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Case Report

Skull Base Osteomyelitis: From Subtle Signs to Severe Complications

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Abstract

Skull Base Osteomyelitis (SBO) is a rare yet severe infection that can be challenging to diagnose, often complicating otomastoid or sinus infections. It is a well-recognized complication that can occur following treatment for head and neck cancer. Early detection is crucial for timely and effective treatment. Unfortunately, mortality rates are high, and the prognosis tends to be poor. Here, we present the case of an 89-year-old woman who underwent parotidectomy and chemotherapy for parotid adenocarcinoma, further complicated by SBO.

Keywords: Skull Base Osteomyelitis; Parotid adenocarcinoma; MRI

Introduction

Skull Base Osteomyelitis (SBO) is a rare but serious infection that primarily affects immunocompromised patients or those with head and neck malignancies. Its insidious onset and nonspecific symptoms often delay diagnosis, increasing the risk of severe complications. Post-surgical changes in head and neck cancer patients can mimic infection, complicating diagnostic efforts. Imaging and microbiological studies are critical to confirming SBO, particularly in cases where malignancy recurrence is suspected. We report the case of an 89-year-old woman with a history of parotid adenocarcinoma treated with parotidectomy and chemotherapy, complicated by SBO.

Case Presentation

We report the case of an 88-year-old woman with a history of adenocarcinoma of the right parotid gland, previously treated with parotidectomy followed by adjuvant chemotherapy. She initially experienced an uneventful post-treatment course; however, over the following months, she developed progressive right-sided facial weakness, associated with ipsilateral hearing loss and otalgia.

Her symptoms gradually worsened, leading to complete right-sided facial paralysis, persistent otorrhea, and trismus, significantly impacting her ability to speak and eat. Additionally, she reported dysphagia and hoarseness, suggesting involvement of the lower cranial nerves. There was no reported history of fever, recent trauma, or immunosuppressive therapy beyond her prior chemotherapy.

On physical examination, she appeared frail but afebrile. Neurological assessment revealed right-sided facial nerve palsy, reduced gag reflex, and diminished hearing on the right. Otoscopic examination showed purulent otorrhea.

There was tenderness over the mastoid region, but no overt signs of fluctuance or erythema.

Contrast-enhanced MRI of the face revealed an abnormal signal intensity centered on the right mastoid bone, extending to the lateral mass of C1, the right lateral clivus, and the petrous apex. The lesion exhibited low signal intensity on T2-weighted and T2 FatSat sequences, with mild enhancement following gadolinium administration.

Additionally, MRI demonstrated thrombosis of the right internal jugular vein, with extension into the sigmoid and transverse sinuses. These findings were consistent with SBO complicated by intracranial extension.

The patient was initially started on intravenous broad-spectrum antibiotics, including piperacillin-tazobactam and vancomycin. Following culture results confirming Pseudomonas aeruginosa as the causative pathogen, the regimen was adjusted to ceftazidime for targeted antimicrobial therapy.

Anticoagulation therapy was initiated to manage the jugular vein thrombosis and prevent further propagation. The patient was initially started on low-molecular-weight heparin (LMWH), with a subsequent transition to oral anticoagulants. Pain management was ensured with acetaminophen and low-dose opioids, providing adequate symptom control. Nutritional support was prioritized to address her frailty and support recovery. Physical therapy focused on facial nerve exercises to improve paralysis and prevent contractures.

