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Managing Skull Base Osteomyelitis: A Case of Concurrent Bacterial and Fungal Infections in the Ear

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Abstract: ***Background: Skull base osteomyelitis is a severe infection with a high risk of complications, particularly in immunocompromised individuals over 65. It often originates from nearby tissue infections and can progress to neuroinfections. In cases of malignant otitis externa, inflammation may extend to the cartilages through Santorini fissures and the tympanomastoid suture, leading to osteitis of the temporal bone. Initial symptoms typically include otalgia, headache, ear discharge, and conductive hearing loss. Involvement of the stylomastoid foramen can cause facial nerve palsy, while inflammation of the jugular foramen may lead to paralysis of lower cranial nerves. Management generally involves prolonged antibiotic therapy and surgical debridement of infected tissues. Observations/Results: A patient with chronic suppurative otitis media in the right ear was diagnosed with bacterial skull base osteomyelitis and fungal skull base osteomyelitis concurrently. Due to the severity of the fungal infection, the patient was urgently referred to a specialized skull base surgeon for surgical debridement. This procedure aims to remove infected tissue and prevent further complications. Discussion and Conclusions: Early and accurate diagnosis is crucial for effective management of skull base osteomyelitis. Timely intervention helps prevent disease progression and reduces morbidity and mortality.***

Keywords: Otology; skull base; osteomyelitis; temporal bone

1. Introduction

Skull base osteomyelitis is a severe condition with a high risk of complications in immunocompromised individuals over 65. It arises from nearby tissue infections and can lead to neuroinfection. In malignant otitis externa, inflammation can spread to cartilages via Santorini fissures and tympanomastoid suture, progressing to osteitis of the temporal bone.

There are several possible origins of skull base osteomyelitis, but the most frequent one is infection that spreads from neighbouring structures such the mastoid, middle ear, or paranasal sinuses.[1] A direct blow to the skull, surgical problems, and infections that spread from a distant sites other possible causes [1].

Particularly vulnerable are immunocompromised people, in whom this condition is commonly seen [2]. Furthermore, this disease's severity and mortality risk are linked to facial nerve paralysis, which is very frequently associated with it [2].

Stylomastoid foramen involvement may cause facial nerve palsy, while jugular foramen inflammation can result in paralysis of lower cranial nerves.

Initial symptoms include otalgia, headache, ear discharge, and conductive hearing loss.

Treatment involves prolonged antibiotic administration and debridement of infected tissues.

Compared to its bacterial counterpart, fungal MOE is more aggressive and has subtler signs. When MOE exhibits resistance to antibacterial medications, a fungal aetiology for MOE may be suspected(3).

The best course of treatment, preferred antifungal medication, and diagnosis are not all fully covered by the current standards. In order to optimise the diagnosis and treatment plan, the multidisciplinary care of this disease should benefit from the description of these uncommon clinical instances.

2. Case Report

A 78-year-old man with no known health issues presented with painless right-sided ear discharge, headaches, occasional giddiness, loss of appetite, weight loss, and recurrent falls. Examination revealed a large central perforation. Imaging initially suggested nasopharyngeal carcinoma, but biopsy results were negative. Ear swab showed pseudomonas, and an 8-week course of INJ MEROPENEM led to improvement.

Three months later, the patient developed foul-smelling, purulent left ear discharge, headaches, and a large central perforation. Culture revealed Candida Auris. MRI confirmed left CSOM with fungal skull base osteomyelitis. The patient received treatment with INJ MEROPENEM and was subsequently referred to a skull base surgeon

MRI brain and CECT neck initially indicated nasopharyngeal carcinoma, prompting diagnostic nasal endoscopy and biopsy, which ruled out malignancy.

Following which CECT OMC was done revealing irregularly enhancing soft tissue density in the nasopharyngeal region, suggestive of skull base osteomyelitis, and a soft tissue density in the right middle ear and mastoid cavity, indicative of chronic suppurative otitis media.

Culture swab from the right ear showed heavy growth of Pseudomonas Aeruginosa.

Repeat MRI brain with contrast showed altered signal intensity and enhancement in the clivus, adjacent prevertebral soft tissue, nasopharynx, and left parenchymal space, along with a peripherally enhancing collection in the prevertebral space.

Culture swab identified Candida Auris resistant to Voriconazole, Fluconazole, and Amphotericin B.

A diagnosis of previously treated chronic suppurative otitis media in the right ear, accompanied by bacterial skull base osteomyelitis, along with a concurrent diagnosis of left chronic suppurative otitis media was made, but with the added complexity of fungal skull base osteomyelitis. Recognizing the severity of the fungal infection and its progressive nature, the patient was promptly referred to a specialized skull base surgeon. The recommended course of action involves surgical debridement to address the advancing fungal skull base osteomyelitis. This intervention aims to remove infected tissue and mitigate further complications, highlighting the urgency and necessity of the surgical approach in managing the patient's condition.

