**Malignant or necrotizing otitis externa complicated by peripheral facial paralysis in an immunocompromised diabetic patient with insulin therapy imbalance.**

**Abstract**

Necrotizing otitis externa is a serious condition that is becoming increasingly difficult to treat. In this paper, we report a case of necrotizing otitis externa complicated by facial paralysis that had resisted medical treatment.

Malignant otitis externa (MOE) or necrotizing otitis is a rare but serious infectious disease of the external auditory canal (EAC) with spread of the infection to the soft tissues and adjacent bone. The predisposing factor is diabetes, with elective onset in elderly diabetic or immunocompromised individuals.

Complementary treatment led to a regression of clinical signs and a cure of the infection.

**Keywords:** Diabetes, Otitis, external, necrotizing, treatment

**INTRODUCTION**

Progressive necrotizing otitis externa (PNOE), also known as malignant otitis externa (MOE), is a rare and serious condition of the external auditory canal (EAC) that occurs mainly in immunocompromised individuals, the elderly, and diabetics. It was first described in 1959 by Meltzer and Kelemer, but it was Chandler's work in 1963 and 1968 that led to it being named malignant otitis externa, due to its severity and often fatal progression.

It is an osteitis of the temporal bone, usually caused by Pseudomonas aeruginosa, which starts in the external auditory canal and then spreads to the base of the skull, causing damage to the cranial nerves. It is therefore a diagnostic and therapeutic emergency that can be life-threatening. Treatment is based on glycemic control, local treatment, prolonged antibiotic therapy, and, less frequently, surgery. (1)

**Case Presentation**

A 52-year-old patient with a history of insulin-dependent diabetes treated with Mixtard®30 and Actrapid®30 for 30 years and amputation of the toes, hospitalized for treatment of malignant external otitis on the right side that had been progressing for 6 months.

The symptoms were ear pain that did not respond to the usual painkillers and antibiotics, hypoacusis, and tinnitus. The clinical examination revealed swelling in the temporomandibular joint region, followed by right peripheral facial paralysis on the same side as the affected ear. Otoscopy revealed a narrowed external auditory canal covered with whitish deposits and black spots. A diagnosis of external otitis was suspected. The patient was hospitalized for treatment.

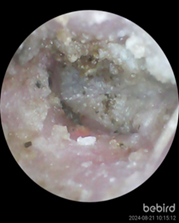
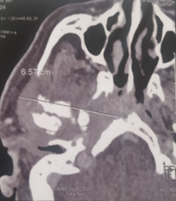
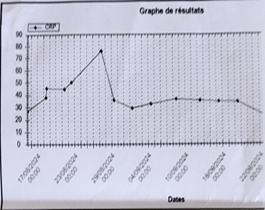
Laboratory tests showed an ESR of 25 Mg/l, which increased gradually despite treatment, hyperglycemia at 28.01 mmol/l, and glycosylated hemoglobin (HbA1c) at 13%. Bacteriological samples showed the presence of staphylococcus.

Tonal audiogram indicated mixed hearing loss in the right ear and a perceptual component in the left ear.

CT and MRI scans indicated acute otitis media of malignant appearance infiltrating the parapharyngeal and periauricular spaces and the right temporomandibular joint.

Bone scintigraphy of the petrous bone revealed active and extensive osteitis in the right petrous bone.

reatment consisted of parenteral dual antibiotic therapy comprising a third-generation cephalosporin (C3G) (ceftriaxone) at 2 g/24 h and a fluoroquinolone (ciprofloxacin 200 mg x 2/24 h). Local treatment consisted of CAE calibration with Pop Otowic k instillation and 5 drops of Antibiosynalar twice daily for 15 days. The condition progressed with persistent symptoms, which led to a change in treatment to Fortum 1 g x3/day and ciprofloxacin 400 mg x2/day and a mastoidectomy to decompress the nerve. After this procedure, the condition improved. The diabetes was managed by diabetologists with Mixtard®30 and Actrapid®30. Oral treatment with ciprofloxacin 1 g/day was continued for 30 days.

The MRI supports a diagnosis of malignant otitis externa infiltrating the peri-auricular space, temporomandibular region, and right parapharyngeal space.

CRT SCAN ROCK in favor of malignant otitis externa with invasion of the temporomandibular region.

Otoscopy of the right ear showing whitish secretions and black spots.

The CRP curve declines from the third week onwards despite treatment.

**Discussion**

The predisposing factor is primarily diabetes (75 to 95%). The occurrence or severity of OEM is not correlated with diabetes imbalance. Bacteriological examination, antibiogram, and medical imaging should be performed systematically. Treatment is based on broad-spectrum antibiotic therapy combined with optimized insulin therapy(2).

OEM is a rare and serious complication of otitis externa. We report a case with a complication of peripheral facial paralysis. This pathology corresponds to cellulitis of the external auditory canal, starting at the osteocartilaginous junction, which leads to perichondritis, then to osteitis of the tympanic bone and temporomandibular joint (3).

