

## Case Report

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# Fungal Malignant Otitis Externa: About Two Cases

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### Introduction

Malignant Otitis Externa (MOE) is a rare but serious infection of the external auditory canal that can extend to the skull base, potentially leading to osteomyelitis and severe neurological complications. MOE occurs more frequently in older adults with diabetes mellitus and in immunocompromised individuals, such as those with HIV or cancer. The disease typically presents with symptoms such as severe otalgia, hearing loss, and purulent otorrhea. While MOE has traditionally been caused by bacterial infections, most commonly *Pseudomonas aeruginosa* there is an increasing incidence of fungal pathogens, which complicates both diagnosis and treatment. This condition is considered a medical emergency due to its potential for high morbidity and mortality, particularly when the infection involves cranial nerves or spreads to intracranial structures. Contributing factors to increased susceptibility in diabetic patients include poor tissue perfusion, immune dysfunction, and altered conditions within the external auditory canal.

### Case Report 1

A 70-year-old man presented to our ENT emergency department with a right-sided headache, severe right-sided otalgia, and otorrhea characterized by a thin, white discharge and associated hearing loss. Notably, there was no purulent ear discharge. His past medical history included long-standing type 2 diabetes mellitus, Dyslipidemia, and systemic hypertension. His medication regimen consisted of insulin and metformin for Glycemic control, as well as amlodipine and valsartan for blood pressure management. The patient had no history of smoking or alcohol use. Family history was significant for diabetes mellitus and hypertension. Prior to referral to the ENT department, he had been treated at a local health center with oral and topical antibiotics, specifically; amoxicillin/clavulanic acid 1 g orally three times daily and ofloxacin ear drops, one instillation twice daily.

The patient was admitted for further evaluation and management. Vital signs were within normal limits. Otoscopic examination

revealed otorrhea, inflammation, and a grossly edematous right external auditory canal. The tympanic membrane appeared erythematous and bulging posteriorly. Cranial nerve examination demonstrated a right-sided House-Brackmann grade IV facial nerve palsy, while the remaining cranial nerves were intact, with no evidence of nystagmus or other cerebellar signs. The right temporomandibular joint was tender on palpation, as well as during mouth opening and mastication. There was no preauricular or postauricular swelling on the affected side. Examination of the head and neck revealed no associated swellings or palpable lymph nodes.

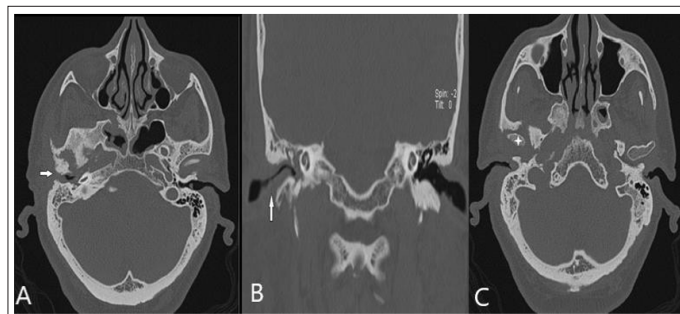


**Figure 1:** CT scan of temporal bones showing destructive middle ear disease surrounding ear canal ring of the right temporal bone, partial right mastoid air cells and middle-ear cavity opacification (A,B) and lytic lesion of the temporomandibular joint (TMJ) (C)

Pure-tone audiometry revealed a right-sided mixed hearing loss with an air-bone gap of 20 dB, along with bilateral presbycusis. Laboratory tests performed during admission showed an elevated erythrocyte sedimentation rate (52 mm at 1 hour, 92 mm at 2 hours) and an elevated C-reactive protein level (114.8 mg/L). White blood cell count was within the reference range. Hemoglobin A1c was 6.0%, kidney function tests were normal, and HIV serology was negative. Electromyography (EMG) of the facial nerve indicated severe axonotmesis of the right facial nerve.

Radiological assessment with a CT scan of the head (Figure 1) demonstrated thickening of the external auditory canal and

middle ear, with an ill-defined soft tissue mass opacifying the right mastoid air cell cavity and causing osteolysis of its walls. There was also a lytic lesion involving the jugular bulb, carotid canal, articular tubercle of the temporal bone, mandibular condyle with subluxation of the temporomandibular joint, zygomatic arch, greater wing of the sphenoid, and the styloid process. These findings are consistent with Malignant Otitis Externa (MOE).

