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Prevalence of Coagulopathy and Its Impact on Outcomes in Isolated Traumatic Brain Injury Patients: A Cross-Sectional Study from a Tertiary Care Centre in Kerala

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ABSTRACT

Background: Traumatic brain injury (TBI) remains a leading cause of mortality and long-term disability worldwide. Among the many complications of TBI, coagulopathy has emerged as a significant prognostic factor, potentially worsening outcomes due to secondary brain injuries and increased risk of haemorrhage. The burden of coagulopathy in isolated TBI without multisystem involvement has not been fully delineated in the Indian context. **Objectives**: This study aimed to determine the proportion of coagulopathy among patients with isolated TBI admitted to a tertiary care centre and to assess its association with in-hospital mortality and clinical outcomes. **Materials and Methods**: A cross-sectional study was conducted over one year at Government Medical College, Thiruvananthapuram. A total of 144 patients with isolated TBI were included. Coagulopathy was defined as any of the following: INR (International normalized ratio) >1.25, PT (Prothrombin time) >14 seconds, APTT (Activated Partial Thromboplastin Time test) >36 seconds, or platelet count <1 lakh/µL. Clinical variables such as Glasgow Coma Scale (GCS), pH, haemoglobin, and pupil response were also analysed. **Results**: Coagulopathy was observed in 39.6% of patients. Among these, 24.6% succumbed to the injury, whereas none of the patients without coagulopathy died ($p<0.01$). A significant association was found between coagulopathy and low GCS scores, anaemia, and metabolic acidosis ($p<0.01$). Road traffic accidents were the most common cause of injury. **Conclusion**: Coagulopathy is a prevalent complication in isolated TBI and is significantly associated with increased in-hospital mortality. Early detection and management of coagulation abnormalities may improve clinical outcomes and should be integrated into routine TBI care protocols.

Keywords: Traumatic brain injury, coagulopathy, INR, PT, APTT, mortality.

INTRODUCTION:

Traumatic brain injuries (TBIs) are a leading cause of morbidity, mortality, disability and socioeconomic losses in India and other developing countries. It is a major public health concern globally and is recognized as a leading cause of morbidity and mortality, particularly among young adults. It is estimated that approximately 69 million individuals sustain a TBI each year worldwide, with road traffic accidents, falls, and interpersonal violence being the most common mechanisms of injury (1). In India, rapid urbanization, increased vehicular traffic, poor enforcement of safety measures, and underdeveloped trauma care systems have contributed to a high burden of TBI, with road traffic accidents alone accounting for a significant proportion of emergency department admissions (2). TBI leads to both primary injury, which results from the initial mechanical impact, and secondary injury, which involves a cascade of pathophysiological events such as inflammation, excitotoxicity, cerebral edema, ischemia, and coagulopathy (3). Among these, coagulopathy has emerged as a particularly important yet often under-recognized factor influencing outcomes in TBI patients. Coagulopathy in TBI refers to a disruption in normal blood clotting processes that may manifest as either a bleeding tendency or thrombotic complications, and it is increasingly being identified as a determinant of increased morbidity and mortality (4). The incidence of TBI-associated coagulopathy has been variably reported in the literature, ranging from 10% to 87.5%,

depending on the definition used, timing of testing, and characteristics of the study population (5,6). The lack of a uniform diagnostic criterion has resulted in inconsistent reporting, though common parameters used include elevated PT, APTT, INR, and low platelet count (7).

The pathophysiological mechanisms underlying TBI-induced coagulopathy are multifaceted. Direct damage to brain parenchyma and blood vessels leads to the release of tissue factor (TF), which activates the extrinsic coagulation pathway, triggering thrombin generation and fibrin formation (8). Simultaneously, there is activation of the protein C pathway, endothelial dysfunction, and systemic inflammatory responses that contribute to dysregulated coagulation and fibrinolysis (9,10). Severe TBI also results in a systemic catecholamine surge, endothelial activation, and the release of microparticles, which further amplify coagulopathy (11). The resulting imbalance may manifest as both hypercoagulability in the early stages and consumptive coagulopathy or disseminated intravascular coagulation (DIC) in later phases, depending on the individual's physiological response. Coagulopathy in TBI is associated with an increased risk of progression of hemorrhagic lesions, increased intracranial pressure, delayed surgical interventions, longer hospital stays, and higher mortality (12,13).

