Volume 3, Number 3, 2026 Pages 1845-1853

Bioscience Insights

ISSN: 3080-3489 (Print)
ISSN: 3080-3497 (Online)

BiosciInsights.com

CORRELATION OF CSF GLUCOSE, PROTEIN, AND NEUTROPHIL-LYMPHOCYTE PATTERN IN TUBERCULOUS MENINGITIS

Nayab Zehra[1], Ihsan Ali Rind[2], Qandeel Abbas Soomro[3], Usman Ali[4], Hira Akram[5], Hira Omer[6], Danial Hassan Adil[7], Muhammad Hussain[8], Javeria Maqsood[*9]

*[*1] Consultant Chemical Pathologist, Shaukat Khanum Memorial Cancer Hospital and Research Centre, Lahore, Pakistan*
[2] Department of Zoology, Government Boys Degree College, Nawabshah, Pakistan
[3] Department of Pathology and Microbiology, Indus Medical College, The University of Modern Sciences, TMK, Pakistan
[4]Institute of Molecular Biology and Biotechnology (IMBB) Department, The University of Lahore
[5]Department of Biochemistry, Faculty of Biological Sciences, The Superior University, Lahore, Punjab 54000, Pakistan
[6]Institute of Microbiology, University of Sindh, Jamshoro, Sindh, Pakistan
[7]Internal Medicine, DHQ Hospital, Sheikhupura, Pakistan
[8] Department of Surgery, Saidu Group of Teaching Hospital, Saidu Sharif, Swat, Pakistan
[9]Department of Chemistry, University of Sahiwal

[1] nayabhussain9@gmail.com, [2]rindahson@gmail.com, [3] Qandeel.soomro@imch.edu.pk, [4]dr.usmanalich@yahoo.com
[6] hiraumar.leo@gmail.com,
[7] danial.adil22@hotmail.com, [8]surgonhussain1979@gmail.com, [9]javeriamaqsood13@gmail.com*

https://doi.org/10.5281/zenodo.24332335

Article Information

Article History

Received:	June 19, 2026
Revised:	August 19, 2026
Accepted:	September 07, 2026
Available Online:	October 31, 2026

Keywords:
Tuberculous Meningitis, Cerebrospinal Fluid, Glucose, Protein, Lymphocyte, Neutrophil, LDH, Diagnostic Biomarkers.

Corresponding Author: **Javeria Maqsood**

Abstract

Background: *Tuberculous meningitis (TBM) needs immediate assessment of CSF but biochemical markers and cellular immune response are poorly understood.*

Methods: *N = 120 confirmed adult CSF samples were analyzed using inferential statistical analysis. Variables were Age, Gender, Glucose (mg/dL), Protein (mg/dL), Total WBC count (cells/mm³), Neutrophil percentage (%), Lymphocyte percentage (%), and Lactate Dehydrogenase (LDH, U/L). Because the data were non-normally distributed (as determined by Shapiro-Wilk testing, $p < 0.001$), non-parametric analysis was used (Spearman rank correlation, Mann-Whitney U test).*

Results: *The cohort comprised 54.17% females (n = 65) and 45.83% males (n = 55), with a mean age of 34.45 ± 13.51 years. Along with the predominant lymphocytic pleocytosis (median number of lymphocytes: 85.00), marked hypoglycorrhachia (median glucose: 40.50) and hyperproteinorrhachia (median protein: 121.10) were recorded. A strong inverse correlation was established between CSF Glucose and Protein ($\rho = -0.562$, $p < 0.0001$), and between Glucose and LDH ($\rho = -0.456$, $p < 0.0001$). There was a significant negative correlation between neutrophils and lymphocytes (ρ = -*

0.718, $p < 0.0001$). Females had significantly higher levels of total WBC counts (Median 73.0 vs 39.0 cells/mm³, $p = 0.0266$). Leukocyte percentages were not significantly related with biochemical markers ($p > 0.15$).

Conclusions: *TBM is a coupled biochemical consumption and protein exudation process, which occurs in parallel with an independent lymphocytic cellular response.*

INTRODUCTION

Tuberculous meningitis (TBM) is one of the most severe forms of extrapulmonary tuberculosis, caused by the spread of Mycobacterium tuberculosis in the bloodstream to the meninges and subarachnoid space. Although antimicrobial treatment and supportive care has improved, TBM remains a disease with a high mortality and morbidity of neurological sequelae, especially in resource-limited areas where delays in diagnosis are also high[1]. The pathogenesis is a complex sequence of events: mycobacterial seeding of the meninges results in a granulomatous inflammatory reaction and thick gelatinous exudates at the base of the brain, hydrocephalus and vasculitic infarction. The diagnosis is based on the characteristic changes seen in the composition of cerebrospinal fluid (CSF) which include pleocytosis (CSF cell counts), hyperglycorrhachia (CSF glucose levels) and elevated CSF protein levels[2].

