



Unusual Presentation of Tuberculous Meningitis: Two Case Reports

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ABSTRACT

Tubercular meningitis can be a great mimicker with varied manifestations. Here we present two cases with extremely unusual presentations. First case is of a 22 year boy who presented with 2 weeks history of headache following a minor accidental fall and blunt injury to head and was diagnosed as tubercular meningitis. Second case is a 42 year old man presented with 6 months history of fever and followed by a stroke involving the left capsuloganglionic region and diagnosed as tubercular meningitis. First case presented without fever or other features of meningitis while second case had prolonged 6 months fever followed by Vasculitic infarcts related to tuberculosis without any other systemic organ involvement.

INTRODUCTION

Tubercular meningitis (TBM) is still one of the common infections of central nervous system (CNS) in developing countries and poses significant diagnostic and management challenge.^[1]

Despite modern antituberculosis chemotherapy, 20% to 50%^[2] of patients still die, and many of the survivors have significant neurological deficits. Death from TBM is strongly associated with delays in diagnosis and treatment.

Global burden of tuberculosis is quite significant in developing countries. Globally, there were an estimated 9.27 million new cases (139 per 100,000 population) of tuberculosis in 2007, and the number of prevalent cases was 13.7 million (206 per 100,000 population).^[3] The incidence of CNS tuberculosis generally reflects the incidence

and prevalence of tuberculosis in the community. About 10% of patients who have tuberculosis develop CNS disease.^[4] HIV infection predisposes to the development of extrapulmonary tuberculosis, particularly tubercular meningitis.^[5] The estimated mortality due to tubercular meningitis in India is 1.5 per 100,000 population.^[6]

CASE REPORT

CASE 1

22 year old man presented with history of gradual onset and progressive headache since 2 weeks. There was no history of fever, neck pain, any other neurological symptoms or systemic symptoms like cough, weight loss or fever. History of fall with insignificant head injury around 1 month back was present. On examination

he was afebrile, there was no neck stiffness or focal neurological deficits. Other system examinations were normal. Routine blood investigations including ESR was normal. CT Brain showed evidence of communicating hydrocephalus. MRI BRAIN with contrast showed hydrocephalus with diffuse meningeal enhancement. In view of possibility of meningitis CSF study was done despite absence of history of fever or neck stiffness. CSF Opening pressure was high (30 cm of CSf) With 330 cells, 90% lymphocytes with raised protein of 95.4 mg/dl and normal blood sugars. CSF ADA was normal and TB PCR was positive. CSF Bacterial and fungal stains were negative. Mantoux was negative and CT Chest and abdomen was normal. He was started on Antituberculous drugs. His headache reduced dramatically in a week. Follow up CT Brain at 4 weeks showed significant resolution of hydrocephalus. CSF was repeated at 3 weeks showed normal opening pressure with 25 cells and normal protein levels.

CASE 2

42 year old patient presented with sudden onset of slurring of speech with facial deviation to right and left upper and lower limb weakness since 2 weeks. He has history of on and off fever since 6 months which was evaluated in many hospitals without a diagnosis and without any response to antibiotics. There was no evening rise of temperature, cough, abdominal pain, headache or any other systemic symptoms. On examination there was no lymphadenopathy or organomegaly. CNS Examination showed a left Upper motor Neuron Facial palsy with left sided grade 3 power in distal and proximal upper and lower limbs with extensor left plantar. Blood investigations like ESR and CRP was normal. VDRL and HIV was nonreactive. MRI Brain with contrast showed left deep basal ganglia infarcts with a enhancing lesion around the left medial temporal lobe probably tubercular granuloma. CSF Study showed 30 cells, 95% lymphocytes with elevated CSF protein 123mg/dl and mildly low CSF

glucose. CSF Fungal stain and cultures were negative. ADA was normal. CSF TBPCR was positive. CT Chest and CT Abdomen was normal and mantoux was negative. He was started on antitubercular agents with steroids in view of vasculitic infarcts. His fever started to resolve in 2nd day and disappeared after 4 days. Patient is on medications with excellent clinic improvement. Plan is to repeat MRI Brain after 6-8 weeks during follow up.

