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Clinical and Cerebrospinal Fluid Profile of Patients with Meningitis: A Descriptive Case Series from a Tertiary Care Centre.

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Abstract

Background: Meningitis is a common cause of acute neurological presentation in India. Tubercular, bacterial and viral aetiologies have overlapping clinical features but characteristic CSF profiles, which help in early empirical management. Along these lines descriptive local data on the clinico-CSF spectrum of meningitis remains useful for the calibration of diagnostic suspicion in low-resource settings. ***Objective:*** To describe the demographic, clinical, and CSF profile of patients presenting with meningitis at a tertiary care centre, and to compare CSF biochemical and cytological parameters across tubercular, bacterial, and viral aetiologies. ***Methods:*** This descriptive case series included a total of 50 consecutive patients who had been evaluated between April 2025 to December 2025 with clinically suspected meningitis. The patients' demographics, presenting complaints, comorbidities, substance-use history, fundoscopy, CSF biochemistry (sugar, protein), cytology, CBNAAT, gram stain, culture, CSF ADA, CSF CRP and neuroimaging were noted. The final aetiological diagnosis given by treating clinicians was tubercular, bacterial, or viral based on clinical, CSF, microbiological and radiological evaluation. The Kruskal-Wallis test was used to compare continuous variables between groups. ***Results:*** Fifty patients (mean age 41.9 ± 15.6 years; 31 male and 19 female) were included. In total, 22 patient (44%) were classified as tubercular meningitis, 20 as viral (40%) and 8 (16%) as bacterial meningitis. The most common complaints were fever (96%), headache (86%) and vomiting (74%). Bacterial (36.4 mg/dL) and tuberculous (35.2 mg/dL) meningitis had the lowest CSF sugar compared to viral meningitis (82.9 mg/dL, p< 0.001). The study showed that CSF protein was significantly higher in tubercular and bacterial cases as compared to viral cases. Lymphocytic pleocytosis was seen as a predominant feature of tubercular meningitis with cellular composition of > 75%. The CSF ADA of patients with TB meningitis was substantial and statistically more when compared with other meningitides and also with the CSF CRP levels – the latter was most in bacterial meningitis, specifically, it was higher when compared with viral meningitis. Only two of the 22 tubercular cases are positive by CBNAAT. In 24 out of 46 patients (52%) examined, neuroimaging was abnormal. ***Conclusion:*** Within this cohort, CSF sugar, protein, differential cytology, ADA, and CRP revealed statistically significant and clinically meaningful distinctions across tubercular, bacterial, and viral meningitis, endorsing their persistent utilisation as initial adjuncts to direct empirical therapy while awaiting microbiological confirmation, which yielded low results in this analysis.

Keywords: Meningitis; Tuberculous meningitis; Cerebrospinal fluid; Adenosine deaminase; CBNAAT; India.

INTRODUCTION

In India, the continued occurrence of meningitis, an inflammation of the protective membranes covering the brain and spinal cord, caused by an infection is an important cause of acute neurological illness and hospitalisation [1]. The urgent treatment and prognosis of the three main infectious causes of meningitis–bacterial, tuberculous and viral–differ widely as do public health implications nevertheless at presentation they often present a similar syndrome of fever headache vomiting and altered sensorium [2]. Since empirical therapy (ATD, broad spectrum antibiotics or supportive care) needs to often get instituted before microbiological diagnosis has been achieved, the clinician places to a large extent, reliance on the cerebrospinal fluid (CSF) biochemistry and cytology for helping triage patient toward a probable aetiology.In tuberculosis endemic countries like India, tuberculous meningitis (TBM) has a substantial mortality and neurological morbidity when treatment is delayed [3]. Diagnosis of TB meningitis is difficult. The acid-fast bacilli smear has a low sensitivity. The CSF culture is more sensitive but takes weeks to result.

The introduction of cartridge-based nucleic acid amplification testing (CBNAAT/GeneXpert MTB/RIF) has considerably improved turnaround time. However, its sensitivity for TBM in published cohorts has been around 50–60% against culture or composite clinical reference standards. A negative CBNAAT cannot, therefore, rule out TBM [4]. The enzyme in the cerebrospinal fluid (CSF) that is released from activated T-lymphocytes during cell-mediated immune responses, adenosine deaminase (ADA) has therefore been evaluated extensively as a rapid, inexpensive adjunct marker for TBM. This has a pooled sensitivity and specificity in meta-analyses in the range of 85–90%. However, the cutoff values vary across populations and assay techniques [5-8].Data from single-centre studies in India comparing the clinical and CSF profiles of tuberculous, bacterial and viral meningitis can serve a useful purpose. They will help to locally validate the classical CSF-based differentiation and also profile the demographic and clinical background against which these aetiologies manifest. We thus decided to conduct this descriptive case series to study the demographic, clinical, and CSF biochemical, cytological and microbiological profiles of meningitis cases in our centre, and to compare these parameters between the three major aetiologies.