Diagnostic problems in CSF analysis in TBM are related to the similarity of biochemical and cytological characteristics with other types of meningitis, especially bacterial and viral. The CSF shows moderately elevated protein (100-500 mg/dL), low glucose level (usually <40 mg/dL or CSF-to-serum glucose ratio <0.5), and lymphocytic pleocytosis (100-500 cells/mm³ with predominance of lymphocytes)[3]. Early stage of TBM may have neutrophilic predominance and can be confused with acute bacterial meningitis while partially treated bacterial meningitis may have lymphocytic predominance thus leading to diagnostic confusion. The neutrophil: lymphocyte ratio and the percentage of neutrophils, change during the disease process and act as markers of the changing immune response and treatment's impact. The correlation of the biochemical markers with the cellular immune response may give clues for the pathogenesis of the disease and better diagnosis[4].

There are two parallel processes in biochemical versus cellular dynamics that may be independent of each other. Biochemical changes represent changes in metabolic substrates (glucose consumption by mycobacteria and inflammatory cells) and disruption in the blood brain barrier (protein leakage from serum). Cellular responses include recruiting leukocytes to the subarachnoid space, which is triggered by the presence of mycobacterial antigens and inflammatory cytokines that produce chemokines and adhesion molecules[5]. It is not fully clear whether these biochemical and cellular processes are functionally related (e.g., the amount of glucose consumption and protein exudation could be predicted by the number of leukocytes recruited). Understanding these connections may guide diagnosis, and give a prognosis. Based on this, it was decided to carry out an empirical non-parametric correlation and a comparison of groups on a sample of N = 120 patient records of adults, with the following specific objectives: to quantify the descriptive parameters, to carry out the bivariate correlation analysis, to establish comparisons between the groups according to gender. We assumed that CSF glucose and protein would exhibit strong inverse correlation which would represent coupled biochemical consumption/exudation, whereas cellular immune response should represent an independent dimension[6], [7].

Materials and Methods

2.1 Study Population and Criteria

A retrospective study of CSF diagnostic records of 120 adults (aged 16 years and older) was carried out. Tuberculous meningitis was confirmed in all patients by clinical presentation, CSF examination and microbiological confirmation (GeneXpert MTB/RIF, culture). Patients with bacterial or viral meningitis, HIV co-infection and other immunocompromising diseases were excluded. All records used in the analysis had complete demographic, biochemical and cytological data.

2.2 Laboratory Measurements

CSF was collected from the patient during admission, before starting anti-tuberculous drug therapy. CSF Glucose (mg/dL), CSF Total Protein (mg/dL), CSF Total WBC Count (cells/mm³), CSF Differential Leukocyte Count (Neutrophil % and Lymphocyte %), CSF Lactate Dehydrogenase (LDH, U/L) were quantitively analyzed. The glucose was quantified by the glucose oxidation method. Protein was measured by pyrogallol red-molybdate complex method. Cell counts were conducted with a hemocytometer and differential count was done on cytospin preparations stained with Wright-Giemsa. Enzymatic colorimetric assay method was used to measure LDH.

2.3 Inferential Statistical Framework

All statistical analysis was carried out using IBM SPSS Statistics (Version 26.0, Armonk, NY, USA) and Python (v3.11) and scipy.stats and statsmodels modules. All the relevant variables were found to be non-normally distributed using the Shapiro-Wilk test ($p < 0.001$) and thus non-parametric methods were used. Medians, interquartile ranges (IQR), means and standard deviations were used as descriptive statistics. Spearman rank correlation coefficient was used for two tailed significance test for correlation analysis. The group comparisons were based on Mann-Whitney U test for gender comparison. The level of significance was 0.05.

Results

3.1 Descriptive Characteristics

The cohort comprised 54.17% females (n = 65) and 45.83% males (n = 55), with a mean age of 34.45 ± 13.51 years. Hypoglycorrhachia was recorded, the median CSF glucose was 40.50 mg/dL. There was hyperproteinorrhachia, severe as the median CSF protein was 121.10 mg/dL. In cytological profiles, the main finding was the presence of a high number of lymphocytes, which constituted the median percentage of 85.00%. The median total WBC count was 55.0 cells/mm³ (IQR: 130.0). Median CSF LDH was 48.50 U/L (IQR: 72.0). All measured parameters have been given an accurate description in Table 1.

