DOI: 10.35990/mk.v9n1.p104-114

Medika Kartika: Jurnal Kedokteran dan Kesehatan

LITERATURE REVIEW

CEREBROSPINAL FLUID ANALYSIS IN TUBERCULOUS MENINGITIS: DIAGNOSTIC, PROGNOSTIC, AND MONITORING PERSPECTIVES

(ANALISIS CAIRAN SEREBROSPINAL PADA MENINGITIS TUBERKULOSIS: DIAGNOSTIK, PROGNOSTIK, DAN MONITORING)

Kiky Monica Soesanto[1], Ni Made Susilawathi[2], A.A. Raka Sudewi[2]

[1]Neurology Specialist Program, Medical of Faculty, Universitas Udayana, Bali, Indonesia
[2]Neurology Department, Medical of Faculty, Universitas Udayana, Bali, Indonesia

Correspondence email: raka_sudewi@unud.ac.id

ABSTRACT

Tuberculous meningitis (TBM) is the most severe form of extrapulmonary tuberculosis, associated with high morbidity and mortality. This review aims to evaluate the role of cerebrospinal fluid (CSF) analysis in the diagnosis and prognosis, particularly in relation to mortality, as well as in therapeutic monitoring of TBM. A narrative literature review was conducted using studies published between 2010 and 2025 on CSF parameters in TBM. Studies reporting associations between CSF biomarkers and clinical outcomes were included. Eighteen eligible studies were analyzed qualitatively. CSF analysis plays a critical role in classifying TBM as definite, probable, or possible according to the Lancet criteria. Elevated CSF protein (≥2 g/L) and reduced glucose (<2.2 mmol/L) levels are strongly associated with mortality, reflecting severe inflammation and blood-brain barrier dysfunction. Additional markers such as CSF lactate, inflammatory cytokines, and CRP further support prognostication. Serial measurements of CSF parameters aid in monitoring treatment response, especially in cases with atypical clinical progress. This review also highlights the diagnostic and monitoring roles of CSF parameters throughout the course of TBM. CSF biomarkers, especially protein and glucose levels, are key to assessing disease severity, predicting outcomes, and guiding treatment strategies in TBM. These findings support the integration of CSF analysis into routine clinical algorithms for managing this life-threatening condition.

Keywords: *cerebrospinal fluid, diagnosis, monitoring, prognosis, tuberculous meningitis*

ABSTRAK

Meningitis tuberkulosis (TBM) merupakan bentuk paling berat dari tuberkulosis ekstraparu dengan angka morbiditas dan mortalitas yang tinggi. Tulisan ini bertujuan mengevaluasi peran analisis cairan serebrospinal (CSS) dalam diagnosis, prognosis, khususnya terkait mortalitas, dan pemantauan terapi TBM. Penelitian ini merupakan tinjauan literatur naratif terhadap studi yang dipublikasikan pada periode 2010–2025 mengenai parameter CSS pada MTB. Studi yang melaporkan hubungan antara biomarker CSS dan luaran klinis dimasukkan dalam analisis. Sebanyak 18 studi yang memenuhi kriteria dianalisis secara kualitatif. Analisis CSS memegang peranan penting dalam klasifikasi TBM sebagai pasti, probable, atau mungkin menurut kriteria

Lancet. Peningkatan kadar protein CSS (≥2 g/L) dan penurunan glukosa (<2,2 mmol/L) sangat berkaitan dengan mortalitas, mencerminkan inflamasi berat dan disfungsi sawar darah-otak. Penanda tambahan seperti laktat CSS, sitokin inflamasi, dan CRP turut mendukung prognosis. Analisis CSS juga penting dalam diagnosis awal dan klasifikasi TBM, terutama di daerah dengan keterbatasan teknologi mikrobiologis. Selain itu, pengukuran serial parameter CSS berperan penting dalam pemantauan respons terapi, terutama pada pasien dengan perkembangan klinis atipikal. Biomarker CSS terutama kadar protein dan glukosa merupakan kunci dalam menilai tingkat keparahan penyakit, memprediksi hasil, dan membimbing strategi pengobatan pada TBM. Peran diagnostik, prognostik, dan pemantauan terapinya menekankan pentingnya integrasi dalam algoritma klinis rutin untuk penatalaksanaan kondisi yang mengancam jiwa ini.

