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Post Traumatic Meningitis Outcomes and Incidences on Mental Health

Papa Midley[1], Kaitlyn Blake[1], Nuha Mohammed[1], Hanan Yusef[1], Banuja Munasinghe[1], Mekdem Bisrat[2], Elizabeth Beyene[2], Syed Fahad Gillani[2], Rawan Elkomi[2], Mrinalini Deverapalli[2], Kalkidan S Alayu[2] and Miriam Michael[2,3]*

[1]Howard University, College of Medicine, Washington DC, USA

[2]Howard University, Department of Internal Medicine, Washington, DC, USA

[3]Department of Internal Medicine, University of Maryland School of Medicine, Baltimore, US

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Author : Dr. Mekdem Bisrat, MD, MPH

Abstract

Background: Traumatic brain injury (TBI) causes substantial morbidity and long-term neuropsychiatric sequelae. Bacterial meningitis may worsen outcomes after TBI, yet its mental-health impact is under characterized. This study examined 90-day neuropsychiatric outcomes following post-traumatic meningitis.

Methods: We performed a retrospective cohort study using the TriNetX Global Collaborative Network. Two propensity-matched cohorts were assembled: severe TBI with subsequent bacterial meningitis and severe TBI without meningitis (2,977 patients each). Outcomes included post-concussional syndrome, amnesia, altered mental status, depressive episodes, anxiety disorders, and non-psychotic mental disorders. We estimated risks, risk ratios, and odds ratios, and generated Kaplan–Meier survival curves and instance-based frequency analyses.

Results: Compared with TBI without meningitis, post-traumatic meningitis was associated with higher risk of amnesia (RR 2.65), altered mental status (RR 5.32), depressive episodes (RR 2.12), and anxiety (RR 2.05). Post-concussional syndrome risk was lower (RR 0.41). No difference was observed for non-psychotic mental disorders. Kaplan–Meier analyses showed significantly lower symptom-free probabilities for most outcomes.

Conclusion: Post-traumatic bacterial meningitis substantially increases early neuropsychiatric complications after severe TBI, highlighting the need for proactive psychiatric evaluation, neurocognitive monitoring, and tailored rehabilitation in this high-risk population. Findings support early infection prevention and integrated neurocritical care pathways implementation.

Keywords: Traumatic Brain Injury; Meningitis; Mental Health Outcomes; Post-Concussional Syndrome; Encephalopathy

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Correspondence:
Dr. Mekdem Bisrat, MD, MPH., Howard University, Department of Internal Medicine, Washington, DC, USA. Tel: 240-425-2256;
E-mail: mekdembisrat21@gmail.com

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Introduction

Traumatic brain injuries (TBIs) remain a leading cause of neurosurgical intervention and neurotrauma-related hospital admissions worldwide. Patients who suffer TBIs are at increased risk for a variety of intracranial complications, including infectious sequelae such as meningitis. When meningitis arises in the context of head trauma, it is often referred to as post-traumatic meningitis (PTM). The reported incidence of hospital-acquired meningitis following TBI is approximately 1.4% [1], though this may vary based on clinical setting and population characteristics. While prior studies have examined factors such as the causative pathogens and demographic trends among PTM patients [2], the current investigation seeks to expand upon this foundation by characterizing the incidence and outcomes of PTM across diverse age groups, racial and sex demographics, and microbiological etiologies. Additionally, we aim to evaluate the long-term neurological and psychiatric sequelae associated with PTM, which remain underexplored in large-scale clinical datasets.

While post-traumatic meningitis (PTM) can occur through various mechanisms, this study focuses specifically on cases in which meningitis developed as a direct consequence of traumatic head injury. The traumatic disruption of the cranial vault or meningeal integrity may create an environment conducive to microbial inoculation and subsequent infection. Prior studies have

noted elevated rates of PTM following neurosurgical interventions commonly associated with severe head trauma, including burr hole drainage, decompressive craniectomy, and the placement of external ventricular drains [3]. We also know that these PTM cases care about the increased risk of poor neurological outcomes [4]. Building on this foundation, the present study further explores the role of demographic factors—specifically sex, gender, race, and age—in influencing the incidence and outcomes of PTM. By integrating both clinical and demographic variables, this research aims to provide a more comprehensive understanding of risk profiles associated with post-traumatic meningitis.