Case 1

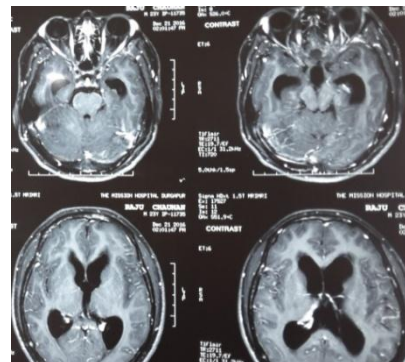


Image 1-MRI Brain T1 Contrast axial Image showing hydrocephalus and diffuse meningeal enhancement

Case 2

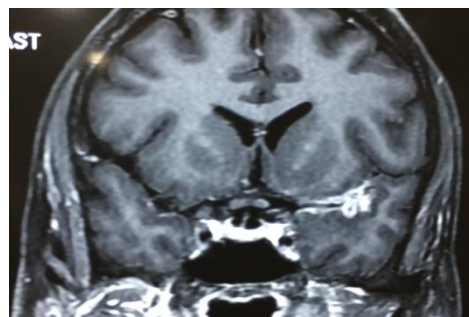


Image 1-MRI Brain T1 Contrast coronal view showing enhancing exudates involving left temporal lobe

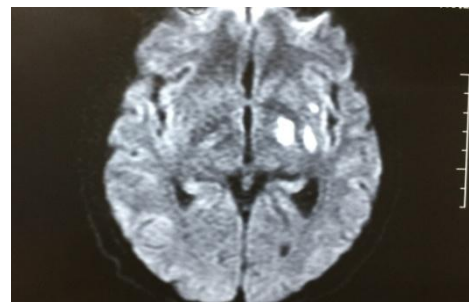


Image 2-MRI Brain diffusion weighted sequence axial view showing acute infarcts in left capsuloganglionic region

DISCUSSION

Tuberculosis (TB) is still faced as a major public and global health issue. India is one of the world's highest TB bearing country according to World health organization (WHO) statistics.^{1,2} It is estimated that 1% of all tuberculosis infected patient will develop intra cranial tuberculosis in time. Definite diagnosis is made by detection of tubercle bacilli in cerebrospinal fluid, which is highly specific but lacks sensitivity, because of this a multidisciplinary approach combining clinical, CSF profile and Neuroimaging help us in making a diagnosis at the earliest. Uncertainty and doubts still dominates all aspect of intracranial TB till date. Its unpredictable natural history and varied clinical manifestation still pose a challenge in the diagnosis, management and prognostication of CNS-TB.

Our first case was atypical because the patient had no history of fever and had only headache as presenting manifestation. Examination did not show any neck stiffness. The history of head injury further delayed the diagnosis. Absence of fever and neck stiffness in Immuno competent young individuals in quite rare and may be seen in 5-10% of cases. The second case presented with prolonged fever of 6 months duration without any lung, gastrointestinal involvement or lymphadenopathy. The presenting manifestation was acute ischemic event which led to diagnosis of underlying tubercular meningitis. Tubercular meningitis presenting as pyrexia of unknown origin and with Vasculitic infarct is unusual. Vasculitis with ensuing infarction is likely caused by several mechanisms: strangulation of the vessels in the thick exudate at the base of the brain, T-cell-mediated inflammation of the adventitia leading to vessel wall necrosis and thrombosis, stretching of vessels by rapidly enlarging ventricles in the setting of acute hydrocephalus, and midbrain and frontal lobe infarction as the brain herniates.⁷

The mortality rate for untreated tuberculous meningitis is 100%.⁸ Even if adequately treated, the mortality rate is still very high when treatment

is initiated after patients have progressed to coma.⁹ Initiation of antituberculosis therapy should not wait for definitive diagnosis, but rather be instated at the first suspicion of tuberculous meningitis.¹⁰

Any patients with new onset headache and pyrexia of unknown origin should be evaluated extensively for CNS Tuberculosis even if inflammatory markers and Mantoux is negative. Patients should be started on ant tubercular agents based on clinical manifestations as definitive diagnosis may not be possible every time.

CONCLUSION

In Endemic countries Tubercular meningitis can present with varied atypical manifestations. High index of clinical suspicion is recommended in order to initiate early therapy which can result in good outcomes.

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