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Surgical Treatment of Malignant Otitis Externa (MOE)-A New Hope Although Challenging

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Case Report

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Abstract

Objective: Malignant external otitis is an aggressive inflammatory condition of the external auditory canal. Most often affects elderly patients with diabetes mellitus. This is a serious disease which spreads in the skull base associated with cranial nerve complications and high morbidity-mortality rate. Malignant otitis externa requires urgent diagnosis and treatment. The primary and most effective treatment is to control the diabetes and to fight infection with the proper antibiotic in adequate dose and duration. If medical treatment fails aggressive surgical management is the only option to save life. We present a short experience in the management of this disease. The aim of this study was to present our experience with the surgical management of malignant otitis externa.

Methods: All patients' records with malignant otitis externa during the last 5 years (2007-2012) were retrieved and reviewed. Diabetes mellitus profile, erythrocyte sedimentation rate, ear swab for culture and sensitivity, computed tomography, were investigated for all patients.

Results: During the last 5 years (2014-2019), 5 patients with the diagnosis of malignant otitis externa were admitted to our hospital for investigation and treatment. All between 60 and 70 years of age, with severe persistent otalgia, purulent otorrhea, granulation tissue in the external auditory canal, and diffuse external otitis, and there were 2 patients with facial nerve palsy. Patients were confirmed to have diabetes. Ear swabs for culture and sensitivity usually revealed Pseudomonas aeruginosa. One patient by systemic antibiotic and two patients were treated by Local debridement and systemic antibiotics were sufficient to control the disease. Two patients were treated by aggressive surgical debridement (skull base debridement) with 360degree Facial nerve decompression. Patients were recovered from dreadful necrotizing infection but with facial paralysis.

Conclusion: Malignant otitis externa is still a serious disease associated with cranial nerve complications and high morbidity-mortality rate. The primary and most effective treatment is to control the diabetes and to fight infection with the proper antibiotic in adequate dose and duration, debridement of necrotic tissue, and sometimes aggressive surgical management. Monitoring of therapy response is done through normalization of erythrocyte sedimentation rate, control of diabetes mellitus.

Keywords: Otitis externa; Malignant otitis externa; Skull base osteomyelitis; Skull base debridement

Abbreviations: MOE: Malignant Otitis Externa; EAC: External Auditory Canal; FN: Facial Nerve.

Introduction

Otitis externa is a common ear infection also known as swimmer's ear. It develops in the ear canal leading to the ear drum. In some cases, otitis externa can spread to surrounding tissues, including bones, jaw & face. This infection is known as Malignant otitis Externa (MOE). If anyone has compromised immune system & aggressive bacteria enter in to his/her ear canal, his/her body will have difficulty warding off infection. If it is left untreated, the infection can spread to his/her brain, cranial nerves & other parts of the body & may cause death.

Malignant otitis externa (MOE) is a rare inflammatory disorder starts in the External auditory canal (EAC). It is an aggressive, rapidly spreading and potentially life-threatening infection of the soft tissues of the external ear and surrounding structures, quickly spreading to involve the periosteum and bone of the skull base [1]. The disease is associated with serious complications with cranial nerve involvement and high mortality and morbidity rate [1-3]. The first reported case was published in 1838 by Toulmouche and the term 'Malignant otitis externa' was coined in 1968 by Chandler et al 3due to its high mortality & aggressiveness, though it is not malignancy or cancer. An alternate name of Malignant otitis externa is Necrotising External Otitis. It can be life threatening if not treated timely. Over 90% of people who develop Malignant otitis externa have diabetes.

It is the end stage of a severe infection that originate from the EAC and progresses through cellulitis> chondritis > periostitis > osteitis and finally osteomyelitis. Once periostitis develops this progress rapidly across the skull base. '*Pseudomonas aeruginosa*' is the most common pathogen [2-4] and is responsible in over 95 percent cases. *Staphylococcus aureus; Proteus mirabilis*; and some species of fungi, such as aspergillus and Candida species, have also been described to cause MOE [5]. It most commonly affects elderly diabetic patients who may have an impaired host response to Pseudomonas.

Malignant otitis externa is a clinical diagnosis made on the basis of pain which get worse when moving head, , exudate(persistent & foul smelling greenish or yellowish discharge), granulation, hearing loss, itching and edema of the EAC and can be strongly supported by some investigations .Management options include aural toileting, long course of topical and systemic antibiotic, hyperbaric oxygen therapy and when all measures fails and if the patient is clinically deteriorating-last option is surgery [6,7]. Here we present a case who needed extensive wound debridement due to unresponsive malignant otitis externa.