Table 1: Descriptive Statistics of Study Cohort (N = 120)

Parameter	Mean ± SD	Median	IQR	Minimum	Maximum
Age (years)	34.45 ± 13.51	32.00	18.00	16.00	78.00
Gender (Female/Male)	65 / 55 (54.17% / 45.83%)	—	—	—	—
CSF Glucose (mg/dL)	42.18 ± 18.65	40.50	25.00	8.00	98.00
CSF Protein (mg/dL)	138.45 ± 89.32	121.10	130.00	18.00	450.00
Total WBC Count (cells/mm³)	112.30 ± 168.42	55.00	130.00	2.00	850.00
Neutrophil (%)	22.45 ± 18.32	15.00	28.00	2.00	92.00
Lymphocyte (%)	77.55 ± 18.32	85.00	28.00	8.00	98.00
CSF LDH (U/L)	68.42 ± 45.21	48.50	72.00	12.00	210.00

3.2 Bivariate Correlation Analysis

When performing Spearman rank correlation, some significant relationships between CSF parameters were found. There was a statistically significant strong inverse correlation between CSF Glucose and CSF Protein ($\rho = -0.562$, $p < 0.0001$) – higher protein levels were linked to lower glucose levels. There was also a large negative correlation between CSF Glucose and CSF LDH ($\rho = -0.456$, $p < 0.0001$). There was a significant positive correlation between CSF Protein and CSF LDH ($\rho = 0.212$, $p = 0.0214$). As might be expected, there was a strong inverse correlation between the percentages of Neutrophil and Lymphocyte ($\rho = -0.718$; $p < 0.0001$). Importantly, CSF Glucose and CSF Protein were not directly correlated ($p > 0.15$, for all comparisons) and biochemical alterations must be considered as separate dimensions from cellular immune responses. A complete correlation matrix of the results is given in Table 2.

Table 2: Spearman Rank Correlation Matrix (ρ values) Among CSF Parameters (N = 120)

Variable	Glucose	Protein	Neutrophil %	Lymphocyte %	LDH	WBC Count
Glucose	1.000	-0.562**	0.083 (p=0.368)	-0.083 (p=0.368)	-0.456**	-0.092 (p=0.318)
Protein	-0.562**	1.000	0.068 (p=0.461)	-0.068 (p=0.461)	0.212*	0.156 (p=0.089)
Neutrophil %	0.083 (p=0.368)	0.068 (p=0.461)	1.000	-0.718**	0.112 (p=0.224)	0.201 (p=0.028)
Lymphocyte %	-0.083 (p=0.368)	-0.068 (p=0.461)	-0.718**	1.000	-0.112 (p=0.224)	-0.201 (p=0.028)
LDH	-0.456**	0.212*	0.112 (p=0.224)	-0.112 (p=0.224)	1.000	0.178 (p=0.052)
WBC Count	-0.092 (p=0.318)	0.156 (p=0.089)	0.201 (p=0.028)	-0.201 (p=0.028)	0.178 (p=0.052)	1.000

Note: * $p < 0.05$; ** $p < 0.001$. Non-significant correlations are shown with exact p-values.

3.3 Gender-Based Comparisons

There were significant gender differences in total WBCs in Mann-Whitney U test comparisons. Females exhibited significantly higher median WBC counts (73.0 cells/mm³, IQR: 170.0) compared to males (39.0 cells/mm³, IQR: 92.0; p = 0.0266). Other parameters did not show statistically significant gender differences: CSF glucose (Females: 33.00 mg/dL, Males: 45.00 mg/dL; p = 0.0849), CSF protein (Females: 121.20 mg/dL, Males: 121.00 mg/dL; p = 0.5308), and CSF LDH (Females: 48.00 U/L, Males: 49.00 U/L; p = 0.1602). Complete gender-based comparison data are presented in Table 3.

