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Case Report

Cranial Nerve Palsy as a Presenting Feature of Tubercular Skull Base Osteomyelitis - A Rare Presentation

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A B S T R A C T

Skull base osteomyelitis (SBO) presents diagnostic challenges due to diverse clinical manifestations mirroring other conditions. Diagnosis relies on imaging like CT and MRI, supplemented by blood cultures and bone biopsies for microbial identification. Pseudomonas aeruginosa is a common causative organism, though TB, albeit rare, has been documented. We present a case of a 22-year-old female who presented with painless left lower neck swelling along with a persistent headache, weight loss, and decreased appetite. Examination revealed a discrete, firm, non-tender, mobile cervical lymphadenopathy. Initial investigations were unremarkable except for an elevated erythrocyte sedimentation rate, prompting further evaluation. Aspiration cytology revealed a suppurative lesion, with no response to treatment with amoxicillin and clavulanic acid. She noticed the development of double vision for which magnetic resonance imaging was done which revealed altered signal intensity in the skull base suggestive of osteomyelitis, with associated pachymeningitis. Cerebrospinal fluid analysis showed lymphocytic pleocytosis. Against the backdrop of the isolation of acid-fast bacilli on lymph node biopsy, a diagnosis of tubercular SBO with cervical lymphadenopathy was made. Initiated antitubercular therapy resulted in gradual resolution of symptoms over a 6-week period, with continued treatment for 12 months leading to complete recovery. This case underscores the need for comprehensive assessment to effectively address unusual clinical presentations.

Keywords: Skull Base Osteomyelitis, Tuberculosis, Diplopia, Lymphadenopathy, Antitubercular Therapy

Introduction

Skull base osteomyelitis (SBO) is an uncommon and possibly critical condition characterised by the contamination of the temporal, sphenoid, or occipital bones because of infections arising from ear, nose or tooth, particularly in immunocompromised patients. Pathogens that can cause it include bacteria, fungi, and even diseases like tuberculosis. SBO is uncommon, with estimates ranging from 0.5 to 1.0 cases per 100,000 people per year.[1]

A high degree of suspicion and focused testing, mainly radiographic, are needed to rule out this rare diagnosis and commence immediate therapy. This unusual clinical manifestation of the illness should not be neglected, particularly in a developing nation like ours, where tuberculosis (TB) is a major public health concern. We hereby present a case of a young female who was diagnosed with skull base osteomyelitis secondary to TB with an atypical presentation.

Case Report

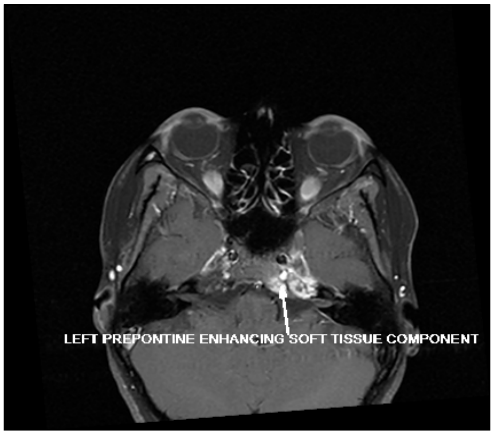
A 22-year-old female student and resident of Uttar Pradesh presented with the chief complaint of swelling in the left lower neck for a month and headache for the previous 15–20 days. The swelling was gradually increasing in size and was painless. It was associated with appetite reduction and weight loss (4 kg in the last one month). She was afebrile and had no history of night sweats or an evening rise in temperature. The patient also complained of a holocranial headache that was insidious in onset and mild to moderate in intensity. It was not associated with nausea or vomiting, nor did it cause any visual symptoms.

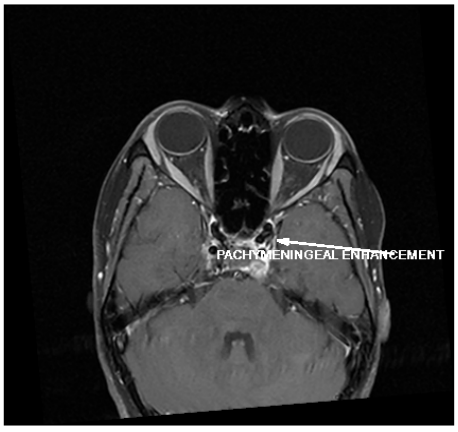
The general physical examination revealed no findings of significance except for lymphadenopathy. On consequent local examination, the swelling was found to be present in the left cervical node region (level III), which was a single discrete swelling measuring 2.5 x 3 cm in size. The swelling was firm on palpation, non-tender, matted and mobile with no overlying skin changes. No nodes were palpable in the contralateral cervical region, neither were any nodes in the axillary or inguinal region. There were no significant findings on systemic examination.

