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Malignant Otitis Externa: A Comprehensive Review

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ABSTRACT

Malignant otitis externa (MOE) is a severe, potentially life-threatening infection of the external auditory canal, most commonly affecting elderly individuals with diabetes mellitus or immunocompromised states. Despite its name, MOE is not a neoplastic process but a progressive and invasive infection, often caused by Pseudomonas aeruginosa. It may extend to involve the skull base and cranial nerves, leading to severe morbidity and mortality. This review discusses the current understanding of the disease's epidemiology, anatomy, pathophysiology, clinical presentation, diagnostic strategies, treatment modalities, and long-term implications. Emphasis is placed on evidence-based approaches, diagnostic imaging advances, emerging antimicrobial resistance, and multidisciplinary management.

Keywords: Malignant Otitis Externa, Skull Base Osteomyelitis, Pseudomonas aeruginosa, Cranial Nerve Palsy, Diabetes Mellitus, Antimicrobial Resistance.

INTRODUCTION

1. Introduction

Malignant otitis externa (MOE), also known as necrotizing external otitis, is an aggressive and potentially fatal infection that primarily involves the external auditory canal and may extend to the skull base. It was first described by Chandler in 1968 [1]. The condition is misnamed, as it is not a malignancy but is termed 'malignant' due to its aggressive behavior and tendency to invade adjacent structures. MOE predominantly affects elderly diabetic patients, but it has also been reported in immunocompromised individuals and occasionally in immunocompetent patients [2].

2. Epidemiology

The incidence of MOE is increasing, largely due to the growing global prevalence of diabetes mellitus and chronic immunosuppressive conditions. MOE primarily affects men over the age of 60 years, with diabetes mellitus being the strongest predisposing factor [3]. It is estimated that over 90% of MOE cases occur in diabetic patients [4]. Other risk factors include malignancy, chemotherapy, HIV infection, and chronic steroid use.

3. Anatomy and Pathophysiology

The external auditory canal (EAC) is composed of an outer cartilaginous portion and an inner bony portion. The epithelium lining the canal is continuous with the skin of the auricle and contains ceruminous and sebaceous glands. In MOE, pathogens breach the epithelial barrier and spread through the fissures of Santorini and foramen of Huschke, leading to involvement of the temporal bone and skull base [5]. Pseudomonas aeruginosa is the most common causative organism due to its ability to thrive in moist environments and its intrinsic resistance mechanisms [6].

4. Clinical Presentation

The hallmark symptoms of MOE include severe otalgia, often out of proportion to physical findings, and persistent otorrhea. Otalgia typically worsens at night and may radiate to the temporomandibular joint. On examination, granulation tissue may be observed at the bony-cartilaginous junction of the EAC. In advanced stages, cranial nerve involvement, particularly facial nerve palsy (CN VII), may occur [7].

5. Diagnosis and Investigations

Diagnosis of MOE is primarily clinical, supported by laboratory markers, imaging, and microbiological evidence. Elevated erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) are nonspecific but useful for monitoring. Definitive diagnosis requires imaging. High-resolution CT shows bone erosion, while MRI better delineates soft tissue involvement. Radionuclide scans such as technetium-99m or gallium-67 can track disease activity [8]. Microbial

cultures from aural discharge guide antimicrobial therapy, though deep tissue biopsy may be needed in refractory cases [9].

6. Staging and Imaging Findings

Staging is based on disease extension. Stage I is limited to the external auditory canal. Stage II involves bone erosion. Stage III includes cranial nerve palsy, and Stage IV denotes intracranial spread [10]. Imaging is crucial: CT detects bony involvement, MRI is best for soft tissue, and gallium scans are ideal for monitoring treatment response. Recent advancements in PET-CT offer improved sensitivity [11].

