

CASE REPORTS

Malignant Otitis Externa (MOE) Causing Cerebral Abscess and Facial Nerve Palsy

John Roberts, MD¹
Línea Larson-Williams, MD¹
Farrah Ibrahim, MD¹
Ali Hassoun, MD²

¹Department of Internal Medicine, University of Alabama-Birmingham, Huntsville Regional Medical Campus, Huntsville, Alabama.

²Alabama Infectious Diseases Center, Huntsville, Alabama.

Disclosure: Nothing to report.

KEYWORDS: cerebral abscess, facial nerve palsy, malignant, otitis externa, pseudomonas.

Malignant Otitis Externa (MOE) is a necrotizing infection of the external auditory canal characterized by extension into nearby soft tissue and bony structures that can potentially lead to mastoiditis, skull base osteomyelitis, cranial nerve palsies, and rarely, intracranial complications. MOE has been classically described as a disease affecting elderly diabetics¹ and has been reported in immunocompromised patients with acquired immune deficiency syndrome (AIDS), malignancy, patients receiving chemotherapy, and neutropenic children.²⁻⁴ The incidence of MOE in the general population is estimated to be quite low and difficult to determine.⁵ However, over the past decades, the number of reported cases has been increasing, suggesting increased awareness of this syndrome by primary care physicians.⁶

Case Report

A 56-year-old white male was brought to the emergency department with altered mental status including decreased level of consciousness, bizarre behavior, headaches, and nausea for several weeks. He had a history of alcohol and cocaine abuse. He was homeless and a smoker. On examination, the patient was lethargic, disoriented with respect to time, place and person. Blood pressure was 107/72 mm Hg, heart rate 85 beats per minute; respirations were 16 per minute and temperature was 97.9°F. Neurological examination was significant for loss of vision of the left eye and left facial peripheral nerve palsy. Examination of the left eye showed yellowish-greenish discharge, a lower lid ectropium with upper lid ptosis, conjunctival erythema, and an 8 mm × 6 mm abrasion in the medial half of the cornea. He had purulent drainage from the left ear with small vesicular lesions on the auricle, and a 3 cm × 4 cm abscess on the right forearm. The remainder of the physical examination was unremarkable.

Laboratory-test results showed white blood cell (WBC) count; 11.27×10^9 cells/L with 76.6% neutrophils. A complete metabolic panel was within normal limits. Human immunodeficiency virus (HIV) and RPR testing were negative. Cerebrospinal fluid (CSF) studies demonstrated a WBC 39 cells/mm³ with lymphocytes predominance (85%), Red blood cell (RBC) 6 cells/mm³, protein 48 mg/dL, and glu-

cose; 63 mg/dL. Computed tomographic scan of the head revealed an area of low attenuation with surrounding edema of the left temporal lobe and fracture of the temporal bone on the superior margin of the mastoid air cells extending into the left mastoid air cells (Figure 1). The fluid draining from the patient's left ear grew 2 different strains of *Pseudomonas aeruginosa*. Magnetic resonance imaging (MRI) of the brain demonstrated a 2.5 cm to 3.0 cm region of multiple loculations and edema in the left temporal lobe representing cerebritis with abscess and complete opacification of left mastoid air cell suggestive of mastoiditis (Figure 2). Piperacillin/tazobactam and tobramycin were initiated for suspected MOE with brain involvement. CSF and blood cultures were negative.

A repeat MRI at 3 weeks of therapy demonstrated interval improvement in the temporal lobe abscess and the edema surrounding the infection. At the time of discharge, after 6 weeks of antimicrobial therapy, the patient was alert and oriented with respect to person, place, and time.

Discussion

Pseudomonas aeruginosa is the most common organism cultured from MOE.^{4,5} Other organisms such as *Staphylococcus aureus*,⁶ *Proteus mirabilis*, *Klebsiella oxytoca*,⁷ and *Aspergillus* species⁸ have been reported as well.

While the exact pathogenesis of MOE is poorly understood, accidental trauma from cotton swabs, exposure to lake water, swimming pool water, and repeated aural lavage have all been implicated as inciting factors.⁹ The current literature suggests that Pseudomonas otitis externa occurs due to abnormal host defense mechanisms rather than enhanced pathogen colonization.¹⁰

MOE typically presents with severe otalgia, headache, auricular tenderness, mastoid tenderness, or persistent otorrhea. The pain of MOE is usually severe, and the classic signs of infection such as fever, leukocytosis and neutrophil predominance ("left shift") may not be present.⁵ The diagnosis can be confirmed by otoscopic exam which will demonstrate granulation tissue at the junction between the bony and cartilaginous tissues in the external auditory canal. MOE can produce certain physical findings that