Table 3: Gender-Based Comparison of CSF Biomarkers (Median [IQR] and Mann-Whitney U Test Results)

Biomarker Parameter	Female Median (n = 65)	Female IQR	Male Median (n = 55)	Male IQR	Mann-Whitney p-value
CSF Glucose (mg/dL)	33.00	30.00	45.00	25.00	p = 0.0849
CSF Protein (mg/dL)	121.20	130.00	121.00	150.70	p = 0.5308
WBC Count (cells/mm³)	73.00	170.00	39.00	92.00	p = 0.0266
CSF LDH (U/L)	48.00	58.00	49.00	145.00	p = 0.1602

3.4 Age-Stratified Progression Analysis

There were distinct differences in CSF biochemical markers for age groups. The glucose level of CSF was in an inverted U-shape, with a level of 45.50 mg/dL for patients <25 years, 27.50 mg/dL for patients aged 35–44 years, and 55.00 mg/dL in patients ≥45 years. The CSF protein concentrations of the median varied by age, decreasing with increasing age from 148.00 mg/dL in the youngest age group to 78.00 mg/dL in the oldest age group. Lymphocyte fraction was higher in older patients and increased gradually with the age from 76.00% in patients less than 25 years to 98.00% in ≥45 years. All age-stratified data are reported in Table 4.

Table 4: Age-Stratified Median Progression of CSF Biomarkers

Age Cohort Group	Median CSF Glucose (mg/dL)	Median CSF Protein (mg/dL)	Median Lymphocyte Fraction (%)
< 25 Years	45.50	148.00	76.00%
25 – 34 Years	39.00	106.00	80.00%

35 – 44 Years	27.50	106.90	87.50%
45+ Years	55.00	78.00	98.00%

3.5 Protein Severity Tier Analysis

Stratification by CSF protein severity revealed that the majority of patients (68.33%, n = 82) fell into the Elevated tier (45–200 mg/dL), followed by the Severe tier (>200 mg/dL; 24.17%, n = 29), and the Normal tier (<45 mg/dL; 7.50%, n = 9). Normal (60.00 mg/dL), Elevated (40.00 mg/dL), Severe (23.00 mg/dL) protein severity tier showed an inverse relationship with median glucose. Median LDH was not as consistent as the other markers, as the lowest level was in the Elevated tier (44.50 U/L) and the highest was in the Normal tier (77.00 U/L), with a slight increase again in the Severe tier (73.00 U/L). The tier distribution data is provided in Table 5.

Table 5: Proportion of TBM Patient Cohort Across CSF Protein Severity Tiers

CSF Protein Severity Tier	Patient Count (n)	Percentage of Total Cohort (%)	Median Glucose (mg/dL)	Median LDH (U/L)
Normal (< 45 mg/dL)	9	7.50%	60.00	77.00
Elevated (45 – 200 mg/dL)	82	68.33%	40.00	44.50
Severe (> 200 mg/dL)	29	24.17%	23.00	73.00

