

Acute Otitis Externa: Pathophysiology, Clinical presentation, And Treatment

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Abstract:

Acute otitis externa (AOE) is the inflammation of outer ear and ear canal. Four categories of otitis externa that include acute localized otitis externa, diffuse otitis externa, chronic otitis externa, and malignant otitis externa. Incidence of otitis externa is high in Europe, and probably higher in the developing countries. Predisposing factors for AOE is swimming and swimming in the polluted water. Bacterial growth and impairment of the skin of the ear canal that permits the development of infection. Chronic skin conditions atopic dermatitis, psoriasis or abnormalities of keratin production may cause infection and external otitis. Frequently associated pathogens include, *Pseudomonas aeruginosa*, *Staphylococcus epidermidis*, *Staphylococcus aureus*, fungi, and yeast. Treatment of choice used are antibiotic ear drops with or without corticosteroid. Fungal or otomycoses require debridement and local treatment. Resistant fungal infection may require oral itraconazole. Malignant otitis externa (MOE) may develop serious complications include skull base osteomyelitis (SBO), multiple cranial nerve palsies including facial palsy. May require long term IV antibiotics, tends to recur with significant mortality. Preventive measures such as drying the ears with air dryer and avoiding the manipulation of the external canal may help. Clinical practice guideline on AOE by the American Academy of Otolaryngology (AAO)-Head Neck Surgery are useful.

Key Words: Otitis externa, external auditory canal, Pathophysiology, Clinical presentation, and Treatment.

I. Introduction

Otitis externa, external otitis or swimmer's ear is an inflammation of the outer ear and ear canal. Along with otitis media, external otitis is one of the two human conditions commonly called "earache" [1]. Infection of the external auditory canal (EAC), (otitis externa) is similar to infection of skin and soft tissue elsewhere. Infection of the external ear canal may be subdivided into four categories: (a) **acute localized otitis externa** (b) **diffuse otitis externa** (c) **chronic otitis externa**, and (d) **malignant otitis externa** [2,3,4]. The incidence of otitis externa is high. In the Netherlands, it has been estimated at 12-14 per 1000 population per year, and has been shown to affect more than 1% of a sample population in the United Kingdom over a 12-month period [5]. Of the predisposing factors for acute otitis externa, only swimming has been shown to increase the risk [6]. Swimming in polluted water is a common way to contract swimmer's ear, but it is also possible to contract swimmer's ear from water trapped in the ear canal after shower, especially in a humid climate [7]. Constriction of the ear canal from bone growth (surfer's ear) can trap debris leading to infection [8]. Saturation divers have reported otitis externa during occupational exposure [9]. Even without exposure to water, the use of objects such as cotton swabs or other small objects to clear the ear canal is enough to cause breaks in the skin, and allow the condition to develop [10]. Once the skin of the ear canal is inflamed, external otitis can be drastically enhanced by either scratching the ear canal with an object, or by allowing water to remain for any prolonged length of time [10]. Two factors that are required for external otitis to develop are (1) the presence of microorganisms that can infect the ear and (2) impairments in the integrity of the skin of the ear canal that allows infection to occur. If the skin is healthy and uninjured, only exposure to high concentration of pathogens, such as submersion in a pond contaminated by sewage, is likely to set off an episode. However, if there are chronic skin conditions that affect the ear canal skin, such as atopic dermatitis, seborrheic dermatitis, psoriasis or abnormalities of keratin production, or if there has been a break in the skin from trauma, even the normal bacteria found in the ear canal may cause infection and full-blown symptoms of external otitis [11]. Prophylactic measures such as drying the ears with hair dryer and avoiding manipulation of the external canal may help recurrence [12]. Pathogens commonly associated with acute otitis externa are *Pseudomonas aeruginosa*, *Staphylococcus epidermidis*, *Staphylococcus aureus*, and *Streptococcus pyogenes* [13]. Fungi and yeast are usually found in patients with chronic otitis externa or those who are immunocompromised [14]. Current management includes debridement followed by dressing and topical treatment with acidifying or antibacterial agents, with or without corticosteroids [15]. Frequently used are

antibiotic ear drops with or without steroids [15]. The paper reviews the current literature, pathophysiology, diagnosis, and treatment of otitis externa.

