Otitis externa: What clinicians need to hear: pathophysiology, management and treatment

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Abstract

Otitis externa is a common inflammatory condition affecting the external auditory canal (EAC), characterised by symptoms such as otalgia (ear pain), pruritus (itching), erythema (redness), and otorrhoea (discharge), with a higher incidence in warm and humid environments. The condition may be classified as acute, chronic, or necrotising, with diagnosis primarily based on clinical evaluation and otoscopic examination. The pathophysiology involves disruption of the protective barrier of the ear canal, subsequent microbial colonisation, and host immune responses. Management strategies include both non-pharmacological measures, such as ear canal care (e.g. dry mopping) and moisture avoidance, as well as pharmacological interventions, including topical antimicrobial and anti-inflammatory agents. Systemic therapy is reserved for severe or complicated infections. Key challenges in the management of otitis externa include antimicrobial resistance, appropriate antimicrobial selection, patient adherence, and limited access to specialist care in resource-limited settings. Further research is required to optimise therapeutic strategies, strengthen antimicrobial stewardship, and improve clinical outcomes.

Keywords: otitis externa, swimmers' ear, pain, pharmacological, non-pharmacological

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Introduction

Otitis externa, commonly known as "swimmer's ear", is characterised by inflammation of the external auditory canal (EAC) and remains a common reason for otologic consultation worldwide.[1,2] It is particularly prevalent among swimmers and individuals exposed to frequent moisture or trauma to the ear canal.[1,3] Moisture retention within the ear canal creates an ideal environment conducive to microbial growth, increasing the risk of infection.[4,5] The condition may arise from infective or non-infective causes; however, it is commonly associated with bacterial or fungal pathogens.[1,6,7]

Epidemiology

Otitis externa is a common condition that affects individuals across all age groups; however, it occurs more frequently in children and young adults.[1,8] It is estimated that approximately 10% of individuals will develop the condition at some point in their lives, the majority of cases, approximately 95%, being acute.[1,5,6,9] The incidence varies geographically and seasonally, often increasing in warm and humid climates where water exposure is common.[1,6]

Aetiology

Bacterial infections, primarily caused by the microorganisms, *Pseudomonas aeruginosa* and *Staphylococcus aureus*, which thrive in the moist, warm environment of the canal are responsible for the majority of acute otitis externa cases, whilst in fungal otitis externa, microorganisms such as *Aspergillus* and *Candida* are frequently implicated, particularly in chronic or recurrent cases.[1,6,7,10,11] Routine laboratory tests and microbiological cultures are not required in uncomplicated cases of otitis externa; however, are recommended in recurrent, or treatment-resistant cases.[1,12]

Risk factors for otitis externa

Several factors increase susceptibility to infection of the EAC by disrupting the ear canal's natural protective barrier and facilitating microbial colonisation. Excessive moisture, commonly resulting from swimming, sweating, or prolonged water exposure, is a key risk factor, particularly in warm and humid environments, where moisture retention promotes microbial growth.[1,3,13] Trauma or mechanical irritation from cotton swabs, earphones, or hearing aids can damage the epithelium of the ear canal and compromise the cerumen barrier.[1,14] Dermatological conditions such as eczema or psoriasis further impair skin integrity, while narrow or stenotic external ear canals may trap moisture and debris, creating a favourable environment for infection.[1,9,15] Additional predisposing factors include obstruction within the ear canal, systemic stress, and immunocompromised states, including diabetes mellitus, and patients undergoing chemotherapy or radiotherapy, all of which weaken local and systemic defence mechanisms and increase vulnerability to infection.[1,8,16,17]

Anatomy and physiology of the external auditory canal

The external ear comprises the auricle (pinna) and the EAC (Figure 1).[18] The auricle is responsible for collecting sound waves and directing them into the EAC, which is a slightly curved, tubular structure.[18,19] The EAC extends from the concha of the auricle to the tympanic membrane (eardrum).[18,20] Its walls are made up of cartilage in the outer third and bone in the medial two-thirds, and are lined by skin containing hair follicles, sebaceous glands,