Kata kunci: cairan serebrospinal, diagnosis, meningitis tuberkulosis, monitoring, prognosis

INTRODUCTION

Tuberculous meningitis (TBM) is the most severe form of central nervous system tuberculosis infection caused by *Mycobacterium tuberculosis*, and it contributes to high morbidity and mortality rates, especially in developing countries.[1,2,3] This condition is characterized by intense meningeal inflammation, disruption of the blood-brain barrier, and dysregulated immune responses within the central nervous system, which may result in serious complications such as hydrocephalus, cerebral infarction, and impaired consciousness.[4] Despite standardized antitubercular therapy and adjunctive corticosteroid use, mortality and long-term neurological disability remain unacceptably high, highlighting the need for improved risk stratification and monitoring strategies.[5]

According to the Global Tuberculosis Report 2023, Indonesia ranks second worldwide in the number of TB cases after India, with an estimated incidence of 969,000 cases, or 354 per 100,000 population.[6] Approximately 5 to 10% of TB cases progress to extrapulmonary disease, among which TBM represents the most devastating and life-threatening manifestation. TBM is associated with high mortality and long-term neurological disability, affecting nearly 50% of patients.[4,7] This burden exceeds that of bacterial meningitis, for which the World Health Organization reports that one in six patients dies and one in five survivors develops severe complications.[8] A study by Feng et al. reported that elevated cerebrospinal fluid (CSF) protein levels and reduced CSF glucose concentrations were independently associated with increased mortality among adult patients with tuberculous meningitis requiring intensive care.[2] The diagnosis of TBM is often delayed because its symptoms are nonspecific and resemble those of other nervous system infections. Therefore, CSF analysis plays a

crucial role in establishing the diagnosis and assessing the patient's prognosis.[9] Two important parameters in CSF examination are protein levels and glucose levels. Increased CSF protein levels reflect impaired blood-brain barrier permeability due to the inflammatory process, while decreased glucose levels indicate increased metabolism by immune cells and pathogenic bacteria.[10,11] Several studies have suggested that high protein levels (>2.5 g/L) and low glucose levels (<2.2 mmol/L) in CSF are closely associated with an increased risk of death in TBM patients.[12,13,14] These biochemical changes have also been found to be associated with a severe immune response and increased levels of inflammatory cytokines such as IL-6 and TNF-α.[15,16]

This systematic review aims to analyze the association between cerebrospinal fluid protein and glucose levels and mortality in patients with tuberculous meningitis. It is expected that this review will provide a basis for clinical decision-making in determining prognosis and guiding treatment strategies for TBM patients in a more timely and effective manner.

METHODS

This study was conducted as a systematic literature review with narrative synthesis to summarize current evidence regarding the diagnostic, prognostic, and therapeutic monitoring roles of cerebrospinal fluid (CSF) parameters in tuberculous meningitis. A structured search of PubMed, Scopus, Web of Science, and Embase was performed to identify relevant articles published between 2010 and 2025 using the keywords “tuberculous meningitis,” “cerebrospinal fluid,” “protein,” “glucose,” “biomarkers,” “mortality,” and “prognosis.” Only English-language studies involving human subjects were included.

The initial search yielded 1,245 records. After removing duplicates and screening titles and abstracts, 132 articles were considered potentially relevant. Full-text assessment was performed for 40 studies, and 18 articles were ultimately included based on their relevance to CSF biomarkers and their association with diagnostic classification, disease severity, mortality, or treatment response in TBM.
Original research articles evaluating CSF biochemical, cellular, or inflammatory parameters in patients with TBM were included. Animal studies, case reports, small case series, conference abstracts without full text, review articles, and non-English publications were excluded. Due to heterogeneity in study design, biomarker thresholds, and outcome definitions, quantitative meta-analysis was not performed. Instead, findings were

synthesized qualitatively to identify consistent patterns between CSF parameters and TBM outcomes.

