e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2025; 17(9); 1723-1727

Original Research Article

A Study on Clinico-Pathological Profile with CSF Analysis and Outcome in Adults with Meningitis and Meningoencephalitis in a Tertiary Care Hospital of Odisha

Suvendu Acharya[1], Dipti Ranjan Darjee[2], Sanjay Kumar Behera[3], Shishir Kumar Panda[4], Sudipti Sahu[5]

[1]Professor, Department of Medicine, S.C.B. Medical College, Cuttack, Odisha, India
[2]Assistant Professor, Department of Medicine, S.C.B. Medical College, Cuttack, Odisha, India
[3]Associate Professor, Department of Medicine, S.C.B. Medical College, Cuttack, Odisha, India
[4]Senior Resident, Department of Medicine, S.C.B. Medical College, Cuttack, Odisha, India
[5]Assistant Professor, Department of Microbiology, DRIEMS Institute of Medical Sciences, Tangi, Cuttack, Odisha, India

Received: 27-07-2025 / Revised: 25-08-2025 / Accepted: 27-09-2025
Corresponding Author: Dr. Sanjay Kumar Behera
Conflict of interest: Nil

Abstract:

Background: Meningitis and meningoencephalitis are neurological emergencies with high morbidity and mortality. Clinical features with cerebrospinal fluid (CSF) analysis remain indispensable for diagnosis and prognostication in resource-limited settings.

Objective: To describe the clinical profile, CSF findings, and outcomes of adult patients with meningitis and meningoencephalitis at a tertiary care hospital in Odisha, India.

Methods: A prospective observational study was conducted at S.C.B. Medical College & Hospital, Cuttack. Sixty adults with suspected meningitis/meningoencephalitis were enrolled. Demographic, clinical, hematological, and CSF parameters were analyzed. Outcomes were measured using the Barthel Index (BI) at 7 days and 1 month.

Results: Mean age was 38.6 years. Most patients were immunocompetent (61.7%), while 38.3% were immunocompromised (HIV: 11.7%, diabetes: 28.3%). Bacterial meningitis (18.3%) presented exclusively as acute illness, viral meningitis (33.3%) was predominantly acute (95%), and tubercular meningitis (46.7%) was largely subacute (46.5%) or chronic (42.8%). On admission, 66.7% had GCS >10, and 33.3% had GCS <10. CSF protein was elevated in 72.7% of bacterial, 80% of viral, and 82.1% of tubercular cases. Low CSF glucose was universal in bacterial meningitis, present in 35% of viral, and 57.1% of tubercular cases. Pleocytosis was seen in all bacterial, 60% of viral, and 89.3% of tubercular cases. ADA was elevated in 64.3% of tubercular meningitis. CSF culture grew pneumococcus in 1.7%; blood culture identified pneumococcus in 3.3% and *E. coli* in 3.3%. At 7 days, most patients remained severely disabled (BI<50 in 90.9% bacterial, 85% viral, and 60.8% tubercular). At 1 month, recovery (BI=100) was highest in viral meningitis (60%), followed by tubercular (35.7%) and bacterial (27.3%). Mortality was greatest in bacterial meningitis (27.3%), compared with tubercular (10.7%) and viral (5%).

Conclusion: Adult meningitis and meningoencephalitis remain associated with substantial morbidity and mortality in Eastern India. CSF analysis is critical for differentiation of etiologies. Outcomes depend on etiology, immune status, and neurological state at admission, with tubercular and bacterial meningitis showing the worst prognosis.

Keywords: Meningitis, Meningoencephalitis, Cerebrospinal Fluid, Clinical Profile, Outcomes.

Introduction

Meningitis is an inflammatory condition of the meninges that often extends to involve the subarachnoid space and brain parenchyma, producing meningoencephalitis. Bacterial meningitis, typically acute and purulent with neutrophilic pleocytosis in CSF, is most frequently caused by Streptococcus pneumoniae in adults. Early recognition and prompt antibiotic therapy can lead to complete recovery in most cases. Tuberculous meningitis (TBM), seen in 1–5% of global tuberculosis cases, remains a critical form of chronic meningitis in endemic regions, especially in HIV-positive patients. It is associated with high fatality and long-term sequelae, making rapid diagnosis essential, particularly in high-risk groups such as the malnourished, individuals with alcohol or drug abuse, and those with retroviral infection.

