Malignant Otitis Externa: Contemporary Approach to Diagnosis and Management

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ABSTRACT

Malignant otitis externa (MOE), also known as necrotizing otitis externa or skull base osteomyelitis, is an aggressive infection originating in the external auditory canal that can extend to the temporal bone and skull base. It predominantly affects older adults with diabetes mellitus or other immunocompromising conditions. Severe persistent otalgia, particularly nocturnal pain disproportionate to local findings, otorrhea, and granulation tissue in the external auditory canal are important clinical warning signs. Pseudomonas aeruginosa remains the most frequently isolated pathogen, although fungal and multidrug-resistant infections are increasingly recognized. Diagnosis requires a combination of clinical assessment, microbiological evaluation, inflammatory markers, and imaging. High-resolution computed tomography is useful for evaluating bony destruction, whereas magnetic resonance imaging provides superior assessment of soft-tissue, skull-base, intracranial, and cranial nerve involvement. Nuclear medicine imaging and FDG-PET/CT may provide additional information regarding disease activity and treatment response. Prolonged culture-directed antimicrobial therapy, meticulous glycemic control, local ear care, and monitoring of disease activity remain the cornerstones of management. Surgery is reserved for selected indications, including diagnostic biopsy, abscess drainage, sequestrated bone, and refractory disease. Cranial neuropathy, extensive skull-base involvement, poorly controlled diabetes, fungal infection, and delayed diagnosis are associated with poorer outcomes. Early recognition and multidisciplinary management are essential to reduce morbidity and mortality.

KEY WORDS

Cranial neuropathy, Diabetes mellitus, Malignant otitis externa, Necrotizing otitis externa, Skull base osteomyelitis

INTRODUCTION

Malignant otitis externa (MOE) is a potentially life-threatening infection that originates in the external auditory canal (EAC) and extends to the temporal bone and skull base. Despite its name, MOE is not a neoplastic disorder. The term “malignant” refers to its historically aggressive clinical course and high mortality. Chandler popularized the term in 1968 after describing progressive external ear infection complicated by skull base osteomyelitis in patients with diabetes mellitus.[1]

MOE predominantly affects elderly individuals with diabetes mellitus and other conditions associated with impaired host immunity. The introduction of effective antipseudomonal antimicrobial therapy has dramatically improved survival; however, delayed diagnosis, extensive skull-base involvement, cranial neuropathy, intracranial complications, antimicrobial resistance, and recurrence remain important clinical challenges.[2,3]

The disease is particularly important for otolaryngologists because its early manifestations may resemble uncomplicated otitis externa. Persistent severe otalgia despite appropriate local treatment should therefore prompt reassessment for skull-base involvement, particularly in elderly patients with diabetes.

This review summarizes the current understanding of the epidemiology, pathogenesis, clinical presentation, diagnostic evaluation, imaging, treatment, monitoring,

and prognostic factors of MOE, with emphasis on practical clinical decision-making.

Epidemiology and risk factors

MOE is relatively uncommon but occurs predominantly among older adults. Diabetes mellitus is the most important recognized risk factor and has been reported in a large majority of affected patients.[2,4] The increasing prevalence of diabetes and an aging population may contribute to the continuing clinical relevance of this disease.

Diabetes predisposes patients to invasive infection through several mechanisms, including microangiopathy, impaired neutrophil function, reduced tissue perfusion, and altered host immune responses.[2,5]

Other recognized risk factors include:

- Advanced age and immunosenescence
- Chronic renal failure
- Hematological malignancies
- Chemotherapy
- Organ transplantation
- HIV infection
- Long-term corticosteroid therapy
- Other causes of immunosuppression

Although diabetes remains the dominant risk factor, MOE should not be considered exclusively a disease of diabetic patients. Clinicians should maintain suspicion in immunocompromised individuals who present with persistent or unusually severe external ear infection.

Etiopathogenesis and pathways of spread

Pseudomonas aeruginosa has historically been the predominant pathogen in MOE and accounts for the majority of bacterial cases.[2,6] Other bacterial organisms, including Staphylococcus aureus, Klebsiella pneumoniae, Proteus mirabilis, and Enterococcus species, have also been reported. Increasing antimicrobial resistance has made microbiological confirmation and susceptibility testing increasingly important.

Fungal MOE, particularly caused by Aspergillus and Candida species, is increasingly recognized, particularly among immunocompromised patients and those who fail to respond to appropriate antibacterial therapy.[7]

The infection initially involves the soft tissues of the external auditory canal and may extend through the osteocartilaginous junction and fissures of Santorini. From there, infection can spread to the temporal bone and skull base.[8,9]

The anatomical pathways of spread can be broadly categorized as

Anterior spread: involving the temporomandibular joint, parotid region, muscles of mastication, temporal fossa, facial nerve, and stylomastoid foramen.