After two weeks of intravenous antibiotic treatment, the patient showed significant clinical improvement, with a marked reduction

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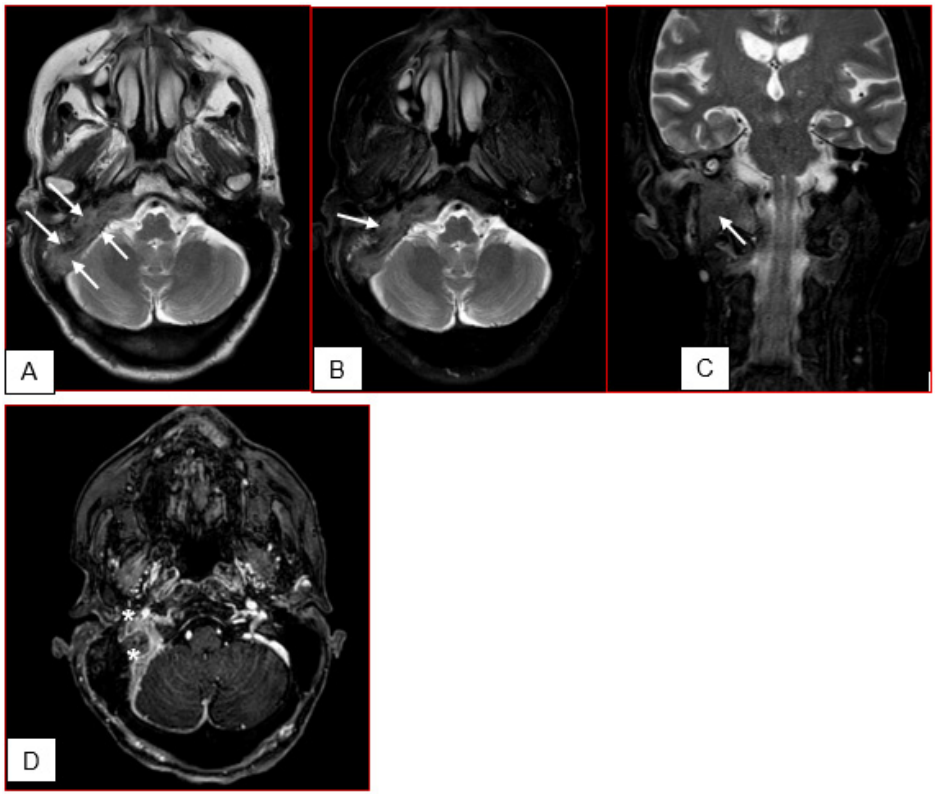

Figure 1: Axial T2-weighted sequence (A), axial and coronal T2 FATSAT sequences (B and C), and post-contrast sequence (D) demonstrate an abnormal signal centered on the right mastoid bone. The lesion shows low signal intensity on T2 and T2 FATSAT sequences (arrows, A and B) and exhibits mild enhancement following Gadolinium administration (D). The signal abnormality extends to involve the right lateral clivus, the lateral mass of C1, the petrous apex, and the jugular foramen. Additionally, there is evidence of thrombosis of the internal jugular vein, extending into the right sigmoid and transverse sinuses (asterisk).

in facial pain and partial recovery of facial nerve function. The facial weakness had diminished, although there was still mild residual asymmetry. Repeat MRI at three weeks revealed a notable decrease in soft tissue inflammation and stable bony lesions, with no evidence of new pathological changes or progression.

After completing six weeks of intravenous antibiotics, the patient was transitioned to oral ciprofloxacin for an additional six weeks. Follow-up imaging during this period showed no signs of recurrent infection or thrombus progression, further confirming the efficacy of the treatment regimen (Figure 1).

At the three-month follow-up, the patient experienced nearly complete resolution of facial paralysis, with only mild residual weakness remaining. She had no new complications, and her neurological and infectious status remained stable.

Discussion

SBO represents a severe infection with a significant potential for life-threatening complications [1]. It predominantly affects the middle cranial fossa, involving structures such as the temporal, sphenoid, and occipital bones. This condition is most frequently encountered in individuals with compromised immune systems.

Diagnosing SBO can be particularly challenging because its symptoms are often nonspecific and may overlap with those of other conditions, such as malignancies, post-surgical complications, or other infectious processes. The clinical presentation typically includes headaches, cranial nerve palsies, facial pain, and otitis. Cranial nerve involvement, especially the facial nerve (VII), trigeminal nerve (V), and glossopharyngeal nerve (IX), is common, and these findings should raise suspicion for SBO, particularly in immunocompromised patients or those with a history of diabetes mellitus, chronic otitis media, or previous head and neck surgeries [2,3].