Case Report

A 70 years male diabetic, nonsmoker patient presented to us with severe pain in left ear for 3 months, discharge from same ear with hearing impairment for 1 month. According to the statement of the patient he was relatively well 3 months back when he developed occasional low-grade pain in left ear that became sever, excruciating and persistent within few days. The pain mostly aggravates at night. The pain was stabbing in nature and felt deep inside the head and often radiates to teeth and jaw. it was so severe that he had to remain asleep overnight for last 1 month. Medication failed to relieve pain. For last 1 month he was noticing scanty, purulent discharge from his left ear that was not foul smelling and not blood tinged. He also noticed progressively developing hearing impairment with noisy sensation in ipsilateral ear. The patient was diabetic and hypertensive, had no vertigo or dizziness. He visited several ENT specialists, took several antibiotics like cefuroxime, ceftriaxone, cefixime, ciprofloxacin etc. over last 3 months (both local and systemic), but no improvement.

On examination

Clinical examination: There was purulent discharge in the EAC completely obscuring the view of TM. It was not foul smelled. A large polypoidal lesion was noted on posterior aspect of EAC just near the TM and obscuring the view of TM. It was non-tender and did not bleeds on touch. Rinne test was negative on left side and weber lateralized to left ear. ABC was equal. Facial Nerve was intact, fistula test was negative.

Lab Investigation: Total count of WBC was 15,500 per liter. Neutrophil count was 12,400 or 80%, ESR 115 mm in 1st hour. High blood sugar (uncontrolled Diabetes)

CT Scan of Temporal bone: Soft tissue density noted within the left EAC as well as within the tympanic cavity and mastoid antrum, there was destruction of mastoid air cell system. The lesion was overlying the facial canal, partially eroded the tegmen tympani plate at the attic region. Ossicles were partially eroded and displaced. Post contrast scan shows no abnormal soft tissue enhancement. Facial nerve, Semicircular canal, cochlea appeared to be normal (Figures 1 & 2).

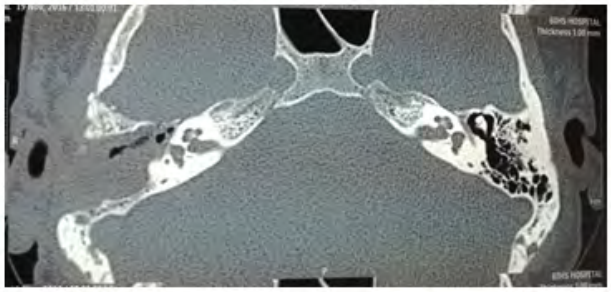

Figure 1

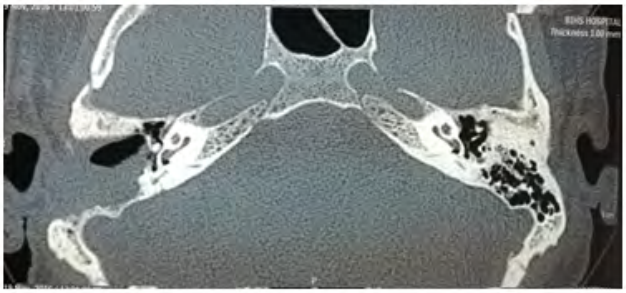

Figure 2

Figures 1& 2: CT Scan of Temporal bone.

Initially we started with injectable ciprofloxacin and antibiotic ear drop prepared with steroid with other symptomatic treatment, later we started ceftazidime injection also. Blood sugar was strictly controlled. We monitored ESR on every 2nd day, monitored patient clinically but there was no improvement rather than deterioration. As the patient was suffering from pain for last 3 months & there was no clinical improvement inspite of medical treatment. So, we made our decision to operate the patient after 18 days of admission with satisfactory control of blood sugar we went for wound debridement.

Findings: there was extensive granulation tissue in the mastoid cavity, attic area, ET area and also extending through the stylomastoid foramen. Tagmen was found partially eroded, incus was absent, the external canal polyp found in continuation with the mastoid antral granulation tissue by eroding the posterior canal wall. Tympanic segment of FN was exposed.

Disease cleared from these areas except stylomastoid foramen area, Specimen was sent for histopathological examination. Preoperatively we had only temporal bone CT-scan, but it was done 10 days before the surgery. We had no neck CT-scan, Frozen section was not available in out institute. So, we had to wait for histopathology. No significant improvement, so after histopathological exclusion of malignancy we re-explored the operated site along with neck. Vascular triangle of neck was explored with adequate neurovascular control. Styloid process was removed.