To provide a more comprehensive understanding of post-traumatic meningitis (PTM), this study also considers broader risk factors associated with the development of meningitis, including both community-acquired and trauma-related cases. In this context, the mechanism and severity of the initial traumatic brain injury (TBI) have been shown to influence susceptibility to subsequent infection [5]. While the primary focus of this study is PTM, it is essential to account for other established risk factors for meningitis, such as underlying immunocompromising conditions including diabetes and HIV/AIDS [6]. By identifying and comparing these clinical and demographic characteristics, we aim to enhance current knowledge surrounding PTM and ultimately improve the timeliness and effectiveness of its recognition and treatment.

Methods

This retrospective cohort study was conducted using the TriNetX Global Collaborative Network, an extensive clinical database. The study aimed to compare the incidence of mental health complications among patients who sustained severe traumatic brain injury (TBI), stratified by the presence or absence of subsequent bacterial meningitis. Patients were identified using ICD-10 codes corresponding to intracranial injury and bacterial meningitis. To ensure temporal relevance, meningitis diagnoses were required to occur within 2 to 90 days following the initial TBI event. To control for confounding variables, 1:1 propensity score matching was performed using key demographic variables including age, sex, race, and ethnicity. The final matched cohorts included 2,977 patients each.

Outcomes of interest included post-concussional syndrome, amnesia, altered mental status, depressive episodes, anxiety disorders, and non-psychotic mental disorders. Patients with documented diagnoses of the outcome prior to the index event were excluded from risk and survival analyses, unless otherwise specified.

Statistical analyses included three main approaches. Risk analysis calculated absolute risk, risk differences, risk ratios, and odds ratios, with significance determined using z-tests and 95% confidence intervals. Kaplan-Meier survival analysis was employed to estimate time-to-event data, with the log-rank test used to assess statistical differences between survival curves. Hazard ratios were derived with proportional hazard testing to confirm model assumptions. Finally, a number-of-instances analysis evaluated the frequency of outcome diagnoses per patient within the follow-up period, using t-tests to compare means across groups. All analyses were performed using the TriNetX analytics engine, which complies with HIPAA and international data privacy regulations.

This study, a secondary analysis of anonymized data, utilized only de-identified patient information in accordance with the HIPAA Privacy Rule. As it involved no direct patient interaction or intervention, this research was exempt from institutional review board approval and informed consent requirements.

Results

After propensity matching for factors of age, sex, and race, both cohorts each had a total of 2,977 subjects. The median days until follow-up was 90 days for both groups. The risk for severe TBI with meningitis (1.0%) was lower than the risk for severe TBI without meningitis (2.4%), with a significant risk difference indicated by a p-value of 0.000. The risk ratio was 0.406 and the odds ratio was 0.400, indicating a lower risk for post-concussional syndrome in the meningitis group. After Kaplan-Meier survival analysis, the survival probability at the end of the time window for the meningitis group was 98.91% and for the non-meningitis group was 97.02%. With a hazard ratio of 0.353 and p-value of 0.247, suggesting no significant difference in how post-concussional syndrome was developed. However, there was a significant difference (p = 0.018) between the number of instances of post-concussional syndrome in each group (Table 1).

For the outcome of amnesia, the risk for the meningitis group was 3.1% and 1.2% for the non-meningitis group. With a risk ratio of 2.6, an odds ratio of 2.65, and a p-value of 0.000, there was a significant difference in risk for amnesia of those in the meningitis group versus those in the non-meningitis group. With a hazard ratio of 2.303 and a p-value of 0.267, there was no demonstrated significance in how amnesia was developed. The number of instances as compared between the meningitis group (91%) versus the non-meningitis group (35%) was not significant with a p-value of 0.057.