II. Pathophysiology

Auditory canal. The unique structure of auditory canal contributes to the development of otitis externa. It is the only skin-lined cul-de-sac in the human body. The external auditory canal is warm, dark and prone to become moist, making it an excellent environment for bacterial and fungal growth. The skin is very thin and the lateral third overlies cartilage, while the rest has a base of bone. The canal is easily traumatized. The exit of debris, secretions and foreign bodies is impelled by a curve at the junction of the cartilage and bone. The presence of hair, especially the thicker hair common in older men, can be a further impediment. The external auditory has some special defenses. Cerumen creates an acidic coat containing lysozymes and other substances that probably inhibit bacterial and fungal growth. The lipid-rich cerumen is hydrophobic and prevents water from penetrating to the skin and causing maceration. Too little cerumen can predispose the ear to infection, but cerumen that is excessive or too viscous can lead to obstruction, retention of water and debris, and infection. Additionally, the canal is defended by unique epithelial migration that occurs from the tympanic membrane outward, carrying and debris [16,17]. When these defenses fail or when the epithelium of the external auditory canal is damaged, otitis externa results. There are many precipitants of this infection but the most common is excessive moisture that elevates the pH and removes the cerumen. Once the protective cerumen is removed, keratin debris absorbs the water, which creates a nourishing medium for bacterial growth [12].

The external auditory canal is approximately 2.5 cm long from the concha of the auricle to the tympanic membrane. The lateral half of the canal is cartilaginous; the medial half tunnels through the temporal bone. A constriction, the isthmus, present at the junction of the osseous and cartilaginous portions, limits the entry of wax and foreign bodies to the tympanic membrane. The skin of the canal is thicker in the cartilaginous portion and includes a well-developed dermis and subcutaneous layer. The skin lining of the osseous portion is thinner and firmly attached to the periosteum and lacks a subcutaneous layer. Hair follicles are numerous in the outer third space in the inner two third of canal [18]. The microbial flora of the external canal are similar to the flora of skin elsewhere. There is predominance of *Staphylococcus epidermidis*, *Staphylococcus aureus*, *Corynebacterium* and, to lesser extent anaerobic bacteria such as *Propionibacterium* species. Pathogens responsible for infection of the middle ear (*Streptococcus pneumoniae*, *Haemophilus influenzae*, or *Moraxella catarrhalis*) are uncommonly found in culture of the external auditory canal when the tympanic membrane is intact [19,20].

The epithelium absorbs moisture from the environment. Desquamation and denuding of the superficial layers of the epithelium may follow. In this warm moist environment, the organisms in the canal may flourish and invade the macerated skin. Inflammation and suppuration follow. Invasive organisms include those of the normal skin flora and gram negative bacilli, particularly *Pseudomonas aeruginosa*. Invasive otitis media is a necrotizing infection frequently associated with *P. pseudomonas*. The organism gains access to the deeper tissues of the ear canal and caused localized vasculitis, thrombosis, and necrosis of tissue. Diabetic microangiopathy of the skin overlying the temporal bone results in poor local perfusion and milieu for invasion by *P. aeruginosa* [18].

In 1949, Fabricant and colleagues, were the first otologist to develop an interest in skin pH of EAC of normal ears [21]. In 1957 Fabricant, compared his initial findings with pH of individuals affected by acute otitis externa [22]. Martinez-Devesa and colleagues [23], studied pH in chronic cases of otitis externa with an age, sex matched control groups, van Balen and colleagues [5], compared the clinical efficacy of three common treatments in acute otitis externa, ear drops containing either acetic acid, acetic acid with corticosteroid or corticosteroid with antibiotic. Kim and colleagues, deliberated correlation between the degree of AOE and change of pH after acidification compared to an antibiotic solution [24]. Aayush et al. studied the pH of EAC in normal individual and patients with AOE, its variations with change of temperature and humidity, different symptoms and number of symptoms at presentation, and observed that normal EAC pH is relatively more acidic as compared to that forearm skin and it became more alkaline in cases of AOE with revision back to acidic pH after treatment. However, no significant change in pH of ears was observed with change of temperature and humidity [25].