A thorough blood work showed a haemoglobin level of 11.3 g/dL, and an acceptable WBC and platelet count. The rate of erythrocyte sedimentation was elevated to 56 mm/hr. Her serum creatinine level was 0.6, while her electrolytes were acceptable. The liver function tests came back normal. Her chest X-ray was unremarkable. The patient was recommended to have fine needle aspiration cytology and biopsy in order to thoroughly investigate the cervical enlargement. The sample's microscopy revealed inflammatory cells on a backdrop of RBCs with no accompanying granulomas, indicating a suppurative lesion.

While we awaited the biopsy results, she was started on amoxicillin with clavulanic acid tablets three times a day. On day 4 of admission, the patient started developing double vision while her headache persisted. The visual acuity was normal in both eyes. The pupils were normal in size and response. Further examination revealed a lateral movement deficit in the left eye with no concomitant weakness in the right eye. Her Glasgow Coma Scale (GCS) was 15/15, and the rest of the nervous system examination was unremarkable. The patient underwent a contrast MRI brain with orbit and lumbar puncture. The white blood cell (WBC) count in the cerebrospinal fluid was 40/mm^3, with differential counts of 98% lymphocytes and 2% neutrophils. The protein level was 50.5 mg/dl and the glucose content was 71 mg/dl. The adenosine deaminase (ADA) levels in the cerebrospinal fluid (CSF) were 5 U/L. Magnetic resonance imaging (MRI) of the brain and orbit (Figure 1) revealed altered signal intensity in the basisphenoid, petrous apex and clivus on the left side with features of soft tissue enhancement in the left prepontine cistern region showing central liquefaction compressing cisternal segment of left VIth cranial nerve. The symptoms were consistent with skull base osteomyelitis with associated pachymeningitis.

On a background of caseating granuloma, Ziehl–Neelsen staining on the lymph node biopsy revealed positive results for acid-fast bacilli as shown in Figure 2.





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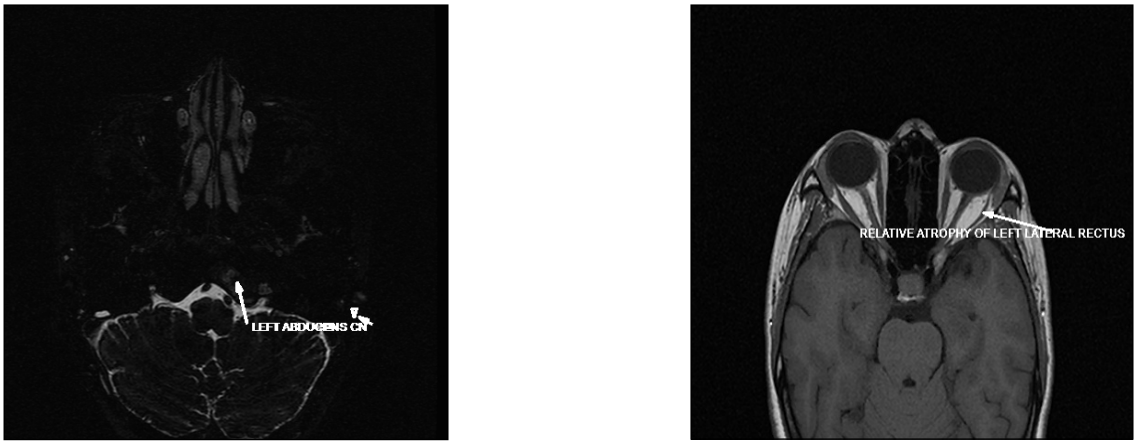

