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Case Report

Tubercular meningitis with altered sensorium in a chronic alcoholic patient: a case report

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ABSTRACT

A 35-year-old Indian male was referred to Vidhatha hospital, Vijayawada, Andhra Pradesh with history of fever, shortness of breath (Grade III), drowsiness, altered sensorium and generalized weakness for 20 days. The patient is chronic alcoholic and smoker. On examination, patient was disoriented and exhibited neck stiffness. Pleural fluid was positive for *Mycobacterium tuberculosis*. CT brain indicated vasogenic oedema, and mild hypodense areas in bilateral periventricular regions. MRI brain suggested mild leptomeningeal enhancement in cortical and sub-cortical regions. CSF analysis revealed lymphocytic pleocytosis (100), lowered glucose level (21 mg/dL) and markedly elevated protein level (312.5 mg/dl). Owing to, a diagnosis of tubercular meningitis was established and antitubercular medication with rifampcin, isoniazid, ethambutal, and pyrazinamide, as well as dexamethasone was initiated. Within two days of treatment, he showed signs of improvement, recognised the surroundings and responded to short conversations. The patient was sensitized regarding the adverse effects of smoking and alcoholism and was advised to continue treatment for additional six months.

Keywords: Altered sensorium, Fever, *Mycobacterium tuberculosis*, Tubercular meningitis, Neuroimaging

INTRODUCTION

Tuberculous meningitis (TBM), a central nervous system infection of *Mycobacterium tuberculosis*, accounts for 70-80% of all central nervous system tuberculosis (TB) cases and is accountable for 20 to 60% of TB associated deaths in children.[1,2] TB is endemic in India with a 25% of world's TB burden, and an estimated incidence of 2.77 million.[3] Headache, fever, vomiting, meningeal signs, focal deficits, vision impairment, cranial nerve paralysis, and intracranial hypertension are the clinical characteristics of TBM, which develop slowly from few days to months.[4] It is an important neuropathy as around 50% of patients with TBM either die or have a disability. The TBM is frequently underreported because of difficulty in clinical diagnosis. Understanding the current status of infection in the population is inevitable to accomplish TB Mukt Bharat Abhiyaan.[5]

Owing to, we report a case of TBM in a 35-year-old Indian male patient, who was presented with fever, drowsiness and altered sensorium. Ill-defined hypodense areas in cerebral lobes on CT and mild leptomeningeal enhancement in cortical and subcortical regions on MRI suggested the TBM, which was confirmed on further CSF analysis.

CASE REPORT

A 35-year-old Indian male was referred to Vidhatha Hospital, Vijayawada from a primary health center with complaints of drowsiness and altered sensorium, following

a 20-day history of fever, mild cough, chest pain, shortness of breath (Grade III), drowsiness, and generalized weakness. The patient is chronic alcoholic and smoker. He has no history of drug abuse. On examination, patient was disoriented and exhibited neck stiffness, but his vitals were stable, with mild pallor. Complete blood and urine analysis was done (Table 1). Liver function tests revealed elevated enzymes. An abdominal and thoracic ultrasound revealed abnormalities in liver and lungs. The patient was closely monitored with continuous oxygen support and a Ryles feeding tube. Pleural fluid analysis was positive for *Mycobacterium tuberculosis*, suggesting tuberculous pleuritis. CT brain showed ill-defined hypodense areas in the right posterior parietal and occipital lobes, suggestive of vasogenic edema, and mild hypodense areas in bilateral periventricular regions (Figure 1).

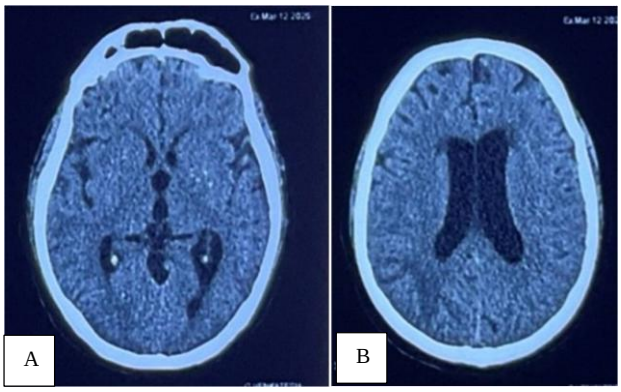

Figure 1 (A and B): Brain computerized tomography showing hypodense areas in right posterior parietal and occipital lobes. Mild hypodense area in bilateral periventricular region.

MRI brain showed ill-defined T2 flair hyperintensities with mild patchy restriction on DWI involving cortex and subcortical white matter in right temperoparietal region typical of neurological event. Post contrast study implied mild leptomeningeal enhancement in this region indicative of meningoencephalitis. Mild prominent bilateral lateral ventricles with periventricular T2 and flair hyperintensity, likely ventriculitis were noticed (Figure 2). On lumbar puncture and subsequent CSF analysis, lymphocytic pleocytosis, lowered glucose level and markedly elevated protein level denoting bacterial meningitis.

In view of the clinical signs, along with laboratory and imaging findings a diagnosis of tubercular meningitis was established. Despite the empirical treatment with IV fluids, Thiamine, and Ceftriaxone, since there was no change in the patient's status, antitubercular medication with rifampcin, isoniazid, ethambutal, and pyrazinamide, together with dexamethasone was initiated as per the national TB management guidelines 2019. Within two days of treatment, he showed signs of improvement, recognized the surroundings and responded to short conversations. He was shifted to ward and finally discharged healthy after two weeks of admission. The patient was sensitized regarding the adverse effects of smoking and alcoholism. Antitubercular treatment with isoniazid, rifampicin, and ethambutol was advised to continue for additional six months.