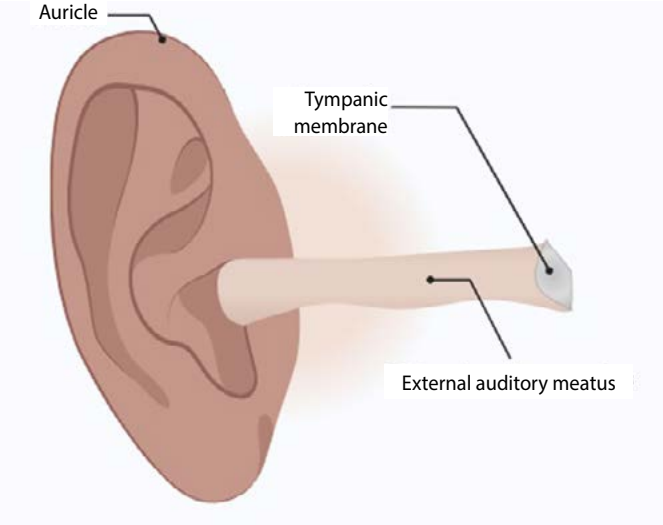

Figure 1: Anatomy of the external auditory canal (Template adapted in https://BioRender.com)

and ceruminous glands.[1,18] The main function of the external ear is to capture sound waves and direct them efficiently toward the tympanic membrane, aiding in hearing.[21] Furthermore, the EAC protects the middle and inner ear from foreign bodies and helps maintain an ideal acoustic environment.[18]

Several mechanisms protect the EAC from injury and infection. Cerumen (earwax), produced by the ceruminous and sebaceous glands, plays an important role in the EAC by trapping dust, debris, and microorganisms, thereby preventing their progression deeper into the ear canal.[22] Cerumen also possesses antimicrobial properties and helps maintain adequate hydration of the canal skin.[23] The presence of hair within the cartilaginous portion of the canal provides a further physical barrier against foreign material.[24] The slightly curved anatomy of the ear canal limits the direct entry of objects, while the epithelial lining undergoes a gradual outward migration, which facilitates the removal of desquamated cells and accumulated debris.[18] Together with reflexive protective mechanisms involving the ear canal and tympanic membrane, these features contribute to the ear's effective mechanical and biological defence system.

Disruption of the external auditory canal barrier

The EAC is protected by keratinised stratified squamous epithelium and cerumen produced from the ceruminous and sebaceous glands.[25] Once the ear's mechanical and biological defence mechanisms have been disrupted or the epithelial lining of the EAC is damaged, otitis externa may develop.[1] The acidic, lipid-rich cerumen, which contains lysozymes, assists in inhibiting fungal and bacterial growth.[4,26] Due to the hydrophobicity of cerumen, water is prevented from entering the canal and penetrating the skin.[4] Consistent cleaning of the ear canal removes the protective cerumen increasing water exposure.[4,27,28] Excessive moisture then leads to maceration, increasing the pH of the canal.[4] Cerumen buildup obstructs the canal leading to the retention of water and debris thereby promoting infection.[4,29] The EAC self-cleans through epithelial migration from the tympanic membrane to the EAC, carrying debris.[29,30-33] Dermatoses, such as eczema, seborrhoeic dermatitis, psoriasis, atopic dermatitis, and allergic dermatitis, as well as acquired or congenital immunodeficiency and diabetes mellitus may increase the risk of developing otitis externa.[4,30,34] Furthermore, the pH may be altered by the use of alkaline eardrops and soapy deposits.[27,28,35,36] Changes in the acidic environment, the lack of cerumen, a buildup of cerumen and trauma to the EAC can lead to a fungal or bacterial infection, resulting in immune and inflammatory responses.[5] Microtrauma from trying to self-clean the ears (cotton swabs, finger nails or other such objects that can scratch the canal) and wearing hearing aids can lead to abrasions within the EAC, permitting microbes into the wounded skin.[28,30,37]