RESULTS AND DISCUSSION

CSF Analysis in TBM Diagnosis

The Lancet consensus criteria (Table 1) provide a standardized framework for the diagnosis of TBM, classifying cases as definite, probable, or possible based on clinical features, cerebrospinal fluid findings, neuroimaging, and microbiological evidence.[1] CSF analysis constitutes a core component of this diagnostic algorithm. Typical CSF abnormalities reported across studies include lymphocytic pleocytosis ranging from 5 to 1000 cells/μL, although early neutrophilic predominance may be observed in the initial phase of the disease.[1,3] Protein concentrations are commonly elevated (45–360 mg/dL), and reduced CSF glucose levels (<2.2 mmol/L) or a CSF-to-serum glucose ratio of <0.5 are frequently documented.[3,9] Elevated opening pressure is reported in approximately half of TBM cases, particularly among patients with hydrocephalus or cerebral edema.[3]

A multicenter evaluation of the Lancet diagnostic scoring system demonstrated a sensitivity of approximately 50% and a specificity exceeding 85%, highlighting the limited diagnostic accuracy of CSF parameters when used in isolation.[1] In clinical practice, the integration of CSF analysis with neuroimaging and molecular diagnostics, such as the GeneXpert MTB/RIF assay, substantially improves diagnostic confidence and classification accuracy.[9,11]

Table 1 Lancet consensus diagnostic criteria for TBM

Diagnostic Category	Clinical Criteria	CSF Criteria	Imaging	Microbiological Confirmation
Definite TBM	Not mandatory	Not mandatory	Not mandatory	Detection of AFB in CSF by smear, culture, or PCR
Probable TBM	≥4 points (if imaging) or ≥5 points (if no imaging), with ≥2 CSF points	Pleocytosis, protein >1 g/L, glucose <2.2 mmol/L	Hydrocephalus, basal meningeal enhancement, etc.	Negative or not done
Possible TBM	6-9 points total	At least one CSF criterion	May or may not be available	Negative or not done

Adapted from: (Marais et al., 2010)

Although conventional CSF parameters form the basis of TBM diagnosis, their standalone diagnostic yield is limited. Ziehl–Neelsen smear microscopy of CSF has low sensitivity due to the paucibacillary nature of TBM, while CSF culture, although considered the microbiological reference standard, is constrained by variable yield and prolonged incubation time.[1,4,17]

Molecular diagnostics have significantly improved early detection of TBM. The Xpert MTB/RIF assay demonstrates moderate sensitivity and high specificity for TBM, and its diagnostic performance improves with larger CSF volumes.[18] The Xpert MTB/RIF Ultra assay further increases sensitivity, particularly in definite and probable TBM, albeit with a small reduction in specificity.[19]

Taken together, these findings support the use of an integrated diagnostic approach that combines clinical assessment, CSF analysis, neuroimaging, and molecular testing to improve diagnostic accuracy and reduce diagnostic delay in TBM.[4]

While these diagnostic criteria provide a structured framework for identifying tuberculous meningitis, cerebrospinal fluid findings also play an important role in assessing disease severity and predicting clinical outcomes. Several CSF biochemical and inflammatory markers have been investigated as potential prognostic indicators in TBM, as summarized in Table 2.

Prognostic Value of CSF Parameters

Multiple studies have consistently reported that routine CSF biochemical parameters are associated with disease severity and clinical outcomes in tuberculous meningitis (Table 2). Elevated CSF protein levels, typically ≥1 g/L, reflect disruption of the blood-brain barrier and intense meningeal inflammation and are associated with more severe disease and increased mortality.[3,4,13] Reduced CSF glucose concentrations (<2.2 mmol/L) or a decreased CSF-to-serum glucose ratio have also been linked to poor neurological outcomes and higher mortality in TBM patients.[1,20]

Additional CSF biomarkers have also been investigated for their prognostic value. Elevated CSF lactate levels (>3.5 mmol/L) have been associated with cerebral hypoxia, vasculitis, and an increased risk of cerebral infarction, which may contribute to poorer survival outcomes.[20,21] Increased concentrations of inflammatory cytokines in the CSF, including interleukin-6 (IL-6), interferon-γ (IFN-γ), and tumor necrosis factor-α (TNF-α), reflect intense intracerebral immune activation and have been correlated with disease severity and adverse neurological outcomes.[9,27]

In addition to biochemical markers, the degree of CSF pleocytosis may also reflect the intensity of the inflammatory response in TBM. Both marked pleocytosis and paradoxically low CSF cell counts have been associated with severe disease and unfavorable outcomes in several patient cohorts.[1,4] Taken together, these findings suggest that no single CSF parameter is sufficient to predict prognosis in TBM. Instead, an integrated interpretation of CSF biochemical, cellular, and inflammatory markers may provide a more reliable assessment of disease severity and mortality risk. Key prognostic CSF biomarkers reported in previous studies are summarized in Table 2.