Medial spread: extending toward the parapharyngeal space, petrous apex, clivus, sphenoid bone, jugular foramen, and lower cranial nerves.

Posterior spread: involving the mastoid region.

Intracranial spread: extending to the dura, cavernous sinus, sigmoid sinus, jugular bulb, internal carotid artery, or jugular vein.[8-10]

The facial nerve is particularly vulnerable because of its anatomical relationship with the temporal bone and stylomastoid foramen. Further medial extension may result in involvement of cranial nerves IX–XII.[15]

Clinical presentation

The classical clinical presentation consists of severe persistent otalgia and otorrhea. The pain is frequently disproportionate to the apparent severity of the external ear findings and characteristically becomes worse at night.[2]

Common clinical findings include:

- Severe persistent otalgia
- Nocturnal pain
- Purulent otorrhea
- Hearing loss
- Aural fullness
- Pruritus
- External auditory canal edema
- Granulation tissue

Granulation tissue at the junction between the bony and cartilaginous portions of the EAC is a particularly important clinical finding and should raise strong suspicion for MOE.[2,14]

The clinical progression of disease is important. Persistent symptoms despite appropriate treatment for presumed otitis externa should trigger investigation for skull-base osteomyelitis.

Cranial neuropathy

Cranial neuropathy is an important marker of advanced disease. The facial nerve is the cranial nerve most commonly involved, followed by the lower cranial nerves in more extensive skull-base disease.[15]

Facial nerve palsy should therefore be considered a marker of disease extension rather than simply an associated facial neuritis. Multiple cranial neuropathies generally indicate

more extensive skull-base involvement and are associated with poorer outcomes.

Advanced disease may also produce intracranial complications, including meningitis, brain abscess, dural venous sinus thrombosis, and other forms of intracranial extension.

Diagnostic evaluation

Diagnosis of MOE requires integration of clinical findings, laboratory investigations, microbiology, and radiological assessment. No single investigation should be interpreted in isolation.

Laboratory investigations

Routine hematological parameters may be normal or only mildly abnormal.[2] Important investigations include:

- ESR
- CRP
- Blood glucose
- HbA1c
- Renal function tests
- Complete blood count where clinically indicated

ESR and CRP are particularly useful because they can provide objective measures of inflammatory activity and may assist in monitoring treatment response.[2]

Assessment of renal function is important when prolonged antimicrobial therapy is anticipated because many commonly used agents require dose adjustment according to renal function.

Microbiological Assessment

Ear discharge should ideally be cultured before antimicrobial therapy is initiated. However, superficial cultures may not always accurately reflect the causative organism in deep skull-base infection.

In refractory, atypical, culture-negative, or suspicious cases, deep tissue sampling and biopsy should be considered.[2]

Biopsy has two important roles

1. Identification of the causative organism.

2. Exclusion of external auditory canal malignancy.

The distinction between MOE and well-differentiated squamous cell carcinoma may occasionally be difficult because reactive epithelial changes and pseudoepitheliomatous hyperplasia can occur in infected tissue.[11-13]

Imaging

Imaging is central to establishing the extent of disease and identifying skull-base and intracranial involvement.

High-Resolution Computed Tomography

High-resolution CT of the temporal bone is particularly useful for demonstrating:

- Bony erosion
- Cortical destruction
- Temporal bone involvement
- Mastoid involvement
- Adjacent soft-tissue changes

CT remains an important initial imaging modality for assessment of osseous disease.[2,9]

However, bony changes may persist after clinical resolution and therefore CT findings should not be interpreted as the sole indicator of active infection.

Magnetic resonance imaging

MRI provides superior soft-tissue characterization and is particularly valuable in advanced disease. It can demonstrate:

- Skull-base involvement
- Soft-tissue extension
- Dural enhancement
- Intracranial extension
- Cranial nerve involvement
- Involvement of adjacent deep spaces

MRI is therefore complementary to CT rather than a replacement for it.[9,10,16]

In patients with cranial neuropathy, severe persistent symptoms, or suspected intracranial extension, contrast-enhanced MRI is particularly important.

Nuclear Medicine Imaging

Tc-99m bone scintigraphy is sensitive for detecting skull-base osteomyelitis and may be useful in establishing the diagnosis. However, increased tracer uptake may persist for months because of continuing bone remodeling and therefore limits its usefulness for monitoring treatment response.[17,18]

Gallium-67 scintigraphy more closely reflects inflammatory activity and may be useful in monitoring response. A reduction in tracer uptake generally correlates with reduction in active inflammation.[17,18]

In-111-labelled leukocyte imaging has also been used to evaluate inflammatory activity, although persistent post-infectious inflammation may result in false-positive findings.[19]

FDG-PET/CT

FDG-PET/CT provides both metabolic and anatomical information and has emerged as a potentially useful modality for evaluating disease activity and treatment response.[20] Its role is particularly attractive when conventional imaging findings remain difficult to interpret.