Coagulopathy in isolated TBI, defined as TBI without associated injuries to other organ systems, presents a unique challenge. In such cases, the bleeding diathesis is believed to arise directly from the brain injury itself, and studying these patients helps isolate the impact of brain trauma on coagulation pathways (14). Understanding the prevalence and consequences of coagulopathy in isolated TBI is essential to developing early diagnostic strategies and evidence-based treatment protocols. Routine coagulation screening is not universally practiced in TBI management, especially in resource-limited settings, due to cost constraints and lack of awareness.

TBI-associated coagulopathy is a complex and clinically significant phenomenon that warrants systematic investigation. While its role in polytrauma has been acknowledged, its impact in isolated TBI remains underexplored. This study was therefore conducted with two key objectives: first, to estimate the proportion of coagulopathy among patients with isolated TBI admitted to a tertiary care hospital, and second, to evaluate the association between coagulopathy and in-hospital outcomes, including mortality. By focusing on routinely available coagulation parameters and clearly defined clinical criteria, this study aims to bridge existing knowledge gaps and contribute to the development of standardized guidelines for early recognition and management of coagulopathy in isolated TBI patients.

MATERIALS & METHODS:

Study Design and Setting

This was a cross-sectional observational study conducted in the Department of General Surgery, Government Medical College, Thiruvananthapuram, over a period of one year. The study was initiated after obtaining clearance from the Human Ethics Committee Trivandrum Medical College (HEC NO:04/21/2023/MCT), adhering to all ethical principles and guidelines for human research.

Study Population and Sampling

The study included adult patients aged 18 years and above with radiologically confirmed isolated traumatic brain injury (TBI) who were admitted within 24 hours of trauma. "Isolated TBI" was defined as brain injury without evidence of other systemic injuries. Patients with traumatic injuries to other organs, known pre-existing coagulopathies, use of anticoagulant drugs, chronic liver disease, diabetes mellitus, systemic hypertension, or sepsis were excluded to reduce confounding variables. A total of 144 participants were enrolled using a consecutive sampling method until the desired sample size was reached. The sample size was calculated based on a prior reported prevalence of coagulopathy in isolated TBI (46%) with a 95% confidence level and 20% relative precision.

Data Collection Tools and Definitions

A structured and pre-tested proforma was used to collect data on demographic characteristics, mechanism and type of injury, Glasgow Coma Scale (GCS) score, pupil reactivity, and CT imaging findings. Laboratory investigations including complete blood count, coagulation profile (prothrombin time [PT], activated partial thromboplastin time [APTT], international normalized ratio [INR], platelet count), arterial blood gas analysis (including pH), haemoglobin, and random blood glucose levels were performed within 12 hours of admission. Coagulopathy was operationally defined as the presence of one or more of the following: PT >14 seconds, APTT >36 seconds, INR >1.25, or platelet count <1 lakh/μL. These thresholds were based on institutional norms and previously published literature.

Exposure and Outcome Variables

The primary exposure variable was the presence or absence of coagulopathy. The primary outcome was in-hospital mortality. Secondary outcomes included associations of coagulopathy with clinical parameters such as GCS score at admission, pupil responsiveness, haemoglobin levels, and acid-base status (pH).

Management Protocol and Follow-up

All patients were managed according to institutional head injury protocols, including neuroimaging, monitoring of vital signs, and supportive measures. Most patients received tranexamic acid as per the CRASH-3 protocol for TBI, a limitation noted due to its influence on fibrinolytic mechanisms. Patients were followed up from the time of admission until discharge or in-hospital death. Data on clinical course, need for neurosurgical intervention, and outcome were collected during hospital stay.

Statistical Analysis

Data entry was carried out using Microsoft Excel and analyzed using IBM SPSS version 27.0. Descriptive statistics were applied to summarize baseline characteristics. Categorical variables were reported as frequencies and percentages, while continuous variables were expressed as mean and standard deviation. The Chi-square test was used to determine associations between coagulopathy and categorical variables like mortality, GCS category, and pupil response. For normally distributed continuous variables such as haemoglobin and pH, Student's t-test was applied; otherwise, the Mann-Whitney U test was used. A p-value of less than 0.05 was considered statistically significant, and $p<0.01$ was considered highly significant.