Findings

There was extensive granulation tissue with mucopus in middle ear cavity involving the hypotympanum, jugular foramen, stylomastoid foramen, perifacial area & petromastiod to styloid process. Facial nerve (FN) was skeletonized from the tympanic segment to the parotid entrance point and trans positioned anteriorly to have a wide exposer. The sinus plate was made paper thin. There was extensive granulation tissue and necrotizing cellulites involving the tendon of Sternomastoid and digastric muscle around the styloid process. All apparent looking diseased tissues were excised. FN was repositioned by making a new canal just deep to the natural canal as it was already dissected. Temporalis fascia was kept over the FN with surgicel over that. Mastoid cavity was obliterated partially by posteriorly based masculoperiosteal flap, surgicel, gel foam and pieces of conchal cartilage (Figures 3 & 4).

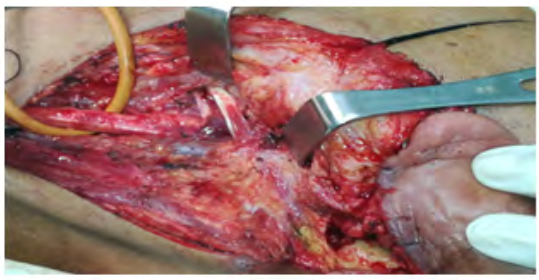

Figure 3

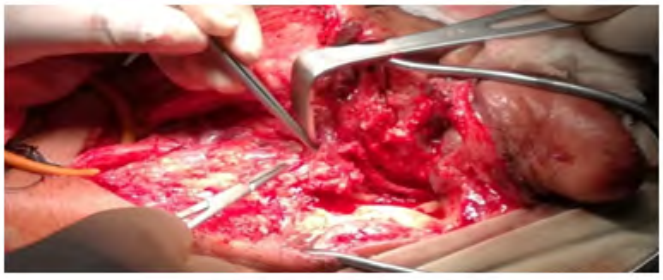

Figure 4

Figures 3& 4: The wound was closed in different layers.

Post-operative recovery was uneventful, we noticed partial weakness on left eye closer with angle of mouth. We kept the patient in ICU for observation. Postoperatively we started with injectable meropenem, ciprofloxacin and steroid with insulin. Stopped steroid on 3rd POD. FN function was same as it was on 1st Post-operative period. He was discharged 1 month after surgery and followed up regularly. 3 months later we re-examined the patient under microscope and noticed little piece of granulation tissue over the sinus plate and advised him to be on contact every 15 days interval for next 3 months (Figures 5 & 6).

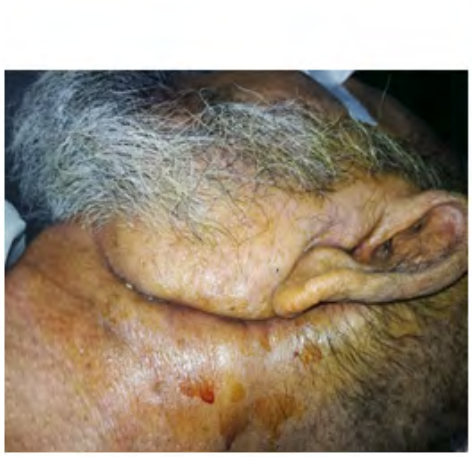

Figure 5

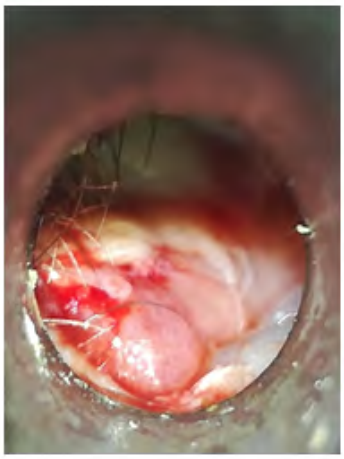

Figure 6

Figures 5& 6: Noticed little piece of granulation tissue over the sinus plate.

Discussion

MOE is a rapidly spreading inflammation of the skin of external auditory canal and spreads to involve the middle ear, mastoid air cell system, skull base and sometimes beyond that. Most cases starts with a simple otitis externa overwhelmed with *Pseudomonas aeruginosa*, into a previously traumatized epithelium of the EAC, exclusively in an elderly diabetic patient [5,6,8]. Severe earache exacerbate at night with or without purulent discharge and pruritus, swollen tender EAC with granulation tissue in the floor of canal at the junction of bone and cartilage, conductive HL and uncontrolled DM, all are the diagnostic criteria of OEM.

