



Tubercular osteomyelitis of the clivus

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ABSTRACT

A 24-year-old girl presented with non-specific headache and diplopia. Nasal endoscopy showed a polypoidal mass in the nasopharynx. Computed tomography and magnetic resonance imaging revealed erosion of the clivus. Nasopharyngeal biopsy clinched the diagnosis of tuberculosis. She was put on antitubercular therapy, but developed seizures, bilateral ptosis and obstructive hydrocephalous, which were relieved by a ventriculo-peritoneal shunt. However, the patient developed infarction of the basal ganglia and thalamus and died due to cardiorespiratory arrest.

Keywords: Clivus; nasopharynx; tuberculosis.

Tuberculosis, a communicable disease, is caused by *Mycobacterium tuberculosis*. With the advent of chemotherapy regimens, the morbidity and mortality has decreased.^[1] Tuberculosis is transmitted by droplet infection and commonly affects the lungs (pulmonary tuberculosis). However, any site, organ or system can be affected by tuberculosis (extrapulmonary tuberculosis) that accounts for 15-20% cases.^[2] Nasopharyngeal tuberculosis is a rare presentation (less than 1%) of head and neck tuberculosis.^[3] Involvement of the clivus is extremely rare with less than 20 cases reported. The clivus is a downward sloping wedge of bone formed by the basisphenoid and basiocciput, extending between the dorsum sellae and foramen magnum. The posterior surface of the clivus is related to the pons, prepontine cistern, VI cranial nerve and basilar artery. This report describes a case of sphenoclivular tubercular osteomyelitis along with nasopharyngeal tuberculosis.

CASE REPORT

A 24-year-old girl was referred from the ophthalmology department with complaints of headache for three months and diplopia for one month with no history of diminution of vision. She also complained of right facial pain that gradually increased over the past month. There was no other nasal or ear complaint. There was no history of tuberculosis contact. Clinical examination revealed left sixth nerve palsy. Nasal endoscopy showed a pale proliferative lesion in the right nasopharynx. The erythrocyte sedimentation rate (ESR) was raised (38 mm). A chest X-ray was normal, and tests for human immunodeficiency virus (HIV), hepatitis B antigen (HBsAg), and hepatitis C virus (HCV) were negative. Magnetic resonance imaging (MRI) of the brain and paranasal sinuses (PNS) revealed a heterogeneously enhancing area involving the clivus causing breach of the antero-inferior and postero-superior cortical margins, extending anteriorly into the right

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nasopharynx (Figure 1). Peripherally enhancing soft tissue with a central non-enhancing necrotic area was seen posterior to the clivus effacing the prepontine cistern. The lesion extended laterally on the left side causing a mass effect on the left cavernous sinus. Hyperintense contents were noted in the sphenoid sinus. These findings suggested osteomyelitis of the clivus. Contrast enhanced computed tomography (CT) of the brain and PNS revealed erosion and lytic destruction of the anterior aspect of the clivus with a well-defined peripherally enhancing collection of 1.1 cm maximum depth noted postero-superiorly, effacing the pre-pontine cistern. A heterogeneously enhancing collection was noted in the right nasopharynx with erosion of the floor of the sphenoid sinus (Figure 2, 3). On the seventh day of presentation, the patient had seizures, projectile vomiting with altered sensorium. There was bilateral ptosis, the pupils were dilated and sluggishly reactive, indicating paralysis of cranial nerve III bilaterally. Repeat non-contrast CT of the head suggested non-communicating hydrocephalous with basal exudates. Fundus examination showed bilateral papilledema. Neurosurgery opinion was obtained