The risk for an altered mental state in the meningitis group was 14.6% and in the non-meningitis group it was 2.8%. The risk ratio was 5.317 and the odds ratio was 6.058. These measurements were significant (p = 0.000), indicating a greater risk of altered mental state in the meningitis group. According to the log-rank test, the time it took for an altered mental status to occur in the meningitis group versus the non-meningitis group was statistically significant (p = 0.000). However, the hazard ratio (5.118) was not statistically significant (p = 0.535) indicating no difference in how altered mental status developed over time. The t-test for the difference in the number of instances between the two groups was also not statistically significant (p = 0.638).

As for depressive episodes, the risk in the meningitis group was 11.1% and the risk in the non-meningitis group was 5.2%. The risk difference was statistically significant with a p-value of 0.000. The risk ratio of 2.115 and odds ratio of 2.254 indicate that there is a higher risk for depressive episodes in the meningitis group as compared to

Table 1: Risks (%) for each outcome in both cohorts, with corresponding risk ratio (RR), odds ratio (OR), and p-value; arrows indicate direction of effect.

Neuropsychiatric outcomes after severe traumatic brain injury with *vs* without post-traumatic bacterial meningitis					
Outcome	**Risk in TBI + Meningitis**	**Risk in TBI Only**	**Risk Ratio**	**Odds Ratio**	***p*-value**
Post-concussional syndrome	1.0%	2.4%	0.41 ↓	0.40 ↓	0.000
Amnesia	3.1%	1.2%	2.60 ↑	2.65 ↑	0.000
Altered mental status	14.6%	2.8%	5.32 ↑	6.06 ↑	0.000
Depressive episodes	11.1%	5.2%	2.12 ↑	2.25 ↑	0.000
Anxiety	10.4%	5.1%	2.05 ↑	2.17 ↑	0.000
Non-psychotic mental disorders	0.3%	0.3%	1.00 —	1.00 —	1.000

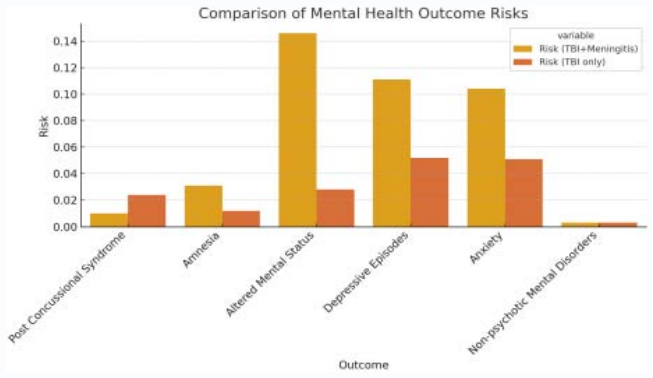

Figure 1: Comparison of mental disorder risk (%) outcomes between traumatic brain injury with and without meningitis.

the non-meningitis group. The log-rank test had a p-value of 0.000 which is statistically significant. However, the hazard ratio was 1.939 which was not statistically significant (p = 0.923). The t-test for the number of instances of depressive episodes in the meningitis group versus the non-meningitis group was not statistically significant (p = 0.203).

For anxiety, the risk ratio for the meningitis group was 10.4% and for the non-meningitis group it was 5.1%. The risk difference was statistically significant with a p-value of 0.000. The risk ratio was 2.046 and the odds ratio was 2.168. The log-rank test showed statistical significance with a p-value of 0.000, but the hazard ratio, which was 1.872, was not statistically significant (p = 0.330). The t-test for the number of instances of anxiety in both groups showed there was a statistically significant difference between the two groups (p = 0.030) For the last outcome of non-psychotic mental disorders, there was no statistical significance in any test between the meningitis and non-meningitis groups. The risk ratio and odds ratio were both 1, with a p-value of 1. The log-rank test had a p-value of 0.615. There was a hazard ratio of 0.715 with a p-value of 0.526. The t-test for the number of instances had a p-value of 0.497 (Figure 1).