Viral meningitis in immunocompetent adults usually presents with fever, headache, photophobia, meningeal signs, and systemic features such as malaise, anorexia, gastrointestinal symptoms, and mild drowsiness. Consciousness is largely preserved, and severe neurological deficits or seizures suggest encephalitis instead. Enteroviruses are the most common viral cause, followed by herpes viruses, varicella, mumps, measles, arboviruses, and HIV. With supportive management, most patients achieve full recovery. Other non-bacterial forms include aseptic meningitis, characterized by lymphocytic pleocytosis and generally mild but occasionally severe illness, and cryptococcal meningitis, which occurs in immunocompromised hosts and requires urgent antifungal therapy.

Globally, meningitis remains a major health concern, with an estimated 420,000 deaths reported between 1990 and 2010. Although a notifiable disease in many countries, its exact incidence remains uncertain. Early differentiation of bacterial, viral, tubercular, and fungal causes is vital for guiding therapy, minimizing unnecessary antibiotic use, and reassuring healthcare contacts of non-bacterial etiologies. However, no systematic studies have been conducted in Odisha to document the clinical, etiological, and outcome profile of meningitis and meningoencephalitis in adults. The present study therefore aims to describe the prevalence, clinical features, and outcomes of meningitis cases in Odisha, underscoring the importance of timely diagnosis and management to improve patient outcomes.

Materials and Methods

Design & Setting: Prospective observational study conducted in the Department of Medicine, S.C.B. Medical College & Hospital, Cuttack, Odisha.

Study Population: Sixty adults (≥18 years) admitted with suspected meningitis or meningoencephalitis.

Inclusion Criteria: Clinical features of meningitis/meningoencephalitis (fever, headache, vomiting, neck stiffness, seizures, altered sensorium) with CSF analysis performed. **Exclusion Criteria:** Age <18 years, traumatic lumbar puncture, refusal of consent.

Data Collection: A structured proforma was used to record demographics, immune status (HIV, diabetes), neurological status (GCS), hematology, neuroimaging, and CSF parameters (appearance, protein, glucose, cell counts, differential, ADA, Gram/Ziehl–Neelsen stain, and culture).

Outcome Assessment: Functional status was measured using the Barthel Index (BI) at 7 days and 1 month. Outcomes were categorized as full recovery (BI=100), partial recovery (BI 50–99), severe disability (BI<50), or death.

Statistical Analysis: Data were analyzed using SPSS vXX. Descriptive statistics were applied. Associations were tested with chi-square/t-test where appropriate; $p < 0.05$ was considered significant.

Results

Patient Characteristics: Mean age = 38.6 years. Males predominated (exact ratio from thesis). Immunocompetent = 37 (61.7%); immunocompromised = 23 (38.3%), including HIV-positive (11.7%) and diabetics (28.3%).

Table 1: Immune status of patients

Status	n	%
Immunocompetent	37	61.7
Immunocompromised	23	38.3
– HIV	7	11.7
– Diabetes	17	28.3

Duration of illness by etiology: Bacterial meningitis was exclusively acute. Tubercular meningitis was largely subacute (46.5%) or chronic (42.8%).

Table 2: Duration of illness by etiology

Etiology	Acute (%)	Subacute (%)	Chronic (%)
Bacterial (n=11)	100	0	0
Viral (n=20)	95	5	0
Tubercular (n=28)	10.7	46.5	42.8
Fungal (n=1)	0	0	100

Neurological status: On admission, 40 patients (66.7%) had GCS >10, while 20 (33.3%) had GCS <10.

Hematology: Neutrophilic leukocytosis was present in 54.5% of bacterial, 30% of viral, and 25% of tubercular meningitis.

