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Clinical, CSF, and Radiological Profile of Tuberculous Meningitis in Adults: A Prospective Study.

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Abstract

Background: A severe and deadly case of TBM extrapulmonary tuberculosis with a high case fatality rate. It is important to diagnose early. The objective of this study was to evaluate clinical features, cerebrospinal fluid (CSF) characteristics and neuroimaging findings in adults. ***Methods:*** A prospective observational study was conducted over 12 months at a tertiary care center. Thirty adult patients were included who were diagnosed to have TBM based on clinical, CSF and radiological criteria. Utilized patient demographics and clinical symptoms, parameters of the CSF, and brain imaging findings (MRI/CT). ***Results:*** The age range of the study population was reported to be 38-50 years, with a mean age of 38.5 years. Most patients had a fever (100%), headache (86.7%), and stiff neck (80%). CSF analysis showed lymphocytic pleocytosis (mean 215 cells/mm^3), elevated protein (mean 190 mg/dL) and hypoglycorrhachia (mean 32 mg/dL). Radiological assessment in patients revealed basal meningeal enhancement in 73.3%, hydrocephalus in 46.7% and tuberculomas in 30%. ***Conclusion:*** Symptoms of TBM in adults usually include fever, headache, and meningeal signs which are classic. The recognition of a condition, together with the characteristic CSF lymphocytic pleocytosis, and basal enhancement, will offer an easy diagnosis and management.

Keywords: Tuberculous meningitis, Cerebrospinal fluid, Magnetic resonance imaging, Hydrocephalus.

INTRODUCTION

Tuberculous meningitis is the most dangerous form of tuberculosis affecting the central nervous system, and it is associated with high mortality and risk of permanent neurological disability among survivors. The disease generally occurs when there is hematogenous dissemination of Mycobacterium tuberculosis into the central nervous system producing meningeal inflammation, exudative basal meningitis, vasculopathy and obstruction of the cerebrospinal fluid pathways [1-3].

The clinical onset of TBM is typically subacute with fever, headache, vomiting, neck stiffness, altered mental status, seizures and focal neurological deficits.

The indistinctness of these symptoms can complicate the differential diagnosis from pyogenic, viral and fungal meningitis; especially early on. As a result, in order to make the diagnosis, clinical assessment should be synthesised with CSF findings, microbiological investigations if available, and neuroimaging [1,4,5].

Classical CSF findings in TBM include lymphocytic pleocytosis, raised protein concentration and reduced glucose concentration. The measurement of CSF adenosine deaminase may provide further supportive evidence, although its diagnostic performance would depend on the clinical scenario, comparator illnesses and cut-off value used [6-8].

Neuroimaging is useful for demonstrating basal meningeal enhancement as well as for detecting hydrocephalus, tuberculomas and cerebral infarcts [1,3,5,6]. During a 12-month period, adult cases of TBM presenting at a tertiary care center were studied to describe their clinical, CSF, and radiological profile.

MATERIALS AND METHODS

Study design and setting

This prospective observational study was conducted at a tertiary care center over a period of 12 months. The study included 30 adult patients aged 18 years or more with clinically suspected TBM.

Eligibility criteria

Patients were included when they had a clinical presentation suggestive of TBM, characteristic CSF findings including lymphocytic pleocytosis, elevated protein, and low CSF glucose relative to plasma glucose, along with neuroimaging findings consistent with TBM. This clinicoradiological approach is consistent with established diagnostic principles for TBM. Patients with an alternative diagnosis, including viral encephalitis, fungal meningitis, pyogenic meningitis, or another neurological disorder, were excluded.

Data collection

Detailed clinical history was recorded and neurological examination was performed for every participant. Demographic data included age and sex. Clinical features assessed were fever, headache, vomiting, neck stiffness, altered sensorium, seizures, and focal neurological deficits.

CSF examination included total leukocyte count, differential cell count, protein, glucose, and ADA level. Neuroimaging by CT or MRI brain was assessed for basal meningeal enhancement, hydrocephalus, tuberculomas, cerebral infarcts, and normal findings.

Statistical analysis

Categorical variables are expressed as frequency and percentage. Continuous variables are expressed as mean with range or predominant pattern, as applicable.

RESULTS

A total of 30 adult patients with TBM were evaluated during the 12-month study period. There were 18 males (60.0%) and 12 females (40.0%), showing mild male predominance. The mean age of the study population was 38.5 years.Fever was present in all patients. Headache was reported by 26 patients (86.7%), while 24 patients (80.0%) had neck stiffness. Vomiting was observed in 20 patients (66.7%). Altered sensorium was present in 15 patients (50.0%), whereas focal neurological deficits and seizures were observed in 8 patients (26.7%) and 6 patients (20.0%), respectively.

Table 1: Clinical Profile of Study Patients

Clinical feature	Number of patients (n=30)	Percentage (%)
Fever	30	100.0
Headache	26	86.7
Neck stiffness	24	80.0
Vomiting	20	66.7
Altered sensorium	15	50.0
Focal neurological deficits	8	26.7
Seizures	6	20.0

The mean CSF total cell count was 215 cells/mm³, ranging from 40 to 550 cells/mm³. A predominant lymphocytic response was observed, with a mean lymphocyte proportion of 85%. Mean CSF protein was 190 mg/dL and mean CSF glucose was 32 mg/dL. The mean CSF ADA level was 14.5 U/L.

