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Original Research Article

Malignant Otitis Externa or Diabetic Ear- A Challange Disease

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Abstract

Malignant otitis externa is a rare but potentially fatal disease of the external auditory canal seen mostly among elderly, diabetic or immune compromised patient. The causative organism is mainly *Pseudomonas aeruginosa*. The disease spreads rapidly, invading surrounding soft tissues, cartilage and bones causing necrosis and even spreading to the cranial nerves. The disease can be fatal if treatment is not started aggressively and timely, especially if spreads outside the external auditory canal with involvement of the cranial nerves. Treatment is mainly medical with antipseudomonal drugs and local debridement. With aggressive treatment the mortality rate of this disease which was 50% in the past has now been reduced to 10-20%. We are our management and results are presented.

Keywords: Malignant external otitis, diabetic, *Pseudomonas aeruginosa*.

Introduction

Malignant otitisexterna(MOE) also known as invasive/granulomatous/necrotizing otitis media is an unusual but potentially fatal infective condition of external auditory canal. Toulmouche was first reported in 1838.Meltzer,1959 reported a case of pseudomonal osteomyelitis of temporal bone. However, first report was published in 1968.The term 'malignant' was first coined by Chandler in 1977, because of invasive infection of external auditory canal and skull base infection which is predominantly occur in uncontrolled elderly diabetic individuals and immune compromised patients. This condition also affects skin and cartilages of ear canal and in severe cases spreads to skull base with involvement of cranial nerves carrying high morbidity and mortality. Uncontrolled diabetes mellitus in elderly individuals is considered as the single most predisposing factor for development of malignant otitisexterna, though it can also in non-diabetic immune compromised patients. The evaluation of extent of disease and response to therapy in malignant or invasive otitisexterna in uncontrolled diabetic patients has always been a problem. The management of MOE has changed since the first case report of Pseudomonas osteomyelitis of temporal bone described by Meltzer, et al. in 1959 and later clinically defined by Chandler in 1968.Traditionally treatment included both surgery and antimicrobial therapy.

Incidence:

The disease is more common in humid and warm climates. Its affects elderly, diabetic (>90%) and immunocompromised patients.

In immunocompromised and AIDS patient, younger age group people may be affected. Males are more affected by MOE than females.

Material and Methods:

All the 12 cases of malignant otitisexterna during a period of 5years from 2006-2011.After proper history and clinical examination, especially predisposing factors, relevant investigations were done including culture and sensitivity of pus, histopathological examination and HRCT with contrast or without contrast. The treatment was started with appropriate antibiotics. Surgery (Modified cortical/Cortical mastoidectomy) was done in most of the cases.Patients were follow up for a minimum of 1 year. Some of the them are still under follow up.We have followed a simplified version of staging from Thakar et al as table I.

Staging: **Stage I**- Necrotising externaotitis {Persistent otalagia, bare bone in EAC, No facial palsy}

Stage II- Limited Skull base osteomyelitis {Lateral to jugular foramen-facial palsy}

Stage III- Extensive skull base osteomyelitis {Jugular foramen and further medially-lower cranial involvement and intracranial extension}

We have also followed a **Nosological criteria**.....

Corey et al,1985(15): Atleast 3 of 5 of the following signs and symptoms:

1. Persistentexternaotitis.
2. Granulation tissue in the EAC.
3. Evidence of mastoiditis or Osteomylitis of the skull base by radiologically or Biopsy.
4. Cranial nerve paralysis.
5. Isolation of Pseudomonas from the EAC or deeper skull site.

Observations:

Total 12 cases studied ,10 cases are males and 2case are female with an average age of 55years. All of them presented to us with partially treated at the primary level with local / parental antibiotics. All of them presented with severe persistent otolagia with purulent discharge, granulation tissue in the EAC, diffuseotitisexterna with sometimes some cases with facial nerve palsy/multiple cranial nerve palsy.

Pathogenesis:

Malignant otitisexterna is an invasive granulomatous infection that invariably originates at the junction of cartilaginous and bony part of the EAC. Its commonly affectly elderly (>50 years) patient who are diabetes or immune compromised. The organisms is mainly pseudomonas aeruginosa. Other organism is staph aureus, proteus species and klebsiella. Diabetic person are more susceptible to this condition irrespective of the type(Insulin dependent or non-insulin dependent, well or poorly controlled)[1].Theories proposed include deficient cell mediated immunity and tissue hypoperfusion secondary to diabetic microangiopathy[2,3].Studies have also shown that of diabetic cerumen has a higher PH and reduced concentration of lysozyme, which could be impair local antibacterial activity.[4]The condition may also originate following syringing of the ear in both diabetic and non-diabetic patients[2,5]. Some reports have shown the condition to be arisen from acute otitis media as seen in 6 out of 15 cases reported by Meyerhoff, Gates and Montaibo in 1977 or also associated with active chronic otitis media as seen in 4out of 11 patients reported by Doroghazi et.al in 1981.[6,7]

The term “Necrotizing” and “Invasive” is used because in most of the infection invades the adjacent deep preauricular tissues into the cartilages and bones resulting in necrosis and osteomyletitis of temporal bone and base of skull. The disease spread from the external auditory meatus rapidly and progressively through

the fissures of santorini and the osseo-cartilagenous junction into the temporal bone and mastoid. The facial nerve lies in close proximity and exits through the styloidmastoid foramen and can easily to be affected. The facial nerve is the most commonly affected nerve;32% incidence has been reported in Chandler's series[8]. Now a day incidence decreased due to recently introduced aggressive medical therapy and does not signify worse prognosis of the disease. From the infection may spread toward the skull base, involving the jugular foramen as well as glossopharyngeal, vagal and spinal accessory nerve. Thepetrous apex may also involved where the abducens and trigemminal nerve may be affected. Although as compared to facial nerve other cranial nerves are involved less frequently, they carry grave prognosis with mortality rate as high as 80-100%. Parotid gland and paranasal sinuses can also be involved by the aggressively spreading infection and also cause venous thrombosis of the jugular vein, leading to cavernous sinus thromobosis. Meningitis is another fatal complication of this disease.