Table 2 Key Prognostic CSF Markers in TBM

Parameter	Abnormal Level	Prognostic Significance	Reference(s)
CSF Protein	Elevated (>1 g/L)	Reflects disruption of the blood-brain barrier and intense meningeal inflammation; associated with higher disease severity and mortality	3,4,13
CSF Glucose	Low (<2.2 mmol/L) or CSF-to-serum glucose ratio <0.5	Indicates high metabolic activity of inflammatory cells and impaired glucose transport; associated with poor neurological outcomes	1,20
CSF Cell Count	Marked pleocytosis	Reflects the inflammatory response; extremely high or paradoxically low counts may indicate severe disease	1,4
CSF Lactate	Elevated (>3.5 mmol/L)	Reflects cerebral hypoxia, vasculitis, and impaired oxidative metabolism; associated with infarction and worse prognosis	20,21
CSF Cytokines (IL-6, IFN-γ, TNF-α)	Elevated inflammatory cytokine levels	Indicates strong intracerebral immune activation and disease severity; may correlate with clinical outcomes	9,27

Adapted from: Marais et al., 2010[1]; Budiman et al., 2018[3]; Wilkinson et al., 2017[4]; Whitworth et al., 2021[9]; Thao et al., 2018[13]; Nuwagira et al., 2022[20]; Manyelo et al., 2021[21]; Dian et al., 2025[27]

CSF Analysis in Monitoring Treatment Response

Serial CSF examinations were reported to be useful in monitoring treatment response during antitubercular therapy. A gradual decline in CSF white blood cell counts was commonly observed among patients who demonstrated clinical improvement, whereas persistent

pleocytosis was associated with poor response or paradoxical reactions.[15,24] Trends toward normalization of CSF protein and glucose levels were reported to correlate with favorable treatment outcomes, while prolonged abnormalities prompted further diagnostic evaluation or treatment adjustments.[24,25,26]

Several studies also evaluated inflammatory and endothelial injury markers in treatment monitoring. Elevated CSF matrix metalloproteinase-9 (MMP-9), particularly when combined with soluble CD163, was identified as an independent predictor of short-term mortality.[26] Neuroimaging findings, when interpreted alongside serial CSF changes, assist in identifying complications such as hydrocephalus, immune reconstitution inflammatory syndrome, and persistent tuberculomas.[27]

The findings of this literature review highlight the central role of CSF analysis in the diagnosis, prognosis, and monitoring of TBM. Characteristic CSF abnormalities reflect the underlying pathophysiology of chronic meningeal inflammation caused by *Mycobacterium tuberculosis*, including disruption of the blood–brain barrier and persistent immune activation.[3,9]

Elevated CSF protein levels indicate increased vascular permeability and tissue injury and have consistently been associated with greater disease severity and poorer clinical outcomes.[12–14] Similarly, reduced CSF glucose levels reflect increased metabolic activity of inflammatory cells and impaired glucose transport across the inflamed blood–brain barrier, supporting their prognostic significance in TBM.[3,9,20]

Additional biomarkers such as CSF lactate and inflammatory mediators further highlight the complexity of TBM pathogenesis and may enhance prognostic stratification when combined with routine CSF parameters.[21,26,27] Nevertheless, heterogeneity in study designs and laboratory methodologies limits direct comparison across studies. Future prospective studies with standardized CSF assessments are required to establish validated biomarker thresholds and integrate CSF parameters into reliable prognostic models for TBM.

From a clinical perspective, routine CSF parameters may provide valuable information for early risk stratification in patients with TBM. Elevated CSF protein, reduced CSF glucose, and increased inflammatory mediators may collectively indicate severe meningeal inflammation and a higher risk of neurological complications or mortality. Because these laboratory parameters are widely available and relatively inexpensive, they may assist clinicians in identifying high-risk patients who require closer monitoring, early neuroimaging evaluation, and more

aggressive management strategies. In resource-limited settings, where advanced molecular diagnostics or biomarker assays are not routinely accessible, careful interpretation of routine CSF findings may therefore remain an important component of prognostic assessment in TBM.

This review has several strengths. First, the literature search was conducted using a PRISMA-guided strategy across multiple major databases, allowing for the comprehensive identification of relevant studies. Second, the review specifically focused on cerebrospinal fluid biomarkers associated with disease severity and mortality, providing clinically meaningful insights for prognostic assessment in TBM. The inclusion of routinely available CSF parameters also enhances the applicability of the findings, particularly in resource-limited settings where advanced diagnostic tools may be unavailable.