A provisional diagnosis of MOE with Skull Base Osteomyelitis (SBO) was made. Although an initial swab from the infected right ear did isolate a germ, the patient was empirically started on intravenous antipseudomonal and anti-anaerobic antibiotics ceftazidime (2 grams three times daily) and metronidazole (500 mg three times daily). Treatment also included regular microsuction and topical treatment consisting of a combination of Coly-Mycin, rifamycin, and corticosteroids (two drops three times daily). In addition, the patient was commenced on hyperbaric oxygen therapy, consisting of 10 sessions of 100% oxygen administered at 2.4 atmospheres of pressure.

After four weeks of treatment, the patient's condition persisted, with worsening headache and progression to House-Brackmann grade V facial nerve palsy, accompanied by elevated C-reactive Protein (CRP) and erythrocyte sedimentation rate (ESR) levels. Consequently, the patient underwent a cortical mastoidectomy for surgical debridement. Multiple microbiological and histopathological samples were obtained from the right external auditory canal, bone biopsies of the temporomandibular joint, and soft tissues at the skull base, revealing no evidence of malignancy or granulomas. A second swab of the infected ear was taken for microbiological culture; extended incubation yielded yeast subsequently identified as *Candida parapsilosis*, isolated from multiple sites. The patient was then started on intravenous voriconazole (V-Fend) at 400 mg twice daily for the first 48 hours, which was subsequently reduced to 200 mg twice daily for two weeks. Following consultation with the microbiology department, the dosage was increased to 200 mg three times daily.

After a nine-week hospital admission, the patient reported resolution of otalgia, and the right ear canal was free of debris. His facial nerve function improved to House-Brackmann grade III. He was discharged on oral voriconazole 200 mg twice daily for eight weeks, with weekly outpatient follow-ups and serial blood tests. Four weeks post-discharge, ESR and CRP levels normalized, and a repeat contrast-enhanced CT scan of the temporal bone demonstrated further reduction and stability of the lesions. Voriconazole was subsequently discontinued. At eight months follow-up, the patient remains disease-free, with facial nerve palsy stable at House-Brackmann grade III.

## Case Report 2

A 70-year-old man presented with severe left-sided otalgia and a mild headache and was admitted to our department. He underwent a six-week course of intravenous antibiotic therapy, which led to both clinical and biological improvement. Upon stabilization, he was discharged on oral antibiotic therapy. However, six months post-discharge, the patient represented with recurrence of the same symptoms and was subsequently readmitted.

The patient's past medical history was notable for long-standing type 2 diabetes mellitus, managed with insulin therapy for 34 years, with a glycated hemoglobin (HbA1c) level of 9% at the time of admission. He also had chronic kidney disease, with an estimated glomerular filtration rate (eGFR) of 45 mL/min/1.73 m<sup>2</sup>, and systemic hypertension managed with Coveram® (perindopril/

amlodipine) 10 mg once daily. Additional history included retinal detachment in 1996. There was no history of auricular trauma, barotrauma, or exposure to ototoxic agents.

The patient's symptoms began three months prior to admission, characterized by progressively worsening unilateral left-sided otalgia, without associated otorrhea or facial nerve palsy. His general condition remained stable, and he was afebrile throughout the course of the disease.

On admission, vital signs were within normal limits. Otoscopic examination revealed a stenotic left external auditory canal, while the right ear appeared normal. Vestibular assessment was unremarkable, with a normal gait, absence of Romberg sign, no spontaneous or provoked nystagmus, and no evidence of a fistula. Cranial nerve examination showed no facial asymmetry or other neurological deficits. The remainder of the physical examination was unremarkable.

A pure-tone audiogram could not be performed due to complete stenosis of the External Auditory Canal (EAC). Laboratory investigations at the time of admission revealed elevated inflammatory markers, including a white blood cell count of 8,300/mm<sup>3</sup>, CRP at 66.62 mg/L, and an ESR of 76 mm/h.

Radiological evaluation with CT scan of the temporal bones demonstrated lytic destruction of the tympanic bone, complete stenosis of the EAC, and an eroded mastoid. MRI confirmed the presence of external otitis with signs of osteitis at the clivus, complicated by a collection within the Eustachian tube (Figure 2).