Figure 1.MRI brain with Orbit (a).Left Prepontine Soft Tissue Enhancement (b).Pachymeningeal Enhancement (c).Abducens Nerve (d).Relative Atrophy of Lateral Rectus Muscle on the Left Side

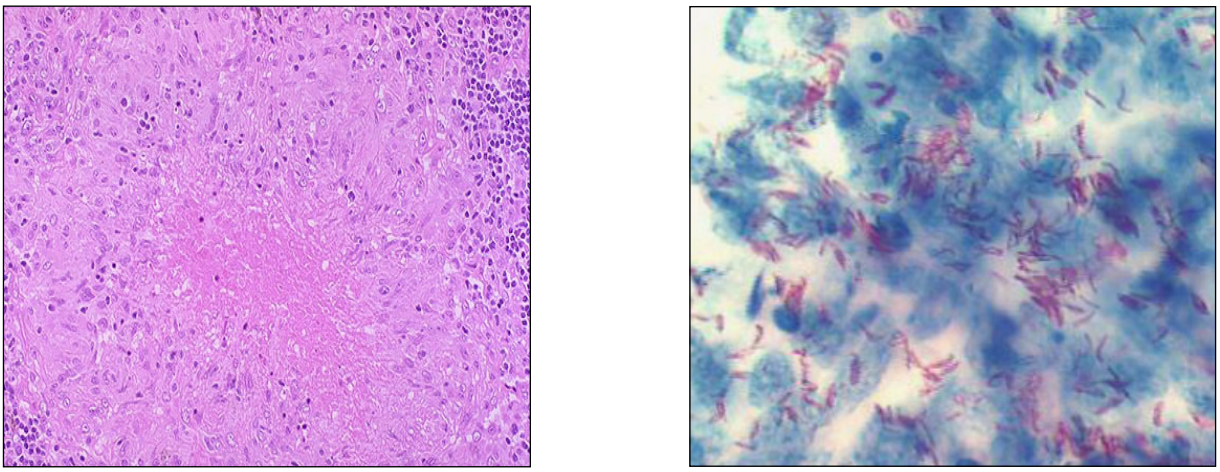

Figure 2.Lymph Node Biopsy (a).200X showing Epitheloid Cell Granuloma with Caseous Necrosis (b).500X showing Acid-fast Bacilli on Ziehl–Neelsen Staining

We arrived at the diagnosis of tubercular skull base osteomyelitis with cervical lymphadenopathy. Antitubercular treatment was started for the patient, and she tolerated it well. After 6 weeks of therapy, her lymph node swelling had subsided, as had her headache and diplopia. The therapy was continued for a period of 12 months. The patient is now doing well, with no new complaints.

Discussion

SBO has the potential to be fatal. SBO pathogenesis is complex and multifactorial, involving both local and systemic factors. Local factors such as trauma, surgery, and chronic inflammation can all lead to bone necrosis and infection.[1] Systemic factors can reduce the body's capacity to fight infection, such as immunocompromised status from conditions like diabetes, HIV, or cancer.[2]

There are two forms of skull base osteomyelitis: typical and atypical. The most prevalent cause of typical skull base osteomyelitis is unregulated infections in the temporal bone area, most often necrotising otitis externa. Atypical SBO (also known as central SBO) develops when there is no apparent temporal bone or external auditory canal infection.[3]

Pseudomonas aeruginosa is a commonly identified organism, however, other bacteria and fungi, such as Staphylococcus aureus and Streptococcus pneumoniae, have also been identified as causal agents.[3,4] Skeletal TB accounts for 1% of tuberculous infection presentations, which implies that the incidence of tubercular skull osteomyelitis is even lower. Involvement of the skull base is much more unusual.[4] TB is a rare cause of this disease but has been reported in the literature.

