



NECROTIZING OTITIS EXTERNA : A RARE PRESENTATION

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KEYWORDS :

INTRODUCTION

Lateral skull base osteomyelitis (SBO), otherwise known as necrotizing otitis externa (NOE), is a severe invasive infection of the external auditory canal, temporomandibular joint, and skull base, which occurs by spread from the external ear canal through the fissures of Santorini and the osseocartilaginous junction. Diagnosis of NOE is based on history, physical examination, inflammatory markers, and radiological studies. CT scanning allows for the identification of bony erosion. Technetium Tc 99m bone scans also allow for diagnosing bony involvement. Although the Gallium scan is the gold standard for following up patients with necrotizing otitis externa, FDG-PET/CT scan has shown to be a reliable alternative when gallium is not available⁽¹⁻²⁾.

Due to its aggressive nature and associated high morbidity and mortality, prolonged treatment with broad- spectrum antibiotics and possible surgical requirement is the mainstay treatment option for skull base osteomyelitis⁽³⁾. In addition, improving the oxygen perfusion of tissues is thought to strengthen host defenses and lead to oxidative damage of pathogens. This can be done by using hyperbaric therapy as an adjunct to the treatment of SBO⁽⁴⁾.

SBO originating from the otitis media is a rare entity. We report a case of SBO occurring in a gentleman with left sided otitis media and mastoiditis and uncontrolled diabetes.

Case Report

A 72 year old male patient with uncontrolled diabetes mellitus presented to our opd with complain of left ear pain and discharge and showed picture of otitis externa. Patient was given symptomatic treatment and was relieved. After 2 weeks patient presented with severe left otalgia and left sided headache. On otoendoscopy external auditory canal had mucopurulent discharge and pulsating tympanic membrane visualised giving a false image of arterial mass. On diagnostic nasal endoscopy left side middle turbinate was seen to having pale purple mucosa with multiple small white patches and in left nasopharynx granulation tissue was seen obliterating the left Eustachian tube orifice which was sent for histopathology and enlarged torus tubaris.

MRI showed features of aggressive left sided skull base osteomyelitis with ipsilateral otitis media and externa along with mastoiditis extending to left half of skull base, left nasopharyngeal region, pharyngeal spaces with extension into left mastication space and left foramen ovale with very minimal intracranial extension. Edema with erosion noted in lateral and anterior wall of Clivus predominantly in left half and adjacent base of left petrous bone around petrous portion of left intracranial internal carotid artery. It also encases the proximal cervical segment of left internal jugular vein. Small necrotic soft tissue is also seen along the left fossa of rosenmuller blocking the eustachian tube.

The granulation tissue around the left ET orifice was removed and sent for histopathology. Patient underwent left endoscopic modified radical mastoidectomy and debris was sent for tissue culture which revealed Staphylococcus epidermidis (CONS) as the causative agent. KOH staining showed no fungal element. On histopathology of nasopharyngeal mass showed granulation tissue and acute inflammatory infiltrate. No organism, caseation or malignancy.

PET scan was done Patient was treated with iv antibiotics meropenem and linezolid, oral ciprofloxacin, insulin to control diabetes.

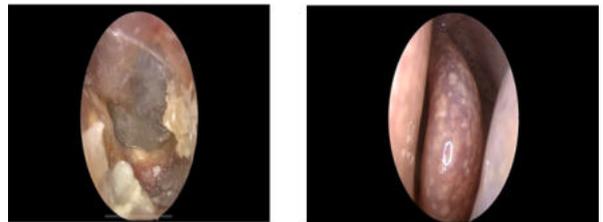


Fig 1 Left Ear- Pulsating TM with edematous EAC

Fig 2 Left Middle Turbinate- Pale mucosa with white patches

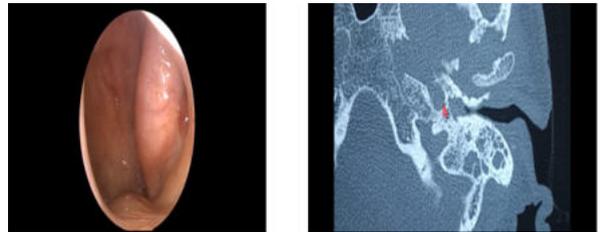


Fig 3 Granulation tissue over the left Eustachian tube orifice and enlarged torus tubaris

Fig4 HRCT temporal bone- Showing dehiscence near carotid giving the pulsation to the tympanic membrane

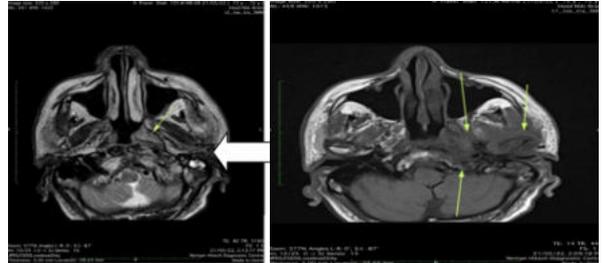


Fig 5 small necrotic soft tissue along left Fossa of Rosemuller, abnormal marrow in left jugular tubercle and hypoglossal canal