MATERIALS AND METHODS

Study design and setting: This was a hospital-based, descriptive observational case series conducted in the Department of General Medicine, Bangalore Medical College and Research Institute, India, between April 2025 and December 2025.

Study population: Fifty consecutive adult patients presenting with a clinical syndrome suggestive of meningitis who underwent diagnostic lumbar puncture were included in the study.

Inclusion criteria:

1. Patients age >18 years.
2. Clinical presentation suggestive of meningitis (presence of at least two of the following: fever, headache, vomiting, altered sensorium, seizures, or neck stiffness).
3. Patients undergoing a diagnostic lumbar puncture with successful CSF extraction.

Exclusion criteria:

1. Contraindications to lumbar puncture (e.g., severe coagulopathy, overlying skin infection at the puncture site, space-occupying lesion with mass effect on neuroimaging).
2. Traumatic lumbar puncture (grossly bloody CSF preventing accurate cytological and biochemical interpretation).
3. History of neurosurgical interventions or traumatic brain injury within the preceding 30 days.
4. Established non-infectious causes of meningeal inflammation (e.g., subarachnoid haemorrhage, neoplastic meningitis, autoimmune encephalitis).
5. Patients who had received more than 48 hours of targeted intravenous antimicrobial or antitubercular therapy prior to presentation.

Data collection: For each patient, the following were recorded on a structured proforma: demographic details (age, sex), presenting complaints, comorbidities, history of substance use (alcohol, smoking, intravenous drug use), fundoscopic examination, and CSF analysis comprising sugar, protein, total cell count with differential (neutrophils, lymphocytes), CBNAAT, Gram stain, culture and sensitivity, ADA, and C-reactive protein (CRP). Contrast-enhanced neuroimaging (CT or MRI brain) was performed where clinically indicated and findings were recorded qualitatively.

Case definition: The final aetiological diagnosis—tuberculous, bacterial, or viral meningitis—was assigned by the treating clinicians based on a composite assessment of clinical presentation, CSF biochemical and cytological profile, microbiological/molecular results, and neuroimaging findings, together with response to empirical therapy where applicable.

Statistical analysis: Continuous variables are expressed as mean ± standard deviation (SD) and median with interquartile range (IQR), and were compared across the three diagnostic groups using the Kruskal–Wallis test in view of the non-normal distribution and small subgroup sizes. Categorical variables are expressed as number (percentage) and were compared using the chi-square test. A two-sided p-value <0.05 was considered statistically significant. Analyses were performed using Python (pandas and scipy libraries).

RESULTS

A total of 50 patients with clinically suspected meningitis were evaluated. The mean age was 41.9 ± 15.6 years (median 41 years, range 18–80 years), and 31 (62%) were men and 19 (38%) were women. Based on composite clinical, CSF, microbiological, and radiological assessment, 22 patients (44%) were diagnosed with tuberculous meningitis, 20 (40%) with viral meningitis, and 8 (16%) with bacterial meningitis (Table 1).

Fever was the most common presenting complaint (96%), followed by headache (86%), vomiting (74%), altered sensorium (44%), and seizures (18%); one patient (2%) presented with a focal neurological deficit. Comorbidities were present in 18 patients (36%), most commonly type 2 diabetes mellitus (16%) and hypertension (10%); four patients (8%) had a prior history of pulmonary tuberculosis. A history of substance use was present in 18 patients (36%), predominantly alcohol use (30%). Fundoscopy was abnormal in 5 of 45 patients in whom it was recorded (11%), including papilloedema, disc hemorrhage, and optic atrophy.

Age and sex distribution did not differ significantly across the three diagnostic groups (Kruskal–Wallis p=0.073 for age; chi-square p=0.71 for sex), although patients with tuberculous meningitis were numerically younger (mean 36.0 years) than those with bacterial (45.6 years) or viral meningitis (47.0 years) (Table 2).