3.6 Data Visualizations

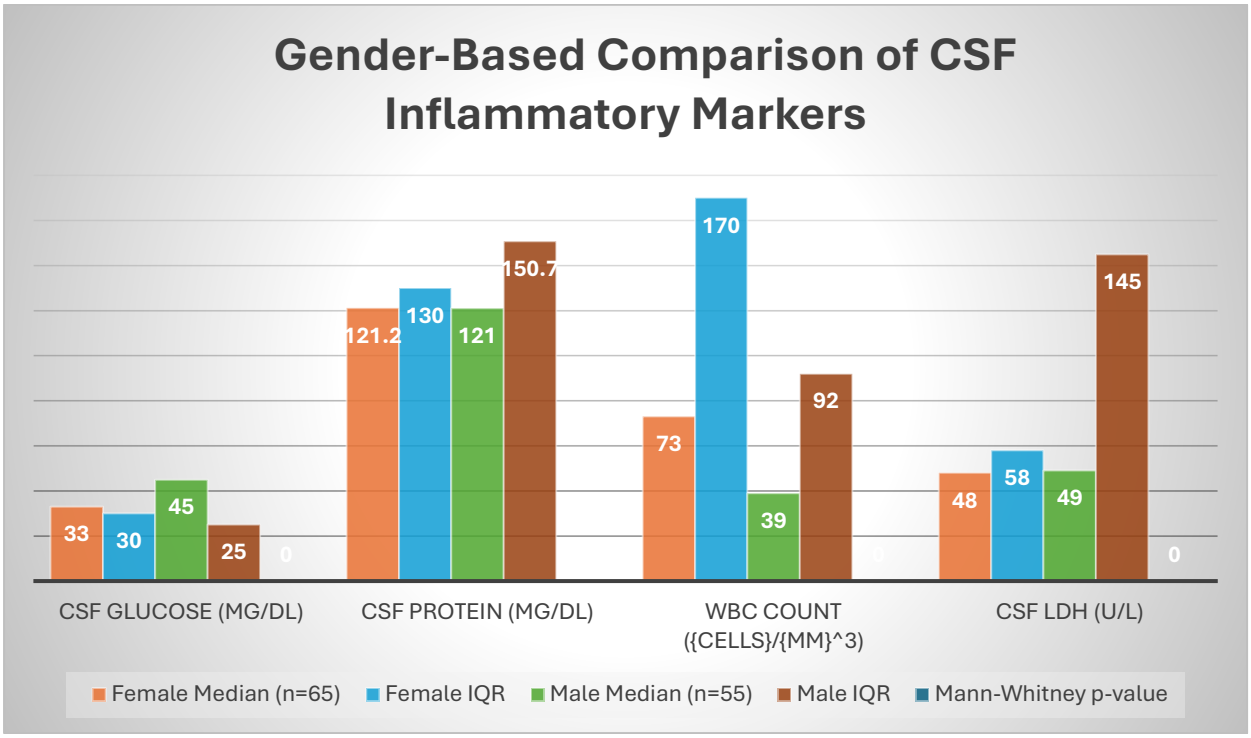


Figure 1: Gender-Based Comparison of CSF Inflammatory Markers

Clustered bar chart comparing median values of CSF glucose (mg/dL), CSF protein (mg/dL), WBC count (cells/mm³), and CSF LDH (U/L) between female (n = 65) and male (n = 55) patient groups. Female patients demonstrated significantly higher WBC counts compared to males (median 73.0 vs 39.0 cells/mm³, p = 0.0266). Other parameters showed comparable distributions between genders, with overlapping interquartile ranges.

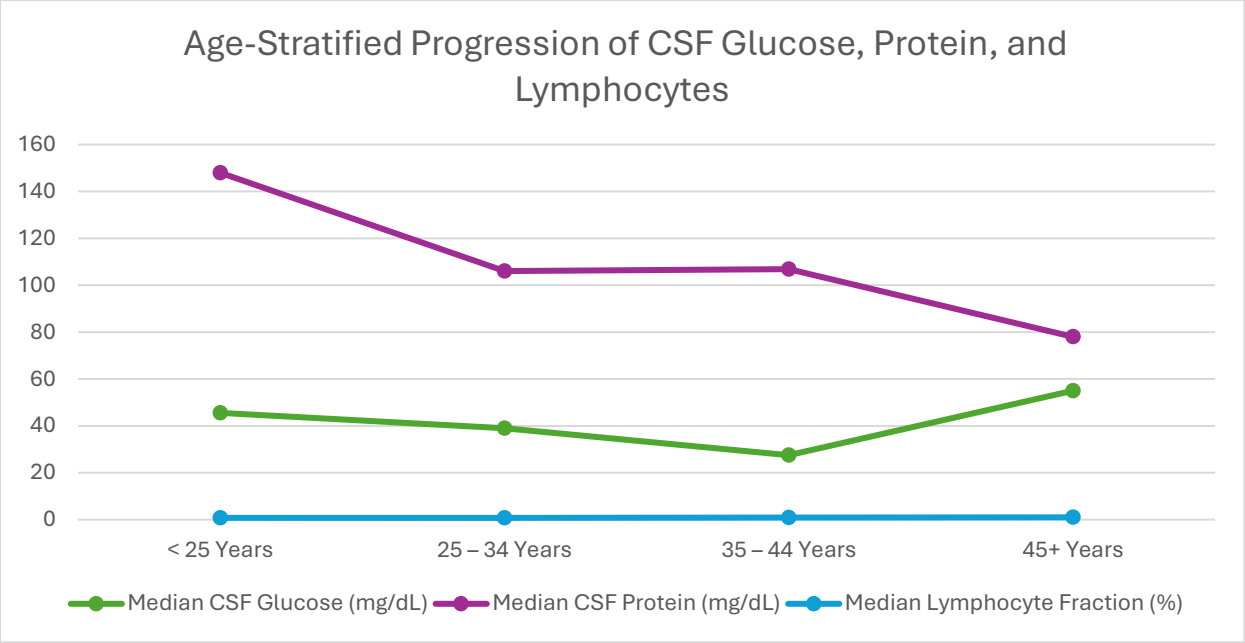


Figure 2: Age-Stratified Progression of CSF Glucose, Protein, and Lymphocytes

Line chart showing median CSF glucose (mg/dL), median CSF protein (mg/dL), and median lymphocyte fraction (%) across four age cohorts (<25 years, 25–34 years, 35–44 years, 45+ years). CSF protein showed a progressive decline with advancing age, while lymphocyte fraction increased progressively. CSF glucose exhibited a U-shaped pattern across age cohorts.