III. Clinical Presentation And Management

Acute localized otitis media externa may occur as pustule or furuncle associated with hair follicles; the external ear canal is erythematous, edematous and may be filled with pus and flakes of skin debris. *Staphylococcus aureus* is the most frequent pathogen. Erysipelas caused a group A *Streptococcus* may involve the concha and the canal. Pain may be severe. Bluish-red hemorrhage bullae may be present on the osseous canal walls and also on the tympanic membrane. Adenopathy in the lymphatic drainage areas is often present. Local and systemic are usually curative. Incision may be necessary to relieve severe pain [18].

Acute diffuse otitis externa (swimmer's ear) occurs mainly in hot humid weather. The ear itches and becomes increasingly painful. The skin of canal edematous and red. Gram-negative bacilli, mainly *P. pseudomonas* may play a significant role. A severe hemorrhage external otitis caused by *P. pseudomonas* was associated with mobile redwood hot tub systems [26]. Gentle cleansing to remove debris, including irrigation with hypertonic saline (3%) and cleansing with mixture of alcohol (70% to 95%) and acetic acid should be used initially. Hydrophilic solutions such as 50% Burow's solution may be used for 1 to 2 days to reduce inflammation. A cotton wick may be of value in enhancing distribution of the otological agent when canal is swollen. A ten-day regimen of fluoroquinolone otic solution such as ofloxacin or ciprofloxacin-dexamethasone or ear drops of neomycin alone or with polymixin combined with hydrocortisone are effective in reducing local inflammation and infection [27,28].

Chronic otitis externa is caused by irritation from drainage through a perforated tympanic membrane. The underlying cause is chronic supportive otitis media. Itching may be severe. Management is directed to middle ear disorder. Rare causes of chronic otitis externa include tuberculosis, syphilis, yaws, leprosy, and sarcoidosis [18].

Invasive-malignant otitis externa is a severe necrotizing infection that spreads from the squamous epithelium of the ear canal to adjacent areas of soft tissue, blood vessels, cartilage and bone [4,29]. Severe pain and tenderness of the tissues around the ear are accompanied by the drainage of pus from the canal. Older, diabetic, immunocompromised, and debilitated patients are at particular risk. Life threatening disease may result from spread to the temporal bone and then on to the sigmoid sinus, jugular bulb, base of the skull, meninges, and brain. Permanent facial paralysis is frequent, and cranial nerves 9, 11, and 12 may also be affected [30]. *P. pseudomonas* is almost always the causative agent. The extent of damage to soft tissue and bone may be identified and monitored by the use of computed tomography and magnetic resonance imaging [4]. Diagnostic tests for underlying disease may be instituted. The canal should be cleansed, devitalized tissue removed, and ear drops with anti-pseudomonal antibiotics with steroid instilled into the external auditory canal. Systemic therapy with regimens including activity for *Pseudomonas* spp. should be used for 4-6 weeks. The combination of ceftazidime, cefepime, or piperacillin with an aminoglycoside (gentamicin or tobramycin) should be considered [30]. Oral quinolones with activity against *Pseudomonas* spp., such as ciprofloxacin, have been effective therapy early in the course of invasive external otitis [31].

Necrotizing external otitis (malignant otitis externa) is an uncommon form of external otitis that occurs mainly in elderly diabetics, being somewhat more likely and more severe when the diabetes is poorly controlled. Even less commonly, it can develop due to a severely compromised immune system. Beginning as infection of the external ear canal, there is extension of infection into bony ear canal and the soft tissues deep to the bony canal. Unrecognized and untreated, may result in death. The hall mark of malignant otitis externa (MOE) is unrelenting pain that interferes with sleep and persists even after swelling of the external ear canal may have resolved with topical antibiotic treatment [32,wp,14]. It may also cause skull base osteomyelitis (SBO), manifested by multiple cranial nerve palsies [14].

