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Tuberculosis Presenting as Base of Skull Osteomyelitis with Stroke: A Case Report

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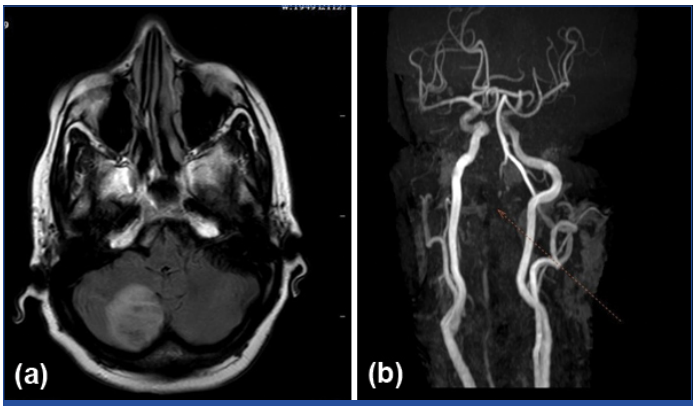
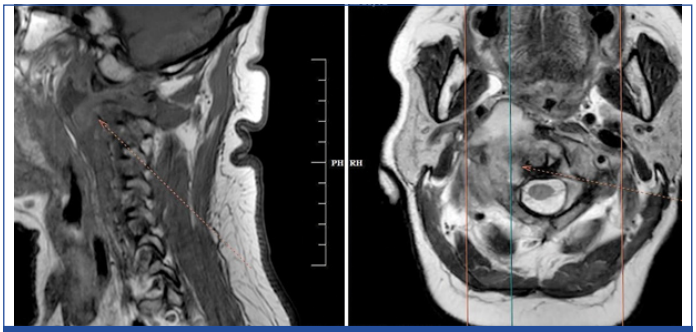
This 70-year-old female patient, a housewife by occupation and a known case of diabetes mellitus on treatment with oral hypoglycaemic agents, presented with a 2-week history of occipital headache and unsteadiness on walking. She was on symptomatic treatment for the same with analgesics only. There was no associated nausea, vomiting or photophobia along with headache. The unsteadiness on walking was more to the right side with no associated slurring of speech. There was no history of fever, limb weakness or seizures. There was no history of evening rise of temperature, cough or weight loss. There was no past history of exposure to tuberculosis. Clinical examination demonstrated normal vital signs. Nervous system examination revealed normal cranial nerves and motor system. Cerebellar signs were positive with significant gait ataxia to the right side. Deep tendon reflexes were well elicited. No clinical signs of meningeal irritation were noted.

Routine blood tests, including blood cell counts, were normal, while blood sugar levels were high (>300 mg%). Magnetic Resonance Imaging (MRI) of the brain showed a right subacute cerebellar infarct with MR angiogram showing right vertebral artery V4 segment occlusion [Table/Fig-1]. In view of the subacute onset of symptoms and imaging findings, a clinical diagnosis of an acute ischaemic stroke involving the right cerebellum was considered as the most likely diagnosis. She was treated with antiplatelet agents, statins, and low molecular weight heparin. Blood sugars were high, requiring insulin therapy. Initial improvement in symptoms was seen and she was discharged after improvement.

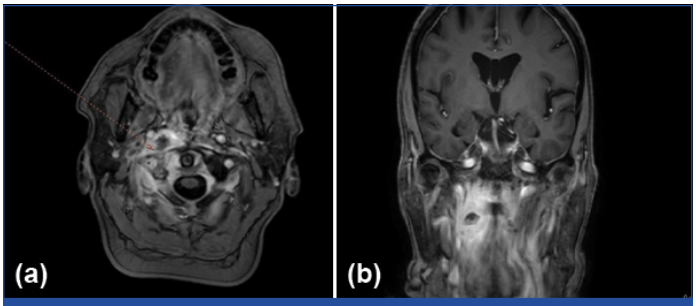


[Table/Fig-1]: a) MRI FLAIR sequence showing subacute right cerebellar infarct with haemorrhagic transformation; b) MR angiogram showing thrombosed right vertebral artery.

She presented after two weeks with severe right-sided neck pain and restricted neck movements. Examination revealed torticollis to the right side and right neck muscle spasm. Blood tests revealed an elevated Erythrocyte Sedimentation Rate (ESR) at 120 mm per hour. Counts, electrolytes and liver functions were normal. On imaging, MRI cervical spine with contrast showed abnormal paraspinal collection on the right side of C1, C2 involving the foramen transversarium and causing bony destruction [Table/Fig-2,3]. There was a thrombosis of the right vertebral artery that was seen. The clinical possibilities considered were skull base osteomyelitis with extension and invasion of the vertebral artery, causing acute cerebral infarction and nasopharyngeal carcinoma with intracranial extension. Considering the possibility of a skull base osteomyelitis, she was started on broad-spectrum antibiotics including Piperacillin-Tazobactam, Levofloxacin and Metronidazole. She underwent a Computed Tomography (CT)-guided aspiration of the neck lesions, revealing pus. Analysis revealed the presence of caseating granulomas and acid-fast bacilli suggestive of tuberculosis. A final diagnosis of tuberculous involvement of the skull base with vertebral artery thrombosis was made She was started on Anti-Tubercular Therapy (ATT) and steroids. The option of a surgical debridement was offered, but the patient was not willing to undergo the same. She had initial improvement in neck pain after starting treatment with ATT. She was continued on ATT and was on regular follow-up.