Table 1. Demographic and clinical characteristics of the study population (N = 50)

Characteristic	n	%
Total patients	50	100.0
Age, mean ± SD (years)	41.9 ± 15.6	—
Male sex	31	62.0
Female sex	19	38.0
Fever	48	96.0
Headache	43	86.0
Vomiting	37	74.0
Altered sensorium	22	44.0
Seizures	9	18.0
Focal neurological deficit	1	2.0
Any comorbidity	18	36.0
Type 2 diabetes mellitus	8	16.0
Hypertension	5	10.0
Prior pulmonary tuberculosis	4	8.0
Cerebrovascular accident	4	8.0
Any substance use	18	36.0
Alcohol use	15	30.0
Smoking	4	8.0
Intravenous drug use	1	2.0
Abnormal fundoscopy (of 45 recorded)	5	11.1

Table 2. Age and sex distribution by final diagnosis

Parameter	Bacterial (n=8)	Tuberculous (n=22)	Viral (n=20)	p-value*
Age, years (mean ± SD)	45.6 ± 16.9	36.0 ± 14.0	47.0 ± 15.2	0.073
Male sex, n (%)	6 (75.0)	13 (59.1)	12 (60.0)	0.71

Kruskal–Wallis test for age; chi-square test for sex.

CSF biochemical and cytological parameters differed significantly across the three groups (Table 3). CSF sugar was markedly lower in bacterial (36.4 ± 25.0 mg/dL) and tuberculous meningitis (35.2 ± 21.6 mg/dL) than in viral meningitis (82.9 ± 50.1 mg/dL, p<0.001). CSF protein was highest in tuberculous meningitis (293.4 ± 295.3 mg/dL) and bacterial meningitis (253.5 ± 113.4 mg/dL), and lowest in viral meningitis (85.9 ± 87.4 mg/dL, p<0.001). Total CSF cell counts were highest in bacterial meningitis (mean 862.2 cells/µL), intermediate in tuberculous meningitis (640.2 cells/µL), and lowest in viral meningitis (227.6 cells/µL, p=0.005). Differential cytology showed the expected pattern: neutrophilic predominance in bacterial meningitis (mean 782.8 neutrophils/µL) versus lymphocytic predominance in tuberculous meningitis (mean 464.4 lymphocytes/µL) and comparatively modest pleocytosis in viral meningitis (p<0.001 for neutrophils, p=0.030 for lymphocytes).

CSF ADA was significantly higher in tuberculous meningitis (mean 16.1 ± 6.8 U/L, median 15.0 U/L) than in bacterial meningitis (mean 6.5 ± 1.5 U/L) or viral meningitis (mean 5.4 ± 3.7 U/L, p<0.001), with minimal overlap between the tuberculous group and the other two groups. CSF CRP followed the opposite pattern, being markedly elevated in bacterial meningitis (mean 3.55 ± 0.76 mg/L) compared with tuberculous (0.51 ± 0.30 mg/L) and viral meningitis (0.38 ± 0.22 mg/L, p<0.001).

Table 3. Cerebrospinal fluid biochemical and cytological parameters by final diagnosis

CSF parameter (normal range)	Bacterial (n=8) Mean ± SD	Tuberculous (n=22) Mean ± SD	Viral (n=20) Mean ± SD	p-value*
Sugar, mg/dL (40–60)	36.4 ± 25.0	35.2 ± 21.6	82.9 ± 50.1	<0.001
Protein, mg/dL (15–45)	253.5 ± 113.4	293.4 ± 295.3	85.9 ± 87.4	<0.001
Total cell count, cells/µL	862.2 ± 628.4	640.2 ± 707.6	227.6 ± 302.2	0.005
Neutrophils, cells/µL	782.8 ± 580.7	176.2 ± 570.5	22.7 ± 27.0	<0.001
Lymphocytes, cells/µL	79.5 ± 52.4	464.4 ± 452.5	204.9 ± 276.6	0.030
ADA, U/L	6.45 ± 1.47	16.10 ± 6.81	5.41 ± 3.72	<0.001
CRP, mg/L	3.55 ± 0.76	0.51 ± 0.30	0.38 ± 0.22	<0.001

Kruskal–Wallis test across the three groups. ADA, adenosine deaminase; CRP, C-reactive protein.

Microbiological yield was low across all groups (Table 4). CBNAAT was positive in only 2 of 22 tuberculous meningitis cases (9%) and negative or not performed in the remainder; Gram stain showed pus cells in 4 of 8 bacterial meningitis cases (50%); and CSF culture was sterile (no growth) in all 36 cases in which it was sent, with no organism isolated in any patient in this series. Neuroimaging was performed in 46 of 50 patients and was abnormal in 24 (52%), most commonly showing leptomeningeal enhancement, tuberculoma, hydrocephalus, or infarction; imaging findings by group are summarised in Table 5.