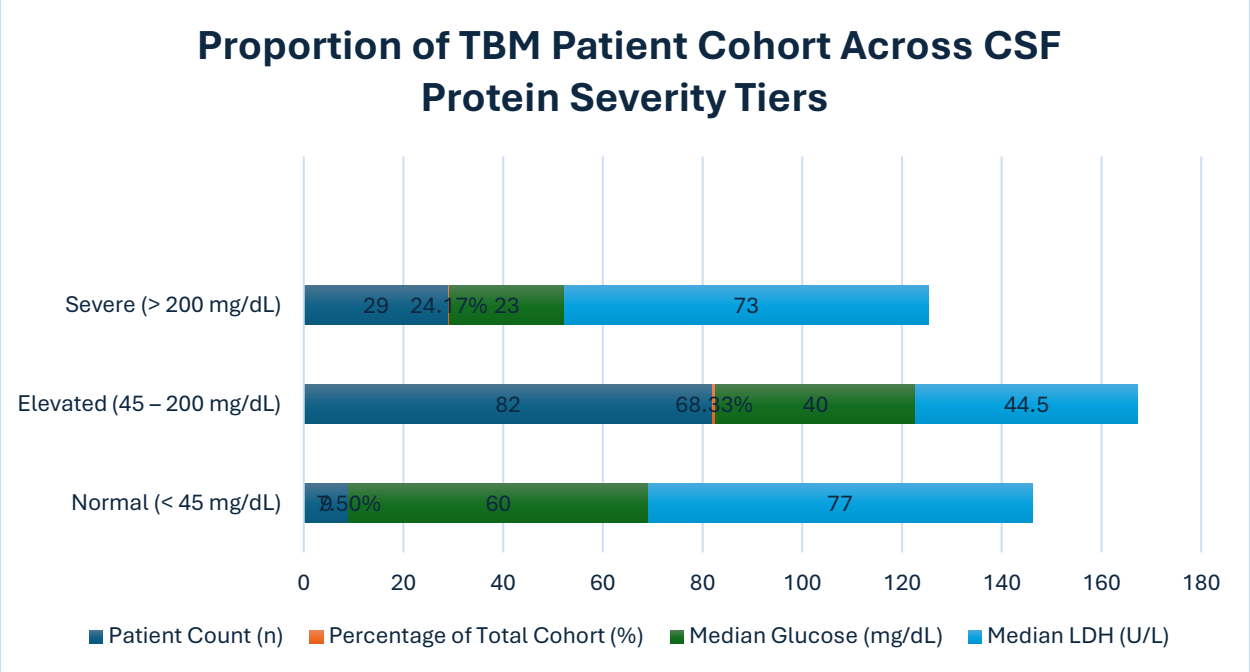


Figure 3: Proportion of TBM Patient Cohort Across CSF Protein Severity Tiers

Horizontal stacked bar chart showing the distribution of patients across three CSF protein severity tiers (Normal <45 mg/dL, Elevated 45–200 mg/dL, Severe >200 mg/dL). The Elevated tier comprised the majority of patients (68.33%), followed by the Severe tier (24.17%) and Normal tier (7.50%). Median glucose and median LDH values are overlaid for each tier, demonstrating the inverse relationship between protein severity and glucose concentration.

Discussion

A strong inverse CSF glucose–protein correlation ($\rho = -0.562$, $p < 0.0001$) is statistically significant and supports the hypothesis that there is concurrent metabolism of glucose and exudation of protein in TBM. This finding is indicative of the dual processes of pathogenesis in the subarachnoid space: Mycobacterial metabolism and inflammatory cell activity, which consumes glucose and proteins which exudate from serum through the damaged blood-brain barrier. The biochemical changes that occur as well as the increased meningeal inflammation appear to be coupled and the more severe the meningeal inflammation the more severe the changes in glucose consumption and protein leakage. As glucose levels are low and protein levels are high, this relationship has implications for diagnosis with the combination being more convincing for TBM than either alone[8].