However, the evidence base remains evolving, and PET/CT should currently be regarded as an adjunct rather than an obligatory investigation for every patient.

Diagnostic criteria and disease extent

The diagnostic criteria proposed by Cohen and Friedman remain widely referenced.[21]

Major criteria include

- Severe otalgia
- Otorrhea
- External auditory canal edema
- Granulation tissue
- Positive bone scan
- Failure of local treatment

Minor criteria include

- Diabetes mellitus
- Cranial nerve involvement
- Advanced age
- Positive radiological findings

Although these criteria remain useful clinically, modern imaging and microbiological techniques have expanded the diagnostic approach.

Carney Clinicopathological Staging

The Carney system provides a useful framework for describing disease extent:

Stage I: Soft-tissue involvement extending beyond the EAC without evidence of bony involvement.

Stage II: Soft-tissue extension beyond the EAC with evidence of skull-base osteomyelitis.

Stage III: Skull-base osteomyelitis with cranial nerve involvement.

- Stage IIIA: Single cranial nerve palsy
- Stage IIIB: Multiple cranial nerve palsies

Stage IV: Intracranial complications such as meningitis, dural venous sinus thrombosis, intracranial empyema, or brain abscess.[22]

The presence of cranial neuropathy should therefore prompt careful evaluation for extensive skull-base disease.

Management

Successful management requires prolonged antimicrobial therapy combined with control of underlying risk factors and careful monitoring.

Antimicrobial Therapy

The cornerstone of treatment is systemic antimicrobial therapy directed against the identified pathogen. Pseudomonas aeruginosa remains the principal bacterial pathogen, although increasing antimicrobial resistance necessitates culture-directed therapy.[2,23]

Antipseudomonal agents used in clinical practice include

- Ciprofloxacin
- Ceftazidime
- Cefepime
- Piperacillin-tazobactam
- Meropenem in selected resistant infections

The choice of antimicrobial agent should be guided by culture and susceptibility results whenever available.

Oral ciprofloxacin historically transformed the treatment of MOE because of its antipseudomonal activity and bone penetration. However, increasing fluoroquinolone resistance has reduced its reliability as empirical monotherapy in some settings.[2,23]

Treatment duration is commonly **6–12 weeks or longer**, depending on clinical response, inflammatory markers, microbiological findings, and the extent of disease.

There is no universal fixed treatment duration applicable to all patients. Therapy should be individualized and continued until there is convincing evidence of clinical and biochemical resolution, with imaging interpreted in the appropriate clinical context.

Glycemic Control

Optimal diabetic control is an essential component of treatment. Hyperglycemia adversely affects host defense mechanisms and tissue perfusion and may contribute to treatment failure.

Blood glucose should therefore be monitored closely, and patients with poor glycemic control may require temporary insulin-based management.

Local Treatment

Local management includes

- Regular aural toilet
- Removal of debris where appropriate
- Appropriate topical antimicrobial therapy
- Management of canal edema
- Adequate analgesia

Pain control is particularly important because severe nocturnal otalgia is a characteristic feature of active disease.

Fungal malignant otitis externa

Fungal MOE should be considered when

• The patient is immunocompromised.

• Bacterial cultures are negative.

• There is inadequate response to appropriate antibacterial therapy.

• Fungal elements are identified histologically or microbiologically.

Aspergillus and Candida species are among the recognized pathogens.[7]

Treatment should be guided by fungal identification and susceptibility testing. Depending on the organism and clinical circumstances, voriconazole, amphotericin B, or posaconazole may be considered.[7]

Fungal infection should not be assumed solely because a patient fails to respond to antibiotics; repeat microbiological assessment and tissue diagnosis are important in refractory disease.

Role of surgery

The role of surgery has diminished with the availability of effective antimicrobial therapy. Extensive debridement is generally avoided because aggressive surgery may result in additional tissue damage and potentially facilitate further spread of infection.[2,23]

Surgical intervention remains appropriate in selected circumstances, including

• Diagnostic biopsy

• Drainage of abscesses

• Removal of sequestrated or necrotic bone

• Management of complications

• Failure of appropriately administered medical therapy

Surgery should therefore complement rather than replace appropriate systemic antimicrobial treatment.

Hyperbaric oxygen therapy

Hyperbaric oxygen therapy (HBOT) has been proposed as an adjunctive treatment for refractory MOE. Potential mechanisms include improved tissue oxygenation, enhancement of leukocyte function, and promotion of angiogenesis.[14]

However, evidence supporting routine HBOT remains limited. It may be considered in selected refractory cases where appropriate facilities and expertise are available, but it should not delay adequate antimicrobial therapy or definitive investigation of persistent disease.