Diagnosis:

The chief complaints will be severe otalagia. Other signs and symptoms are otorrhoea, temporal headache as well as neurological involvement in some cases. On examination inflammatory changes seen in external auditory canal and preauricular soft tissue. The pain may be mimic diffuse otitisexterna or furunculosis. There may be referred pain to TM joint present indicates granulation tissue at the osseo-cartilagenous junction at the floor of external auditory canal is virtually pathognomic of MOE. Necrosis of the cartilage and bony may be seen in some cases. The tympanic membrane in most of cases are intact.

Investigation reveals high leukocytes count, elevated ESR, with average 88mm/hr[9]. Known diabetic need an evaluation of the serum chemistry to determine if the infection affecting their baseline glucose intolerance. Culture and sensitivity from the external auditory canal will identify Pseudomonas aeruginosa as the predominent causative organisms(95%).

Imaging study for determining the presence of disease, extent as well as response to therapy. CT and MRI are helpful to determining the extent of soft tissue, bone erosions, abscess formations and intracranial complications. However, CT scan may not detect early osteomyletitis, as 30-50% of bone destruction may be required for CT to be detect osteomyletitis. MRI provides poor bone resolution. Technetium-99 and Gallium-67 scan are more sensitive but not specific. Indium-111 has same sensitivity as Gallium-67 scan but more specific in cases of inflammatory processes[10,11,12].

Biopsy may help to rule out to maligancy or other pathologies. Nadal described histopathology of 2 temporal bones affected by MOE and determined the infection did not spread through the pneumatized air cells tracts of the temporal bone[13]. Rather infection may spread via vascular and fascial planes on the exiting the temporal bone through the external auditory canal osseo-cartilagenous junction of fissures of Santorini. The condition may be misdiagnosed as acute otitisexterna or otitis media with complication, other differential diagnosis include squamous cell carcinoma, Wegener granulomatosis, eosinophilic granuloma, glomusjugulare, and meningeal carcinoma.

In child (MOE) has no true pathognomic features of MOE. Rubin reported 15 cases of MOE in 1988 which is predominant risk factor was immune dysfunction, not a diabetes. They usually present with sudden onset of earache, ear discharge and hearing loss. Levenson, Corey, Benecke, and Davis have all proposed staging system for MOE based on soft tissue and bony involvement

or neurological involvement, however staging systems are not widely accepted.

Treatments: The guidelines of treatment are as follows-

1. Control of blood sugar level and improve the general condition of the patient.
2. Aural toilet and local derbidement of granulation tissue.
3. Aggressive treament with suitable antimicrobial therapy (Topical and systemic).

We used ciprofloxacin and in some casescephelosporin both iv for a short period and then switching over to oral ciprofloxacin. In according to culture and sensitivity report showing sensitivity of the organism to ciprofloxacin, the drug was tried and came out successful. Therapy was given for prolonged period of about 3months, the end point of treatment mainly decided by clinical correlations.

Surgical debridement was done in most of the cases, which amounted to the removal of all granulation tissue and drilling away of all diseased and necrosed tissue. A canal wall down mastoidectomy, exposing the antrum, lowering the posterior canal wall, exposing the facial nerve upto the stylomastoid foramen and was found to be in one case with vertical segment of facial nerve was involved, facial nerve decompression done. Lastly procedure was followed by wide meatoplasty done.

Results: There was 2 mortalities in our case, died due to the extensive disease involvement. Some cases are followed up till now they are symptomatically better. 2 patient showed show facial nerve palsy after surgical intervention, but not fully recovered. Extremely severe earache in some cases was fully relieved even after treatment.

Discussion:

In our patients all were diabetics and also other comorbities in some cases like chronic kidney disease and hypertension. Microangiopathy in diabetes and vascular abnormalities are in chronic renal disease. Though prolonged antibiotic treatment and surgical debridement could reduce the mortality and morbidity of earche. Good results in terms of resolution of pain are reported only for diagnosed in satge I.

All of our patients were diagnosed before the development of facial palsy, only one patient was presented with the pain and facial nerve palsy. Rest of them presented to us after treated at primary care level including physician, general practitioners and physicians treating diabetes. Greater awareness of this condition will help in early diagnosis. Very high index of suspicion in elderly diabetes or otherwise immunocompromised patients presenting with earache can be help in early diagnosis before the development of facial palsy and thus decreasing the morbidity.

Prognosis:

With adequate and aggerssive antibiotic treatment use the cure rates have improved from 55% in1968 to 74-91% in 1985 to 1989[9]. Chandler had initially reported mortality of 50% which has been decreased, now decreased to 20% due to improved imaging studies, early intervention with antibiotic and surgical process. Most recent studies report a mortality rate of <10% except for those patients with cranial nerve involvement other than facial nerve and those with intracranial complications. Disease recurrence rate is reported in 9-27% of the cases usually seen with inadequate therapy. Early diagnosis is essential. Prognosis to be related directly to the stage that the disease has reached at the onset of treatment. This requires great awareness of the condition. Mainstay of treatment is antipseudomonal antibiotics and surgical debridement.

Conclusion:

An early diagnosis with an aggressive treatment with wide local surgical excision

if required, debridement and prolonged antibiotic therapy usually proves successful.

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