7. Treatment Strategies

Table 2: Medical and Adjunctive Therapies for MOE

Treatment is multimodal. Empirical antipseudomonal antibiotics like ciprofloxacin or ceftazidime are started, then tailored to culture. Fungal MOE (e.g., Aspergillus) needs antifungals like voriconazole. Antibiotics may be required for 6–12 weeks. Hyperbaric oxygen therapy (HBOT) may aid refractory cases by enhancing oxygenation and immune response [12]. Surgery is reserved for abscess drainage or necrotic bone removal [13].

8. Complications and Prognosis

MOE may lead to skull base osteomyelitis, cranial neuropathies, thrombophlebitis, meningitis, or brain abscess. Facial nerve involvement occurs in up to 50% of cases and is a predictor of poor outcome. Mortality is high in delayed or inadequately treated cases, but early aggressive therapy improves prognosis [14].

9. Clinical Implications

Clinicians should maintain a high index of suspicion in elderly diabetic patients with refractory otitis externa. Early imaging, deep cultures, and prolonged antimicrobial therapy are critical. Multidisciplinary teams including ENT, radiology, infectious diseases, and neurology improve outcomes [15].

10. Discussion and Conclusion

MOE represents a diagnostic and therapeutic challenge. Timely diagnosis with appropriate imaging and aggressive prolonged treatment are paramount. Emerging pathogens, antimicrobial resistance, and atypical presentations necessitate vigilant follow-up. Future directions include biomarker development, standardized treatment algorithms, and further validation of prognostic models. Personalized therapy based on immune status, culture, and imaging is the cornerstone for improved patient outcomes [16].

11. Differential Diagnosis

Differential diagnoses of MOE include chronic suppurative otitis media, cholesteatoma, neoplasms of the external auditory canal, tuberculous otitis, and fungal infections. Differentiation is essential because these conditions may mimic the symptoms of MOE, including persistent otorrhea and granulation tissue. Imaging and microbiology help distinguish MOE from these conditions [17].

12. Risk Factors and Comorbidities

Apart from diabetes mellitus, other risk factors include chronic renal failure, malnutrition, prolonged corticosteroid use, and HIV/AIDS. These conditions impair immune function and predispose individuals to chronic infections. Poor glycemic control has been directly linked with poor prognosis in MOE patients [18].

13. Prognostic Indicators

Several indicators predict poor outcomes in MOE: presence of cranial neuropathies, fungal etiology, intracranial extension, immunosuppressive therapy, and delayed initiation of treatment. Elevated inflammatory markers and persistent symptoms beyond 6 weeks are associated with increased morbidity [19].

Table 1: Staging of Malignant Otitis Externa

Stage	Extent	Clinical Features
Stage I	External auditory canal	No bony erosion
Stage II	Bone erosion of skull base	Confirmed via CT
Stage III	Cranial nerve involvement	Facial nerve palsy common
Stage IV	Intracranial spread	Meningitis, abscess, vascular involvement
Therapy	Agents	Notes
Antibiotics	Ciprofloxacin, Ceftazidime	6–12 weeks; guided by culture
Antifungals	Voriconazole, Amphotericin B	For Aspergillus and resistant fungi
Adjuncts	Hyperbaric oxygen therapy	Useful in refractory cases
Surgery	Debridement, mastoidectomy	Only if abscess or necrosis

Table 2 : Differential Diagnosis of MOE

Condition	Distinguishing Features
Chronic Suppurative Otitis Media	Middle ear involvement, tympanic membrane perforation
Cholesteatoma	Erosion of ossicles, presence of keratin debris
Carcinoma of EAC	Biopsy shows malignancy, non-responsive to antibiotics
Tuberculous Otitis	Painless otorrhea, multiple tympanic membrane perforations
Fungal Otitis	Black or white fungal debris, less aggressive course

Table 3 : Prognostic Indicators of Poor Outcome in MOE

Indicator	Implication
Cranial nerve involvement	Increased risk of complications and mortality
Fungal infection	Poor response to antibacterial therapy
Delayed diagnosis	Higher risk of bone and intracranial spread
Immunosuppression	Reduced host defense against pathogens
Persistent inflammation	May indicate treatment failure or resistance

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