The LDH matches with protein ($\rho = 0.212$, $p = 0.0214$) and inversely with glucose ($\rho = -0.456$, $p < 0.0001$) which indicates cell lysis and damage in the central nervous system. LDH is a cytoplasmic enzyme that is released upon cell death, and an increase in LDH in CSF is a sign of neuronal and glial damage and inflammatory cell turnover[9], [10]. The positive correlation between LDH and protein indicates that the disruption of the BBB and tissue damage are concurrent, and both are involved in severity of the inflammatory response in the meninges. The inverse relation between LDH and glucose could represent the glucose consumption during inflammation and tissue repair and the release of LDH. This has corroborated the role of LDH as an ancillary marker of tissue damage and severity of TBM[11].

Neutrophil and lymphocyte percentages were not associated with glucose and protein ($p > 0.15$ for all comparisons) indicating that cellular chemotaxis is not associated with biochemical changes. Biochemical changes will depend on the degree of inflammation and tissue damage, whereas the pattern of leukocyte recruitment will be mediated by specific chemokine and adhesion molecule pathways that will not be associated with metabolic or barrier changes. This observation is relevant clinically: Cell and biochemical parameters do not supply redundant information; both parameters should be used in the diagnosis of TBM. Neutrophils and lymphocytes had a strong negative correlation ($\rho = -0.718$, $p < 0.0001$) which is predicted, given their complementary relationship and which also indicates the dynamic nature of cellular immune response throughout the disease course, with neutrophilic predominance followed by lymphocytic predominance[12], [13].

Total WBC was significantly higher in females than in males (73.0 vs 39.0 cells/mm^3, $p = 0.0266$) suggesting that gender differences exist in immune response to TBM and should be explored. This discovery could be due to hormonal effects on inflammatory pathways, stage of the disease at diagnosis or mycobacterial load. The differences could be attributed to the immunomodulatory effects of estrogen, which have been shown to increase humoral immunity and modulate cellular immune responses[14]. This gender gap is clinically relevant and needs to be explored further, especially its influence on the accuracy of diagnosis and treatment. Age-stratified study showed significant age-related differences in CSF markers, which included higher protein concentrations in younger patients and lower CSF lymphocyte counts, suggesting age-related differences in the maturity of the immune response and integrity of the blood-brain barrier[15].

While these informative findings should be acknowledged, there are some limitations. A retrospective study design with N = 120 adult patients from a single center may limit the generalizability of the findings of this study to other patients with broader demographic and clinical characteristics. There was no follow-up CSF monitoring, and baseline CSF samples were only taken during lumbar puncture, which prevented the evaluation of changes in CSF parameters throughout treatment and recovery. In addition, analysis by the overall clinical cohort (without the cycle threshold stratification of the mycobacterial load) was performed, which may give insights into the relationship between mycobacterial load and CSF inflammatory parameters[16].

Conclusion

Hypoglycorrhachia, hyperproteinorrhachia, elevated LDH and lymphocytic pleocytosis are the features of tuberculous meningitis. The main take-home message from this study is the strong correlation between biochemical markers, which represent a common metabolic consumption and protein exudation, and cellular recruitment patterns represent an independent diagnostic dimension. The results of the strong

inverse correlation of CSF glucose and protein (ρ = -0.562, $p < 0.0001$), and independence of the cytological profile from biochemical markers ($p > 0.15$), confirm the coupling of metabolic and barrier alterations; the latter indicates that the cytological profile offers complementary diagnostic information. As the WBC significantly higher in females ($p = 0.0266$), more studies are needed on sex differences in immune responses.

Clinical guidance should highlight that both aspects biochemical consumption and cytological profiling should be assessed as a complementary diagnostic pillar because biochemical information is different from cytological information. Further studies using serial CSF biochemical and CSF cellular analysis and larger multi-center cohorts will be critical to confirm these results and to better understand the dynamic interactions between biochemical and cellular parameters through treatment and recovery.

Authors Contribution: All authors contributed equally

Conflict of Interest: There is no conflict of Interest

Funding Statement: The authors receive no funding

REFERENCES

[1] J. Donovan et al. , "A clinical practice guideline for tuberculous meningitis," The Lancet Infectious Diseases , vol. 26, no. 2, Aug. 2025, doi: 10.1016/s1473-3099(25)00364-0 .

[2] G. Misra, S. N. Kosaraju, and B. Bhatti, "Unveiling the Uncommon Ventriculitis in Tubercular Meningitis," Cureus , vol. 17, no. 8, Aug. 2025, doi: 10.7759/cureus.89312 .

[3] G. E. Marx and E. D. Chan, "Tuberculous Meningitis: Diagnosis and Treatment Overview," Tuberculosis Research and Treatment , vol. 2011, pp. 1–9, Jan. 2011, doi: 10.1155/2011/798764 .