The patient was initially treated with a reduced-dose intravenous antibiotic regimen due to underlying chronic kidney disease, consisting of ciprofloxacin 400 mg/day and a third-generation cephalosporin at 3 g/day. After four weeks of treatment, there was no clinical or biochemical improvement, with persistently elevated CRP levels ranging from 60 to 100 mg/L and ESR values between 70 and 120 mm/h. Procalcitonin testing was negative, effectively ruling out an active bacterial infection.

A mastoidectomy was subsequently performed, which revealed an eroded, inflamed mastoid bone that bled upon contact. Multiple microbiological and histopathological samples were obtained from the left external auditory canal, mastoid bone, and soft tissues at the skull base. Culture results confirmed the presence of *Candida orthopsilosis* from multiple anatomical sites.

The patient was then initiated on intravenous voriconazole (Vfend®), administered at a loading dose of 400 mg twice daily for the first 48 hours, followed by a maintenance dose of 200 mg twice daily for the subsequent two weeks.

After ten days of antifungal therapy, the patient developed an altered level of consciousness and was transferred to the Intensive Care Unit (ICU), where he required endotracheal intubation and mechanical ventilation. Despite intensive supportive care, he experienced a cardiac arrest and died on the third day of ICU admission.

This case illustrates the fatal progression of fungal malignant external otitis in an elderly patient with long-standing diabetes mellitus and chronic kidney disease, despite extended courses of both antibacterial and antifungal therapy. The aggressive clinical course, delayed identification of the fungal etiology, and the presence of significant comorbidities likely contributed to

the poor outcome. This case underscores the importance of early recognition and prompt antifungal treatment in patients with refractory or atypical presentations of malignant external otitis, particularly in immunocompromised individuals.

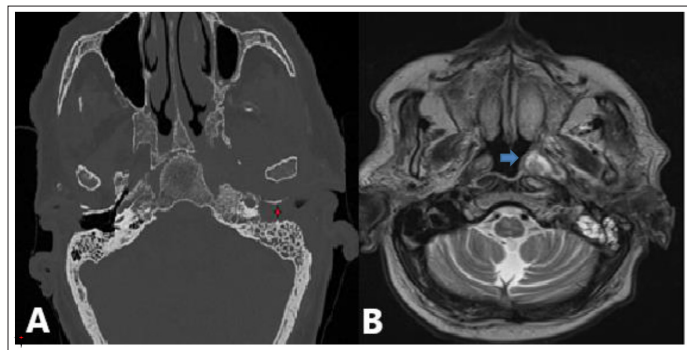


**Figure 2:** A: CT scan of temporal bones showing destructive middle ear disease surrounding ear canal ring (+) of the right temporal bone, partial right mastoid air cells and middle-ear cavity opacification. B: MRI of temporal bones T2- weighted imaging with signs of osteitis, complicated by a collection in the Eustachia tube (→).

### Discussion

Malignant Otitis Externa (MOE) is a rare but life-threatening infection involving the external auditory canal, mastoid, and skull base, first formally described by Chandler in 1968 [1]. It predominantly affects immunocompromised individuals, particularly elderly patients and those with diabetes mellitus. The condition is most commonly caused by *Pseudomonas aeruginosa* and can rapidly extend to adjacent structures, including cranial nerves and intracranial compartments, resulting in serious and potentially fatal complications [2,3].

While the introduction of antipseudomonal antibiotics initially led to improved outcomes, recent years have witnessed an increase in treatment failures attributed to the emergence of multidrug-resistant *Pseudomonas* strains and Methicillin-Resistant *Staphylococcus aureus* (MRSA) [4].

Fungal Malignant External Otitis (MEO) is of particular concern, primarily affecting elderly or immunocompromised individuals. The first reported case of fungal involvement in MOE was published in 1985 [5]. Although less common than bacterial forms, fungal MEO most frequently caused by *Aspergillus* and *Candida* species is typically more insidious and associated with a more severe clinical course [6]. Otomycosis, or fungal otitis externa, accounts for approximately 9% of all otitis externa cases, with the majority of the remainder being bacterial in origin [7,8]. Due to frequent delays in diagnosis and the often-refractory nature of the infection, fungal MEO carries a significantly poorer prognosis than its bacterial counterpart. Early clinical suspicion, microbiological confirmation, and the initiation of targeted systemic antifungal therapy are essential to improving outcomes in affected patients.