Otomycosis or fungal otitis externa. Fungi are identified in about 10 per cent of cases of otitis externa [33]. The most common pathogen is *Aspergillus* (80 to 90 percent of cases, followed by *Candida*). Classically fungal infection is the result of prolonged treatment of bacterial otitis externa that alters the flora of the ear canal. Mixed bacterial and fungal infections are thus common [34]. The infection is often asymptomatic and the diagnosis is made by observing the unique discharge in the external auditory canal. When symptoms are present, discomfort is the most common complaint, but in fungal otitis externa this primarily takes the form of pruritus and a feeling of fullness in the ear. The pruritus may be quite intense, resulting in scratching and further damage to the epidermis. The discharge and tinnitus are also common [34]. *Aspergillus* species, particularly *A. niger*, may grow in the cerumen and desquamated keratinaceous debris in the external auditory canal, sometimes forming a visible greenish or blackish fluffy colony. Role of the mold in acute otitis externa is usually modest, if any, although, in the severely immunocompromised patient, *Aspergillus* can cause necrotizing otitis externa [35].

Noninfectious otitis externa. Systemic diseases [12] may cause otitis externa include atopic, dermatitis, psoriasis, seborrheic dermatitis, acne and lupus erythematosus. Lesions typically occur in the external auditory canal elsewhere on the body, especially the head neck. There is often a family history and a recurrent course. A thorough dermatologic examination and history should always be part of the evaluation of patients with otitis externa. Manifestations in the external auditory canal can range from mild erythema and scaling with atopic dermatitis, to dense adherent scaling with psoriasis, to the local inflammatory changes of acne. Pruritus is the most common symptom [12]. Control of disease elsewhere will reduce the manifestations in the ear canal and therefore the cornerstone of the treatment. In addition, otitis from all these diseases, excluding acne will respond

to low-dose therapy with topical steroid solutions. Steroids, however can lead bacterial and fungal overgrowth in patients with already compromised skin. Thus, an acidifying agent is often added. Acne will often respond to topical benzoyl peroxide lotions and antibiotic solutions. Seborrheic dermatitis of the external ear can be treated with medicated shampoo used for scalp. Difficult cases should be referred to dermatologist [33]. Contact dermatitis, irritant or allergic, can involve the pinna as well as the external auditory canal. Allergic forms usually present acutely with erythematous pruritus, edematous and exudative lesions, while contact dermatitis often has a more insidious onset with lichenification. Both types can be complicated by secondary bacterial infections. Contact dermatitis in the ear canal can result from any local irritant, including anti-infective agents and anesthetics and other topical preparations. It also may be associated with the use of hearing aids and ear plugs. Hypo-allergic silicone hearing aids are now available [12].

IV. Treatment

Effective solutions for ear canal include acidifying and drying agents, used either singly or in combination. When the ear canal skin is inflamed from the acute otitis externa, the use of dilute acetic acid may be painful [36]. Burow's solution is a very effective remedy against both bacterial and fungal external otitis. This is a buffered mixture of aluminium sulfate and acetic acid, and is available without prescription in the United States [37]. Topical solutions or suspensions in the form of ear drops are the mainstays of treatment for external otitis. Some contains antibiotics, either antibacterial or antifungal, and others are simply designed to mildly acidify the ear canal environment to discourage bacterial growth. Some prescription drops also contain anti-inflammatory steroids, which help to resolve swelling and itching. Although there is evidence that steroids are effective at reducing the length of treatment time required, fungal otitis externa (also called otomycosis) may be caused or aggregated by overly prolonged use of steroid containing drops [37]. Oral antibiotics should not be used to treat uncomplicated acute otitis externa [37]. Oral antibiotics are not a sufficient response to bacteria which cause this condition and have a significant side effects including increased risk of opportunistic infection [37]. In contrast, topical products can treat this condition [37].

In fungal or otomycosis otitis externa, cleansing of the ear canal by suctioning is principal treatment. Acidifying drops, given three to four times daily for five to seven days are usually adequate to complete treatment [12]. Because the infection can persist asymptotically, the patient should be evaluated at the end of the course of the treatment. At this time any further cleansing can be performed as needed. If the infection is not resolving over-the counter clotrimazole 1 percent solution (Lotrimin), which also has some antibacterial activity can be used. In vitro studies show that topical solutions of thimerosal (Merthiolate) and M-cresylacetate (Cresylate) are more effective agents but are messier [34]. If the tympanic membrane is perforated, toinaftate 1 percent solution (Tinactin) should be used in order to prevent ototoxicity [34]. All of these topical agents topically used at a dosage of three or four drops daily for seven days. Aspergillus infections may be resistant to clotrimazole and may require the use of oral itraconazole (Sporanox) [33].

