

A Case Report: Necrotizing Otitis Externa

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ABSTRACT

Necrotizing Otitis Externa (NOE) is a rare infection of the ear canal with frequent bone erosion. It is also known as Malignant Otitis Externa (MOE), a life threatening infection which affect the external auditory canal and skull base, the infection which also invade the stylomastoid and jugular foramina. Treatment includes antibiotic therapy, hyperbaric oxygen therapy, and surgical therapy. It has a lifetime prevalence of 10% and can arise in acute, chronic and necrotising forms. A 72 year old male was admitted with complaints of anxiety for two days, cough on and off. He has a known complaints of Type II Diabetes Mellitus, Systemic Hypertension, Chronic Kidney Disease (CKD) and Coronary Artery Disease (CAD) and is on treatment.

KEY WORDS

NOE, MOE, CKD, CAD

I. INTRODUCTION

Malignant Otitis Externa is an uncommon potentially lethal infection of the temporal bone primarily affecting elderly diabetic patients. It starts in the external auditory canal and then extends into the temporal bone and adjacent structure^[1]. It is non cancerous, but it can quickly progress in patients auditory canal and has been associated with high mortality rate. The condition have 10% lifetime prevalence and it can be developed as acute, chronic and necrotizing forms^{[2],[3]}. Most common cause of NOE are gram

negative bacteria like *Pseudomonas aeruginosa*. Aged patients are vulnerable to this infection, especially males having Diabetes Mellitus. Other risk factors include HIV, malignancies, and chemotherapy^[4].

NOE usually starts as simple otitis externa with soft tissue infection in otitis and External Auditory Canal (EAC). In this sense, NOE then spreads through the fascial planes and venous sinuses, like otitis media, which follows the pneumatic cavities of the temporal bone. Spread of infection causes bone erosion and stretching of adjacent tissues, and as the condition progresses, it can cause damage to the base of the skull, cranial nerves and intracranial structures^[5]. The infection usually spreads through the osteocartilaginous junction of the EAC and through the santorini fissure, which opens into the cartilage on the lateral side of the EAC. As a result, the stylomastoid and jugular foramina, as well as the hypoglossal canal, become weak.

Infection in this area can cause cranial neuropathy with clinical symptoms such as facial weakness, dysphagia, hoarseness, shoulder weakness, and tongue weakness. If the lesion is moderately advanced, it can include the cavernous sinus, causing trigeminal paralysis and nerve entrapment. This development indicates a poor prognosis due to the large number of diseases that may affect this nerve. Distension of the infratemporal fossa causes stretching of the retrocondyles and parapharyngeal

fat, temporomandibular joints, and muscles responsible for mastication^[6].

Treatment of NOE is multimodal and may include systemic and local antimicrobial therapy, close glucose control, surgical debridement, and hyperbaric oxygen therapy. Surgery is performed when non-surgical treatment has proven ineffective and usually includes procedures such as local debridement, abscess drainage, or removal of bone sequelae^{[7],[8],[9]}.

II. CASE REPORT

A 72 year old male patient was admitted in Nephrology department with complaints of anxiety for two days, cough on and off and earache radiating to jaw. He has a known complaints of Type II Diabetes Mellitus and Systemic Hypertension for 29years, Chronic Kidney Disease for 1.5years, and Coronary Artery Disease for 5 years and on treatment with T.GLIPIZIDE, 5mg, P/O 1-1/2-0, T.DAPAGLIFLOZIN, 10mg, P/O, 1-0-0, for 1.5 years and Insulin used for controlling Diabetes, T. METOPROLOL, 50mg, P/O, 1-0-0 for 1.5years used to treat Hypertension, T. SODIUM BICARBONATE, 500mg, P/O, 1-1-1 and T. FOLIC ACID, 5mg, P/O, 0-0-1 for 1.5years used for management of Chronic kidney disease, T. ASPIRIN + ATORVASTATIN, 75mg+ 10mg, P/O, 0-0-1 for Coronary artery disease. He had a surgical history of Angioplasty (PCI) 3 years ago. He had a social history of alcohol intake and smoking habit but stopped 15 years and 25 years back respectively.

He was conscious, oriented and afebrile. On examination, chest was clear, he was able to move all limbs and GI was soft, non-tender. During the admission time, he had an elevated respiratory rate of 30 breaths/ min, pulse rate of 76 beats/ min and blood pressure on D2 (150/90mmHg). On monitoring the laboratory reports, an elevated parameters such as ESR (78 mmHr), RBS (285 mg/dL), Urea (D1- 62 mg/dL, D2-62 mg/dL, D3-52 mg/ dL), Creatinine (D1-2.4mg/ dL, D2-2.4mg/ dL, D3-2.2 mg/ dL) and declined parameters of Creatinine Clearance (D1-18.92ml/min, D2-18.92ml/min, D3- 20.64 ml/min) and Sodium (129 mEq/L) were observed. His Plain HRCT temporal bone report reveals an abnormal hypodensity filling the left mild ear cavity and partially in aditus/ mastoid antrum/ air cells with sclerosis of mastoid part of temporal bone, minimal erosion of stupum with extension into external auditory canal with mild wall thickening. Faint dehiscence on wall of horizontal part of facial nerve canal - CSOM with

mastoiditis and otitis externa for clinical correlation. Soft tissue density node of size ~ 20 X 15 X 20 mm (upto 50Hu) within the deep lobe of parotid gland. His echo showed regional wall motion abnormalities of left atrial dysfunction territory and overall fair left ventricular dysfunction.