Discussion

Our findings reveal that post-TBI meningitis significantly elevates the risk for several neuropsychiatric outcomes within a 90-day follow-up period. Patients who developed meningitis after TBI were over six times more likely to experience **altered mental status** compared to those who did not (Odds Ratio [OR] 6.06, p < 0.001). This suggests a synergistic effect where direct brain trauma [7] and the neuroinflammatory consequences of bacterial meningitis exacerbate brain injury. Meningitis likely contributes to secondary injury through mechanisms such as blood-brain barrier disruption, cytokine storm, cerebral edema, and the accumulation of neurotoxic metabolites, all of which can impair cognition, consciousness, and behavior [8].

Furthermore, **depressive episodes** and **anxiety disorders** were significantly more prevalent in patients with post-TBI meningitis (ORs 2.25 and 2.17, respectively). These results align with existing literature indicating that inflammation and central nervous system infections increase vulnerability to mood disorders [9], potentially via microglial activation, hippocampal atrophy, or dysregulation of neurotransmitter systems like serotonin and glutamate. The early onset of these conditions within the 90-day window highlights a potential for underdiagnosed acute neuropsychiatric needs in this patient population.

Amnesia was also more frequently observed in the meningitis cohort (OR 2.65), potentially reflecting compounded damage to limbic structures such as the hippocampus [10], exacerbated by post-infectious encephalopathy. Interestingly, **post-concussional syndrome** was *less* common in the meningitis group (OR 0.40, p < 0.001) [11]. This paradoxical finding might be attributed to differences in clinical surveillance, coding practices, or diagnostic overshadowing due to more prominent acute symptoms like delirium or altered mental status [12]. Conversely, no statistically significant difference was found for **non-psychotic mental disorders**, likely due to the rarity or broad heterogeneity of this diagnostic category.

From a clinical perspective, these findings emphasize the critical need for **proactive neuropsychiatric screening and support** for patients who develop bacterial meningitis after TBI [13]. Early psychiatric consultation, neurocognitive assessment, and tailored rehabilitation programs have the potential to mitigate long-term disability and enhance quality of life. Moreover, infection prevention strategies, including early antibiotic therapy, sterile surgical technique, and vaccination, could serve as indirect interventions to reduce the neuropsychiatric burden in TBI survivors [14].

Limitations

Several limitations warrant consideration. First, the study is observational and retrospective, precluding causal inference. Second, despite propensity score matching, unmeasured confounders (e.g., severity of injury, ICU care variability) may still influence outcomes. As mentioned previously, during the creation of our cohort groups we added TBI's without loss of consciousness to our exclusionary variables. This was done to target only severe cases of TBI. Third, diagnosis codes may under capture or misclassify certain mental health conditions, especially in under-resourced or overwhelmed clinical settings. We mentioned this in our discussion section and made reference to how symptoms of post-concussional syndrome may be overshadowed by broader mental disorders such as delirium or altered mental status. Additionally, follow-up was limited to 90 days, which may underestimate long-term neuropsychiatric sequelae.

Conclusions

Post-traumatic bacterial meningitis significantly amplifies the risk of neuropsychiatric complications in TBI patients, particularly altered mental status, amnesia, and mood disorders. These findings highlight the dual burden of trauma and infection on the brain and call for integrated neurocritical care pathways that incorporate infection control, mental health surveillance, and early rehabilitation. Further prospective studies are needed to elucidate the mechanistic pathways and to explore targeted interventions for this high-risk population.

Patients who develop bacterial meningitis after a traumatic brain injury (TBI) face a significantly higher risk of cognitive and emotional complications within the first 90 days, including altered mental status, depression, anxiety, and memory issues. This may be due to an intensified neuroinflammatory response triggered by the combined effects of the initial trauma and the infection. Interestingly, we observed a lower rate of post-concussional syndrome in this group, which could reflect either overlapping symptoms being overlooked or distinct underlying mechanisms. These findings highlight the need

for early psychiatric support, careful neurocognitive assessment, and a multidisciplinary rehabilitation approach for TBI patients with secondary central nervous system infections. Further research is needed to better understand the long-term outcomes and biological pathways involved.

Declaration of Interest Statements

Competing Interests: The authors declare no competing financial interests or personal relationships that could have influenced the work reported in this manuscript.

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