Ethical Considerations

Written informed consent was obtained from all participants or their legal guardians in cases of altered sensorium or impaired consciousness. Confidentiality of patient data was strictly maintained, and participation in the study did not affect the course of clinical management. No additional investigations or financial burdens were imposed on patients as all tests were part of standard clinical care. The results of the study were used solely for academic and research purposes.

RESULTS:

The study included 144 patients with isolated traumatic brain injury (TBI). The majority of traumatic brain injury (TBI) patients in the study belonged to the age group of 18–30 years (31.9%), reflecting the high-risk behaviour and increased exposure to road traffic accidents in younger adults. The mean age of the sample was 43.4 ± 19.3 years, suggesting a relatively younger to middle-aged population affected by TBI. Regarding sex distribution, there was a marked male predominance (85.4%), consistent with global trends indicating higher risk-taking behaviours and occupational exposure among men **(Table 1)**. Among the 144 patients with traumatic brain injury (TBI), road traffic accidents (RTAs) emerged as the leading cause, accounting for 78.5% of cases. This underscores the significant public health impact of motor vehicle-related injuries. Assault (11.8%) and slip and fall incidents (7.6%) were other notable contributors, while giddiness-related falls (2.8%) were more common among the elderly population, suggesting age-related balance issues or underlying comorbidities. These findings emphasize the importance of improving road safety measures and fall prevention strategies, particularly in vulnerable age groups **(Figure 1)**. The majority of TBI cases (61.1%) presented with extra-parenchymal injuries, including subdural hematoma (SDH), epidural hematoma (EDH), and subarachnoid hemorrhage (SAH), which are commonly associated with blunt trauma and acceleration-deceleration mechanisms. Intra-parenchymal injuries, such as contusions and intraparenchymal hemorrhage, were noted in 16.7% of the patients. Notably, 20.8% had combined extra- and intra-parenchymal hemorrhages, indicating more severe or diffuse brain trauma. A minority (1.4%) presented with pneumocephalus, likely secondary to basal skull fractures or open cranial injuries **(Figure 2).**

Among the 144 patients with traumatic brain injury (TBI), 20.1% had an elevated INR (>1.25), indicating the presence of coagulopathy at admission. The majority of patients (79.9%) had INR values within the normal range (≤1.25). An increased INR reflects impaired extrinsic pathway function, which may predispose to or worsen intracranial bleeding **(Table 2)**.

Among the coagulation parameters, 20.1% of patients exhibited an elevated INR (>1.25), while 23.6% had a prolonged prothrombin time (PT >14s), both indicating significant disruption in the extrinsic coagulation pathway. Although activated partial thromboplastin time (APTT >36s) was elevated in only 8.3% of patients, its presence still contributed to the overall coagulopathy profile. Thrombocytopenia (platelet count <1 lakh/μL) was observed in 11.1%, which is clinically important as platelets play a central role in clot formation and prevention of progressive intracranial hemorrhage. Anemia (Hb ≤10 g/dL) and metabolic acidosis (pH ≤7.2) were each noted in 8.3% of patients, both of which were statistically associated with coagulopathy in this cohort. These findings support the interplay between tissue hypoxia, systemic shock, and impaired coagulation mechanisms. From a neurological standpoint, 9% of patients presented with severe TBI (GCS <8), and this group had a high prevalence of coagulopathy, consistent with previous studies linking injury severity to haemostatic dysfunction. Additionally, abnormal pupillary findings were seen in 10.4% of patients 6.9% with anisocoria and 3.5% with absent reactivity suggesting elevated intracranial pressure or brainstem dysfunction **(Table 3)**.

