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Case Report

Unexpected Remission of Facial Palsy Complicating Malignant External Otitis

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ABSTRACT

Background: Malignant external otitis (MEO) is a severe, potentially fatal infection of the external auditory canal (EAC) and skull base, predominantly affecting immunocompromised patients, particularly those with diabetes mellitus. While Pseudomonas aeruginosa remains the most common causative organism, fungal etiology - especially Aspergillus species - is increasingly recognized, notably in patients who fail to respond to antibacterial therapy. Facial nerve palsy (FNP) is the most frequent cranial nerve complication and is associated with significant morbidity.

Case Presentation: We report a 61-year-old insulin-dependent diabetic male presenting with severe otalgia and purulent otorrhea. Otoscopy revealed an inflammatory polyp obstructing the EAC. Computed tomography (CT) demonstrated necrotic lesions of the EAC with extension to the middle ear and adjacent tissues. Magnetic resonance imaging (MRI) confirmed an infiltrating process involving the anterior and posterior walls of the EAC, extending to the middle ear, deep cervical spaces, and pre- and retrostyloid spaces, with extensive bone lysis. The patient subsequently developed House-Brackmann Grade IV facial nerve palsy. Laboratory tests showed HbA1c of 10.9%, CRP of 21 mg/L, and WBC of 8,420/mm^3. Bacteriological culture isolated Enterococcus spp. Despite appropriate antibiotic therapy and glycemic optimization, no clinical improvement was observed. Mycological culture returned negative; however, empirical voriconazole therapy was initiated based on clinical suspicion, resulting in complete remission of facial nerve palsy from Grade IV to Grade I.

Conclusion: This case highlights the critical importance of considering fungal MEO in diabetic patients refractory to antibacterial therapy, even in the absence of mycological confirmation. Negative mycological culture should not preclude empirical antifungal therapy. Voriconazole can induce complete remission of facial nerve palsy, even in advanced MEO with skull base involvement.

Keywords: Malignant external otitis; Necrotizing external otitis; Facial nerve palsy; Voriconazole; Fungal otitis; Diabetes mellitus; House-Brackmann grading; Skull base osteomyelitis

Introduction

Malignant external otitis (MEO), also termed necrotizing external otitis (NEO), is a severe, potentially life-threatening infection of the external auditory canal (EAC) and adjacent skull base structures. First characterized by Chandler in 1968[1], MEO predominantly affects elderly patients with diabetes mellitus and immunocompromised individuals, in whom impaired host defenses allow progressive invasion of the temporal bone and surrounding tissues.

The etiology of MEO is classically attributed to Pseudomonas aeruginosa, which accounts for more than 90% of bacteriologically confirmed cases. However, an increasing body of evidence highlights the growing importance of fungal pathogens - particularly Aspergillus species - as causative agents, especially in patients with prior antibiotic exposure, prolonged antibacterial therapy, or those refractory to conventional treatment[2,3]. Other fungal pathogens, including Candida species, Mucorales, and Scedosporium apiospermum, have been reported in isolated cases[4].

The clinical course of MEO is characterized by progressive extension from the EAC to the temporal bone, skull base, and adjacent soft tissues, potentially leading to life-threatening complications including cranial nerve palsies, meningitis, and dural sinus thrombosis. Facial nerve palsy (FNP) represents the most frequently affected cranial nerve complication, occurring in approximately 20-30% of advanced MEO cases. Its occurrence indicates involvement of the stylomastoid foramen region and carries significant prognostic implications[5].

The diagnostic workup of fungal MEO presents unique challenges. Mycological cultures frequently yield false-negative results, and histopathological findings may be non-specific. This diagnostic difficulty can lead to delayed initiation of appropriate antifungal therapy, with potentially catastrophic consequences. Voriconazole, a second-generation triazole with broad-spectrum activity against Aspergillus species, has emerged as the treatment of choice for invasive aspergillosis and is increasingly employed in fungal MEO[6].

We report an unusual and instructive case of MEO in a poorly controlled insulin-dependent diabetic patient, complicated by House-Brackmann Grade IV facial nerve palsy, in whom bacteriological culture isolated Enterococcus spp. - an atypical MEO pathogen - while mycological cultures returned negative. Despite failure of targeted antibiotic therapy, empirical voriconazole administration led to complete resolution of otalgia and full remission of facial nerve palsy, strongly suggesting an underlying fungal etiology.