Inflammatory and immune responses

Inflammatory and immune responses are initiated once the acidic environment within the ear has been lost and the protective barrier has been compromised.[1] When cerumen is removed from the ear, the acidic environment and protective barrier are lost.[1] Cerumen provides protection from infection and provides a barrier against foreign bodies by providing microbial protection

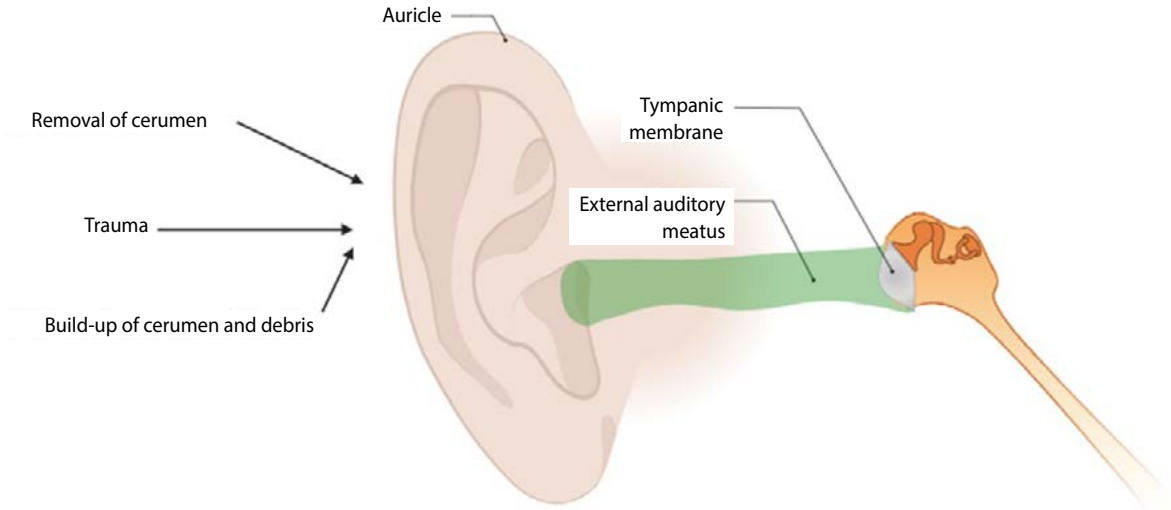

Figure 2: An overview of the pathogenesis of otitis externa. The removal of cerumen, trauma, and buildup of cerumen and debris all contribute to the growth of bacteria, resulting in otitis externa (Template adapted in https://BioRender.com).

to the epithelial lining of the EAC.[23,38] If the epithelial lining is damaged, local inflammation, the colonisation of pathogens and a decrease in the microbiota diversity may result.[39] The inflammatory response is initiated with pain, swelling, redness and warmth.[40] Sensory nociceptors distribute across the skin and upon injury secrete neuropeptides to induce a local inflammatory response including protein extravasation and vasodilation.[40,41] Keratinocytes, a cell type in the epidermis, form a passive physical barrier to pathogens and initiates primary inflammation once danger has been detected.[42] Additionally, keratinocytes respond to inflammatory mediators that are released by immune cells to maintain or amplify inflammation of the skin.[42]

Mechanism of pain and tissue damage

Pain and tissue damage in otitis externa can be attributed to inflammatory immune responses and otalgia.[1] Otalgia is a multifaceted condition involving cranial nerves V, VII, IX, and X and cervical plexus branches C2 and C3.[43] The tympanic membrane and ear canal receive sensory input from these nerves.[43] Cranial nerves IX and X, specifically contribute to otalgia through the larynx, oropharynx and distant organs.[43] Disease progression usually increases erythema and oedema and pain is further exacerbated due to pressure on the tragus and movement of the auricle.[29] Pain intensifies with an increase in severity of infection.[29] If there is obstruction of the canal lumen and adenopathy, auricular cellulitis or parotitis can occur.[4,29,44] Tissue damage may result in severe cases such as malignant otitis externa especially in immunocompromised and diabetic patients.[16] Tissue damage occurs by coagulative necrosis resulting from microangiopathy of the small blood vessels.[45]