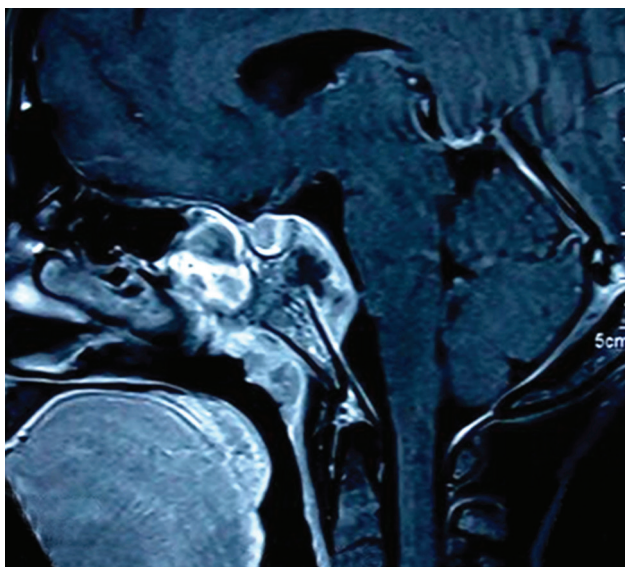


Figure 1. Post gadolinium magnetic resonance imaging of the brain shows a heterogeneously enhancing area involving the clivus and breaching its antero-inferior and postero-superior cortical margins. Peripheral enhancing soft tissue with a central non-enhancing necrotic area was seen posterior to the clivus effacing the prepontine cistern.

and the patient was started on intravenous antibiotics (ceftriaxone 2 g 12 hourly), anti-epileptics (phenytoin 100 mg tablet eight hourly), intracranial tension lowering drugs (mannitol, 20% 100 mL intravenous eight hourly) and a ventriculo-peritoneal shunt was placed. She became oriented but cranial nerve paralysis persisted. Endoscopic biopsy was taken under general anesthesia from the right nasopharynx and caseous material was removed from the

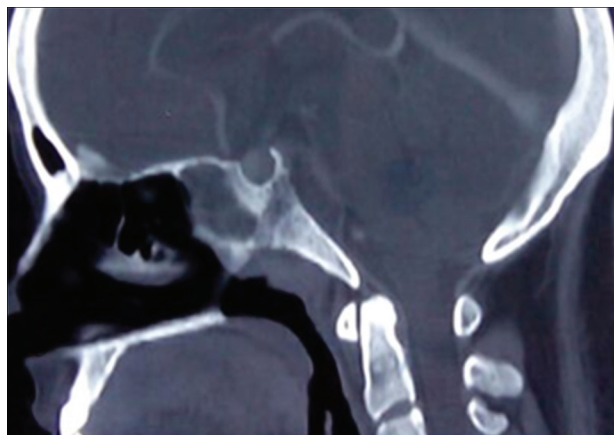


Figure 2. Contrast enhanced computed tomography paranasal sinuses and brain, sagittal section showing erosion and lytic destruction of the anterior aspect of the clivus with a well-defined peripherally enhancing collection of maximum depth 1.1 cm noted along its postero-superior aspect. The collection was seen projecting into the prepontine cistern and reaching up to the posterior wall of the sella.



Figure 3. Contrast enhanced computed tomography paranasal sinuses and brain, coronal section, showing polypoidal mucosal thickening involving the sphenoid sinus with erosion of the floor of the sinus and extension into the right nasopharynx.

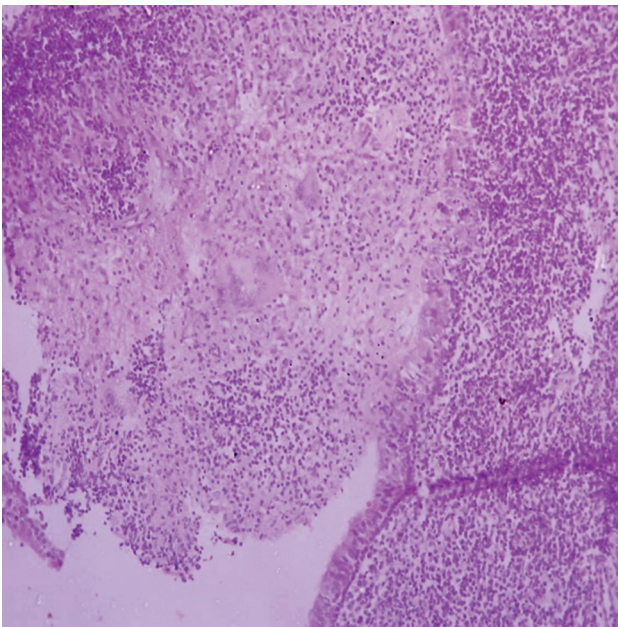


Figure 4. Histo-micrograph showing moderate to dense chronic inflammation with epithelioid granuloma and giant cells (H-E×100).