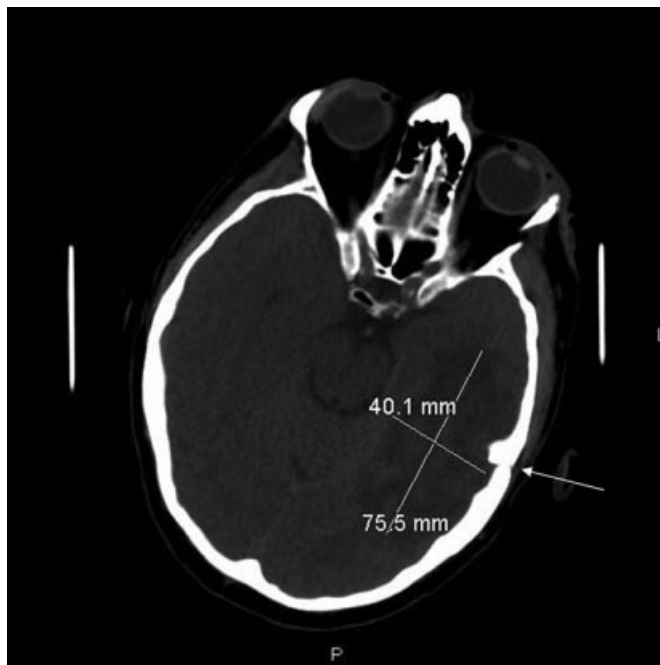


FIGURE 1. CT head revealed area of low attenuation, edema of the left temporal lobe and fracture of the temporal bone on the superior margin of the mastoid air cells extending into the left mastoid air cells.

should raise red flags for local extension. Temporomandibular joint pain in the susceptible patient with otalgia could indicate MOE with joint invasion. Cranial nerve involvement, most commonly involves the seventh cranial nerve which results in a facial palsy. Other cranial neuropathies have been reported in MOE such as the glossopharyngeal, vagal, spinal accessory, and hypoglossal nerves.¹¹ Confusion and nuchal rigidity should arouse suspicion of intracranial extension of the infection.

The diagnosis of MOE is usually made by a constellation of clinical, microbiological and radiological features. The first attempt at defining diagnostic criteria for MOE was in 1987, when Cohen and Friedman named several obligatory signs such as pain, exudate, edema, granulation tissue in the external ear canal, the presence of microabscesses if surgery is performed, and either a positive Tc-99m bone scan or failure of local treatment for 1 week.¹²

Recent literature reviews emphasize that a positive bacterial or fungal culture of the external ear canal can help make the diagnosis of MOE.⁴ A recent study looking at diagnostic criteria noted that MOE may be present even without meeting all of these major criteria (clinical, microbiological, and radiological features).¹³ Some authors suggest that ear biopsy be considered if malignancy is a reasonable possibility.⁹

Other laboratory data can be within normal limits, such as a WBC count or differential and metabolic profiles.⁴ Erythrocyte sedimentation rate (ESR), while non-specific, has been reported to be markedly increased in the setting of MOE.⁵ It is recommended that a baseline ESR be obtained, and then used to follow the response to treatment.¹⁴

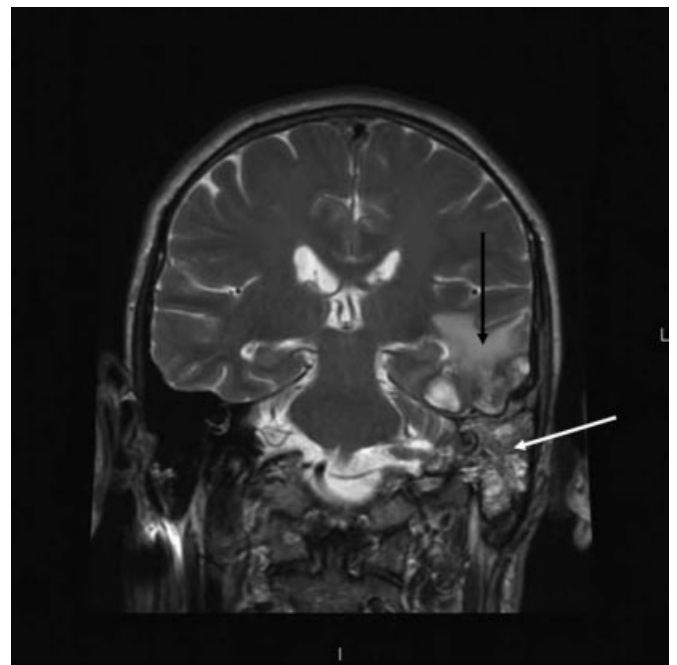


FIGURE 2. MRI of the brain demonstrated a 2.5 cm to 3.0 cm region of multiple loculations and edema in the left temporal lobe brain representing cerebritis with abscess and complete opacification of left mastoid air cell suggestive of mastoiditis.