Out of 144 patients with isolated traumatic brain injury (TBI), 90.3% were successfully discharged, while 9.7% died during their hospital stay. Although the majority of patients recovered, the in-hospital mortality rate of nearly 10% underscores the severity of isolated TBI and its potentially fatal complications. Mortality was observed to be significantly associated with coagulopathy, particularly among patients with elevated INR, prolonged PT, thrombocytopenia, metabolic acidosis, low GCS scores, and abnormal pupillary response. These findings suggest that early identification and correction of these derangements could be crucial in reducing preventable TBI deaths **(Figure 3)**. Among those without coagulopathy, all 87 patients (100%) were discharged, and none expired. In contrast, among patients with coagulopathy, 24.6% died while receiving intensive care, and only 75.4% were discharged. The chi-square test yielded a χ^2 value of 23.67 with a p-value < 0.01, indicating that the difference in outcomes between patients with and without coagulopathy is highly statistically significant. This confirms that coagulopathy is a strong predictor of adverse outcome in isolated TBI and should be used as a critical early marker in patient triage and management decisions **(Figure 4)**.

Clinical and laboratory variables revealed significant associations between coagulopathy and several risk factors in patients with isolated traumatic brain injury (TBI). Among the variables studied, Glasgow Coma Scale (GCS) showed a strong inverse relationship with coagulopathy. While only 30.8% of patients with mild TBI (GCS 13–15) had coagulopathy, the proportion increased to 72.7% in those with moderate TBI (GCS 8–13) and peaked at 92.3% among patients with severe TBI (GCS <8). This trend was statistically significant ($\chi^2 = 24.01$, $p < 0.01$), indicating that lower GCS scores at

presentation are strongly predictive of coagulation abnormalities. Similarly, arterial pH showed a meaningful association with coagulopathy. Patients with metabolic acidosis (pH ≤7.2) had an 83.3% prevalence of coagulopathy compared to only 35.6% among those with normal pH values (>7.2), a difference that was highly significant ($\chi^2 = 10.48$, $p = 0.001$). Haemoglobin levels also correlated significantly with the presence of coagulopathy; 75% of patients with anaemia (Hb ≤10 g/dL) exhibited coagulopathy, compared to 36.4% in those with normal haemoglobin levels, with statistical significance ($\chi^2 = 6.87$, $p = 0.009$) **(Table 4).**

DISCUSSION:

Traumatic brain injury (TBI) remains a leading global cause of mortality and morbidity, particularly among young adults. Sixty-nine million (95% CI 64-74 million) individuals worldwide are estimated to sustain a TBI each year. The proportion of TBIs resulting from road traffic collisions was greatest in Africa and Southeast Asia (both 56%) and lowest in North America (25%) (15). It is acknowledged that 12% of all deaths in India are caused by injuries and the resulting deaths, which constitute a public health concern (16). The eighth most common cause of mortality worldwide and the top cause of death for youths between the ages of 15 and 29 is road traffic injuries (RTI). 2. Unless immediate action is taken, current trends indicate that traffic-related deaths will rank as the fifth most common cause of death by 2030 (17).

Our study contributes to the existing literature by examining the prevalence of coagulopathy and its impact on clinical outcomes in patients with isolated TBI admitted to a tertiary care center in Kerala.

Several studies from high-income countries have recommended using coagulation markers such as PT, APTT, INR, platelet count, and thromboelastography (TEG) or rotational thromboelastometry (ROTEM) to identify patients at risk for poor outcomes (17,18). However, these advanced tests are not widely available in most Indian tertiary centers. Our study adopts practical and commonly available laboratory parameters to assess coagulopathy. Defining coagulopathy as PT >14 seconds, APTT >36 seconds, INR >1.25, or platelet count <1 lakh/μL, this investigation seeks to provide data relevant to routine clinical settings. Additionally, it evaluates associated clinical predictors such as Glasgow Coma Scale (GCS), pupillary response, pH, and haemoglobin levels to understand the risk factors contributing to coagulopathy. A key finding of our study was the significant association between coagulopathy and in-hospital mortality. Among patients without coagulopathy, no deaths were recorded, whereas 24.6% of coagulopathic patients died during hospitalization. Another study stated the similar finding where coagulopathy is a strong independent predictor of mortality in TBI patients (6,13). According to some studies, the mechanism might entail diminished cerebral perfusion, elevated intracranial pressure, and progressive haemorrhagic damage (12).