### Risk Factors and Pathophysiology

Although fungal pathogens are infrequently implicated in MOE, when present, they are predominantly associated with severely immunocompromised patients, particularly those with poorly controlled diabetes mellitus. As demonstrated in multiple studies and in both of our cases, diabetes mellitus is the most commonly reported comorbidity, with fungal MOE occurring in 75% to 100% of cases [9]. Additional risk factors include chronic renal failure,

malignancies, immunosuppressive therapies, acute leukemia, and HIV infection. Fungal MOE remains a clinical enigma, as it is uncertain whether it develops as a sequela of inadequately treated bacterial otitis externa or as a primary fungal infection [10].

The pathogenesis of fungal MOE, particularly infections caused by *Aspergillus* species, is multifactorial and not yet fully understood. *Aspergillus* demonstrates several virulence factors that contribute to its pathogenicity, including the secretion of proteolytic enzymes such as proteases and phospholipases, as well as the production of mycotoxins like aflatoxins. These fungi thrive optimally at human body temperature (~37°C), facilitating tissue invasion and dissemination. While *Aspergillus* is the most commonly implicated fungal pathogen in MOE, *Candida* species also play a significant role, especially in immunocompromised patients. *Candida* species form biofilms that act as physical barriers to host immune responses and antifungal agents, complicating treatment and eradication efforts [11-13].

A key factor contributing to fungal overgrowth and pathogenicity in the external auditory canal is the disruption of normal bacterial flora, often resulting from prior or concurrent use of broad-spectrum antibiotics. Prolonged intravenous antibiotic therapy, particularly in the treatment of bacterial MOE, may predispose patients to secondary fungal infections by eliminating competitive microbial flora and impairing mucosal immunity. Furthermore, aural water exposure such as irrigation during cerumen removal is considered a potential iatrogenic factor that can facilitate fungal colonization [14].

Patients with diabetes mellitus are particularly susceptible to both bacterial and fungal infections of the external auditory canal. This increased vulnerability is attributed to multiple factors, including diabetic microangiopathy resulting in impaired tissue perfusion, alterations in cerumen pH, and compromised immune function [15,16].

Interestingly, cases of fungal otitis externa have also been reported in immunocompetent patients [17]. Further studies suggest that even in individuals without overt immunosuppression, a deficient adaptive immune response may contribute to the development of fungal skull base osteomyelitis. Netea et al. reported that patients without known immunodeficiency who developed *Aspergillus*-induced skull base osteomyelitis exhibited significantly reduced production of interleukins IL-17 and IL-22 by peripheral blood mononuclear cells upon stimulation with *A. fumigatus* and *Candida albicans*, indicating an impaired Th17-mediated immune response [18].

### Clinical Presentation

Patients with MOE, particularly of fungal origin, typically present with severe otalgia often worsening at night accompanied by purulent otorrhea, conductive hearing loss, and persistent headache. A systematic review of 197 patients identified headache and otorrhea as the most common presenting symptoms, in contrast to other studies that emphasize otalgia as the predominant clinical feature [9]. Moreover, the absence of purulent ear discharge has been reported as a sensitive predictor (91%) of fungal involvement in cases initially presumed to be bacterial MOE [19]. Additional functional symptoms may include trismus, tinnitus, dizziness, and low-grade fever. On physical examination, common findings include edema of the external auditory canal, the presence of aural polyps, and, less frequently, features of coexisting chronic suppurative otitis media.

In advanced stages, the disease may involve the skull base, leading to cranial neuropathies. As was the case for both of our patients, the facial nerve (cranial nerve VII) is most commonly affected, typically at the level of the stylomastoid foramen. Additional cranial nerves, such as the glossopharyngeal (IX), vagus (X), and spinal accessory (XI), may be involved at the jugular foramen, while the hypoglossal nerve (XII) can be affected at the hypoglossal canal. Rarely, petrous apex involvement may lead to deficits in the trigeminal (V) and abducens (VI) nerves.