Monitoring response to treatment

Clinical response remains central to assessing treatment effectiveness.

Important parameters include

• Reduction in otalgia

• Resolution of otorrhea

• Improvement in canal granulation and edema

• Recovery or stabilization of cranial nerve function

• Reduction in ESR and CRP

• Improvement in glycemic control

• Microbiological response where appropriate

Serial ESR and CRP measurements can be particularly useful during prolonged treatment.[2]

Radiological findings must be interpreted cautiously. Persistent bone abnormalities on CT or uptake on bone scintigraphy may reflect residual remodeling rather than active infection. Therefore, clinical status and inflammatory markers should be integrated with imaging rather than relying on a single radiological parameter.

FDG-PET/CT and gallium imaging may provide additional information regarding persistent metabolic or inflammatory activity in selected patients.[17,20]

Prognosis and recurrence

The prognosis of MOE has improved substantially since the introduction of effective antipseudomonal therapy.[2] Nevertheless, morbidity remains significant, particularly in patients with advanced skull-base involvement.

Factors associated with poorer outcomes include

• Advanced age

• Poorly controlled diabetes

• Cranial neuropathy

• Multiple cranial nerve involvement

• Extensive skull-base osteomyelitis

• Delayed diagnosis

• Fungal infection

• Antimicrobial resistance

• Intracranial complications

Cranial neuropathy is particularly important because it indicates extension beyond the external auditory canal.[15]

Recurrence remains a significant clinical problem and emphasizes the need for adequate treatment duration and follow-up. Patients should be monitored clinically and, when appropriate, with serial inflammatory markers and targeted imaging.

Current challenges and future directions

Despite advances in diagnosis and treatment, several challenges remain.

First, early recognition continues to be difficult because the initial clinical presentation may resemble uncomplicated otitis externa. Persistent severe nocturnal pain in an elderly diabetic patient should therefore be regarded as a major diagnostic warning sign.

Second, antimicrobial resistance is increasingly important. Reliance on empirical fluoroquinolone therapy without microbiological confirmation may be problematic in regions with high antimicrobial resistance. Culture-directed therapy should be prioritized whenever feasible.

Third, fungal MOE is increasingly recognized, particularly in immunocompromised patients and those with refractory disease.[7]

Fourth, monitoring treatment response remains challenging because radiological abnormalities may persist after clinical resolution. Advanced imaging modalities, including FDG-PET/CT, may improve assessment of disease activity, although further evidence is needed to define their optimal role.[20]

Finally, management of MOE is best undertaken through a multidisciplinary approach involving otolaryngologists, infectious disease physicians, radiologists, and clinicians managing diabetes and other underlying systemic conditions.

CONCLUSION

Malignant otitis externa is an aggressive skull-base infection that predominantly affects elderly and diabetic patients. The combination of severe persistent or nocturnal otalgia, otorrhea, and granulation tissue in the external auditory canal should prompt early consideration of the diagnosis.

The disease should be regarded as a spectrum of infection extending from the external auditory canal to the temporal bone and skull base, with cranial neuropathy and intracranial complications representing advanced disease. CT and MRI provide complementary information regarding osseous and soft-tissue involvement, while microbiological sampling is important for targeted antimicrobial therapy and identification of resistant or fungal pathogens.

Practical clinical approach

A practical approach to a patient with suspected MOE can be summarized as follows:

Elderly/diabetic patient with severe persistent otalgia ± otorrhea

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Examine the EAC for edema and granulation tissue

↓

Obtain microbiological samples and assess ESR/CRP, glucose and HbA1c

↓

HRCT temporal bone ± contrast-enhanced MRI depending on suspected extent

↓

Assess for skull-base osteomyelitis and cranial neuropathy

↓

Start appropriate systemic antipseudomonal therapy

↓

Modify therapy according to culture and susceptibility

↓

Optimize glycemic control + aural toilet + analgesia

↓

Monitor clinical symptoms and inflammatory markers

↓

If poor response: reassess diagnosis, repeat culture/ biopsy, evaluate for fungal disease, resistant organisms, malignancy, abscess or intracranial extension

↓

Consider selected surgical intervention or adjunctive HBOT in refractory/complicated disease

Prolonged culture-directed systemic antimicrobial therapy, strict glycemic control, local ear care, adequate analgesia, and close monitoring remain the foundations of treatment. Surgery has a selective role, while HBOT may be considered in refractory disease. Early recognition and multidisciplinary management are essential to reduce cranial neuropathy, intracranial complications, recurrence, and mortality.

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