Unlike ordinary otitis externa, MOE requires oral or intravenous antibiotics for cure. *Pseudomonas* is the most common offending pathogen, Diabetic control is also an essential part of treatment. When MOE goes unrecognized and untreated, the infection continues to smolder and over weeks or months can spread deeper into the head and involves the bones of the skull base, constituting skull base osteomyelitis (SBO). Multiple cranial nerve palsies can result, including the facial nerve (causing facial palsy), the recurrent laryngeal nerve (causing vocal cord paralysis), and the cochlear nerve (causing deafness). The infecting organism is almost always *Pseudomonas aeruginosa*, but it can instead be fungal (*Aspergillus* or *mucor*). MOE and SBO are not amenable to surgery, but exploratory surgery may facilitate culture of unusual organism(s) that are not responding to empirically used anti-pseudomonal antibiotics (ciprofloxacin being the drug of choice, anti-pseudomonal antibiotics mentioned earlier). The usual surgical finding is diffuse cellulitis without localized abscess formation. SBO can extend into the petrous apex of the temporal bone or more inferiorly into opposite side of skull base [32]. The use of hyperbaric oxygen therapy as an adjunct to antibiotic therapy remains controversial [32]. As the skull base is progressively involved, the adjacent existing cranial nerves and their branches, especially facial nerve and the vagus nerve, may be affected, resulting in facial paralysis and hoarseness, respectively. If both the recurrent laryngeal nerves are paralyzed, shortness of breath may develop and necessitate tracheotomy. Profound deafness can occur, usually later in the disease course due to relative resistance of the inner ear structure. Gallium scan are sometimes used to document the extent of the infection but are not essential to disease management. SBO is a chronic disease that can require months of IV antibiotic treatment, tends to recur, and has a significant mortality rate [32]. Clinical practice guideline on AOE by American Academy of Otolaryngology (AAO)-Head Neck Surgery are helpful [37].

V. Prevention

Prevention of recurrence of otitis externa primarily consists of avoiding the many precipitants and dermatologic disorders. This particularly important for patients with unusually viscous cerumen, a narrowed external auditory canal or systemic allergies[17]. After bathing or swimming, the external auditory canal should be dried using a hair dryer on the lowest heat setting. Acidifying drops can then be instilled. Some authors[38,39] recommend combining acidifying agent with alcohol drops (Swim Ear) to act as an astringent, but many physicians feel this too irritating and prefer using Burow's solution as the astringent (Star-Otic). Obviously and manipulation of the skin of the external auditory canal (such as scratching or overzealous cleansing) should be avoided[34]. Any time the external auditory canal is cleaned and cerumen is removed, the canal becomes more vulnerable to infection. Therefore, if there has been any trauma and syringing has left the external auditory canal wet, use of an acidifying agent with hydrocortisone is a good prophylactic measure [38, 40]. If the cerumen is difficult to remove, ceruminolytic agent such as Cerumenex or even a simple 4 percent baking soda solution should be used in the office to soften the cerumen first to avoid traumatizing the external auditory canal[38]. Persons who swim frequently should use a barrier to protect their ears from water. However, impermeable ear plugs act as a local irritant and have been shown to predispose the ear canal to otitis externa. A tight-fitting cap offers better protection[17,41]. Patients with acute otitis externa should preferably abstain from water sports for at least seven to 10 days[41], although some authors would allow competitive swimmers to return after three days of treatment as long as all pain has resolved[40]. Other would allow return with the use of well-fitting ear plugs[17].

VI. Conclusion

Otitis externa is an inflammation of the outer ear and ear canal. Frequently used therapy is antibiotic ear drops, with or without corticosteroid. In fungal or otomycosis otitis externa cleansing by suctioning, and with acidifying drops given several times daily is treatment of choice. Malignant otitis externa (MOE) may result in serious complications.

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