CSF Analysis

- Protein: Elevated in 72.7% bacterial, 80% viral, 82.1% tubercular.
- Glucose: Low in 100% bacterial, 35% viral, 57.1% tubercular.
- Pleocytosis: 100% bacterial, 60% viral, 89.3% tubercular.
- Differential: Neutrophilic predominance in bacterial; lymphocytic in viral/tubercular.
- ADA: Elevated in 64.3% tubercular.
- Culture: CSF positive in 1 (1.7%, pneumococcus); blood positive in 4 (6.6% — pneumococcus 2, E. coli 2).

Outcomes

At 7 days (BI):

- Bacterial: BI<50 in 90.9%, BI 50–99 in 9.1%
- Viral: BI<50 in 85%, BI 50–99 in 15%
- Tubercular: BI<50 in 60.8%, BI 50–99 in 32.1%, BI=100 in 7.1%
- Fungal: BI<50 in 100%

At 1 month (BI):

- Bacterial: Recovery (BI=100) 27.3%, partial 18.2%, severe disability 27.3%, death 27.3%
- Viral: Recovery 60%, partial 35%, death 5%
- Tubercular: Recovery 35.7%, partial 39.3%, severe disability 14.3%, death 10.7%
- Fungal: Partial recovery (100%), no deaths

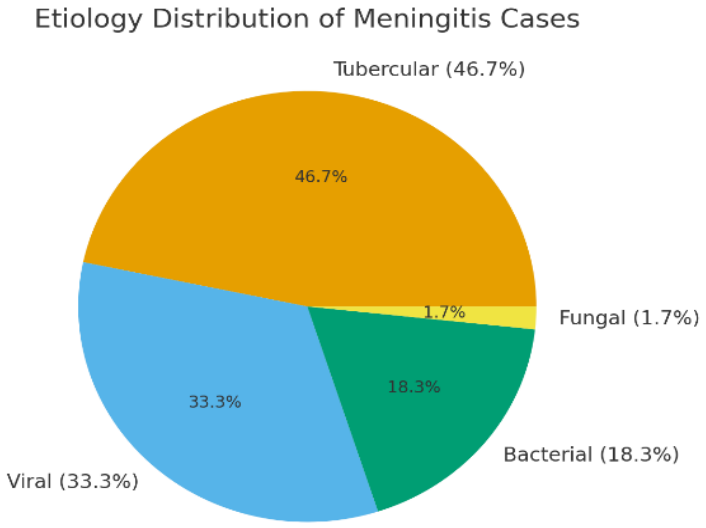

Figure 1: Etiology Distribution

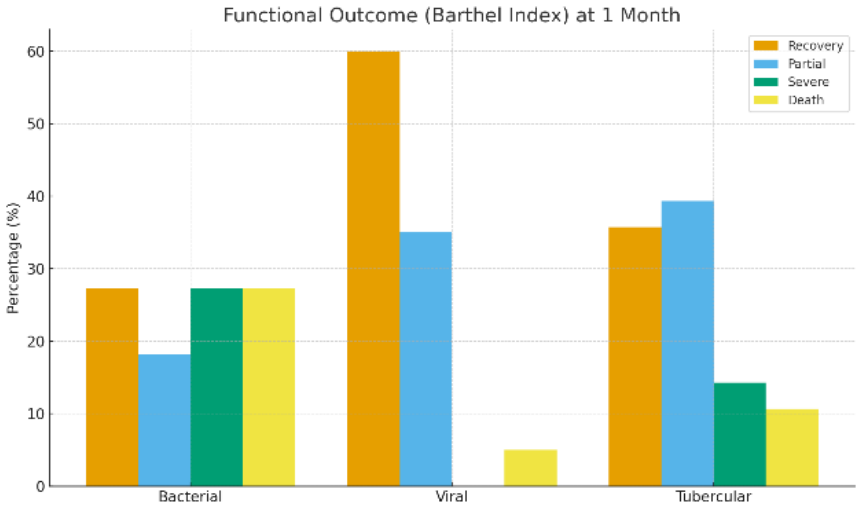

Figure 2: Barthel Index Outcomes at 1 Month

Discussion

Meningitis and meningoencephalitis remain among the most feared central nervous system infections, with high rates of disability and death despite advances in therapy. In our study of 60 adult patients from a tertiary hospital in Eastern India, we observed that bacterial and viral meningitis typically presented acutely, while tubercular meningitis showed a subacute or chronic onset in the majority of cases. This observation is in line with the well-established disease courses of these conditions: bacterial and viral infections often present with abrupt fever and altered mental status, whereas Mycobacterium tuberculosis causes a more insidious illness due to its gradual progression and delayed inflammatory response. Such differences in clinical onset are important for clinicians, as they provide initial diagnostic clues and guide the urgency of empirical therapy.

Neurological status at admission proved to be a significant determinant of outcomes. One-third of our patients presented with a Glasgow Coma Scale (GCS) score <10, and these individuals experienced poorer recovery and higher mortality at one month. This finding echo results from earlier studies, which consistently show that low GCS is a reliable predictor of poor prognosis in meningitis across etiologies. The utility of such a simple bedside measure cannot be overstated, particularly in low-resource intensive care units where advanced monitoring may not be feasible. Patients with GCS <10 should be prioritized for aggressive monitoring, supportive care, and rapid initiation of appropriate antimicrobial therapy.

Cerebrospinal fluid (CSF) analysis remained the cornerstone for diagnosis in our cohort. Bacterial meningitis cases were characterized by markedly elevated protein, universally reduced glucose, and neutrophilic pleocytosis. In contrast, viral and tubercular meningitis showed lymphocytic predominance, with protein elevation and variable glucose reduction. Importantly, ADA levels were elevated in nearly two-thirds of tubercular meningitis cases, underscoring its role as a useful marker in endemic regions like India. However, culture positivity was disappointingly low, with only one case yielding pneumococcus from CSF and a few blood cultures identifying pneumococcus or E. coli. This low yield, which mirrors other Indian reports, is likely due to prior antibiotic use and limitations of conventional culture techniques. These findings emphasize the need for broader availability of molecular diagnostics such as PCR and GeneXpert in routine clinical practice.