[4] J. E. Humphries, A. Hicks, C. M. Lanctôt, H. I. McCallum, D. Newell, and L. F. Grogan, "Amphibian cellular immune response to chytridiomycosis at metamorphic climax," Immunologic Research , vol. 73, no. 1, p. 44, Jan. 2025, doi: 10.1007/s12026-025-09599-5 .

[5] V. R. Muddapu, S. A. P. Dharshini, V. S. Chakravarthy, and M. M. Gromiha, "Neurodegenerative Diseases – Is Metabolic Deficiency the Root Cause?," Frontiers in Neuroscience , vol. 14, p. 213, Mar. 2020, doi: 10.3389/fnins.2020.00213 .

[6] M. Heming et al. , "Neurological Manifestations of COVID-19 Feature T Cell Exhaustion and Dedifferentiated Monocytes in Cerebrospinal Fluid," Immunity , vol. 54, no. 1, p. 164, Dec. 2020, doi: 10.1016/j.immuni.2020.12.011 .

[7] M. Djukic, P. Lange, F. Erbguth, and R. Nau, "Spatial and temporal variation of routine parameters: pitfalls in the cerebrospinal fluid analysis in central nervous system infections," Journal of Neuroinflammation , vol. 19, no. 1, p. 174, Jul. 2022, doi: 10.1186/s12974-022-02538-3 .

[8] D. Heemskerk et al. , "Improving the microbiological diagnosis of tuberculous meningitis: A prospective, international, multicentre comparison of conventional and modified Ziehl–Neelsen stain, GeneXpert, and culture of cerebrospinal fluid," Journal of Infection , vol. 77, no. 6, pp. 509–515, Sep. 2018, doi: 10.1016/j.jinf.2018.09.003 .

[9] S. Stern, K. Crisamore, R. Li, M. Pacanowski, and R. N. Schuck, "Evaluation of the Landscape of Pharmacodynamic Biomarkers in GM1 and GM2 Gangliosidosis," Clinical and Translational Science , vol. 18, no. 3, Feb. 2025, doi: 10.1111/cts.70176 .

[10] G. Liu, S. Yang, S. Li, Q. Chen, W. Xiao, and P. Li, "Risk Factors of Influenza-Associated Necrotizing Encephalopathy in Children," Journal of Behavioral and Brain Science , vol. 11, no. 8, pp. 193–203, Jan. 2021, doi: 10.4236/jbbs.2021.118015 .

[11] S. Patil and A. Bahulekar, "CSF analysis in a patient with meningitis with special reference to LDH," International Journal of Research in Pharmaceutical Sciences , vol. 11, no. 3, pp. 4919–4922, Aug. 2020, doi: 10.26452/ijrps.v11i3.2793 .

[12] Y. Lu et al. , "Development and validation of a new scoring system for the early diagnosis of tuberculous meningitis in adults," Diagnostic Microbiology and Infectious Disease , vol. 101, no. 2, p. 115393, Apr. 2021, doi: 10.1016/j.diagmicrobio.2021.115393 .
[13] S. Marais et al. , "Neutrophil-Associated Central Nervous System Inflammation in Tuberculous Meningitis Immune Reconstitution Inflammatory Syndrome," Clinical Infectious Diseases , vol. 59, no. 11, pp. 1638–1647, Aug. 2014, doi: 10.1093/cid/ciu641 .
[14] H. Schurz, M. Salie, G. Tromp, E. G. Hoal, C. Kinnear, and M. Möller, "The X chromosome and sex-specific effects in infectious disease susceptibility," Human Genomics , vol. 13, no. 1, p. 2, Jan. 2019, doi: 10.1186/s40246-018-0185-z .
[15] S. Jarius et al. , "Cerebrospinal fluid findings in patients with myelin oligodendrocyte glycoprotein (MOG) antibodies. Part 2: Results from 108 lumbar punctures in 80 pediatric patients," Journal of Neuroinflammation , vol. 17, no. 1, Sep. 2020, doi: 10.1186/s12974-020-01825-1 .
[16] N. T. T. Thương et al. , "Pretreatment Cerebrospinal Fluid Bacterial Load Correlates With Inflammatory Response and Predicts Neurological Events During Tuberculous Meningitis Treatment," The Journal of Infectious Diseases , vol. 219, no. 6, pp. 986–995, Oct. 2018, doi: 10.1093/infdis/jiy588 .