Signs and symptoms of otitis externa

A clinical diagnosis of otitis externa requires a complete history and physical examination of the auricle, surrounding skin, and lymph nodes, as well as pneumatic otoscopy.[1,10] The clinical presentation may vary depending on the stage and severity of the condition, with symptoms often including otalgia (ear pain) (often disproportionate to the physical examination findings), pruritus (itching), erythema (redness), and otorrhoea (discharge), which may sometimes lead to hearing impairment and significantly affect the patient's quality of life.[1,29] Systemic symptoms occasionally include a fever accompanied by general malaise.[1] The condition is often classified according to severity into i) mild, characterised by pruritus, slight discomfort, and oedema of the ear canal, ii) moderate, with additional swelling, where the ear canal is partially occluded or blocked, and iii) severe, characterised by complete occlusion or blockage of the ear canal as a result of oedema accompanied by intense pain, swollen or enlarged lymph nodes, and a fever.[1]

Classification of otitis externa

Otitis externa is classified into various categories (Table I) based on the duration of symptoms, extent of inflammation, underlying aetiology (bacterial, fungal, or dermatological), and spread of infection. This classification system assists healthcare professionals in diagnosing the condition, distinguishing between its different clinical forms, and guiding appropriate management and treatment strategies.

Table I: Classification and description of otitis externa

Classification	Description
Acute diffuse otitis externa[1,8,46]	• Most common form of otitis externa • Acute and short-lived, typically lasting days to weeks • Usually bacterial in origin • Involves diffuse inflammation of the entire external ear canal • Frequently associated with swimming and prolonged moisture exposure • Commonly referred to as "swimmer's ear"
Acute localised otitis externa (furunculosis)[8,13,47]	• Localised infection of a hair follicle (furunculosis) • Typically bacterial in origin • Presents as a boil within the external ear canal • Causes severe, localised ear pain • Pain is often exacerbated by movement of the pinna or jaw
Chronic otitis externa[1,8,13]	• Similar in presentation to acute diffuse otitis externa • Persists for more than 3 months despite treatment • Itching is often seen as the predominant symptom
Malignant (necrotising) otitis externa[8,13,48]	• Severe, progressive infection originating in the EAC • Extends into adjacent soft tissues and bone (skull base osteomyelitis) • Occurs predominantly in immunocompromised adults, particularly those with diabetes mellitus • Associated with significant morbidity and requires urgent, aggressive management
Eczematous otitis externa[8,13,49]	• Associated with underlying dermatological or allergic conditions • Itching and inflammation are common symptoms
Otomycosis[8,13,50]	• Fungal infection of the EAC • Most commonly caused by *Aspergillus* or *Candida* species • Characterised by itching, discomfort, and debris within the ear canal

Complications of otitis externa

The most frequent complications of otitis externa include malignant otitis externa and periauricular cellulitis.[13] Additional complications include myringitis, perichondritis, facial cellulitis, and osteomyelitis of the temporal bone.[1,13]

Diagnostic criteria

Otitis externa is diagnosed based on the symptoms of inflammation present in the EAC.[8] Symptoms usually develop within 48 hours and last up to three weeks; and symptoms of ear canal inflammation include itching, ear pain and a sense of

fullness, which may be with or without hearing loss or jaw pain; and signs of ear canal inflammation include tenderness of the tragus or pinna with movement or erythema or oedema of the ear canal, with or without tympanic membrane erythema, otorrhoea, local lymphadenitis or cellulitis of the pinna.[8,10,27] Evaluation includes a history of presenting and associated symptoms, absence of cerumen, local trauma, water exposure, diabetes, local radiotherapy, ear surgeries and inflammatory skin disorders.[10] Physical examinations include an otoscopy of the ear canal, examination of the auricle and surrounding lymph nodes, skin examination and verification of an intact tympanic membrane via pneumatic otoscopy to differentiate between otitis externa from otitis media as well as determining if the tympanic membrane has been perforated as this affects treatment options.[8,10] If the tympanic membrane is not visible, hearing screening tests or audiological examinations can be performed while tone threshold audiogram and tuning-fork examinations are performed to test for conductive hearing loss.[6] Secretions may be swabbed for cultures and undergo pathogen resistance testing.[6] Patient presentation can range from itching with mild discomfort and minimal oedema to complete obstruction of the EAC usually involving the pinna and adjacent skin and severe pain.[10] A mild fever may occur, but temperatures greater than 38.3 °C (101 °F) suggest the spread of infection beyond the EAC.[10] Pain correlates to the severity of disease.[10,51]