Table 2: Cerebrospinal Fluid (CSF) Findings

CSF parameter	Mean value	Range/predominance
Total cell count (cells/mm³)	215	40–550
Lymphocyte percentage (%)	85	60–100
Protein (mg/dL)	190	80–450
Glucose (mg/dL)	32	15–45
Adenosine deaminase (U/L)	14.5	9.0–22.0

Basal meningeal enhancement was the most frequent imaging finding, observed in 22 patients (73.3%). Hydrocephalus was detected in 14 patients (46.7%). Tuberculomas and infarcts were identified in 9 patients (30.0%) and 6 patients (20.0%), respectively. Neuroimaging was normal in 3 patients (10.0%).'

Table 3: Radiological (MRI Brain) Findings

Radiological finding	Number of patients (n=30)	Percentage (%)
Basal meningeal enhancement	22	73.3
Hydrocephalus	14	46.7
Tuberculoma	9	30.0
Infarcts	6	20.0
Normal scan	3	10.0

DISCUSSION

The present prospective study evaluated 30 adults with TBM and found mild male predominance, with males constituting 60.0% of the cohort. The mean age was 38.5 years, indicating that TBM mainly affected adults in the economically productive age group. Kaur et al., in a prospective study of 55 adult TBM patients from North India, reported a similar male predominance, with 61.8% of patients being male and the largest proportion belonging to the 21–40-year age group [9].Fever was universal in the present cohort, followed by headache, neck stiffness, vomiting, and altered sensorium. The high frequency of headache and vomiting may reflect meningeal inflammation and increased intracranial pressure. Kaur et al. reported fever, headache, neck rigidity, altered sensorium, and vomiting as common clinical manifestations in adults with TBM [9]. Thwaites et al. also demonstrated that duration of illness and clinical features, combined with laboratory parameters, help differentiate TBM from bacterial meningitis [5,9].The CSF profile in this study was typical of TBM, showing lymphocytic pleocytosis, high protein concentration, and low glucose concentration. The mean CSF cell count was 215 cells/mm³, with an average lymphocyte predominance of 85%. Mean CSF protein was 190 mg/dL, whereas mean glucose was 32 mg/dL. Such findings are characteristic of TBM and reflect chronic meningeal inflammation, increased blood-CSF barrier permeability, and impaired glucose transport or utilization [1,3,5,9].

Kaur et al. reported mean CSF cell count, protein, and glucose values of 303.21 cells/mm³, 170.2 mg/dL, and 38.3 mg/dL, respectively, which are broadly comparable with those of the present cohort [9].The mean CSF ADA concentration was 14.5 U/L in this study. Kaur et al. reported a nearly identical mean CSF ADA level of 14.44 U/L among adult TBM patients [9]. A meta-analysis by Xu et al. found that CSF ADA has useful diagnostic accuracy for TBM, especially with cut-off values around 10 U/L. However, CSF ADA should not be considered independently diagnostic because overlap may occur with other inflammatory and infectious meningitides [8,10]. In the present study, the ADA finding was therefore interpreted as supportive in conjunction with clinical, conventional CSF, and imaging features [8-11].Basal meningeal enhancement was the most common radiological abnormality, seen in 73.3% of patients. This reflects basal inflammatory exudates, a characteristic pathological and imaging feature of TBM [1,3,12]. Kaur et al. reported basal exudates and meningeal enhancement among common CT/MRI findings [9], while Marais et al. described meningeal enhancement, hydrocephalus, infarcts, and parenchymal granulomas as characteristic radiological features of TBM [1,3,12].Hydrocephalus was identified in 46.7% of patients, making it the most frequent complication in the present study. It results from impaired CSF circulation due to basal meningeal exudates and may contribute to vomiting, altered sensorium, and neurological deterioration [13].

Gupta et al. reported hydrocephalus as a common complication of TBM that may occur in a large proportion of affected patients and requires early recognition because of its prognostic and therapeutic implications [1,13].Tuberculomas were observed in 30.0% of patients, while infarcts occurred in 20.0%. Tuberculomas result from focal granulomatous inflammation, whereas infarcts are typically related to TBM-associated vasculitis or vasculopathy affecting perforating cerebral arteries. These complications may contribute to focal neurological deficits, which were present in 26.7% of participants in the current study [3,12].The study has several limitations.

First, the sample size was small and findings may not be generalizable to all populations. Second, microbiological confirmation data, HIV status, disease severity staging, treatment details, and clinical outcomes were not available. Third, the study was descriptive and could not assess associations between CSF abnormalities, imaging findings, and patient outcomes. Nevertheless, the prospective design and combined evaluation of clinical, CSF, and radiological parameters provide a clinically relevant profile of adult TBM at a tertiary care center.

CONCLUSION

Tuberculous meningitis still presents in adults with severe clinical signs. Doctors must stay alert to prolonged fever and headache as potentially an indication of an infectious disease. The first parallel lymphocytic pleocytosis with low glucose level in CSF and basal enhancement on neuroimaging forms the mainstay of early presumptive diagnosis. Recognition based on this clinical, CSF, and radiological profile is timely and necessary to initiate early treatment and prevent the serious morbidity and mortality caused by the disease.

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