

# MALIGNANT EXTERNAL OTITIS

Massoud Moghaddam\*, MD

\* Assistant Professor, Tehran University of Medical Sciences, Faculty of Medicine, Amir Alam Hospital.

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## SUMMARY

Two case reports of malignant external otitis in the elderly diabetics and their complications and management with regard to our experience at Amir Alam Hospital, Department of ENT will be discussed here.

**KEY WORDS:** *Elderly diabetics; Facial paralysis; Microorganism; Necrotising external otitis; Pseudomonas aeruginosa; Sigmoid sinus thrombosis.*

Necrotising external otitis, commonly referred to as malignant external otitis, is an infection which begins from the external auditory canal. It is uniformly caused by the gram-negative *Pseudomonas aeruginosa* and mainly affects elderly diabetics. It spreads to the soft tissues beneath the temporal bone and, if not properly treated, will lead to facial paralysis, mastoiditis, sepsis, osteomyelitis of the base of the skull, sigmoid sinus thrombosis, multiple cranial nerve paralysis, and ultimately death.

Medical treatment is recommended through hospitalization and intravenous carbenicillin and gentamycin. Minor surgical debridement is helpful. All patients should be treated medically as long as improvement is achieved, reserving surgical intervention only if a plateau is reached or symptoms and signs worsen under treatment. With or without a major surgical procedure, it is imperative to continue treatment for at least seven days after apparent improvement in order to avoid the disease recurrence possibly at a site distant from the canal.

A classic description of malignant external otitis and its pathogenesis was given in 1959 by Meltzer and Kelemen.

The disease should be suspected in any patient with an external otitis found to be non-responsive to

the usual methods of treatment. The diagnosis should not be made on the basis of only one examination.

Diabetes mellitus is a chronic disorder of metabolism, especially of carbohydrates, characterised by hyperglycemia and glycosuria. Notwithstanding this, the general concept of most physicians is that diabetic patients are indeed susceptible to many serious infections, presumably due to increased blood sugar levels, decreased activity of formed blood elements such as white blood cells, etc., in response to antigen stimulation and lower state of cellular nutrition.

One of the primary pathologic changes of diabetes is microangiopathy, a term which reflects its effect on the small blood vessels. This takes the form of arteriosclerosis, which consists of a hyaline thickening of the capillary walls primarily due to thickening of the basement membrane and intima. This process is rather a generalised one.

*Pseudomonas aeruginosa* is a ubiquitous opportunistic gram-negative organism not ordinarily found on normal skin or in the external auditory canal. However, with excessive moisture or minor trauma such as scratches caused by foreign objects, wearing a hearing aid, mold, etc, it colonizes in the external auditory canal; and thus, it is responsible

for most cases of the acute external otitis. It cannot usually gain access to the tissues outside the canal but, under certain conditions, it results in a cellulitis or furuncle. As deeper structures become involved, they gain the capacity for selective vasculitis with invasion of arteriolar, capillary and venular walls with or without hemorrhage accompanied by thrombosis with focal coagulation necrosis of surrounding tissues. The necrosis may lack the usual inflammatory cell response.

These characteristics of the organism in combination with the basic pathologic changes of diabetes discussed above, may account for the pathogenesis and relentless course of what is usually a benign and self-limited disease in other individuals.

The incidence of facial paralysis in the patients with malignant external otitis has slightly been decreased.

Facial paralysis, by itself, is primarily due to a physiologic conduction block because of the infection around the nerve in the soft tissues just at its way out from the stylomastoid foramen. If this infection is not controlled, actual necrosis of the nerve will occur. Return of the facial function is not possible in such instances and should not be expected.

Patients with multiple cranial paralyses especially 10th or 11th cranial nerve are considered to be at risk for a thrombosis of the dome of the jugular bulb and sigmoid sinus. Arteriovenographic studies demonstrate a block in the sigmoid sinus and dome of the jugular bulb. These patients have a poor prognosis.

Although the infection usually extends through the floor of the canal into the soft tissues at the base of the skull, it may and frequently extend posteriorly into the bony wall of the external auditory canal and through the mastoid cortex or its tip. In other words, necrosis and sequestration of bone permit the direct extension of the infection into the pneumatized air cells of the mastoid process. This was seen in many patients.