Management and treatment of otitis externa

Effective management of otitis externa requires a multifaceted approach that combines both non-pharmacological and pharmacological interventions. A thorough understanding of the aetiology, risk factors, and pathophysiology of this condition is essential for accurate diagnosis, effective treatment, and prevention of complications. The main goals of treatment for otitis externa include treating the infection, pain management, returning the skin of the ear canal to its normal state and promoting cerumen production.[5,6] The use of aural toilet, topical antibiotics and steroids support treatment goals.[5] Topical treatment aims to reduce the chronic inflammation associated with otitis externa.[6]

Non-pharmacological interventions

Otitis externa may result from a build-up of debris in the ear canal, and removal of this debris may relieve symptoms. The EAC becomes more susceptible to trauma as a result of the inflammation and as a result curettes and cerumen spoons should not be used.[4] Suctioning under direct visualisation is the best form of cleansing.[4] The aural toilet is recommended when dry mopping or gentle suctioning is performed under microscopic or otoscopic visualisation.[10] This removes any debris or material obstructing the ear canal.[10] A lavage should be used only when the tympanic membrane is known to be intact, and should be avoided on patients with diabetes.[10] An operating or open otoscope paired with a 5 or 7 Fr Frazier malleable suction tip at low suction works best.[4] Cotton swabs with the cotton fluffed out can be used to clean the EAC.[4] The EAC can swell and the use of a cotton wick can be used for drainage and assist topical applications.[4] Tympanic membranes that are red and visible should be assessed via a tympanometry or pneumatoscope, to assist with determining if associated otitis externa is present.[4]

Pharmacological management

Initial therapy for diffuse, uncomplicated acute otitis externa should be with topical preparations due to their efficacy and safety when compared to placebo.[6,46] Treatment is appropriate in patients without middle ear disease, abscess formation, osteitis or recurring infection.[28] Topical treatments are administered between 7–10 days.[8]

Topical antibiotics

Topical antibiotics such as eardrops treat *Pseudomonas aeruginosa* and *Staphylococcus aureus*, the most common pathogens associated with otitis externa.[6] They are considered to be first-line therapy as they are effective and inexpensive.[10] It is important to consider resistance and culture results, though routine cultures are usually reserved for refractory or complicated cases.[6] As the drugs are administered topically, high local concentrations of drugs can be achieved without the side-effects associated with systemic treatment.[6] Quinolones (ciprofloxacin) are highly effective and do not cause local irritation; however, prolonged treatment can lead to resistance.[6] Preparations of fluoroquinolones require twice-daily dosing and some may be used with a non-intact tympanic membrane.[29] Ofloxacin and ciprofloxacin/dexamethasone can be used if the tympanic membrane is not intact as it has been approved for use in the middle ear.[10] Aminoglycosides (neomycin and gentamycin), while effective, can be ototoxic and should only be administered if the eardrum is intact.[6,29] Neomycin is sensitising in 5–18% of patients and can lead to contact dermatitis.[29, 52-56] The ototoxicity associated with this group has been linked to open middle-ear spaces or prolonged use.[29] Polymyxins (polymyxin B) can also be used.[6] These drugs result in lower recurrence and rapid symptomatic relief of otitis externa compared with placebo.[6,27] Topical antibiotics should not be used for longer than necessary, and ototoxic drugs should not be administered if there is perforation of the eardrum.[6]