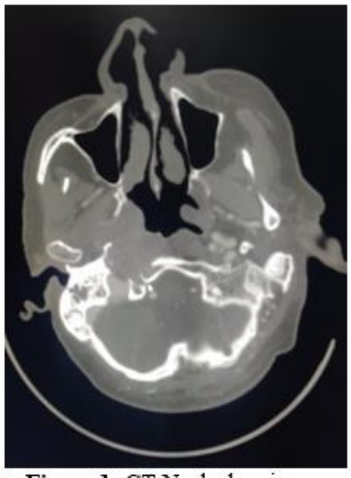

Figure 1: CT Neck showing irregular lesion involving the right lateral wall & posterior wall of nasopharynx with bony erosions

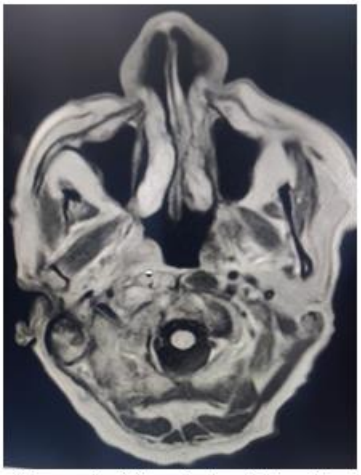

Figure 2: Altered signal intensity involving right lateral wall of nasopharynx and right parapharyngeal space

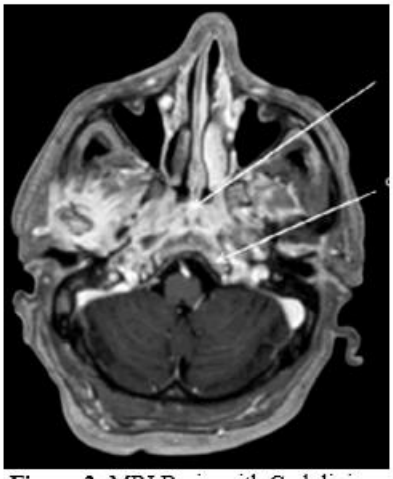

Figure 3: MRI Brain with Gadolinium enhancement involving the nasopharynx, retropharyngeal space, clivus, and masticator space.

3. Discussion

The initial documentation of progressive temporal bone osteomyelitis was by Toulmouche in 1838.[6] Meltzer and Kelemen first described otogenic SBO in 1959 [7]. The term "malignant otitis externa" is a misnomer since it does not denote a neoplastic disease [8] and is used interchangeably with SBO. Chandler introduced this term in 1968 to reflect the aggressive destruction caused by Pseudomonas aeruginosa in the ear canal and its extension to the skull base [9]. Petrak et al. reported the first case of fungal malignant otitis externa in 1985 [10]. SBO risk factors include age over 60, diabetes mellitus, and an immunocompromised state, with diabetes being a significant comorbidity in up to 90-100% of SBO patients [11]. Other predisposing conditions include HIV infection, chemotherapy-induced aplasia, and chronic leukemia [10, 11]. In fungal SBO cases, over 70% have diabetes mellitus [5]. Fungal infections in SBO can spread via several routes:

a) Through the external ear canal, moving through the Santorini fissures to the tympanomastoid suture [9, 11]
b) Via the internal acoustic meatus in fungal meningitis cases
c) Through the Eustachian tube
d) Through hematogenous spread, with very rare cases following a paranasal sinus fungal infection [9].

SBO development in diabetic patients is linked to endarteritis and microangiopathy causing small vessel obliteration [11].

Aspergillus fumigatus is the most common fungal agent for SBO [9, 11], though cases involving Candida species have also been reported [4, 12]. A definitive SBO case is characterized by skull base infection with localized symptoms, radiologic bone erosion, and organism isolation from the affected bone, while a probable SBO is defined by organism isolation from a non-bone source, such as an ear swab [5].