The factors that contribute to the development of coagulopathy were also examined in the study. The Glasgow Coma Scale (GCS) score showed a strong negative connection with coagulopathy. The prevalence of coagulopathy increased significantly to 72.7% in moderate TBI cases and 92.3% in severe TBI cases, whereas it was only 30.8% in mild TBI patients. This result is consistent with other studies where they stated that systemic coagulation abnormalities are caused by the activation of inflammatory and endothelial pathways as the severity of brain injury increases [3,4]. Acidosis (pH ≤7.2) was another important factor linked with coagulopathy. Our study found that 83.3% of patients with acidosis developed coagulopathy, a statistically significant finding. This supports the widely accepted concept of the "lethal triad" in trauma acidosis, coagulopathy, and hypothermia – which together significantly worsen outcomes (11-16). Similarly, anemia (Hb ≤10 g/dL) was found in 75% of patients with coagulopathy, compared to only 36.4% in non-coagulopathic patients. This highlights how reduced oxygen delivery may exacerbate secondary brain injury and impair hemostasis (7).

APTT abnormalities were present in only 8.3% of the cohort, their presence alongside elevated PT and INR reinforces the need for a comprehensive coagulation profile upon admission. Some studies have similarly stressed the importance of evaluating both intrinsic and extrinsic coagulation pathways in TBI patients (10,11). Also, studies have proved thrombocytopenia correlates with poor neurological outcomes in head injury (14,16,18). Our study also supports the recommendation of early correction of coagulation abnormalities in neurosurgical and trauma patients to prevent deterioration (17,21).

However, our study has several limitations. First, it was conducted in a single tertiary care center, which may limit generalizability. Second, the observational design precludes causal inference. Third, advanced coagulation diagnostics such as thromboelastography (TEG) were not available, which could have provided a more dynamic understanding of coagulation status. Also, the use of tranexamic acid based on CRASH-3 protocol in most patients may have influenced the natural progression of coagulopathy, potentially masking its full impact. Finally, we did not assess long-term functional outcomes post-discharge, which could offer more comprehensive insight into the burden of TBI-related coagulopathy. Despite these limitations, the study highlights the high prevalence and prognostic value of coagulopathy in isolated TBI, and underscores the importance of integrating basic coagulation tests such as PT, INR, APTT, hemoglobin, and pH in routine evaluation. These markers, along with clinical indicators like GCS and pupillary response, can guide early intervention strategies and improve patient outcomes.

CONCLUSION:

In conclusion, this study has provided valuable insights into the prevalence of coagulopathy among isolated TBI patients admitted to a tertiary care centre and its implications for patient outcomes. Coagulopathy is a common and clinically significant complication in patients with isolated TBI. It is strongly associated with poor outcomes, including in-hospital mortality. Early identification and correction of coagulation abnormalities, particularly in patients with low GCS, acidosis, or anemia, can be crucial in reducing TBI-related morbidity and mortality. Further multicentric studies with long-term

follow-up and advanced diagnostics are warranted to develop standardized protocols for managing TBI-associated coagulopathy.

DECLARATIONS

Conflicts of interest: The authors declare that they have no conflicts of interest.

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Ethical approval: The study was approved by the Human Ethics Committee of Government Medical College Thiruvananthapuram (HEC NO:04/21/2023/MCT).

Consent to participate: Written informed consent was obtained from all participants or their legally authorized representatives.

Consent for publication: Informed consent for publication was obtained from all participants.

Availability of data and material: The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request.

Authors' contribution:

- AS: Conceptualization, Study design, Data collection, Drafting of manuscript
- AS, JS, NA: Statistical analysis, Interpretation of results, Critical revision
- JS, NA, AS: Supervision, Final approval of manuscript

All authors have read and approved the final version of the manuscript.

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REFERENCES:

1. Dewan MC, Rattani A, Gupta S, Baticulon RE, Hung YC, Punchak M, Agrawal A, Adeleye AO, Shrime MG, Rubiano AM, Rosenfeld JV, Park KB. Estimating the global incidence of traumatic brain injury. J Neurosurg. 2018 Apr 27;130(4):1080-1097. doi: 10.3171/2017.10.JNS17352. PMID: 29701556.
2. Gururaj G. Epidemiology of traumatic brain injuries: Indian scenario. Neurol Res. 2002 Jan;24(1):24-8. doi: 10.1179/016164102101199503. PMID: 11783750.
3. Maas AIR, Stocchetti N, Bullock R. Moderate and severe traumatic brain injury in adults. Lancet Neurol. 2008 Aug;7(8):728–41.
4. Harhangi BS, Kompanje EJ, Leebeek FW, Maas AI. Coagulation disorders after traumatic brain injury. Acta Neurochir (Wien). 2008 Feb;150(2):165–75.
5. Chhabra G, Sharma S, Subramanian A, Agrawal D, Sinha S, Mukhopadhyay AK. Coagulopathy as prognostic marker in acute traumatic brain injury. J Emerg Trauma Shock. 2013 Jul;6(3):180-5. doi: 10.4103/0974-2700.115332. PMID: 23960374; PMCID: PMC3746439.
6. Yuan Q, Yu J, Wu X, Sun Y, Li Z, Du Z, et al. Prognostic value of coagulation tests for in-hospital mortality in patients with traumatic brain injury. Sci Rep. 2016;6:38752.
7. Chhabra G, Sharma S, Subramanian A, Agrawal D, Sinha S, Mukhopadhyay AK. Coagulopathy as a prognostic marker in acute traumatic brain injury. J Neurosci Rural Pract. 2013 Jan;4(Suppl 1):S19–23.
8. Epstein DS, Mitra B, Cameron PA, Fitzgerald M, Rosenfeld JV. Acute traumatic coagulopathy in the setting of isolated traumatic brain injury: definition, incidence and outcomes. Br J Neurosurg. 2014;28(4):452–6.
9. Pathak A, Dutta S, Marwaha N, Singh D, Varma N, Mathuriya SN. Change in tissue thromboplastin content of brain following trauma. Neurol India. 2005;53(2):178–81.
10. Greuters S, van den Berg A, Franschman G, Viersen VA, Beishuizen A, Peerdeman SM, et al. Acute and delayed mild coagulopathy are related to outcome in patients with isolated traumatic brain injury. Crit Care. 2011 Jan;15(1):R2.
11. de Oliveira Manoel AL, Neto AC, Veigas PV, Rizoli S. Traumatic brain injury associated coagulopathy. Neurocrit Care. 2015 Feb;22(1):34–44.
12. Stein SC, Spettell C, Young G, Ross SE. Delayed and progressive brain injury in closed-head trauma: radiological demonstration. Neurosurgery. 1993 Jan;32(1):25–30.
13. Talving P, Benfield R, Hadjizacharia P, Inaba K, Chan LS, Demetriades D. Coagulopathy in severe traumatic brain injury: a prospective study. J Trauma. 2009 Nov;66(1):55–61.
14. Kalkan E, Bayir A, Kocak S, Ak A, Erol FS, Akdemir H. Fibrinolytic markers and neurologic outcome in traumatic brain injury. Neurol India. 2006;54(4):363–5.
15. Non-communicable Diseases: County profile (India), World Health Organization, 2014. https://doi.org/10.1371/journal.pone.0157705
16. World Health Organization, Global Status Report on Road Safety, Supporting a Decade of Action, 2013.
17. West KL, Adamson C, Hoffman M. Prophylactic correction of the international normalized ratio in neurosurgery: a brief review of a brief literature. J Neurosurg. 2011 Jan;114(1):9–18.
18. Greuters S, Boer C, Franschman G, Peerdeman SM, Vries GJ, Vries HJ, et al. Acute coagulopathy is associated with progressive hemorrhagic injury in patients with traumatic brain injury. J Neurotrauma. 2012 Jan;29(1):137–42.

19. Lustenberger T, Talving P, Kobayashi L, Barmparas G, Inaba K, Demetriades D. Time course of coagulopathy in isolated severe traumatic brain injury. Injury. 2010 Sep;41(9):924–8.
20. Mitra B, Mori A, Maini A, Churilov L, Chan P, Fitzgerald M, et al. Early prediction of acute traumatic coagulopathy using clinical and laboratory parameters in trauma patients. J Am Coll Surg. 2018 Oct;227(4):453–62.
21. Schochl H, Maegele M, Solomon C, Gorlinger K, Voelckel W. Early and individualized goal-directed therapy for trauma-induced coagulopathy. Scand J Trauma Resusc Emerg Med. 2012 Mar;20:15.