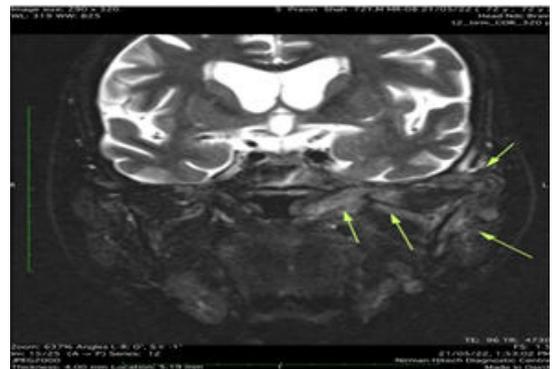


Fig 6 ill defined marrow edema with erosion in basisphenoid, left posterolateral wall of sphenoid sinus, petrous portion of left

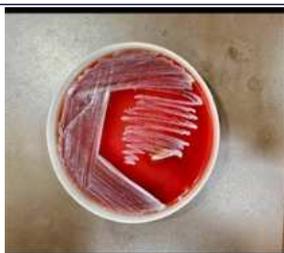


Fig 7 Tissue culture on Blood and MacConkey Agar: Staphylococcus epidermidis

DISCUSSION

Skull base osteomyelitis (SBO) is an inflammatory process that may involve the frontal, sphenoidal, temporal, and occipital bones, caused by an infection or direct head trauma (injuries and iatrogenic trauma)^[5]. Temporal bone involvement usually occurs secondary to necrotizing otitis externa (NOE) or chronic mastoid infections^[6]. The major risk factor for the development of NOE is well-known to be uncontrolled diabetes mellitus, typically in an elderly patient. Both risk factors were present in our patient. The diagnosis of skull base osteomyelitis should be considered in any elderly diabetic patient who presents with otitis media or mastoiditis refractory to a standard course of antibiotic therapy. Men are more commonly affected than women^[7,8]. *Pseudomonas aeruginosa* is the causative pathogen in at least 95% of NOE cases. Patients who have NOE commonly complain of severe otalgia that is not comparable to the clinical findings, and otorrhea that does not respond to first-line topical treatment given for otitis externa^[1]. Even though the patient had prolonged ear pain that did not respond to initial therapy, his symptoms were not always associated with otorrhea and he presented with a pulsating tympanic membrane and ear effusion. His tissue culture from mastoid also did not grow *Pseudomonas aeruginosa*, an expected culprit in SBO originating from otitis externa and rather had *Staphylococcus epidermidis* as the causative agent.

Radiologic features. Imaging modalities used for the diagnosis of skull base osteomyelitis include CT, MRI, technetium bone scans, gallium scans, and indium111-labeled leukocyte scans. CT can define the location and extent of disease at the initial evaluation. However, CT will show bony destruction only when demineralization of 30% or more occurs; once demineralization occurs, CT findings rarely return to normal, and thus it is of little value for monitoring therapeutic effect^[9]. MRI is helpful in determining the full anatomic extent of soft-tissue disease during the initial evaluation of skull base osteomyelitis. It is available for immediate interpretation, whereas most nuclear medicine scans take several days for results to return. However, MRI does not show any bony involvement, and thus it is ineffective as a single radiologic modality. MRI does serve as a valuable guide to the interpretation of technetium and gallium scans, and thus it is extremely useful in the treatment of skull base osteomyelitis^[10].

Treatment and outcomes. Patients with skull base osteomyelitis often experience multiple medical problems associated with poorly controlled diabetes, and they should be carefully monitored for strict blood glucose control. Treatment has evolved from the initial radical surgical approach described by Chandler^[11] in 1968 to the current practice of prescribing long-term, culture-directed antibiotic therapy. Three regimens make up the mainstays of treatment: (1) an aminoglycoside and a beta-lactamase antibiotic, (2) a third-generation cephalosporin such as ceftazidime, and (3) an oral quinolone such as ciprofloxacin. In summary, an absence of otitis externa does not preclude a diagnosis of skull base osteomyelitis, as this condition may originate from any inadequately treated infection near the skull base, including otitis media and mastoiditis. Features suggestive of skull base osteomyelitis include headache in an elderly diabetic man who presents with otitis media or mastoiditis that is refractory to treatment. Investigative radiologic modalities include CT to look for bony destruction and MRI to evaluate soft-tissue involvement. The current treatment of choice for skull base osteomyelitis is long-term culture-directed antibiotic therapy, with the response to therapy being gauged by clinical resolution of symptoms and signs and improvement seen on imaging.

CONCLUSIONS

Skull base osteomyelitis resulting from otitis media is an emerging entity that needs to be further studied in detail. The involvement of

atypical pathogens makes it a more difficult disease to treat compared to necrotizing otitis externa. F-18 FDG-PET/CT should be considered to diagnose questionable necrotizing otitis externa, evaluate the disease extent, and assess response to antimicrobial therapy.

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