Topical corticosteroids and combination therapy

Corticosteroids are administered to lessen oedema and may have minor antimicrobial and antifungal effects, but their main role is anti-inflammatory.[6,57] High-potency corticosteroids are more effective against swelling, inflammation and pain than low-potency corticosteroids.[6,58] Corticosteroid-containing preparations provide rapid symptomatic relief.[10] Corticosteroid use alone lacks strong evidence of effectiveness, but is beneficial when combined with antibacterial agents.[9] Combinations of topical antibiotics and topical corticosteroids reduce swelling, secretions and erythema better than topical antibiotics alone.[6]

Antifungal agents

Otitis externa caused by fungal infections is usually mild and a 2% acetic acid or a 90–95% alcohol solution can be used.[29] Established infections require topical treatment such as 1% clotrimazole or tolnaftate.[4,29,59,60] Fungal otitis externa can be treated by soaking strips in an antifungal drug such as miconazole, nystatin, clotrimazole or ciclopirox, and placing them in the ear canal.[6] Systemic therapy may be considered if topical therapy is unsafe due to perforation, but not solely based on resistance testing in routine fungal otitis externa, since fungal cultures are not routinely done.[6]

Analgesic and adjunctive therapies

Pain management is one of the main goals in the management of otitis externa. Ear pain usually results from inflammation involving the sensitive periosteum of the bony ear canal.[6] The use of analgesics is recommended to give patients rest, comfort and to allow them to resume their daily activities.[28] Mild to moderate pain can be treated with nonsteroidal anti-inflammatory drugs (NSAIDs) such as ibuprofen or acetaminophen/paracetamol.[6,28] Topical local anaesthetics can be used if the tympanic membrane is intact or if a myringotomy tube has been inserted.[6] However, topical local anaesthesia can mask progression of infection and should be reviewed after 48 hours so that the effect of treatment can be evaluated.[6,28] Opioids may be given when symptoms become severe.[10]

Systemic therapy

Topical therapy should be supported by systemic antibiotics if the infection has spread beyond the EAC into the pinna, into deeper tissues causing necrotising otitis externa, when otitis media is present or if the patient has diabetes, human immunodeficiency virus (HIV) or acquired immunodeficiency syndrome (AIDS).[4,28] It is important to note that oral antibiotics are associated with significant adverse effects including diarrhoea, altered nasopharyngeal flora, vomiting, allergic reactions and rashes.[28,35,61,62] Systemic antibiotic therapy also increases the risk of pathogen resistance and disease recurrence.[10]

Management of malignant otitis externa

Malignant otitis externa is an aggressive infection, and management of this condition has been challenging due to the need for long-term therapy and consistent monitoring.[63,64] Additionally, there is a lack of a standardised care protocol.[63] Management includes assessment of clinical signs and symptoms, evaluation of laboratory assays for inflammatory markers, and radiological imaging.[63] Treatment is based on oral antibiotics and pathogen-specific parenteral antibiotics administered for 4–6 weeks, guided by sensitivity and resistance testing.[6,65] If sensitivity and resistance testing does not yield definitive results, empirical antibiotic therapy targeting *Pseudomonas aeruginosa* should be initiated, depending on the severity of the disease.[6] Adjunctive, topical antimicrobial therapy using antimicrobial medicated strips placed in the ear canal can be administered.[6] Surgical removal of bone sequestra and necrotic tissue may be required and strict glycaemic control should be maintained.[6] Hyperbaric oxygen therapy may improve cure rates; however; regular radiological and clinical follow-ups are necessary to monitor treatment response and detect recurrence.[6,60] In cases where malignant otitis externa is diagnosed at an advanced stage, intravenous antibiotic therapy is required.[63]

Conclusion

Otitis externa is a common, potentially complex inflammatory condition of the external ear. Although the majority of cases are acute, the condition encompasses a wide clinical spectrum, ranging from mild, self-limiting disease to severe, life-threatening forms. Management is focused on topical therapy, supported by non-pharmacological interventions such as aural toilet, moisture avoidance, and patient education. Topical antimicrobials, often in combination with corticosteroids, are highly effective for uncomplicated disease, whereas systemic therapy is reserved for severe, complicated, or refractory cases. Further research is required to refine therapeutic approaches, address emerging resistance patterns, and develop standardised protocols for the management of this condition.

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