The infection was mainly managed with antibiotic GENTAMICIN + DEXAMETHASONE EAR DROPS, 3^o-3^o-3^o, A/U and the earache of patient was primarily treated with T.TRAMADOL + PARACETAMOL, P/O, (37.5mg+325mg). Other medical condition of the patient was managed with his own medications such as T.GLIPIZIDE, 5mg, P/O, 1-1/2-0, T. DAPAGLIFLOZIN, 10mg, P/O, 1-0-0, T. METOPROLOL, 50mg, P/O, 1-0-0, T. SODIUM BICARBONATE, 50mg, P/O, 1-1-1 and T. FOLIC ACID, 5mg, P/O, 0-0-1, T. ASPIRIN + ATORVASTATIN, 75mg+ 10mg, P/O, 0-0-1. The patient got symptomatically improved and discharged with medications like T. GLIPIZIDE, 5mg, 1-1/2-0, T. DAPAGLIFLOZIN, 10mg, P/O, 1-0-0, T. METOPROLOL, 50mg, 1-0-0, T. SODIUM BICARBONATE, 500mg, 1-1-1 and T. FOLIC ACID, 5mg, P/O, 0-0-1, T. ASPIRIN + ATORVASTATIN, 75mg+ 10mg, P/O, 0-0-1 for 15 days.

III. DISCUSSION

Necrotizing Otitis Externa commonly developed from external auditory canal and may progressed as cellulitis, chondritis, periostitis, osteitis and osteomyelitis. When the infection affect the bone, it is referred as "Skull Base Osteomyelitis"^[10]. Diabetes Mellitus is a major risk factor, which result in poor vascular supply and decreased tissue perfusion^[11]. Infection in the temporal bone may be extended to cranium and leads to cranial nerve palsies. The secretion of neurotoxins or the compressive effect of destructive process through the foramina can be the justifications of occurring palsies. Initially facial nerve get affected due to the anatomical location of the temporal bone. The recovery of patient from the disease with cranial involvement is difficult. Mostly death may occur due to intracranial and treatment complications. Intracranial complications include sigmoid sinus thrombosis and long-term antibiotic therapy induced bone marrow suppression is an example of treatment complications^[12].

Sami Horani, Shiva P.Daram et.al presented a case report on Malignant Otitis Externa. A case of 11 year old male presented with two day history of right otalgia, purulent otorrhea and swelling after hot tub exposure. The right ear

canal contain purulence with relevant erythema, edema, and tenderness along the lobule, tragus and preauricular skin on physical examination. CT scan demonstrated a soft tissue phlegmon, subcutaneous emphysema, gas along pinna, edema around the mastoid tip and skull base and bony changes indicates Malignant Otitis Externa. Initially it was managed with intravenous Meropenem due to bony involvement. He was improved and discharged on the third day although he was returned after one week because of worsening of pain and drainage. A combination of intravenous Meropenem, Clindamycin, Amphotericin was started. He received four serial debridements of the tragus, concha and lateral external auditory canal over 21 days with repeated cultures growing *Actinomyces* and discharged on day 4. Oral Doxycycline of six month course was initiated along with local wound care in every 2 weeks in a clinic. After 2 months, he was identified with external auditory canal stenosis with over 50% obstructions in the lumen of external auditory canal. The patient required a 6 months of Merocel ear wick stenting and it changed weekly, Ciprofloxacin-Dexamethasone ear drops twice daily used along the period. At the fourth month, a Keloid of the right lobule was evolved and managed with Kenalog steroid injections at month 5 and 7. During the follow-up of 8 months, there were no signs of middle ear effusion or external auditory canal narrowing, and audiometric examinations confirmed intact hearing and normal function of the tympanic membrane^[13].

A case report by Kaushik Guha depicts a case of seventy year old man was admitted with complaints of weakness on left side of his face along with pain in the left ear for 1 month, while travelling. He was referred to E.N.T consultation for management. CT elucidated mild age related cerebral atrophy with prominence of cortical sulci and supratentorial ventricular system. MRI interpreted left maxillary sinusitis and left mastoiditis. Nerve conduction velocity test identified right sided axonal type of facial neuropathy. The patient was diagnosed with Malignant Otitis Externa with left side Bell's Palsy of grade VI (complete paralysis) according to the House- Brackmann Classification of Facial Function A.K.A Facial Nerve Grading System. The medical treatment and physiotherapy for Bell's Palsy was started. Galvanic stimulation was given to the left side of the patient. Additionally the patient undergo mirror feedback exercise twice daily twenty times per sitting for face as home programme^[14].

In our case, the disease was diagnosed with physical examination and HRCT temporal bone report. Microbial culture sensitivity test was not conducted to identify the causative organism. Primarily the patient was managed with antibiotic ear drops and analgesic for pain. He was discharged with the following own medication.

IV. CONCLUSION

NOE is a medical condition which requires expertise medical care due to its poor diagnosis. In-order to manage the disease, the clinicians have to assess clinical symptoms and signs as well as radiological imaging and inflammatory markers. Elderly patients should be considered in the same as immunocompromised and diabetic patients in the context of NOE. This case report underscores the importance of clinical suspicion, early intervention, and inter disciplinary collaboration in optimizing patient outcomes.

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