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Case Series

Malignant otitis externa: our experience

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ABSTRACT

Malignant otitis externa (MOE) is a severe and potentially life-threatening infection that originates in external auditory canal. Infection begins in external auditory canal, spreading through fissure of Santorini to the surrounding structures. The clinical course is varied and outcome prediction is difficult. This review aims to explore the clinical presentation, treatment, and follow up of patient with MOE. Three patients who were known cases of type 2 diabetes mellitus (T2DM) presented to the Outpatient department with complaints of intractable otalgia and otorrhea. Some patients had cranial nerve palsy. Radiological investigations were used as adjunct for diagnosis. Clinically they were diagnosed with MOE and were treated with a 6 week course of intravenous ciprofloxacin and piperacillin-tazobactam. Response to treatment was assessed using improvement in signs and symptoms and level of inflammatory markers.

Keywords: Malignant otitis externa, Type 2 diabetes mellitus, Otalgia, HRCT-temporal bone, MRI skull base, IV antibiotics

INTRODUCTION

Malignant otitis externa (MOE) is an invasive infection that can spread from the External auditory canal to the skull base.[1] This condition often affects elderly patients who are diabetic or immunocompromised.[1] MOE is most commonly caused by *Pseudomonas aeruginosa* but also by *Staphylococcus epidermidis*, *Klebsiella* and *Proteus mirabilis*, as well as by fungi, *Aspergillus*.[2] Patients typically present with severe otalgia, hearing loss, and purulent otorrhea. Otoscopic examination may show oedema of external auditory canal and granulation tissue in bony cartilaginous junction of external auditory canal.[3] Invasion of the base of skull may lead to paralysis of facial nerve (in most cases) and other cranial nerve (IX, X, XII).

According to literature MOE should be suspected in high-risk groups, such as elderly with diabetes, patients on chemotherapy, or in immunocompromised patients. Early and long-term use of appropriate antibiotics will reduce the morbidity and mortality. Surgery can be used only for draining micro abscesses, removing sequestrum suggesting a limited role in management of MOE.[4]

According to literature, for MOE surgery should be avoided whenever possible but should not be delayed if there is cranial nerve involvement. Surgery is the best option for cases which are refractory to medical therapy.[5]

CASE SERIES

Case 1

A 74-year-old male patient with T2DM reported worsening nocturnal otalgia in his left ear and left ear discharge for six months. Clinical examination revealed left ear canal congestion and oedema with granulation tissue occupying the floor of the left ear canal (Figure 1 A). Cranial nerve examination was clinically normal. Laboratory tests showed uncontrolled diabetes (as evidenced by monitoring of HbA1c status) and elevated

levels of inflammatory parameters [white blood cell count (WBC), erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP)]. An ear swab was negative for bacterial growth. CT scan of the temporal bone and MRI of the skull base showed soft tissue thickening in the left ear canal and petrous temporal bone, suggesting the possibility of skull base osteomyelitis involving the posterior skull base (Figure 1 B and C). PET-CT scan was performed, which showed a metabolically active lesion involving the left middle and posterior skull base, eroding the petrous and mastoid parts of temporal bone, the body of the sphenoid bone, and the Basi occiput (Figure 1 D).