The initial work up of any patient hospitalized for treatment of malignant external otitis should include mastoid x-rays. This will disclose any unsuspected changes and provide a baseline record for possible

progressive disease which would mandate surgical intervention. Even when there is no clinical evidence of middle ear effusion or mastoiditis, x-ray may reveal slight clouding of what is usually a well-pneumatized mastoid process. When the organism gains access to the mucosal lining of the air cells, there is more clouding and in some instances evidence of bone destruction.

Even an elderly diabetic with an external otitis should be given the benefit of our doubt and treated as an outpatient with careful cleaning of various liquids and ointment medicaments. With a severe pain, abscess formation and/or cellulitis, oral or systemic antibiotic therapy is usually indicated. Most instances of external otitis will be cured promptly with or without such treatment. In those instances that a dehiscence appears in the canal accompanied by some granulation tissue, it can be removed gently, and the local treatment and usual supportive measures can be continued. If the infection removes within the several weeks of standard treatment, it is not certainly the malignant variety of the external otitis. On the other hand, if it is proved that it is non-responsive to such usual methods of treatment and if the granulation tissue persists or increases in a size along with the local pain and purulent discharge, malignant external otitis must be considered as a possible diagnosis. The discharge substance should be taken from the canal for culture and sensitive studies. If there is no history of diabetes, a urinalysis and glucose tolerance test should be done to reveal its presence or absence. In the presence of a positive culture for *P.aeruginosa* and the absence of response after two weeks of intensive local therapy, admission to the hospital for intensive intravenous anti-microbial therapy is indicated.

Hospital work up should include mastoid x-ray, routine urine analysis and blood counts, blood urea nitrogen (BUN), creatinine and blood sugar levels, and an electrocardiogram (ECG). In the absence of any history suggestive of allergy to penicillin, treatment should be instituted promptly with 5 gr. carbenicillin intravenously every four hours.

A continuous intravenous infusion of saline or dextrose in water is necessary to provide access for drug administration. One of the newer semisynthetic

penicillin derivatives such as ticarcillin or piperacillin may be substituted. In addition to carbenicillin, an aminoglycoside antibiotic should be added. Gentamycin is the drug with which we have had the most experience.

In the absence of the renal disease and presence of normal blood creatinine levels, it should be given in the dosage of 80 mg every eight hours; it may be given intravenously as a bolus but should not be mixed with the carbenicillin. Tobramycin, which has a similar spectrum of anti-bacterial activity, and/or amikacin or some other aminoglycoside derivatives may be substituted. It is important to use such a drug together with carbenicillin, as it may prevent the development of the organism resistance to carbenicillin.

In the event that the patient has a history of sensitivity to penicillin, treatment may begin with only intravenous gentamycin. The clinical course of the disease and further bacteriological and laboratory studies will be efficacious. If the infection is in the advanced stage of the disease, perhaps with mastoiditis and/or facial paralysis, the patient should be desensitized to carbenicillin.

## CASE REPORT

We have had some cases of external otitis caused by *P.aeruginosa* in the diabetic patients in the past ten years, two of which were of malignant variety.

The first patient was a 72-year-old man with the right facial paralysis, severe swelling in the mastoid area, and extensive infection in the right external auditory meatus.

Mastoid x-ray showed severe destruction in mastoid air cells with erosion in the sinus plate and tympanic tegmen.

This patient suffered from pain in the neck and irregular fever that revealed sigmoid sinus thrombophlebitis.

Biochemical and hematologic tests were done. Fast blood sugar (FBS) was elevated in the diabetic patient who has previously been treated with anti-diabetic medications.

Bacteriologic tests of the external ear discharge revealed *P.aeruginosa*. Because of the generalised weakness and cardiopulmonary disease, surgery was

not performed and medical therapy with gentamycin began, but the patient died on the fifth day of the treatment.

The second patient was an 80-year-old man, visited for the first time, with discharge because of his external otitis, and pain that had not been resolved with different antibiotics.

Bacteriologic tests repeatedly showed *P.aeruginosa* in the external ear.

Treatment with gentamycin for the period of ten days showed some healing, but after the discontinuation of the treatment, ear discharge increased at first, the 6th cranial nerve paralysis occurred two days later followed by facial weakness of the right side. Nothing was evident in the mastoid x-ray and CT Scan of the temporal bone, but granulation tissue was demonstrated in the inferior and posterior walls of the external auditory canal without any malignant changes.

Treatment with amikacin continued for fifteen days. Infection and facial paralysis were recovered, but the 6th nerve paralysis was still present. In the following six months the patient showed no sign of infection.

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