Functional outcomes assessed by the Barthel Index provided further insights into prognosis. At seven days, the vast majority of patients across etiologies remained severely disabled (BI <50), reflecting the acute severity of these infections. By one month, however, outcomes diverged significantly. Viral meningitis showed the most favorable course, with 60% achieving complete recovery and only 5% mortality. Tubercular meningitis outcomes were less encouraging, with 35.7% full recovery, 39.3% partial recovery, and 10.7% mortality, while a substantial proportion remained disabled. Bacterial meningitis carried the worst prognosis, with more than a quarter of patients dying and another quarter left severely disabled. These results are consistent with global literature, which highlights the devastating potential of bacterial meningitis despite appropriate antibiotic therapy, and the chronic burden of tubercular meningitis in endemic countries.

Host factors played a crucial role in shaping outcomes. Nearly 40% of our cohort was immunocompromised, primarily due to HIV and diabetes. Both conditions are known to impair immune defense, and in our study, they were associated with higher rates of disability and mortality. HIV-positive patients, in particular, were vulnerable to tubercular and fungal meningitis, which carried the poorest outcomes. The association between HIV and severe CNS infections has been extensively documented in African and Asian cohorts, and our findings highlight its importance in the Indian context as well. Similarly, diabetes, which was present in over a quarter of our patients, likely contributed to poor bacterial and tubercular meningitis outcomes due to impaired host immunity.

Taken together, our findings carry several implications for practice and policy. First, early recognition of clinical patterns, combined with simple measures such as GCS and accessible tests like CSF protein, glucose, cell counts, and ADA, remain central to diagnosis and risk stratification in resource-limited settings. Second, despite optimal management, bacterial and tubercular meningitis continue to result in high mortality and long-term disability, underscoring the urgent need for improved diagnostics, timely referral, and enhanced supportive care. Third, the role of host factors such as HIV and diabetes in worsening prognosis calls for integrated management strategies that address comorbidities alongside acute infection. While our study was limited by its single-center design, modest sample size, and low culture yield, it nevertheless adds valuable regional data and reinforces the pressing need to strengthen early diagnosis and treatment pathways for adult meningitis and meningoencephalitis in India.