Computed tomography (CT) scanning is considered the appropriate initial imaging study, however, there are mixed reports about whether a CT scan alone is enough to evaluate disease severity and its complications.¹⁵ While CT scan is quite sensitive for demonstrating bony destruction associated with MOE, MRI is better at detecting the soft tissue changes associated with MOE and more useful for following disease resolution after treatment.¹⁶

The treatment of MOE has evolved over time. Before the introduction of effective anti-pseudomonal antibiotics, MOE had an associated mortality near 50%, and surgery was the recommended therapy.¹⁷ Currently, given the availability of effective anti-pseudomonal therapy, there is usually no indication for surgery as a primary treatment in MOE. For sensitive pseudomonal species, oral Ciprofloxacin therapy for 6 to 8 weeks is considered the treatment of choice for MOE.¹⁸ Clinicians should be aware of the emergence of quinolone resistance in *P. Aeruginosa*,¹⁹ and antibiotic sensitivities should be performed on the culture to guide further treatment. In the setting of resistant or multi-species infection, one should obtain external ear canal biopsy with wound debridement and begin long-term intravenous therapy with an antipseudomonal beta-lactam plus an aminoglycoside.¹⁸ In the setting of extension into the cranium causing cerebritis or abscess, it is recommended that a neurosurgeon be consulted immediately to determine whether the lesion is amenable to aspiration, excision, or watchful waiting with imaging follow up.

This case illustrates a serious complication from MOE, with ipsilateral facial nerve palsy, ophthalmitis, mastoiditis, and cerebral abscess that was successfully treated with conservative medical management.

Address for correspondence and reprint requests:

Ali Hassoun, MD, Alabama Infectious Diseases Center, 420 Lowell Drive, suite 301, Huntsville, AL 35801; Telephone: 256-265-7955; Fax: 256-265-7954; E-mail: ali_hasoun@yahoo.com Received 23 August 2009; revision received 12 October 2009; accepted 23 November 2009.

References

1. Chandler JR. Malignant external otitis. *Laryngoscope*. 1968;78:1257.
2. Ress BD, Luntz M, Telischi FF, et al. Necrotizing external otitis in patients with AIDS. *Laryngoscope*. 1997;107:456.
3. Nir D, Nir T, Danino J, Joachims HZ. Malignant external otitis in an infant. *J Laryngol Otol*. 1990;104(6):488–490.
4. Rubin J, Branstetter BF 4th, Yu VL. The changing face of malignant (necrotizing) external otitis: clinical, radiological, and anatomic correlations. *Lancet Infect Dis*. 2004;4:34.
5. Rubin J, Yu VL. Malignant external otitis: insights into pathogenesis, clinical manifestations, diagnosis, and therapy. *Am J Med*. 1988;85:391.
6. Bayardelle P, Jolivet-Granger M, Larochelle D. Staphylococcal malignant external otitis. *Can Med Assoc J*. 1982;126:155.
7. Garcia Rodriguez JA, Montes Martinez I, Gomez Gonzalez JL, et al. A case of malignant external otitis involving *Klebsiella oxytoca*. *Eur J Clin Microbiol Infect Dis*. 1992;11:75.
8. Gordon G, Giddings NA. Invasive otitis externa due to *Aspergillus* species: case report and review. *Clin Infect Dis*. 1994;19:866.
9. Handzel O, Halperin D. Necrotizing (malignant) external otitis. *Am Fam Physician*. 2003;68(2):309–312.
10. Salit IE, Miller B, Wigmore M, Smith JA. Bacterial flora of the external canal in diabetics and non-diabetics. *Laryngoscope*. 1982;92:672.
11. Schwarz GA, Blumenkrantz MJ, Sundmaker WL. Neurologic complications of malignant external otitis. *Neurology*. 1971;21:1077–1084.
12. Cohen D, Friedman P. The diagnostic criteria of malignant external otitis. *J Laryngol Otol*. 1987;101:216–221.
13. Peleg U, Perez R, Raveh D, Berelowitz D, Cohen D. Stratification for malignant external otitis. *Otolaryngol Head Neck Surg*. 2007;137(2):301–305.
14. Rubin J, Stoehr G, Yu VL, Muder RR, Matador A, Kamerer DB. Efficacy of oral ciprofloxacin plus rifampin for treatment of malignant external otitis. *Arch Otolaryngol Head Neck Surg*. 1989;115:1063–1069.
15. Sudhoff H, Rajagopal S, Mani N, Moumoulidis I, Axon PR, Moffat D. Usefulness of CT scans in malignant external otitis: effective tool for the diagnosis, but of limited value in predicting outcome. *Eur Arch Otorhinolaryngol*. 2008;265(1):53–56.
16. Ismail H, Hellier WP, Batty V. Use of magnetic resonance imaging as the primary imaging modality in the diagnosis and follow-up of malignant external otitis. *J Laryngol Otol*. 2004;118:576.
17. Slattery WH 3rd, Brackmann DE. Skull base osteomyelitis. Malignant external otitis. *Otolaryngol Clin North Am*. 1996;29(5):795–806.
18. ouie TJ. Ciprofloxacin: an oral quinolone for the treatment of infections with gram-negative pathogens. Committee on Antimicrobial Agents. Canadian Infectious Disease Society. *Can Med Assoc J*. 1994;150(5):669–676.
19. Bernstein JM, Holland NJ, Porter GC, Maw AR. Resistance of *Pseudomonas* to ciprofloxacin: implications for the treatment of malignant otitis externa. *J Laryngol Otol*. 2007;121:118–123.