### **Etiologic Agents**

Compared to bacterial MOE, especially cases caused by *Pseudomonas aeruginosa*, fungal MOE tends to exhibit a more aggressive clinical course with a higher incidence of cranial nerve palsies and more extensive skull base osteomyelitis, as well as persistent headache. Therefore, it is essential to maintain a heightened level of clinical vigilance, to reassess and expand our diagnostic framework, with particular emphasis on identifying atypical clinical manifestations that deviate from the classical presentation outlined by Cohen and Friedman [20]. In our cases, the most relevant clinical signs were severe otalgia, headache, and stage V facial nerve Palsy; as for the second patient, it was severe otalgia but had a minor headache.

FMOE is a rare but increasingly recognized condition, accounting for approximately 10% of MOE cases [21]. The most common etiological agents are species from the *Aspergillus* genus, particularly *Aspergillus fumigatus*, followed by *A. flavus* and *A. niger*. *Candida albicans* is the second most frequent fungal pathogen implicated, and cases of mixed bacterial-fungal or dual fungal infections have also been reported [22,23].

*Aspergillus* species are ubiquitous saprophytic molds commonly found on decaying organic matter. Among them, *Aspergillus flavus* is notable for its broader pathogenic potential, ranging from hypersensitivity reactions to invasive infections such as osteomyelitis, particularly following trauma or direct inoculation. It exhibits greater virulence and higher resistance to antifungal agents compared to *A. fumigatus*, and is more prevalent in hot, arid climates [24]. FMOE typically involves the middle ear and mastoid regions rather than the external auditory canal. While *A. fumigatus* is the most frequently implicated species, rare cases involving other fungi such as *Alternaria* have been reported [25]. Advances in diagnostic methods have also led to increased recognition of *Candida* species as causative pathogens in MOE [26]. In our cases, *Candida parapsilosis* and *Candida orthopsilosis* were identified as the responsible fungal agents.

### **Diagnostic Approach**

The diagnoses of patients are established through comprehensive clinical evaluation, integrating symptomatology, failure to respond to first-line therapies, the presence of predisposing factors, and radiologic findings. Computed tomography (CT), favored for its rapid acquisition and widespread availability, was employed to assess the extent of disease, including involvement of osseous structures and the skull base. Radiographic features such as mastoid cortical erosion, extracapsular soft tissue edema, and inflammatory changes in adjacent structures, including the temporomandibular joint (TMJ) supported the diagnosis. In contrast, magnetic resonance imaging (MRI) offers superior sensitivity for initial diagnosis, disease staging, and the detection of complications, including vascular and meningeal infiltration, soft tissue involvement, cerebral parenchymal extension, and disease spread [27,28].

For monitoring treatment response, CT remains the imaging modality of choice due to its ability to track morphological changes over time. Although technetium-99m bone scintigraphy is highly sensitive for detecting malignant otitis externa (MOE), it lacks specificity. Gallium-67 citrate scintigraphy demonstrates increased uptake in areas of active infection and returns to baseline upon resolution, making it a useful tool for assessing therapeutic efficacy [29]. Additionally, positron emission tomography-computed tomography (PET-CT) is emerging as a highly sensitive modality for both the diagnosis and follow-up of MOE, particularly in evaluating the extent of disease and monitoring response to treatment [30,31].

With regard to radiological findings in fungal malignant otitis externa (MOE), Peled et al. (2021) reported that temporomandibular joint (TMJ) involvement, observed in one of our cases, is more frequently associated with fungal etiologies. This observation supports the early initiation of antifungal therapy in patients presenting with TMJ related symptoms and sterile cultures consistent with MOE [30]. Furthermore, Maxim Moniot et al. proposed the “ink-smudge” sign characterized by a signal void on T2-weighted MRI sequences as a potential radiologic marker specific to fungal MOE, based on findings from their case report [32].

Diagnosis of fungal MOE requires formal tissue sampling from the external ear canal or the middle ear to identify the causative agent. Fungal and bacterial cultures may be negative at initial presentation, with positive cultures arising only later in the treatment process [14]. ESR may be elevated and, although not diagnostic, is a useful prognostic marker [29].

### **Therapeutic Strategies**

The cornerstone of treatment for Fungal Malignant Otitis Externa (FMOE) is prolonged systemic antifungal therapy, often in conjunction with antibiotic therapy, due to the frequent presence of coexisting bacterial infections. In cases of bacterial MOE, intravenous antibiotics such as aminoglycosides, antipseudomonal  $\beta$ -lactams, and fluoroquinolones are commonly used, with selection guided by microbiological cultures and antimicrobial susceptibility profiles. For FMOE, first-line antifungal agents include amphotericin B, voriconazole, and itraconazole. Triazoles such as fluconazole and voriconazole are frequently used either alone or in combination with amphotericin B during the initial phase of treatment [33-35].