Several limitations should be acknowledged. The included studies demonstrated heterogeneity in study design, patient populations, laboratory methods, and biomarker cutoff values, which limited direct comparison between studies. Most studies were observational and retrospective, which introduces potential selection bias and confounding. In addition, variability in the timing of CSF sampling and treatment initiation may have influenced the reported associations. These limitations highlight the need for prospective studies with standardized CSF assessment protocols to validate and refine the prognostic utility of CSF biomarkers in tuberculous meningitis.

CONCLUSION

Cerebrospinal fluid (CSF) analysis remains a fundamental component in the management of tuberculous meningitis. Routine CSF parameters and selected biomarkers provide important diagnostic, prognostic, and therapeutic monitoring information. Elevated CSF protein levels, reduced CSF-to-serum glucose ratios, and the degree of pleocytosis have been associated with disease severity, mortality, and long-term neurological disability. Therefore, a comprehensive and serial interpretation of CSF parameters is essential for risk stratification and assessment of treatment response. Integrating CSF findings with clinical evaluation, neuroimaging, and microbiological testing may improve early diagnostic accuracy and patient outcomes, particularly in high tuberculosis-burden countries such as Indonesia.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

ACKNOWLEDGMENTS

The authors would like to express their sincere appreciation to the clinical staff and fellow residents of the Department of

Neurology, Faculty of Medicine, Universitas Udayana, who contributed to this study and the overall research process.

REFERENCES

1. Marais S, Thwaites G, Schoeman JF, Török ME, Misra UK, Prasad K, et al. Tuberculous meningitis: a uniform case definition for use in clinical research. Lancet Infect Dis. 2010 Nov;10(11):803–12.
2. Feng B, Fei X, Sun Y, Zhang X, Shang D, Zhou Y, et al. Prognostic factors of adult tuberculous meningitis in intensive care unit: a single-center retrospective study in East China. BMC Neurol. 2021 Dec 10;21(1):308.
3. Budiman ATM, Suraya N, Faried A, Parwati I. Characteristics of cerebrospinal fluid in tuberculous meningitis patients with hydrocephalus. Int J Integr Health Sci. 2018;6(2):57–62.
4. Wilkinson RJ, Rohlwink U, Misra UK, Van Crevel R, Mai NTH, Dooley KE, et al. Tuberculous meningitis. Nat Rev Neurol. 2017;13(10):581–98.
5. Stadelman AM, Ellis J, Samuels THA, Mutengesa E, Dobbin J, Ssebambulidde K, et al. Treatment outcomes in adult tuberculous meningitis: a systematic review and meta-analysis. Open Forum Infect Dis. 2020;7(7):ofaa257.
6. WHO. Global Tuberculosis Report 2023 [Internet]. World Health Organization. 2023 [cited 2025 Dec 4]. Available from: https://www.who.int/teams/global-programme-on-tuberculosis-and-lung-health/tb-reports/global-tuberculosis-report-2023
7. Isaiah AR, Williams M, Loots DT, Williams AA, Chegou NN, van Furth MT, et al. Potential biomarkers for tuberculous meningitis diagnosis using metabolomics and proteomics: a systematic review. BMC Infect Dis. 2025;25(1):1298.
8. World Health Organization. Meningitis [Internet]. Geneva: World Health Organization; 2025 [cited 2025 Dec 24]. Available from: https://www.who.int/news-room/fact-sheets/detail/meningitis
9. Whitworth LJ, Troll R, Pagán AJ, Roca FJ, Edelstein PH, Troll M, et al. Elevated cerebrospinal fluid cytokine levels in tuberculous meningitis predict survival in response to dexamethasone. Proc Natl Acad Sci U S A. 2021 Mar 9;118(10).
10. Wang MG, Luo L, Zhang Y, Liu X, Liu L, He JQ. Treatment outcomes of tuberculous meningitis in adults: a systematic review and meta-analysis.

BMC Pulm Med. 2019 Dec 6;19(1):200.