SBO has a diverse clinical appearance that can mirror other illnesses, making diagnosis difficult. Headache, fever, and nasal congestion are the most typical presenting symptoms. Additional signs and symptoms include face discomfort, periorbital puffiness, and blurred vision. In some cases, otitis media, hearing loss, and cranial nerve palsies may be present. SBO can cause meningitis or cerebral abscess development in rare cases.[5,6]

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Clinical features of SBO may change depending on the site and extent of the disease. Nasal congestion, rhinorrhoea, and epistaxis are common symptoms of anterior skull base involvement, while headache and neck discomfort are common symptoms of posterior skull base involvement. Temporal bone involvement can cause otalgia, otorrhoea, and facial nerve palsy, whereas sphenoid sinus involvement can cause visual abnormalities, cranial nerve palsies, and pituitary dysfunction. Clivus involvement can cause cranial nerve palsies, dysphagia, and neck discomfort. A comprehensive history and physical examination are required, as is a strong index of suspicion, particularly in patients with SBO risk factors or chronic or worsening symptoms.[3,4]

Imaging tests are essential in the diagnosis of SBO. A CT scan can detect skeletal damage and changes in bone density, but an MRI can detect soft tissue involvement, such as abscess development and inflammation. A contrast-enhanced MRI is preferable when there is a suspicion of intracranial involvement or consequences. MRI can also be used to assess therapy response.[5,6] Laboratory procedures such as blood cultures and bone biopsies may be required in addition to imaging techniques to identify the causative organism. Just a small fraction of blood cultures is positive, and a negative result does not rule out the diagnosis of SBO. A bone biopsy is the most reliable method for diagnosis, and histology and microbiological culture of the biopsy samples can identify the pathogenic microbe and help guide the therapy with appropriate antibiotics.[1,7]

Endoscopic assessment can also help with the diagnosis and treatment of SBO. Endoscopic examination can help guide biopsy and debridement operations by identifying regions of mucosal ulceration, necrosis, and granulation tissue. It can also help identify problems including carotid artery involvement and cranial nerve dysfunction.[4]

In the diagnosis and therapy of SBO, a multidisciplinary approach is required, and coordination between experts such as infectious disease physicians, neurosurgeons, and otolaryngologists is crucial. A lengthy course of antibiotics is usually prescribed, with surgical debridement and reconstruction saved for cases of chronic or progressing illness. The course of antimicrobial treatment varies according to the extent and severity of the condition, but 6–8 weeks of intravenous antibiotics are advised as a minimum. In some circumstances, oral antibiotics can be taken for long-term suppression treatment. Surgery may include necrotic bone debridement, abscess drainage, or even excision of the affected bone. In certain circumstances, hyperbaric oxygen treatment, particularly for recalcitrant infections, may be useful.[1,3,4,7]

Case reports of skull base osteomyelitis are limited, given its rare incidence. TB as the cause of SBO is extremely rare, and on literature search, we found a few cases. In a case report by Iyer et al., a 12-year-old girl presented with sudden onset right-sided weakness, numbness, and facial deviation to the left. The patient reported headaches and neck pain for a month, which resolved with painkillers. Physical examination showed right-sided hemiparesis, alongside facial palsy. A computed tomography (CT) scan revealed substantial destruction in the anterior and posterior clinoid processes and occipital protuberances, as well as encircling ill-defined peripherally enhancing soft tissue, implying an infective origin, most likely TB osteomyelitis of the skull base. The patient improved and completed 12 months of antitubercular therapy.[3] Kumar et al. described a 32-year-old male with a suboccipital headache and a low-grade fever. Sphenoid sinusitis was initially diagnosed, and endoscopic sinus surgery was performed. He was readmitted, however, due to deteriorating symptoms. The MRI revealed occipital bone and clivus osteomyelitis. The patient underwent drainage of abscesses and tubercular aetiology was identified. He received antitubercular medication as well as further debridement of abscesses. His recovery after surgery was unremarkable, and at the three-month mark, he displayed full symptom remission. Antitubercular treatment was continued for 18 months.[4] Although uncommon, TB should be considered among the possible causes of skull base osteomyelitis, particularly in individuals with a history of TB or who are immunocompromised. Early detection and treatment are vital for preventing complications and improving results.

Despite extensive literature search, we could not find a case report which presented a case with concomitant cervical lymph node and cranial nerve palsy secondary to tubercular skull base osteomyelitis.

Conclusion

To summarise, SBO is an uncommon and possibly fatal disorder that can be difficult to identify. Making an early diagnosis requires a high index of suspicion, a comprehensive history and physical examination, and suitable imaging investigations. A multidisciplinary approach to therapy is required, and treatment typically consists of a protracted course of antibiotics, with surgical debridement reserved for individuals with chronic or progressive infection.

Conflict of Interest: None

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