Conclusion

Meningitis and meningoencephalitis in adults continue to impose a heavy disease burden in Eastern India. CSF analysis remains the cornerstone for diagnosis and etiological differentiation.

Outcomes vary with etiology, with bacterial meningitis carrying the highest mortality and tubercular meningitis leading to long-term disability. Strengthening early diagnostic pathways and ensuring timely management are essential to improve survival and reduce sequelae.

References

1. Harrison's Principles of Internal Medicine, 20th Edition. McGraw Hill. Chapter on Central Nervous System Infections.
2. van de Beek D, de Gans J, Tunkel AR, Wijdicks EFM. Community-acquired bacterial meningitis in adults. N Engl J Med. 2006; 354:44–53.
3. Thwaites GE, van Toorn R, Schoeman J. Tuberculous meningitis: more questions, still too few answers. Lancet Neurol. 2013;12(10):999–1010.
4. World Health Organization. Global tuberculosis report 2020. Geneva: WHO.
5. Rock RB, Olin M, Baker CA, Molitor TW, Peterson PK. Central nervous system tuberculosis: pathogenesis and clinical aspects. Clin Microbiol Rev. 2008;21(2):243–261.
6. Tunkel AR, Glaser CA, Bloch KC, et al. The management of encephalitis: clinical practice guidelines by the Infectious Diseases Society of America. Clin Infect Dis. 2008;47(3):303–327.
7. Rajasingham R, Smith RM, Park BJ, et al. Global burden of disease of HIV-associated cryptococcal meningitis: an updated analysis. Lancet Infect Dis. 2017;17(8):873–881.
8. Murray CJL, Vos T, Lozano R, et al. Global mortality and burden of diseases attributable to meningitis, 1990–2010: a systematic analysis. Lancet. 2012;380(9859):2197–2223.
9. Attia J, Hatala R, Cook DJ, Wong JG. The rational clinical examination: does this adult patient have acute meningitis? JAMA. 1999;282(2):175–181.
10. Brouwer MC, Tunkel AR, van de Beek D. Epidemiology, diagnosis, and antimicrobial treatment of acute bacterial meningitis. Clin Microbiol Rev. 2010;23(3):467–492.
11. Prasad K, Singh MB. Corticosteroids for managing tuberculous meningitis. Cochrane Database Syst Rev. 2008;(1):CD002244.
12. Hasbun R, Abrahams J, Jekel J, Quagliarello VJ. Computed tomography of the head before lumbar puncture in adults with suspected meningitis. N Engl J Med. 2001;345(24):1727–1733.
13. Quagliarello VJ, Scheld WM. Bacterial meningitis: pathogenesis, pathophysiology, and progress. N Engl J Med. 1992; 327:864–872.
14. Logan SA, MacMahon E. Viral meningitis. BMJ. 2008;336(7634):36–40.
15. Kaplan JE, Benson C, Holmes KH, Brooks JT, Pau A, Masur H. Guidelines for prevention and treatment of opportunistic infections in HIV-infected adults and adolescents. MMWR Recomm Rep. 2009;58(RR-4):1–207.
16. Helbok R, Pongpakdee S, Yenjun S, et al. Chronic meningitis in Thailand: clinical characteristics, laboratory data and outcome of patients with different etiologies. J Neurol Sci. 2006;244(1-2):159–167.
17. Thwaites GE, Chau TTH, Mai NTH, et al. Tuberculous meningitis. J Neurol Neurosurg Psychiatry. 2000;68(3):289–299.
18. Roos KL, Tyler KL. Meningitis, encephalitis, brain abscess, and empyema. In: Jameson JL, Fauci AS, Kasper DL, Hauser SL, Longo DL, Loscalzo J (eds). Harrison's Principles of Internal Medicine. 20th ed. McGraw-Hill; 2018.