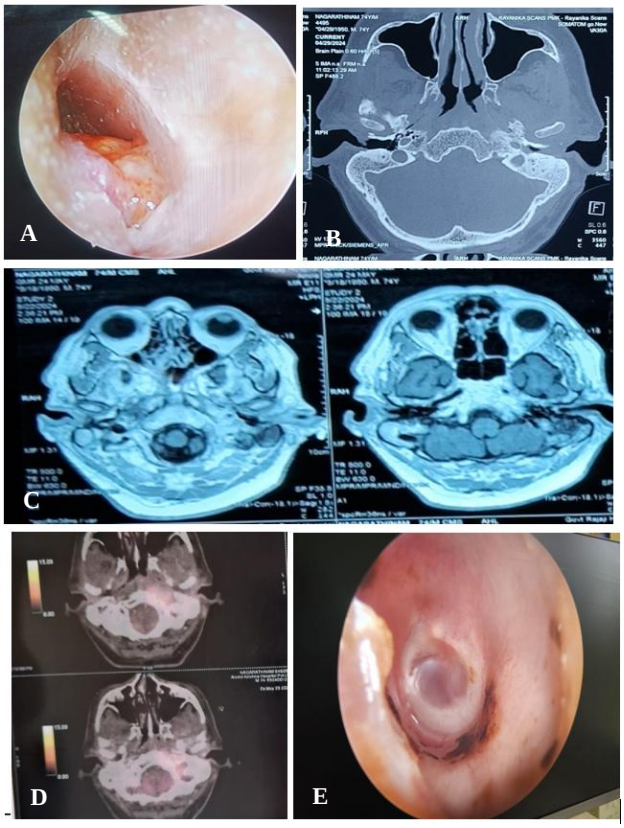

Figure 1 (A-E): Oto-endoscopy (admission), CT temporal bone, MRI skull base, PET-CT and post treatment oto-endoscopy of case 1.

The patient was treated with dual antibiotic therapy consisting of intravenous ciprofloxacin (400 mg every 12 hours) plus piperacillin/tazobactam (4.5 grams every 12 hours) for six weeks, along with local debridement of the granulation tissue. Histological examination of the granulation tissue confirmed its inflammatory nature. Glycaemic control was achieved with titrated insulin therapy, and serial monitoring of inflammatory markers (ESR and CRP) was conducted. The patient showed clinical improvement with complete resolution of nocturnal otalgia and a reduction in inflammatory parameters. Edema and granulation tissue in the ear canal improved (Figure 1 E). The patient was discharged on oral ciprofloxacin 750 mg twice daily with instructions for regular follow-up. At the one-year follow-up, there was no evidence of recurrence.

Case 2

A 47-year-old white male with T2DM presented with left ear pain, which worsened at night, and left ear discharge for seven months. Clinical examination revealed a congested and oedematous left ear canal with granulation tissue on the anterior, posterior, and superior walls of the left ear canal (Figure 2 A). Cranial nerve examination was clinically normal. Blood tests showed uncontrolled diabetes (as evidenced by monitoring of HbA1c status) and elevated levels of inflammatory parameters, including WBC count, ESR, and CRP. An ear swab was negative for the growth of any organism. CT scan of the temporal bone and MRI of the petrous bone were performed, which showed soft tissue thickening in the left ear canal with bony erosion of the anterior and posterior walls of the left ear canal and the mastoid process.

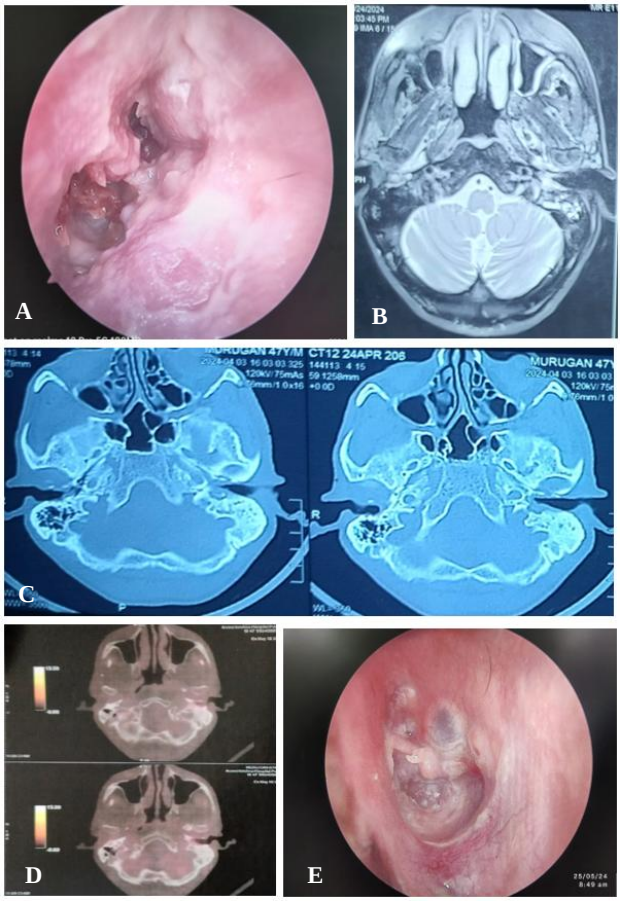

Figure 2 (A-E): Oto endoscopy (admission), MRI skull base, CT temporal bone, PET-CT, post treatment oto-endoscopy of case 2.