sphenoid sinus. Histopathology revealed moderate to dense chronic inflammation with epithelioid granuloma and giant cells (Figure 4). The diagnosis of tubercular osteomyelitis of the clivus with nasopharyngeal tuberculosis was made. The patient was started on category I anti-tubercular treatment, ATT, (isoniazid 5 mg/kg, rifampicin 10 mg/kg, pyrazinamide 30 mg/kg, ethambutol 20 mg/kg) but on the fourth day of starting ATT, she again developed altered sensorium, headache and seizures. Another plain CT of the head showed infarcts in bilateral basal ganglia, thalamic region and cerebral peduncle. The patient expired due to cardiorespiratory arrest.

DISCUSSION

Extrapulmonary tuberculosis of the head and neck region comprises of 10% of cases. Isolated tubercular lymphadenitis is most common (73.3%) followed by laryngeal tuberculosis (14.5%), tuberculous otitis media (2.4%), parotid (1.8%), oral cavity (5%), nose (1%) and TM Joint (1%).^[4]

Tuberculosis can spread via hematogenous, lymphatic and direct inoculation/invasion.

Tubercular bacteria may primarily inoculate the upper respiratory tract (tonsil, adenoid, and nasopharynx) being rich in lymphatic tissue as happened in present case. Involvement of bone in tuberculosis occurs mainly via the hematogenous route but direct invasion can also occur and cause bone erosion as observed in the present case.

Nasopharyngeal tuberculosis presents with nasal discharge, post-nasal drip, nasal obstruction, aural fullness and cervical lymphadenopathy.^[3] Skull base involvement can occur as primary involvement or as direct extension from a lesion in the nasopharynx or atlanto-occipital joint.^[5] In both scenarios, hematogenous spread appears to be important. Involvement of the clivus and sphenoid sinus is extremely rare with only few cases reported.^[6]

An erosive lesion of the clivus needs to be differentiated from chordoma, osteomyelitis, metastases, and fungal granuloma. In the present case, the patient presented with symptoms of skull base and intracranial involvement. Headache and diplopia pointed towards a space occupying lesion with false localizing signs.^[6,7] The patient developed raised intracranial pressure due to non-communicating hydrocephalus. Imaging revealed basal exudates that are characteristics of tubercular meningitis. Witcombe and Cremin^[8] reported tubercular meningitis, involvement of lungs, chest wall and limbs in four cases of spheno-clival tuberculosis. In the present case, erosion of the clivus extended anteriorly into the nasopharynx. Richardus et al.^[5] and Shenoy and Raja^[7] reported nasopharyngeal involvement in clival tuberculosis. The patient later developed bilateral ptosis, seizures, bilateral thalamic and cerebral peduncle infarcts which resulted in death. Richardus et al.^[5] and Selvapandian and Chandy^[9] reported multiple cranial nerve palsies, and our case also had 3rd and 6th cranial nerve palsy. Most commonly affected cranial nerves are II, III, IV, VI, and VII. In our case, cranial nerves III and VI were bilaterally involved.

Sencer et al.^[10] reported hemiparesis and seizures with involvement of the petrous apex in clival tuberculosis. Infectious vasculitis of skull base vessels leads to bilateral thalamic and cerebral peduncle infarcts. Tubercular meningitis

can lead to vasculitis-like syndrome and infarct either due to direct vessel wall invasion by mycobacteria or may result from secondary extension of adjacent arachnoiditis. To the best of our knowledge, this is the first case report with such extensive involvement in clival tuberculosis. All the above-mentioned case reports responded well to anti-tubercular therapy with complete or partial resolution of symptoms.

Declaration of conflicting interests

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