Case Presentation

A 61-year-old male patient with a known history of insulin-dependent type 2 diabetes mellitus presented to our otorhinolaryngology department with a several-week history of severe right-sided otalgia and purulent otorrhea. The patient reported no preceding auricular trauma, swimming, or recent upper respiratory tract infection. Systemic review was otherwise unremarkable.

Physical examination revealed active purulent otorrhea. Otoscopic examination demonstrated an inflammatory polyp completely occupying the external auditory canal, precluding visualization of the tympanic membrane. No cervical lymphadenopathy or parotid swelling was identified. The patient developed right-sided peripheral facial nerve palsy, graded as House-Brackmann Grade IV[7].

Computed tomography (CT) of the temporal bones was performed and demonstrated areas of necrosis within the external auditory canal, with soft tissue density filling the canal lumen and associated erosion of the bony canal walls. The process extended to involve the middle ear cavity and adjacent soft tissues, consistent with aggressive necrotizing osteomyelitis **(Figure 1)**. The ossicular chain was partially involved.

Magnetic resonance imaging (MRI) of the skull base provided further characterization of the extent of disease, revealing an infiltrating process involving the anterior and posterior walls of the EAC, with extension to the middle ear cavity, deep spaces of the face, and the pre- and retrostyloid spaces. Extensive bone lysis of the EAC walls and tympanic cavity was confirmed **(Figure 2)**. No intracranial extension or sigmoid sinus thrombosis was identified.

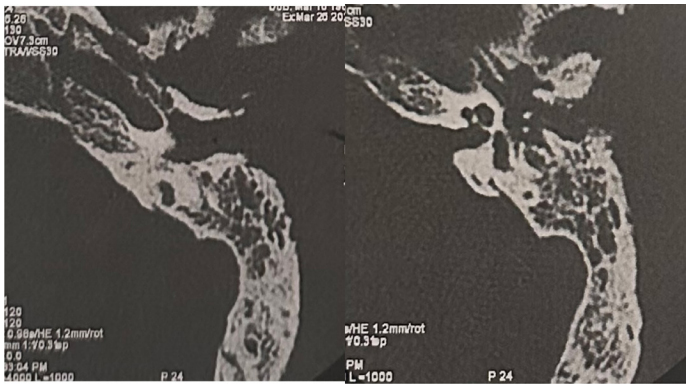

Figure 1: Computed tomography (CT) of the temporal bone (axial view) demonstrating soft tissue opacity within the external auditory canal with extensive bony erosion of the canal walls, extending to the middle ear cavity.

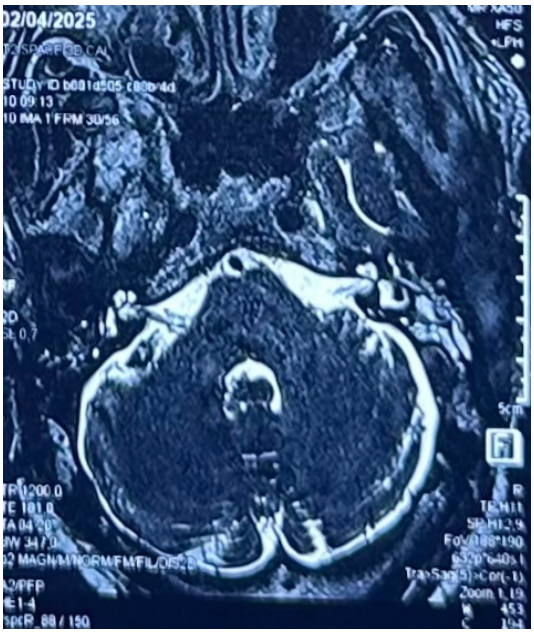

Figure 2: Magnetic resonance imaging (MRI) of the skull base (axial view) demonstrating an infiltrating process involving the anterior and posterior walls of the external auditory canal (EAC), with extension to the middle ear, deep spaces of the face, and pre- and retrostyloid spaces.

Laboratory investigations revealed a glycated hemoglobin (HbA1c) of 10.9%, reflecting poor long-term glycemic control. Inflammatory markers demonstrated a C-reactive protein (CRP) of 21 mg/L and a white blood cell count of 8,420/mm³. Bacteriological culture of the otorrheal discharge isolated Enterococcus spp., which was sensitive to amoxicillin-clavulanate, imipenem, and vancomycin on antibiogram testing. Biopsy of the inflammatory polyp revealed a nonspecific inflammatory infiltrate without granuloma formation, fungal hyphae, or malignant cells.