Fungal SBO typically presents more rapidly (average presentation time of 8 weeks) compared to bacterial SBO (26 weeks) [5]. Common presentation includes a patient over 60 with diabetes, experiencing persistent earache and headache, often without fever, and potentially swelling in the preauricular area [7]. Blood tests usually show no leukocytosis but elevated erythrocyte sedimentation rate (ESR) [11–13]. Severe cases may involve cranial nerve palsy, commonly affecting the facial nerve (cranial nerve VII) due to its proximity to the stylomastoid foramen, and less commonly, the glossopharyngeal, vagus, and spinal accessory nerves (cranial nerves IX, X, XI) [5, 11, 12]. Clinical examination often reveals granulation tissue in the ear canal. Our case mirrored others where Candida albicans was cultured from the ear swab

and biopsy of granulation tissue was malignancy-negative [7, 12, 13]. Granulation tissue presence suggests a favorable treatment outcome in fungal SBO [10], though squamous cell carcinoma of the temporal bone must be considered in persistent cases unresponsive to treatment [8]. Thus, tissue biopsy under general anesthesia may be necessary for probable SBO cases.

Fungal SBO typically originates from the middle ear cavity or mastoid air cells [9–11]. Our HRCT imaging showed middle ear destruction and soft tissue density in the mastoid air cell system [12, 13], but did not affect the ossicles or scutum. While HRCT detects bony erosions well, it is inadequate for monitoring treatment response, necessitating additional imaging like Technetium 99m-methyl diphosphonate (Tc-99m Mdp) bone scans and Gallium scans to diagnose and monitor SBO [11].

Treatment involves prolonged antibiotic therapy with quinolones due to their good bioavailability and bone penetration [9], and strict blood sugar control. For fungal SBO, systemic and topical antifungals are essential [12, 13]. Surgical treatment is limited to biopsy, debridement, and abscess drainage [5, 10, 11]. Hyperbaric oxygen therapy has been suggested as an adjunct treatment but is not commonly practiced due to limited evidence and availability [5, 12]. Long-term survival depends more on disease severity than on specific treatments [10], with prognosis heavily influenced by the patient's immune status [9].

4. Conclusion

The effective management of skull base osteomyelitis necessitates meticulous attention to detail to ensure an early and accurate diagnosis. This proactive approach is crucial in implementing timely interventions, thereby preventing the further spread of the disease. By addressing skull base osteomyelitis promptly, healthcare professionals can mitigate the associated morbidity and mortality, underscoring the importance of swift and comprehensive medical attention in enhancing patient outcomes.

References

[1] Osteomyelitis: diagnostic and therapeutic challenges among various presentations. Asian J Neurosurg. 2018, 13:959-70. 10.4103/ajns.AJNS_90_17
[2] Rothholtz VS, Lee AD, Shamloo B, Bazargan M, Pan D, Djalilian HR: Skull base osteomyelitis: the effect of comorbid disease on hospitalization. Laryngoscope. 2008, 118:1917-24. 10.1097/MLG.0b013e31817fae0d
[3] Di Lullo AM, Russo C, Grimaldi G, et al. Skull Base Fungal Osteomyelitis: A Case Report and Review of the Literature. Ear, Nose & Throat Journal. 2021;100(10_suppl):1089S-1094S. doi:10.1177/0145561320936006
[4] Volsky PG, Hillman TA. Fungal skull base osteomyelitis: Emerging microbial identification techniques. Laryngoscope 2017; 127(1): E5–E7.
[5] Blyth C, Gomes L, Sorrell T, Da Cruz M, Sud A, Chen SA. Skull-base osteomyelitis: fungal vs. bacterial infection. Clinical Microbiology and Infection 2011; 17(2): 306–11.
[6] Mortazavi MM, Khan MA, Quadri SA, et al. Cranial Osteomyelitis: A Comprehensive Review of Modern Therapies. World Neurosurgery 2018; 111: 142–53.
[7] Meltzer PE, Kelemen G. Pyocyaneous osteomyelitis of the temporal bone, mandible and zygoma. Laryngoscope 1959; 69(10): 1300–16.
[8] GleesonMJ,ClarkeRC.Scott-Brown'sOtorhinolaryngology:Headand Neck Surgery. 7th Ed. CRC Press 2008.
[9] Stodulski D, Kowalska B, Stankiewicz C. Otogenic skull base osteo- myelitis caused by invasive fungal infection. European Archives of Oto-Rhino-Laryngology and Head & Neck. 2006; 263(12): 1070–6.
[10] Yao M, Messner AH. Fungal malignant otitis externa due to Sce- dosporium apiospermum. Annals of Otology, Rhinology & Laryngol- ogy 2001; 110(4): 377–80.
[11] Carfrae MJ, Kesser BW. Malignant otitis externa. Otolaryngologic Clinics of North America 2008; 41(3): 537–49.
[12] Bowles PF, Perkins V, Schechter E. Fungal malignant otitis externa. BMJ Case Reports 2017; 2017: bcr2016218420.
[13] Shelton JC, Antonelli PJ, Hackett R. Skull base fungal osteomyelitis in an immunocompetent host. Otolaryngology – Head and Neck Sur- gery 2002; 126(1): 76–8.