropriately treated.[4] The persistence of infection and granulation tissue despite treatment will require a biopsy to exclude malignancy.

Chronic Otitis Externa

Chronic otitis externa occurs when there is incomplete resolution of the acute infection or where there is a persistent low-grade infection and inflammation for more than 3

months. There is less pain but more profound itching and persistent discharge. Examination reveals thickening of the canal skin of the ear canal with no wax. The chronic scaling and itching in the canal predispose the patient to manipulation of the canal, excoriation and repeated episodes of acute otitis externa. With time, the canal becomes completely obliterated by the hypertrophic skin.

The goal of treatment is to restore the external auditory canal skin to its original healthy state and to promote the reproduction of cerumen.[2] These consist of frequent aural toilet and instillation of an acidifying and drying ear drops (vinegar and water or ethyl alcohol and water). The authors have also found the painting of gentian violet to be a useful adjunct in the treatment of chronic otitis externa. The use of topical antibiotics and steroids may be required during acute exacerbations. Surgery is rarely indicated and is limited to enlarging and resurfacing the external auditory canal (canalplasty).

Furunculosis

The pathology of furunculosis is an infection of a hair follicle by *S. aureus*. The main symptom is otalgia and there is generally no otorrhoea or deafness. The patient complains of pain on tugging the pinna and compression of the tragus. Examination reveals a localised swelling with surrounding erythema within the hair-bearing area of the external ear canal. If the furuncle is not pointing, an antibiotic (e.g., Triderm®, Bactroban®)-impregnated wick is inserted into the canal over the furuncle for about 3 to 5 days. If it is pointing, an incision and drainage will be required. A course of antibiotics (cloxacillin 250 mg qds for a period of 1 week) is also prescribed.

Otomycosis

Otomycosis, a fungal infection of the external ear canal, is more frequently encountered in the tropical countries as compared to temperate countries as a result of the greater heat and humidity. Other predisposing factors include long-term topical antibiotic therapy, previous medical history of diabetes mellitus or an immunocompromised state.[2,12] *Aspergillus* species (60% to 90%) and *Candida* species (10% to 40%) are most often implicated.[7,13] Patients usually complain of pruritus and thickened otorrhoea. Examination reveals black, gray, bluish green, yellow, or white fungal growth and debris in the external ear canal. *Aspergillus* has a distinctive appearance consisting of small black conidiophores, with a long white filamentous hypha.

Treatment consists of thorough aural toilet and altering the environment of the external auditory canal with acidifying/drying agents and antifungal topical eardrops such as clotrimazole.

Malignant Otitis Externa (MOE)

First described by Chandler in 1968,[14] this is an invasive pseudomonas infection of the external ear canal, which may lead to osteomyelitis of the temporal bone, multiple cranial nerve palsy and death. The term "malignant" does not imply malignancy but refers to the high mortality associated with the disease when left untreated. It is primarily but not exclusively seen in elderly diabetics. Any immunocompromised patient, including those with acquired immunodeficiency syndrome (AIDS) and those on chemotherapeutic or immunosuppressive medications, can be affected.[15] The organism involved in MOE is invariably *P. aeruginosa*, though fungal MOE (*Aspergillus* species) has been described.[16] The *Pseudomonas* species has a propensity to invade soft tissue and bone (osteomyelitis) through the secretion of enzymes.[17] If left unchecked, the infection spreads towards the skull base, involves the lower cranial nerves and brainstem, ultimately leading to septicaemia and death.

The initial presentation is that of diffuse otitis externa. It does not, however, respond to conventional topical measures. The true nature of the condition emerges as the patient develops severe pain, which is out of proportion with the clinical signs.[15] The pain is progressive, worsens at night and interferes with sleep and the ability to function. The patient may look toxic. Purulent otorrhoea is seen in more than half of the cases while hearing is not affected in most cases.[18] Examination reveals a swollen and tender external auditory canal with an intact eardrum. Granulation tissue is classically seen on the floor on the canal at the bony-cartilaginous junction.[16] The most commonly affected cranial nerve is the facial nerve, with cranial nerves IX to XI less frequently involved.[18] Hence, an external ear infection with ipsilateral lower motor neuron facial nerve palsy is MOE until proven otherwise.

The severity can be classified into 3 stages.[19] Stage I represents necrotising infection limited to soft tissue and cartilage, stage II exhibits soft tissue involvement and bony erosion of the temporal bone, and stage III involves bony erosion of the skull base extending beyond the confines of the temporal bone or intracranial extension.

Diagnosis is based primarily on clinical finding. A high index of suspicion is necessary as early diagnosis with early treatment reduces morbidity and mortality. It should be suspected in any patient (particularly diabetics) with disproportionate otalgia and granulation tissue deep in the external meatus that does not settle with standard conservative treatment.