There was erosion of the vertical segment of the bony facial canal (Figure 2 B and C). PET-CT scan showed a metabolically active lesion involving the left ear canal

and middle ear with minimal bony erosion and opacification of the left mastoid air cells (Figure 2 D).

The patient received dual antibiotic therapy with intravenous ciprofloxacin (400 mg every 12 hours) plus piperacillin/tazobactam (4.5 grams every 12 hours) for six weeks, along with local debridement of the granulation tissue. Histological examination of the granulation tissue confirmed the inflammatory nature of the disease. Glycaemic control was achieved with titrated insulin therapy, and serial monitoring of inflammatory markers was conducted. The patient showed clinical improvement with complete resolution of nocturnal otalgia and a reduction in inflammatory parameters. Clinical examination of the ear canal showed reduced inflammation (Figure 2 E). The patient was discharged on oral ciprofloxacin 750 mg twice daily with instructions for regular follow-up. A recent follow-up at one year showed no evidence of recurrence.

Case 3

A 60-year-old white male with T2DM presented with right ear pain that worsened at night and right ear discharge for four months. Clinical examination revealed a congested and oedematous right ear canal with granulation tissue on the floor and anterior wall of the right ear canal (Figure 3A). Cranial nerve examination was clinically normal at admission. Blood tests showed uncontrolled diabetes (as evidenced by monitoring of HbA1c status) and elevated levels of inflammatory parameters, including WBC count, ESR, and CRP. An ear swab was negative for the growth of any organism. CT scan of the temporal bone and MRI of the petrous bone were performed, which showed soft tissue thickening in the right ear canal with bony erosion in the posterior wall of the ear canal and soft tissue thickening in the right middle ear cavity extending into the aditus and mastoid antrum (Figure 3 B and C). PET-CT scan showed a metabolically active lesion involving the right ear canal and middle ear with erosion of the adjacent petrous and tympanic portions of temporal bone (Figure 3 D).

The patient received dual antibiotic therapy with intravenous ciprofloxacin (400 mg every 12 hours) plus piperacillin/tazobactam (4.5 grams every 12 hours) and underwent local debridement of the granulation tissue. Histological examination of the granulation tissue confirmed the inflammatory nature of the disease. Glycaemic control was achieved with titrated insulin therapy, and serial monitoring of inflammatory markers was conducted.

This patient, who initially responded to intravenous ciprofloxacin and piperacillin/tazobactam therapy with a lowering trend in ESR and CRP, developed worsening otalgia and right facial nerve palsy (Grade 4 of the House-Brackmann grading scale) during the course of treatment. The patient was then scheduled for surgery. Subtotal petrosectomy was performed.

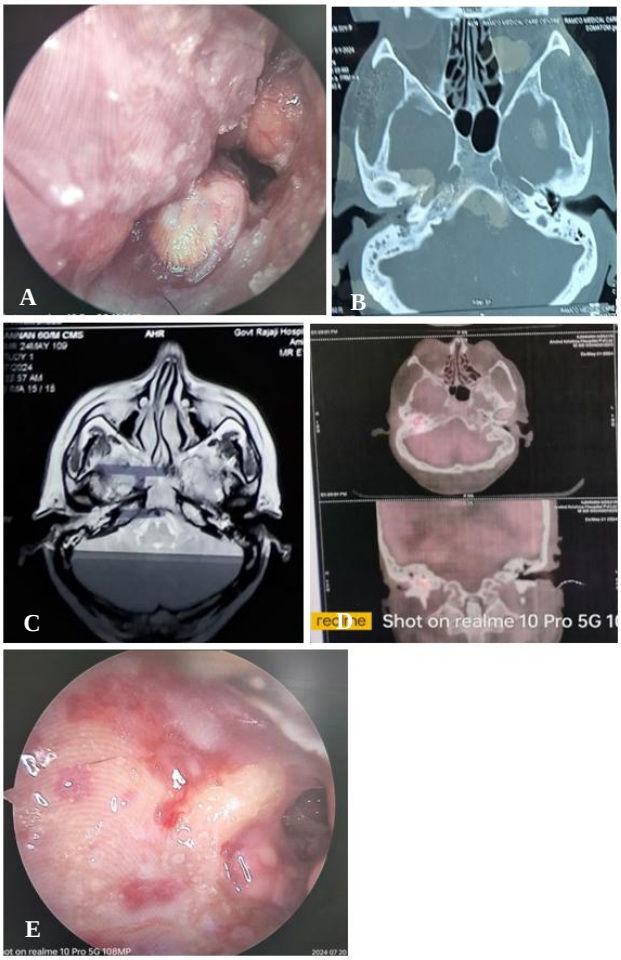

Figure 3 (A-E): Oto-endoscopy (admission), CT temporal bone, MRI skull base, PET-CT and post operative of case 3.

