

Skull Base Osteomyelitis with Extensive Bony Erosion Complicated by Venous Sinus Thrombosis

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Skull Base Osteomyelitis with Bony Erosion

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INTRODUCTION

Skull base osteomyelitis (SBO) involves an inflammatory process implicating the temporal, sphenoid, and occipital bones that commonly presents with severe otalgia, purulent otorrhea, headache, and conductive deafness from Eustachian tube dysfunction¹. Left untreated, SBO leads to significant mortality with the

development of meningitis, intracranial abscess, and venous sinus thrombosis². Treatment requires an extended course of culture-directed antibiotics and possible surgical debridement³. SBO may present similarly to malignancy thereby presenting a diagnostic dilemma. Herein, we present a case of SBO with extensive bony erosion diagnosed by computed tomography (CT) guided core biopsy and complicated by cerebral venous sinus thrombosis in a patient lacking traditional risk factors.

CASE

An otherwise healthy 70-year-old male with a history remarkable for 30 pack-years of cigarette smoking presented to the emergency department (ED) with worsening right-sided otalgia and otorrhea. The patient initially began experiencing otalgia three months before presentation after undergoing a dental procedure. The otalgia improved slightly with intermittent antibiotics but became increasingly severe prompting ED presentation. On arrival, he also reported frontal-to-right temporal headaches and worsening dysphagia, initially to solids and progressing to include liquids resulting in a 20-pound weight loss over the span of three weeks. He denied any changes to his hearing or vision.

On examination, the patient had a hoarse voice, right palatal weakness, and tenderness along the right lateral neck and peri-auricular region. The tympanic membranes (TM) were intact. There was scant otorrhea with mild edema of the right external auditory canal (EAC). There was no facial weakness, and the tongue was midline. He was afebrile and his labs were notable only for mildly elevated white blood cell count ($13.51 \times 10^9/L$) and C-reactive protein (15.34 mg/L).

Computed tomography (CT) of the head and neck was obtained and demonstrated a significant infiltrating soft tissue mass of the right upper neck and skull base (Figure 1). There was accompanying bony destruction and invasion of the occipital bone with extension to the right hypoglossal canal, jugular bulb, and stylomastoid foramen. Magnetic resonance imaging (MRI) T1 Dixon sequence imaging demonstrated hyperintensity at the right skull base with infiltration of the occipital bone (Figure 1). The right TM was moderately thickened with a narrowed EAC (Figure 2). Mild meningeal enhancement and thickening were present without findings of intracranial abscess.

Imaging findings were concerning for skull base infectious process or invading malignancy. The patient was initially started on empiric intravenous vancomycin, cefepime, voriconazole, and metronidazole. Corticosteroids were also given which improved cranial nerve dysfunction and lateral neck pain. A CT-guided core biopsy of the soft tissue mass was obtained. Pathology was negative for neoplastic processes while aerobic culture grew pan-sensitive *Pseudomonas aeruginosa*. The antibiotic regimen was subsequently narrowed to culture-directed cefepime.

The patient's hospitalization was complicated by sigmoid and transverse sinus thrombosis, upper gastrointestinal bleeding, and sepsis. Venous thrombosis was diagnosed by magnetic resonance venography, and the patient was started on a low-intensity intravenous heparin, which was later transitioned to oral anticoagulation. After appropriate interventions, the patient was safely discharged to home with a peripherally inserted central catheter (PICC). Oral anticoagulation and intravenous cefepime was continued at discharge.

Six weeks after discharge, cranial neuropathies and headaches had completely resolved. The patient was transitioned to oral ciprofloxacin for an additional three months with complete resolution of infection and normalization of CRP. Oral anticoagulation was continued for two months for continued prophylaxis against venous thrombosis which did not recur.

DISCUSSION

SBO is an uncommon condition that can lead to significant morbidity including cranial neuropathies and intracranial complications. Cranial nerve (CN) dysfunction can frequently occur with extension to the jugular foramen (CNs IX, X, and XI), stylomastoid foramen (V), and petrous apex (V and VI) leading to dysphagia, dysphonia, and facial weakness¹. Many pathogens, including *Pseudomonas*, *Streptococcus*, and fungal species, can lead to SBO¹. A leading cause of SBO remains malignant otitis externa (MOE) with a *Pseudomonas* infection occurring in > 90% of cases, but SBO can also occur from odontogenic, otogenic,

sinogenic, and mastoiditis infections ⁴. The pathogenesis of SBO remains unclear, however, cerumen pH alternations and impaired immune response and vascular perfusion may provide the necessary environment for SBO as evidenced by most patients being diabetic, elderly, or immunocompromised.

Clinically, SBO may demonstrate signs of overt infection including fever, leukocytosis, or elevated inflammatory markers. Imaging studies, commonly utilized for early diagnosis for localization and extent of infection, are often non-specific but may demonstrate soft tissue infiltration and bony erosion of the skull base. Compared to CT, MRI has been shown to have better sensitivity for characterizing disease infiltrate ⁵. SBO can frequently be misdiagnosed for malignancy which makes biopsy with histopathology necessary. However, depending on the extension and localization of the infection, obtaining a biopsy may be difficult.

Treatment for bacterial SBO involves an extended course of culture-directed intravenous antibiotics for a minimum of six weeks to achieve complete resolution ³. Surgical intervention for debridement of infected tissue may be beneficial for source control but can lead to unnecessary morbidity. If left untreated, SBO leads to significant mortality with the development of meningitis, intracranial abscess, and venous sinus thrombosis. Overall, the long-term prognosis of SBO was reported to be 90.5% within 18 months with 31% developing neurologic sequelae, though certain medical conditions such as diabetes have demonstrated worse prognosis ¹.

For the patient in the present case, aside from advanced age, there were no known risk factors for SBO as hemoglobin A1c was normal, and human immunodeficiency virus (HIV) testing returned negative. It was hypothesized the SBO infection was secondary to MOE that may have been a result of odontogenic seeding with subsequent extension to the skull base. Nonetheless, *Pseudomonas* is an uncommon odontogenic infiltrate making it an unlikely presentation. The case underlines the importance of recognizing SBO versus similarly presenting malignancy especially in patients lacking traditional risk factors for prompt culture-directed antibiotic treatment leading to a favorable outcome.

CONCLUSION

SBO mimics the presentation of various conditions, notably skull base malignancies. For optimal prognosis, prompt diagnosis with core biopsy for culture and pathology may be necessary that informs antibiotic selection. Although patients with diabetes or that are immunocompromised are more susceptible to SBO, not all patients have these risk factors.

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Figure 1: Axial computed tomography (A) and T1 Dixon magnetic resonance imaging (B) demonstrating right central skull base bone erosion (asterisk) with infiltrating soft tissue mass (arrows).

Figure 2: Temporal bone axial computed tomography showing right tympanic membrane (arrows) thickened with narrowed external auditory canal (A) compared to the normal left tympanic membrane and external auditory canal (B).