Antifungal therapy is typically prolonged, with treatment durations ranging from 6–8 weeks to several months, depending on clinical response and radiologic findings. In certain cases, lifelong oral antifungal therapy may be required for disease control. Management must also include strict control of underlying risk factors, particularly glycemic regulation in diabetic patients [14,29]. The presence of skull base osteomyelitis (SBO) or central nervous system (CNS) involvement necessitates the use of antifungal agents with good tissue and CNS penetration. Amphotericin B, flucytosine, and azoles fulfill this requirement. In contrast, echinocandins (e.g., caspofungin, anidulafungin) have limited CNS penetration data but may be considered in refractory or invasive *Candida* infections [36,37]. Antifungal Therapy requires careful monitoring of hepatic and renal function due to the potential toxicity of antifungal agents. Adverse effects may include hepatotoxicity, nephrotoxicity, electrolyte imbalances, and visual disturbances, particularly with agents like voriconazole.

Surgical intervention is no longer considered the primary treatment modality for MOE. Its role is now generally limited to obtaining bone biopsy samples and tissue cultures to establish a definitive diagnosis. In selected cases particularly those with extensive disease or infections refractory to medical therapy surgical debridement may be necessary [38]. However, following the findings reported by Lucente et al., clinicians became increasingly aware that initiating surgical management without prior disease stabilization through conservative medical therapy may result in unfavorable outcomes [39].

While there are no established guidelines specific to fungal MOE or Skull Base Osteomyelitis (SBO), the Infectious Diseases Society of America (IDSA) provides recommendations for *Candida* osteoarticular infections that may offer clinical guidance. The IDSA recommends surgical debridement followed by lipid-based amphotericin B or an echinocandin for an initial period of two weeks, with subsequent transition to oral fluconazole for a duration of 6 to 12 months, depending on clinical response [40].

Hyperbaric Oxygen Therapy (HBOT) has been reported as a useful adjunctive treatment in selected cases of Malignant Otitis Externa (MOE) [41,42]. HBOT enhances oxygen delivery to hypoxic tissues, thereby promoting angiogenesis, collagen synthesis, and improved phagocytic activity—processes that are critical for effective soft tissue and bone healing. In cases of MOE, particularly those caused by *Aspergillus* species, vascular invasion may lead to thrombosis and tissue hypoxia, making HBOT a potentially beneficial therapeutic option.

Although HBOT is not considered standard of care due to limited availability and variable supporting evidence, it has demonstrated clinical utility in refractory cases. It may be considered in patients with advanced disease, including those with skull base osteomyelitis or intracranial extension [43-45]. Its implementation remains institution-dependent and should be individualized based on disease severity and patient response.

In our study, in addition to systemic antifungal therapy and surgical debridement, the first patient received adjunctive HBOT and achieved successful clinical resolution.

The prognosis of MOE depends on early management and the extent of disease; if not detected and treated adequately early, it can have irreversible consequences such as persistent cranial nerve abnormalities and a high mortality rate. The second patient in our study succumbed to fungal infection before surgery could be considered, due to probable cerebral involvement, despite antifungal treatment being instituted after failure of antimicrobial treatment.

## Conclusion

These two cases highlight the severity of Fungal Malignant Otitis Externa (FMOE), a rare but life-threatening condition primarily affecting immunocompromised individuals. One patient survived following early diagnosis, antifungal therapy, surgical debridement, and HBOT, while the other died before surgical treatment and HBOT could be considered, emphasizing the importance of early diagnosis and management.

FMOE often mimics bacterial MOE, delaying appropriate management. High clinical suspicion, especially in patients unresponsive to antibiotics, should prompt early biopsy, fungal cultures, and imaging. *Aspergillus flavus*, though less common, poses a greater treatment challenge due to its higher virulence and antifungal resistance.

Voriconazole remains a first-line agent, and prolonged antifungal therapy, sometimes combined with surgical debridement or hyperbaric oxygen therapy, may be required. Timely diagnosis and species-specific therapy are crucial to improve outcomes and reduce mortality in FMOE.

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