Treatment was initiated with intravenous antibiotherapy guided by antibiogram results (amoxicillin-clavulanate), combined with rigorous optimization of glycemic control through insulin therapy titration. Strict monitoring of clinical evolution, laboratory parameters, and neurological status was maintained throughout hospitalization. However, despite several days of appropriate antibacterial therapy and correction of metabolic status, no clinical improvement was observed. Otalgia persisted at severe intensity, otorrhea continued, and facial nerve palsy showed no signs of recovery.

In view of this therapeutic failure, a mycological study was pursued. Both direct examination and fungal culture of the otorrheal discharge returned negative results. Despite the absence of mycological confirmation, the overall clinical scenario raised strong clinical suspicion for an underlying fungal etiology. Empirical antifungal therapy with oral voriconazole was therefore initiated at a dose of 400 mg/day (200 mg twice daily) after a loading dose, with regular hepatic and ophthalmological monitoring.

Following the initiation of voriconazole, a progressive and clinically significant improvement was observed. Otalgia regressed substantially within the first two weeks of therapy. Otorrhea diminished progressively. Most notably, facial nerve function demonstrated a remarkable and sustained recovery, ultimately achieving complete remission with restoration to House-Brackmann Grade I **(Figure 3)**. The patient was discharged on maintenance voriconazole therapy with close outpatient follow-up.

Discussion

The case described herein illustrates the diagnostic and therapeutic complexity of malignant external otitis in a diabetic patient, with several distinctive features deserving careful analysis. MEO is a serious infectious disease predominantly affecting elderly diabetics, in whom impaired neutrophil function, compromised microvascular supply to the EAC cartilage, and altered cerumen composition collectively create a permissive environment for aggressive pathogens[1,2].

The pathophysiology of MEO involves a progression from superficial infection of the EAC to deep invasion of the cartilaginous and bony canal walls, with subsequent extension to the mastoid, middle ear, petrous apex, and skull base foramina. This process of progressive osteomyelitis characteristically affects the fissures of Santorini, through which the infection gains access to deeper structures. The CT and MRI findings in our patient - demonstrating necrosis within the EAC with extension to the middle ear, deep face spaces, and pre- and retrostyloid spaces, along with extensive bone lysis - were fully consistent with this established pathological pathway.

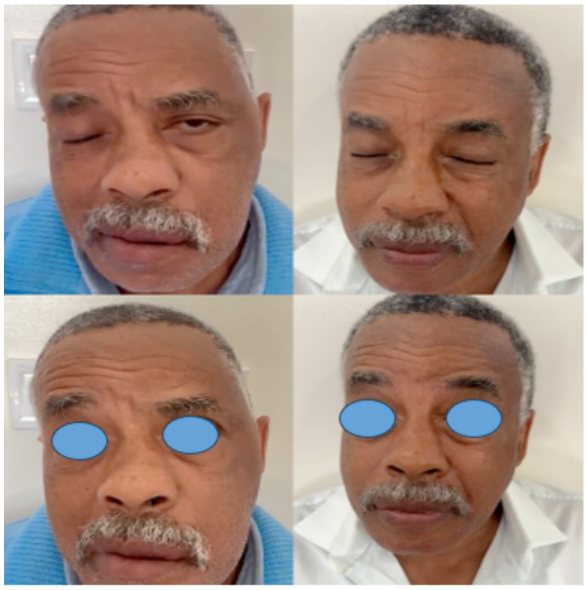

Figure 3: Sequential clinical photographs demonstrating the evolution of facial nerve palsy. Left panels: Initial presentation with House-Brackmann Grade IV facial nerve palsy, showing incomplete eye closure, lagophthalmos, and marked facial asymmetry. Right panels: Complete remission of facial palsy (House-Brackmann Grade I) with restoration of facial symmetry and full eye closure.

An atypical microbiological feature in the present case was the isolation of Enterococcus spp. from the otorrheal culture. While Pseudomonas aeruginosa represents the causative organism in the overwhelming majority of MEO cases, Enterococcus is not a recognized primary pathogen in this context. The isolation of this organism most likely reflects secondary bacterial colonization superimposed on a primarily fungal infection. The complete absence of clinical response to antibiotic therapy guided by antibiogram, targeting the isolated Enterococcus spp., further supports this interpretation.