Table 4. Microbiological findings by final diagnosis

Investigation / result	Bacterial (n=8)	Tuberculous (n=22)	Viral (n=20)
CBNAAT positive	0 (0.0%)	2 (9.1%)	0 (0.0%)
CBNAAT negative	7 (87.5%)	16 (72.7%)	17 (85.0%)
CBNAAT not done	1 (12.5%)	4 (18.2%)	3 (15.0%)
Gram stain — pus cells seen	4 (50.0%)	2 (9.1%)	1 (5.0%)
Gram stain — no pus cells / negative	4 (50.0%)	18 (81.8%)	17 (85.0%)
Gram stain — not done	0 (0.0%)	2 (9.1%)	2 (10.0%)
Culture — no growth / sterile	7 (87.5%)	15 (68.2%)	14 (70.0%)
Culture — not sent	1 (12.5%)	7 (31.8%)	6 (30.0%)

Table 5. Neuroimaging findings by final diagnosis

Neuroimaging finding	Bacterial (n=8)	Tuberculous (n=22)	Viral (n=20)
Abnormal	6 (75.0%)	11 (50.0%)	7 (35.0%)
Normal / no significant abnormality	1 (12.5%)	0 (0.0%)	2 (10.0%)
Not performed / not recorded	1 (12.5%)	11 (50.0%)	11 (55.0%)

DISCUSSION

The study was conducted on 50 patients with clinically suspected meningitis which showed that the single most common aetiology was tuberculous meningitis (44%) followed by viral meningitis (40%) which was close second. Bacterial meningitis was the final aetiology with a mere (16%)

The strong agreement of our distribution with other Indian tertiary-care series', whereby viral and tuberculous meningitis together comprise the large majority of cases and bacterial meningitis is comparatively less common, likely reflects India's endemic tuberculosis burden as well as the probable prior partial antibiotic exposure attenuating culture yield in bacterial cases [2].

This cohort replicated the classical CSF-based differentiation of the aetiologies of meningitis. According to the expectation, viral meningitis had preserved CSF sugar, modest elevation of protein and relatively low total cell count. Tuberculous and bacterial meningitis showed hypoglycorrhachia and markedly raised protein; but these findings alone do not separate tuberculous from bacterial disease. Differential cytology provided useful discriminatory value: bacterial meningitis was strongly dominated by neutrophilic pleocytosis while in the case of tuberculous meningitis there was lymphocytic predominance which are in keeping with the pyogenic versus granulomatous nature of meningeal inflammation.

ADA level in CSF is significantly elevated in TB meningitis (mean 16.1 U/L) as compared to bacterial and viral meningitis with comparable degree of separation as reported in literature. Using the often-cited cutoff of 10 U/L, published series report a sensitivity of approximately 71–95% and specificity of 84–95% for CSF ADA for the diagnosis of TBM [3, 5, 8, 9]. However, at least one large prospective study found a lower optimal cutoff (5.5 U/L) with more modest specificity and

cautioned against over-reliance on ADA as a stand-alone confirmation test [6]. Our results show that ADA is useful in a tuberculosis-endemic setting such as ours. Our findings should be interpreted alongside clinical and other CSF findings rather than in isolation. CSF CRP was consistently higher in bacterial meningitis, compared to the other two groups. CSF CRP is an acute-phase reactant likely driven by pyogenic bacterial inflammation. It may be a useful rapid adjunct, in the case where CRP assays are more available than ADA or molecular testing choice. However, it is not specific and can be influenced by systemic infection.

Microbiological confirmation in this series was rare, with only 9% of patients in the tuberculous meningitis category having a positive CBNAAT result; no organism was cultured from any patient, even those with bacterial meningitis. This shows the well-known paucibacillary nature of TBM and the limited sensitivity of the CBNAAT, which literature i.e. published cohorts report to lie roughly between 50% and 72% even against culture or composite clinical reference standards i.e. negative CBNAAT does not exclude diagnosis [4]. The complete lack of positive bacterial cultures from this cohort likely results from a combination of small sample size, prior antibiotic exposure before lumbar puncture, and limited culture sensitivity, and highlights the continued reliance on composite clinical–CSF criteria for real-world diagnosis and treatment decisions in this setting. Neuroimaging was abnormal in just over half of patients in whom it was performed, with leptomeningeal enhancement, tuberculoma, and hydrocephalus among the more common findings, broadly in keeping with published radiological correlates of tuberculous and bacterial meningitis, and again reinforces the complementary role of imaging with CSF analysis when microbiological confirmation is unavailable or delayed.

CONCLUSION

Through this descriptive case series, it was found that the common most aetiology was tuberculous and viral meningitis, the standard CSF biochemical and cytological parameters sugar, protein, and differential cell counts along with CSF ADA and CRP showed a statistically significant and clinically meaningful difference in tuberculous, bacterial and viral meningitis. These budget-friendly CSF parameters that are available rapidly continue to provide early diagnostic support in resource-limited, tuberculosis-endemic settings where, as in this series, microbiological yield is often low. There is a need for larger, multicentric studies that are prospective in nature and using standard case definition and clinical outcomes to validate findings and refine local diagnostic algorithms.

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