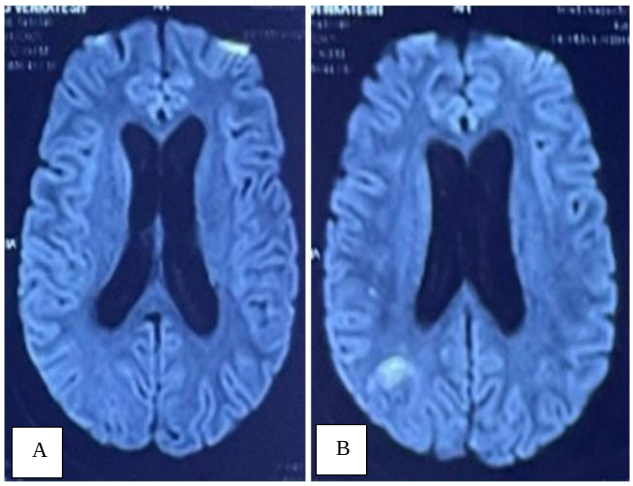

Figure 2 (A and B): MRI revealing mild leptomeningeal enhancement in cortical and subcortical of right temperoparietal region. Mild prominent bilateral lateral ventricles.

Table 1: Clinical and diagnostic features of patient.

Clinical/diagnostic features	Results	Note
Fever	Present	
Headache	Absent	
Altered sensorium	Present	
Neck stiffness	Present	
Blood		
Hb (13.5-17.5 g/dl)	10.2 g/dl	
WBC (4-11×10^3/µl)	6.5 × 10^3/µl	
Polymorphs (55-70% of WBCs)	87.5%	Slightly increased
Haematocrit (41-50%)	28.7%	Anaemia
ESR (0-15 mm/hr)	160 mm/hr	Elevated
Amylase (23-85 U/l)	105 U/l	Slightly elevated
Urine analysis	Normal	
Malaria and typhoid antibody test	Negative	

Continued.

Clinical/diagnostic features	Results	Note
Liver function tests		
AST (8-48 U/l)	101 U/l	Elevated
ALT (7-55 U/l)	86 U/l	
ALP (40-129 U/l)	248 U/l	
Ultrasound of the abdomen	Hepatomegaly with altered echo texture	
Thoracic ultrasound	Bilateral pleural effusion, left lower lobe consolidation of lung	
Pleural fluid analysis	*Mycobacterium tuberculosis* +ve	Tuberculous pleuritis
CT brain	Ill-defined hypodense areas of cerebral lobes	Evidence of oedema/infarcts
MRI brain	Leptomeningeal enhancement	Meningitis
CSF analysis		
Total leucocyte count (0-5)	10	Bacterial meningitis
Neutrophils (0-5)	100	
Glucose (50 to 80 mg/dl)	21 mg/dl	
Protein (15 to 45 mg/dl)	312.5 mg/dl	

DISCUSSION

TB is a typical cause of meningitis in the developing countries which are characterized by the high number of pulmonary TB cases.[4] Tubercular meningitis accounts for about 5% of extrapulmonary TB cases and is considered as a rare neuropathy because of its unusual symptoms and deferred laboratory examinations. Early accurate diagnosis and interventions is crucial, though challenging, due to its neurologic impairment, morbidity and mortality rates. Delayed diagnosis can cause cranial nerve impairment and interfere with normal vision that can be permanent.[6] The clinical features of TBM are difficult to differentiate from other category of meningitis. Specific features for instance chronic illness, for more than a week can predict TBM.[7] Accordingly, the present case (20 days) and all TBM cases (minimum 14 days) in north India had long history of fever, headache and stiff neck.[8] Hepatomegaly and markedly elevated liver enzymes, especially AST in this case could be due to chronic alcoholism.[9]

The definitive diagnosis of TBM is a complex of clinical, imaging and CSF analysis parameters. Including classical signs, the patient exhibited altered sensorium. Both CT and MRI scans along with CSF analysis were valuable for the validation of the TBM. The mild hypodense areas in the brain on CT scan, suggesting white matter oedema or small infarcts, are related to an increased chance of future stroke.[10] Vascular infarction is the mechanism responsible for many diverse clinical neurological abnormalities in TBM patients and accounts for a significant portion of irreversible neurological sequelae.[11] Similar to the present case Botha et al indicated ventriculomegaly, leptomeningeal enhancement, periventricular infarcts, and tuberculomas as the neuroimaging features of TBM. In healthy persons the lifetime risk of TB increases in HIV, under nutrition, diabetes and habits such as alcohol use and smoking.[12-14] Age (>30 year), gender (being male), living in urban area, and previous exposure were associated with TB infection in India while smoking and alcoholism were not, though alcohol use and smoking contributed significantly to TB cases globally as in the present case. Following treatment with antitubercular drugs the condition of patient was improved.[15] The option of anti-TB drugs is affected by the intensity of liver disease.[16] In the present case, except mild hepatomegaly and raised liver enzyme levels, no other parameters were indicative of liver disease. In India, the TB program provides counseling and support to the patients with smoking and alcohol use, considering the need for community sensitization regarding their adverse effects.[17]

CONCLUSION

Rapid and accurate diagnosis of tubercular meningitis is crucial for early intervention and better prognosis. With this context, the successful treatment of a 35-year-old male with fever, headache, stiff neck and altered sensorium due to tubercular meningitis, was reported.

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