The most commonly isolated pathogen in SBO is Pseudomonas aeruginosa, accounting for a significant proportion of cases. This pathogen is particularly associated with chronic middle ear infections or post-surgical infections in the head and neck region. The infection often spreads to the skull base via vascular and neural sheaths, exploiting areas of bone weakness, such as the mastoid air cells, and can extend into adjacent structures like the nasopharynx and facial fat spaces. The involvement of these anatomical regions increases the complexity of the disease and contributes to the often-delayed diagnosis [4].

Imaging plays a crucial role in the diagnosis of skull base osteomyelitis (SBO), with CT and MRI being the primary modalities employed. CT scans are vital for detecting bone erosion and demineralization, particularly around theexternal auditory meatus, stylomastoid foramen, and the skull base bones. The imaging protocol involves acquiringthin, high-resolution slices of the temporal bones and skull base, along with axial and coronal reconstructions. However, it is important to note that a normal CT scan does not definitively rule out the diagnosis of SBO, as early changes may not be detectable [4].

MRI is the imaging modality of choice for diagnosing SBO, assessing the extent of infection, and monitoring treatment response.

A standard MRI protocol includes T1-weighted sequences prior to contrast injection, followed by multiplanar T1-weighted sequences with gadolinium enhancement and fat saturation (Fat Sat), along with T2-weighted sequences in both axial and coronal planes. This comprehensive approach enables detailed visualization of soft tissue involvement, evaluation of vascular or neural spread, and allows for longitudinal assessment of treatment efficacy.

MRI findings typically reveal altered bone marrow signals, characterized by a loss of fatty hyperintensity and enhancement following gadolinium administration on fat saturation sequences. These findings are often indicative of soft tissue infiltration around the external auditory meatus, frequently extending into the peripharyngeal fat spaces. Further evaluation is necessary to assess the extent of infection, particularly involving the skull base foramina. This includes evaluating enhancement of the facial nerve in its second and third segments, enhancement of mixed cranial nerves at the jugular foramen, and potential infiltration of the hypoglossal nerve within the hypoglossal canal. Additionally, MRI may reveal meningeal enhancement near the clivus or temporal meninges, which are critical in assessing the intracranial extension of the infection [4,5].

SBO can lead to severe complications, including septic thrombosis, particularly affecting the lateral and cavernous sinuses. Intracranial extension of the infection often originates from meningeal involvement, resulting in further complications. These can include bacterial meningitis, extradural or subdural empyemas, pre-suppurative encephalitis, and abscess formation, all of which significantly increase morbidity and mortality if not identified and managed promptly.

The differential diagnosis of SBO primarily includes tumoral pathologies, such as nasopharyngeal carcinoma, squamous cell carcinoma of the head and neck, and lymphoma, which can closely mimic both the clinical and radiological presentation of SBO. As a result, histological examination is essential for confirming the diagnosis, distinguishing infectious osteomyelitis from neoplastic processes. This emphasizes the crucial role of tissue biopsy in accurately identifying the underlying pathology and guiding the implementation of targeted treatment strategies for patients with skull base lesions.

The management of SBO has significantly improved with the advent of advanced antibiotic therapies, which have reduced the need for surgical intervention to a select number of cases. This progress has allowed for more conservative treatment approaches, with a focus on targeted antibiotic therapy based on culture and sensitivity results, while reserving surgery for cases with complications such as abscess formation or intracranial extension. 5. Conclusion

SBO is a rare yet serious infection that presents significant diagnostic challenges. Imaging techniques, particularly MRI, play a crucial role in accurately diagnosing SBO and assessing the extent of the infection. Early and precise imaging allows for the timely initiation of appropriate treatment, ultimately improving patient outcomes and prognosis.

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