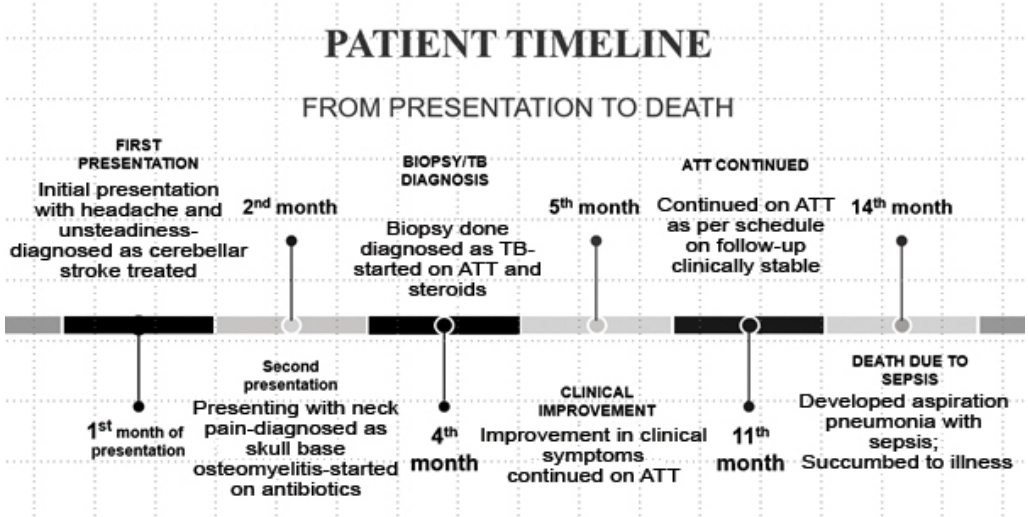
[Table/Fig-2]: MRI T2 sagittal and axial cervical spine images showing paravertebral collection with bony destruction and loss of right vertebral artery flow void.



[Table/Fig-3]: MRI STIR and post-contrast images showing soft-tissue involvement with enhancement and central necrosis causing bony destruction of the lateral mass of C2 vertebra.

At a subsequent visit, she was admitted with features of aspiration pneumonia and sepsis. Despite adequate treatment, she succumbed to her secondary infection [Table/Fig-4].

Skullbase osteomyelitis is an uncommon condition that can be life-threatening if not diagnosed and treated early in the course of illness [1]. Most patients are immunosuppressed with predisposing conditions including diabetes mellitus, otitis media, malignancy and steroid use. Pseudomonas aeruginosa is one of the most common organisms identified in these cases. Atypical cases arise due to non-otogenic causes and can be due to many processes.



[Table/Fig-4]: Patient timeline from diagnosis to death extending over a span of 14 months from the time of diagnosis.

At present, base of skull osteomyelitis is rare and mainly involves the temporal and the sphenoidal bones [2]. Skull base osteomyelitis commonly occurs as a complication of malignant otitis externa involving the temporal bone or secondary to sphenoidal sinusitis. A rarer form is the central or atypical form, which may begin with otitis externa [3]. This entity primarily affects the sphenoidal bone and the occipital bone in proximity to the clivus.

Bacteraemia or fungaemia may also be responsible for osteomyelitis, typically in immunocompromised patients, including those who have prolonged neutropenia, leukaemia, corticosteroid use, critical illnesses in an Intensive Care Unit (ICU) setting, cancer chemotherapy, Human Immunodeficiency Virus (HIV) patients and uncontrolled sugars [4].

Clinical features include severe ear pain due to the involvement of temporal, parietal, postauricular and retro-orbital areas, intermittent fevers, and ear discharge, which is usually purulent [5]. Atypical presentations with pulsatile headache and tinnitus can be seen and the diagnosis is often missed in these cases. Spread of infection upwards leads to early cranial neuropathies, commonly abducens palsy, if not treated or diagnosed early [6]. Prognosis remains poor in many cases.

For the imaging of the skull base, MRI scans are superior to CT imaging in view of better differentiation of soft-tissue structures, medullary cavity of bone and meninges. Findings of T2 hyperintense and T1 hypointense lesions are said to be characteristic [7]. Elevation of the ESR is seen in many cases and may guide towards the diagnosis. Other rare causes include Wegener's granulomatosis, tuberculosis, Paget's disease and fibrous dysplasia. Tuberculosis with bony destruction is a rare but important differential diagnosis [8]. This patient developed a stroke secondary to infection, causing vertebral artery thrombosis. Primary tuberculous osteomyelitis, both of the skull vault and skull base, is a common cause of cranial osteomyelitis in many regions of the world [9]. Tissue diagnosis by means of a CT-guided biopsy is required in all cases for a definitive diagnosis. Treatment is through the initiation of ATT along with steroids in the initial stages. Surgical debridement may be needed in case of poor response to medical therapy or in case of bony instability. This patient had initial improvement but succumbed to secondary infection. Surgical management was advised, but the relatives were not willing to undergo the same. Tuberculosis should be considered in the differential diagnosis of skull base osteomyelitis, especially with stroke presentation. Marked elevation of ESR can be a clue to the same. Early tissue diagnosis may help in these cases.

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