Investigations are aimed at confirming diagnosis and determining the extent of the disease. Culture and sensitivity should be performed on the discharge and biopsy of granulation tissue taken to rule out malignancy. Elevated

erythrocyte sedimentation rate (ESR) is commonly seen at initial diagnosis and will normalise in response to successful treatment.[20] Leukocytosis is uncommon and not contributory to diagnosis and management.[20]

High-resolution computed tomography (CT) of the temporal bone is performed to determine the extent and severity of soft tissue involvement and bony destruction.[20] As more than 30% of affected bone need to be demineralised to appear eroded, early findings are limited to soft tissue inflammation.[21] The disadvantages of CT scan are its inability to distinguish MOE from malignancy, and its inability to evaluate response to treatment because remineralisation of afflicted bone may never occur despite resolution of infection.[22]

A magnetic resonance imaging (MRI) scan is superior to a CT scan in delineating soft tissue involvement and is recommended when central skull base invasion is seen on CT scan.[21] It is, however, unable to detect bony destruction, and because changes on MRI do not resolve with disease, it is also not useful for evaluating the course of treatment.[22]

Technetium phosphate radionucleotide scan (bone scan) is used to detect bony involvement when there is an absence of destruction on CT scans. Signal enhancements are seen in areas of increased osteoblastic activity. It is non-specific and can also occur in infection, inflammation, neoplasm, trauma or postoperative conditions.[20] As bone demineralisation need not be present, it allows for earlier diagnosis of osteomyelitis than with other radiographic techniques.[16] Evaluation at 4 hours and 24 hours post-injection with an increased uptake between the 2 periods is the most sensitive marker of temporal bone osteomyelitis.[23] As bone repair may occur long after the infection has cleared, it remains positive for months or years after the infection has resolved and hence cannot be assessed for response to treatment.

Gallium-67 citrate binds to leukocytes and forms a complex with lactoferrin.[21] It will be positive in soft tissue and bone infection. The main advantage is that it returns to normal sooner once the infection is resolved and can be used to assess response to treatment.[16] It can be repeated every 4 weeks to monitor antibiotic response to antibiotic therapy, which is continued until the scan is normal.[20] However, it should be noted that some patients may have persistence of uptake despite clinical evidence of resolution.[24]

Treatment

All patients with suspected or confirmed MOE are admitted to hospital. The aim is to start intensive pharmacological therapy, monitor progression (or resolution) and to control the predisposing condition (diabetes, immunodeficiency). An anti-pseudomonal antibiotic (ciprofloxacin or ceftazidime alone or in combination with an aminoglycoside) is started intravenously empirically until sensitivity results are available.[20,24-26] There has been controversy over the most appropriate antibiotic regimen such as oral versus parenteral and monotherapy versus multiple agents. Since the 1970s, a combination of an anti-pseudomonal antibiotic and an aminoglycoside has been the standard of treatment.[16,18,20,24] Ciprofloxacin has been used since the late 1980s as it is effective against *Pseudomonas*, has strong bone penetration, rapid accumulation in tissue with oral administration, and a mild side effect profile.[20] There have been reports of successful treatment with oral ciprofloxacin but reports of the susceptibility of ciprofloxacin to resistance in the *Pseudomonas* species have moved some authors to caution against using oral ciprofloxacin as the primary antibiotic of choice.[15,17,27,28] Currently, ceftazidime appears to be effective in treating ciprofloxacin-resistant cases. This may change in the face of resistance to ceftazidime and combination therapy may be required again in the future.[17]

Daily debridement of the external ear canal with microsuction is carried out until all granulation tissue has resolved. Strict control of blood sugar and pain relief are also important.

The use of topical antibacterial eardrops is controversial, as it changes the bacterial flora and alters future culture results.[18] MOE is primarily a medical condition and surgical intervention is limited to patients who are refractory to medical treatment, requiring debridement of sequestra (devitalised tissue) within the external canal and facial nerve decompression when indicated.[24]

Hyperbaric oxygen has been used as an adjuvant therapy for advanced disease with significant skull base or intracranial involvement, recurrent cases, and infections refractory to antibiotic treatment.[29] The increase in oxygen partial pressure and oxygen tension aids in the polymorphonuclear cells' ability to kill aerobic pathogens. However, the cost, duration of treatment and side effects such as oxygen toxicity and barotrauma have limited its use.[20]

Antibiotic therapy should be instituted for a minimum of 6 weeks.[4] Resolution of otalgia, decreased drainage, a falling ESR and improvement in facial nerve palsy all indicate a response to the treatment.[20] After an initial 2-week course of intravenous chemotherapy and upon cessation of all symptoms, intravenous antibiotics can be converted to oral ciprofloxacin.[4] At the end of the treatment, follow-up gallium-67 scanning can be performed to confirm complete resolution of disease. Recurrences have been known to occur up to 1 year from the time of clinical resolution, so patients need to be informed and followed closely.[20]

Conclusion

Otitis externa remains a common condition in the otolaryngology practice. The majority of cases can be managed with a combination of meticulous and regular aural toilet, eardrops and preventive measures. The diagnosis of MOE must be considered in all patients with persistent otalgia and granulation tissue in the external ear canal. Early diagnosis will lead to reduced morbidity and mortality.

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