In the absence of effective treatment, this infection spreads to the base of the skull, reaching the infratemporal fossa, the parapharyngeal space, the nasopharynx, and the intracranial compartment. The facial nerve is primarily affected in the stylomastoid foramen. The spread of infection to the jugular foramen can lead to thrombophlebitis of the lateral sinus and paralysis of other cranial nerves (IX, X, XI, XII). Our patient had facial involvement, stage IV with Charles Bell's sign, for which an ophthalmological opinion was sought in favor of exposure keratitis and additional treatment (4).

Several authors report a male predominance. The conditions predisposing to infection are mainly elderly diabetic patients who are poorly balanced or immunocompromised. Diabetes is the main predisposing factor for the development of OEM, with a prevalence of 75 to 95%. According to Rubin et al., hyperglycemia is not a factor in the pathogenesis of the disease, but rather the vascular complications of diabetes (microangiopathy) (5).

Histopathological examination of the capillaries in the skin and subcutaneous tissue around the temporal bone showed thickening of the subendothelial basement membrane in diabetics, resulting in tissue hypoperfusion and reduced resistance to infection, which spreads gradually. (5)

Pseudomonas aeruginosa is the germ responsible in more than 95% of cases. It is a commensal germ of the skin, which colonizes the CAE when swimming in a pool or washing the ear. It becomes pathogenic when the immune system is compromised or after a skin breach. Other bacteria may also be involved, such as Staphylococcus epidermidis, which tested positive in bacteriological studies, and, more rarely, fungal agents such as Aspergillus fumigates. (6)

Biological tests generally show elevated CRP levels. This is a non-specific inflammatory marker for diagnosing the disease, but it can be used in follow-up as an indicator of response to treatment(7).

**IMAGERY**

CT and MRI scans can confirm the diagnosis and assess the extent of the lesions, particularly in the bones (temporal bone, base of the skull, temporomandibular joint). It was performed on the patient. It showed thickening of the soft tissues of the EAC and filling of the mastoid cells in all cases, as well as mastoid lysis. However, its usefulness in follow-up is limited, as lesions involving demineralization and erosion of the cortical bone appear late and disappear slowly after healing. Furthermore, the lesions identified by CT are not specific to OENP and can also be seen in cases of malignant tumor pathology of the EAC.

Scintigraphy or PET scanning are more effective. Both of these tests were performed on the patient. (8)

The treatment of OEM is essentially medical and must be initiated as early as possible in a specialized setting. This treatment consists of three essential components: correction of immunodeficiency or glycemic balance with diabetes control, daily local treatment, and prolonged effective systemic antibiotic therapy. The switch to insulin therapy is mandatory regardless of diabetes control, with discontinuation of all oral antidiabetic drugs. Our patient's care was consistent with these guidelines, with intensified insulin therapy based on repeated blood glucose cycles. Local treatment is administered several times a day, with cleaning, calibration, debridement of the external auditory canal, and instillation of antibiotic drops (9).

Currently, the antibiotic therapy recommended by most authors is the parenteral combination of a fluoroquinolone (ciprofloxacin or ofloxacin) with C3G (ceftazidime or ceftriaxone), followed by oral quinolone once clinical improvement is observed.

Anti-Pseudomonas molecules are prescribed as first-line treatment, and the treatment will be adjusted based on the results of microbiological samples [4]. In limited forms of OENP, some authors recommend monotherapy with oral ciprofloxacin at a dose of 1.5 g/day in two doses for 6 to 8 weeks. However, given the increasing frequency of ciprofloxacin-resistant Pseudomonas, the addition of an aminoglycoside or third-generation cephalosporin is essential.

Surgery has a limited role in the treatment of osteitis of the skull base. Some authors believe that it is indicated in cases of unfavorable progression under medical treatment and should be limited to purely local procedures in order to prevent the spread of lesions to healthy bone. It consists of excision of bone sequestra, debridement of infected tissue, and drainage of purulent collections. For others, however, it involves performing a mastoidectomy or even a subtotal petrectomy (depending on the extent of the lesions) with decompression of the facial nerve in cases of facial paralysis. We performed a mastoidectomy due to the persistence of symptoms despite appropriate medical treatment.

The outcome was favorable in the patients in our study.

**CONCLUSION:**

OEN is a rare and serious infection of the EAC that commonly occurs in elderly diabetic or immunocompromised patients. Its diagnosis and management pose a challenge for ENT physicians.

Pseudomonas aeruginosa is responsible for more than 95% of OEM cases.

Its prognosis has improved significantly since the advent of antibiotics effective against this germ and involves several aspects: correction of immunodeficiency or control of diabetes, local treatment of the EAC, prolonged parenteral antibiotic therapy, and mastoidectomy in some cases.

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