Fungal MEO is increasingly documented in the literature, particularly in patients with prior antibiotic courses, prolonged antibacterial therapy, or those failing conventional treatment[3,8]. Aspergillus fumigatus and other Aspergillus species are the most frequently implicated fungal pathogens, although Candida species, Mucorales, and Scedosporium have also been reported[4]. The clinical and radiological presentation of fungal MEO is generally indistinguishable from its bacterial counterpart, making mycological diagnosis particularly challenging.

Crucially, mycological cultures carry a well-documented false-negative rate in fungal MEO, estimated at 30–50% in some series[8,9]. This diagnostic limitation arises from multiple factors: the paucity of fungal elements in tissue samples obtained from deep bone lesions, the requirement for specific culture media and prolonged incubation periods, the degrading effects of prior antifungal exposure, and sampling errors in heterogeneous necrotic tissue. Molecular diagnostic approaches, including PCR-based fungal detection and serum galactomannan assay, may improve sensitivity for Aspergillus detection; however, their use in the otological context remains limited and was not available in our setting.

Facial nerve palsy in MEO is considered a marker of advanced disease, indicating spread of infection to the region of the stylomastoid foramen and the vertical segment of

the facial nerve within the temporal bone. Historically, the prognosis for recovery of facial nerve function in MEO has been considered guarded, particularly in cases with complete palsy[5]. However, accumulating evidence suggests that favorable outcomes - including complete recovery - are achievable with early diagnosis, appropriate antimicrobial therapy, and rigorous glycemic optimization.

Voriconazole, a second-generation broad-spectrum triazole antifungal, is currently endorsed as the first-line treatment for invasive aspergillosis by major international guidelines[6]. Its pharmacological profile is particularly well-suited for MEO: it achieves high tissue concentrations in bone and soft tissues, excellent oral bioavailability (>90%), and satisfactory central nervous system penetration - critical properties given the invasive nature of fungal MEO with skull base extension. The dramatic and sustained clinical response to empirical voriconazole in our patient - including complete remission of House-Brackmann Grade IV facial nerve palsy - provides compelling indirect evidence for a fungal, likely Aspergillus, etiology as the primary pathogen.

Glycemic optimization constitutes a cornerstone of MEO management in diabetic patients. Persistent hyperglycemia impairs host immune defenses, promotes pathogen proliferation, and compromises the delivery of antimicrobial agents to infected tissues. In our patient, the HbA1c of 10.9% at presentation reflected severely uncontrolled diabetes, underscoring the importance of aggressive metabolic management as an integral component of the treatment strategy. The combination of antifungal therapy and glycemic optimization likely synergistically contributed to the favorable clinical outcome.

This case carries several key clinical lessons for practicing otorhinolaryngologists and infectious disease specialists: (1) Fungal MEO should be actively considered in diabetic patients who fail to respond to appropriate antibacterial therapy, regardless of mycological culture results; (2) A negative mycological culture does not exclude fungal etiology and should not delay the initiation of empirical antifungal therapy when clinical suspicion is high; (3) Empirical voriconazole therapy can achieve complete remission of facial nerve palsy, even in advanced MEO with extensive skull base involvement; and (4) Rigorous glycemic control is an indispensable and irreplaceable component of MEO management.

Conclusion

Fungal malignant external otitis is a life-threatening condition that poses formidable diagnostic and therapeutic challenges, particularly in diabetic and immunocompromised patients. The present case demonstrates that a negative mycological culture result should not exclude the possibility of fungal etiology when the clinical context is suggestive and bacteriological cultures yield atypical organisms or when antibacterial therapy fails to produce improvement.

Empirical antifungal therapy with voriconazole, combined with rigorous glycemic optimization, led to complete remission of facial nerve palsy in this challenging case of advanced MEO with extensive skull base involvement. This outcome underscores the importance of maintaining a high index of clinical suspicion for fungal etiology in refractory MEO and supports the early initiation of empirical antifungal therapy, even in the absence of mycological confirmation.

A multidisciplinary approach integrating otorhinolaryngology, infectious disease, diabetology, and neuroradiology expertise is strongly recommended for optimal management of these complex cases. Prospective multicentric studies are needed to better define the role of empirical antifungal therapy, optimal duration of treatment, and the use of molecular diagnostic tools in improving outcomes for fungal MEO.

Ethics Statement and Patient Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. The patient was fully informed of the nature of the publication and consented to the use of clinical photographs and radiological images. Identifying information has been appropriately managed to protect patient privacy.

Conflict of Interest

The authors declare no conflict of interest.

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