11. Seid G, Alemu A, Dagne B, Gamtesa DF. Microbiological diagnosis and mortality of tuberculosis meningitis: Systematic review and meta-analysis. PLoS One. 2023 Feb 16;18(2):e0279203.
12. Kagimu E, Bangdiwala AS, Kasibante J, Kabahubya M, Gakuru J, Timothy M, et al. Predictors of early mortality in HIV-associated tuberculous meningitis. Open Forum Infect Dis. 2023;10(Suppl 2):ofad500.1490. doi:10.1093/ofid/ofad500.1490.
13. Thao LTP, Heemskerk AD, Geskus RB, Mai NTH, Ha DTM, Chau TTH, et al. Prognostic Models for 9-Month Mortality in Tuberculous Meningitis. Clin Infect Dis. 2018 Feb 1;66(4):523–32.
14. Wang MS, Zhao M, Liu XJ. Risk factors for poor outcome in childhood tuberculous meningitis. Sci Rep. 2021 Apr 21;11(1):8654.
15. Van Laarhoven A, Dian S, Ruesen C, Hayati E, Damen MSMA, Annisa J, et al. Clinical Parameters, Routine Inflammatory Markers, and LTA4H Genotype as Predictors of Mortality Among 608 Patients With Tuberculous Meningitis in Indonesia. J Infect Dis. 2017 Apr 1;215(7):1029–39.
16. Koeken VACM, Ganiem AR, Dian S, Ruslami R, Chaidir L, Netea MG, et al. Cerebrospinal fluid IL-1β is elevated in tuberculous meningitis patients but not associated with mortality. Tuberculosis (Edinb). 2021;126:102019. doi:10.1016/j.tube.2020.102019.
17. Stadelman AM, Ellis J, Samuels THA, et al. Cerebrospinal fluid AFB smear in adults with tuberculous meningitis: a systematic review and diagnostic test accuracy meta-analysis. Lancet Microbe. 2022;3(8):e594–e603. doi:10.1016/S2666-5247(22)00067-1.
18. Luo Y, Yang Y, Zhang Y, Liu Z, Zhang Y, Wang J. Diagnostic accuracy of Xpert MTB/RIF for tuberculous meningitis: a systematic review and meta-analysis. J Clin Microbiol. 2020;58(11):e01119-20. doi:10.1128/JCM.01119-20.
19. Donovan J, Phu NH, Dung VTM, Quang TP, Nghia HDT, Oanh PKN, et al. Xpert MTB/RIF Ultra versus Xpert MTB/RIF for the diagnosis of tuberculous meningitis: a prospective, randomised, diagnostic accuracy study. Lancet Infect Dis. 2020;20(3):299–307.

20. Nuwagira E, Huppler Hullsiek K, Jjunju S, Rutakingirwa M, Kasibante J, Tadeo KK, et al. Diagnostic and Prognostic Value of Cerebrospinal Fluid Lactate and Glucose in HIV-Associated Tuberculosis Meningitis. Microbiol Spectr. 2022;10(4):e01071-22.
21. Manyelo CM, Solomons RS, Walzl G, Chegou NN. Tuberculous Meningitis: Pathogenesis, Immune Responses, Diagnostic Challenges, and the Potential of Biomarker-Based Approaches. J Clin Microbiol. 2021;59(3):e01771-20.
22. Le THN, van Abeelen KCJ, Ardiansyah E, Avila-Pacheco J, Dian S, Carstens G, et al. Pre-treatment untargeted cerebrospinal fluid metabolomic profiling in tuberculous meningitis uncovers pathways associated with mortality. Med (N Y). 2025;6(9):100703.
23. Lourens R, Singh G, Arendse T, Thwaites G, Rohlwink U. Tuberculous Meningitis Across the Lifespan. J Infect Dis. 2025 Jun 2;231(5):1101–11.
24. He H, Xu J, Peng Q, Li Y, Huang Y, Zhang YL, et al. The application value of cerebrospinal fluid immunoglobulin in tuberculous meningitis. Microbiol Spectr. 2024;12(6):e01234-24.
25. Rohlwink UK, Figaji AA, Wilkinson RJ. Tuberculous meningitis in children and adults: new insights into pathogenesis and host-directed therapies.
Lancet Neurol. 2023;22(3):267–80.
26. Imron A, Hermanto Y, Rizal A, Yunivita V, Ruslami R. Cerebrospinal fluid analysis in tuberculous meningitis: A literature review. Surg Neurol Int. 2025 Jun 20;16:246.
27. Dian S, Koeken VACM, Ardiansyah E, Ganiem AR, van Abeelen K, Aguirre-Gamboa R, et al. Inflammatory markers in the cerebrospinal fluid linked to mortality in tuberculous meningitis. Brain Commun. 2025;7(4):fcaf224.