The patient showed clinical improvement with complete resolution of nocturnal otalgia and a reduction in inflammatory parameters (Figure 3 E). The patient was discharged on oral ciprofloxacin 750 mg twice daily with instructions for regular follow-up. A recent follow-up at one year showed no evidence of recurrence.

In our case series, all patients were treated with dual antibiotic therapy consisting of intravenous ciprofloxacin and piperacillin-tazobactam for a minimum period of six weeks. Strict glycaemic control was achieved with insulin management as per the diabetologist's suggestion. Pain management was attained with analgesics under the guidance of the pain clinic. Local debridement of granulation tissue was performed. Out of the three cases, two patients were found to have normal cranial nerve functions. The other patient, who initially responded to antibiotic therapy with lowering levels of ESR and CRP, developed worsening otalgia and right facial nerve palsy while on antibiotic treatment. Then, surgery was planned for that patient, and a subtotal petrosectomy was performed.

Table 1: Pre and post treatment inflammatory markers level.

Case	ESR		CRP	
	Admission	**Post treatment**	**Admission**	**Post treatment**
1	72 mm/hr	14 mm/hr	36 mg/dl	4 mg/dl
2	64 mm/hr	8 mm/hr	24 mg/dl	3 mg/dl
3	84 mm/hr	18 mm/hr	46 mg/dl	6 mg/dl

DISCUSSION

MOE is an invasive and potentially fatal condition[1]. It is characterized by challenging management due to the need for long-term therapy requiring regular monitoring.[6] Misdiagnosed MOE can involve the skull base and cause major complications such as thrombosis of the lateral sinus or internal jugular vein, meningitis, Bezold's abscess, and cranial nerve palsies.[7] There is no standardized protocol for the management of MOE.

The diagnostic criteria of MOE are divided into two categories: obligatory and occasional. The obligatory criteria are: pain, edema, exudate, granulations, micro abscess (when operated), positive bone scan or failure of local treatment often more than 1 week, and possibly pseudomonas in culture. The occasional criteria are diabetes, cranial nerve involvement, positive radiograph, debilitating condition and old age. All of the obligatory criteria must be present in order to establish the diagnosis. The presence of occasional criteria alone does not establish the diagnosis.[8]

When the index of clinical suspicion is high for MOE, CT and MRI facilitate diagnosis and evaluation of disease progression. PET-CT scan helps in assessing the extension of disease and response to treatment. To monitor for residual disease after treatment, radionuclide scans like Gallium-67 or Indium-111 can be used.[1]

Treatment for MOE usually involves antipseudomonal antibiotics (like cephalosporins and fluoroquinolones) for atleast a period 6-8 weeks, local debridement of granulation in EAC and surgery. Some patients don't respond to this treatment because of fungal infection (e.g. *Aspergillus* spp, *Candida* spp) and unnoticed malignancy. Those who are not responding to medical therapy, surgery will be proposed.[1,9]

Literature suggests that because of chance of high recurrence, patients with MOE should be followed up for at least 1 year after treatment. In our case series no recurrence of MOE has been seen after 1 year followup.[10]

CONCLUSION

Treatment of MOE needs multidisciplinary approach which includes strict glycemic control should be attained and Antibiotic treatment of minimum of 6 weeks should be given. Monitoring the progress of the disease and treatment outcome should be based on clinical evaluation like Alleviation of nocturnal otalgia, Disappearance of granulation tissue from the external auditory canal, Improvement in cranial nerve functions if involved, return of the CRP and ESR to near normal values. HRCT temporal bone, MRI and PET-CT are useful adjunct for diagnosis. Surgery should be preserved for special cases, when there is failure of adequate medical treatment.

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