Cranial nerve involvement, particularly if multiple, is associated with extremely high mortality [3,9]. Frequently these patients comes with complications specially FN paralysis, after involvement of FN at stylomastoid foramen other cranial nerves can also be affected as a consequence. Granulation tissue is formed in the floor of EAC, mostly at the bony cartilaginous junction and spreads to the skull base via fissures of Santorini and through tympanomastoid suture to involve the stylomastoid foramen and jugular foramen. Trismus may occur secondary to involvement of temporomandibular joint and masseter myositis. It causes necrosis of adjacent muscles and tendons around the styloid process and periosteum may lead to form micro-abscess.

Persistent granulation tissue in the external auditory canal in an elderly, diabetic or immunocompromised patient in-spite of adequate medical treatment is the single most important clue to the diagnosis [3,6]. Biopsy for histopathological examination to rule out malignancy and a culture at that time to confirm presence of pseudomonas is almost diagnostic. Elevated ESR is common and has a Prognostic value as it normalizes after treatment. CT scan helps to evaluate the extension of bony destruction and MRI to identify soft tissue damage. In bone scintigraphy, demineralization is not essential to visualize the lesions; more helpful in monitoring MOE is the scanning with gallium 67 than with technetium 99 [10].

Management options includes medical and surgical. Although there are no unified guidelines regarding the treatment of MOE and the optimal duration of treatment is unknown, most authors advise oral or intravenous antibacterial therapy usually administered for 4-6 weeks [5,7,11]. Medical management includes early detection, strict control of diabetes and intravenous antibiotics, preferably 3rd generation cephalosporin (Ceftazidime) or Piperacillin+tazobactum with or without amynoglycosides to clear the disease process. In some nonresponsive cases voriconazole also advocated by Isa kaya, et al. [5] in their study. In special cases IV metronidazole is also added initially to cover anaerobes.

Surgery should be preserved for special cases when there is failure of adequate medical treatment. Raines JM, et al. [6] recommended radical surgery in patient in whom there is sign of acute infection for 2 weeks after institution of medical therapy along with persistent granulation tissue in EAC and cranial neuropathy during treatment. Strict control of diabetes and improvement in immunocompromised status is necessary. Early debridement of granulation tissue and osteitic bone in the external canal, middle ear, mastoid and skull base areas, drainage of abscess pockets and necrotic tissue can relieve pain and shorten the antibiotic therapy and hasten healing process [6,8].

In our case, patient received multiple 3rd generation cephalosporin's, ciprofloxacin for 3 months before getting admission in our hospital. We did not start amynoglycoside to avoid possible ototoxicity to only hearing ear. In our patient the pain was so intolerable that he used to hit his head to wall several times even after admission. We started injectable ciprofloxacin 12 hourly and also ceftazidime for 18 days but no improvement. Patient had received 3 months course of different systemic antibiotic but no improvement occurs. So, we operated him with the intention of maximum wound debridement and removal of bony sequestrum (so called skull base wound debridement). Post operatively pathedine was kept for 2 days with meropenum as antimicrobial. Pathedine was stopped on 2nd POD, diclofenac suppository was given for another 2 days. His pain was significantly decreased from 1st POD onwards. From 5th POD only paracetamol tablet was enough. Post operatively there were some complications like electrolyte imbalance and grade IV facial palsy. All were treated accordingly but there was permanent grade III facial palsy. Our patient was satisfied with that as he was pain free. Later, we offered facial reanimation procedure for facial asymmetry, but he denied any further surgery.

Immediate postoperative care of resulting mastoid cavity, local application of broad spectrum antibiotic steroid ointment to the meatal opening and drops into cavity for 7 to 10 days is very important. Prevention of contamination of cavity by placing cotton ball soaked in methylated spirit and long use of vinegar solution diluted equally with normal saline as drops into the cavity will help quick healing and prevents occurrence of perichonditis. Multivitamin with trace elements like zinc can hasten wound healing.

Conclusion

Malignant otitis externa is a notorious disease that gives significant morbidity to the patient. Early diagnosis and adequate medical treatment can save a patient from these unfortunate consequences. A high index of suspicion is mandatory to diagnose these patients at their early onset. Surgery should be avoided whenever possible but should not be delayed if there is evidence of cranial nerve involvement. If adequate current medical therapy fails to improve the condition within an expected period, only surgery is the best option as that we had done in this case.

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