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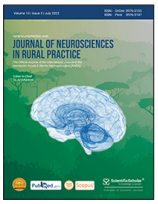

Original Article

Risk factors of deterioration in patients of head injury with non-operative management on first neurosurgical consultation

Jayati Churiwala[1], Mahadeo Namdeo Garale[1], Juhi Kawale[2], Saswat Kumar Dandpat[4], Amit Mahore[3]

Departments of [1]General Surgery, [2]Internal Medicine, and [3]Neurosurgery King Edward Memorial Hospital and Seth Gordhandas Sunderdas Medical College, Mumbai, Maharashtra, [4]Department of Neurosurgery, All India Institute of Medical Sciences, Kalyani, West Bengal, India.

ABSTRACT

Objectives: In most of the emergency trauma intensive care units (ICUs) of India, neurosurgical opinion is sought for patients presenting with head trauma after earliest possible resuscitation to determine the further line of management. This study aimed to identify common risk factors, leading to neurological deterioration in conservatively managed patients of traumatic brain injury (TBI).

Materials and Methods: This retrospective study analyzed patients admitted with acute TBI and traumatic intracranial hematoma under emergency trauma care ICU who did not require neurosurgical operation within 48 h of trauma. The recorded data were analyzed to determine the predictors of neurological deterioration using univariate and binary logistic regression analysis in SPSS-16 software.

Results: Medical records of consecutive 275 patients of acute TBI presenting to the emergency department were studied. One hundred and ninety-three patients were afflicted with mild TBI (70.18%), 49 patients had moderate TBI (17.81%), and 33 had severe TBI (12%). In the outcome, 74.54% of patients were discharged, and operative decision was made on 6.18% of patients and 19.27% died. Severe TBI is the independent predictor of neurological deterioration during their stay in ICU. Progressive hemorrhagic injury (PHI) showed neurological deterioration in 86.5% of patients. Systemic inflammatory response syndrome (SIRS) was present in 93.5% of patients who had deteriorated neurologically. Dyselectrolytemia was the biochemical derangements seen in 24.36% of cases.

Conclusion: This study revealed severe TBI, PHI, and SIRS to be strong and independent risk factors of neurological deterioration.

Keywords: Traumatic brain injury, Hemorrhage, Indian intensive care unit, Risk factors

INTRODUCTION

Traumatic brain injury (TBI) is a leading cause of death and disability worldwide with India being one of the largest contributors to the numbers.[1-3] In most of the tertiary care centers in India, neurosurgical opinion is sought with earliest possible resuscitation of a patient presenting with head trauma, to determine the further line of management. Early detection of non-neurological complications following TBI is very common which eventually determines the outcome.[4,5] A computed tomography (CT) scan of the brain is the standard diagnostic tool in the determination of intracranial injury. Depending on the neurological examination and findings on CT scan of brain, the need of surgery is decided for acute TBI.[6-8] The patients who do not require neurosurgical procedures initially are subjected to close observation under intensivists and neurosurgeons. Patients who do not improve with medical management or deteriorate clinically are routinely subjected to repeat CT scan and laboratory investigations to determine the cause of neurological deterioration.

We performed a retrospective analysis of risk factors, leading to neurological deterioration in conservatively managed TBI patients.

MATERIALS AND METHODS

This is a single-center retrospective analytical study. The study had been initiated after approval from the Institutional Ethics Committee. This study analyzed 275 patients with acute TBI and traumatic intracranial hematoma (tICH) on initial CT brain within 48 h of trauma, admitted under emergency trauma care intensive care unit (ICU) who did not require neurosurgical operation. The tICH included epidural hemorrhage, subdural hemorrhage (SDH), intraparenchymal

***Corresponding author:** Amit Mahore, Department of Neurosurgery, King Edward Memorial Hospital and Seth Gordhandas Sunderdas Medical College, Mumbai, Maharashtra, India. amit2mahore@yahoo.co.in

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hemorrhage, and subarachnoid hemorrhage (SAH). The study was conducted in King Edward Memorial Hospital, Mumbai, which is largest tertiary care center in West India.

Patients with following criteria were excluded: (1) Age <12 years, (2) on antiplatelets/anticoagulant medications, (3) head injury with normal CT study of brain, (4) patients with penetrating head injuries, and (5) admissions >48 h after the traumatic event. The criteria for non-operative management were primarily based on the clinical and radiographic findings on admission, including alert mental status, absent lateralizing signs, basal cistern effacement or obliteration, and midline shift <5 mm. Initial neurosurgical intervention was defined as an operation done immediately while the patient was in the emergency department's ICU. Delayed neurosurgical intervention was defined as an operation done after the failure of non-operative management.

Clinical assessment

Data of the participants were recorded on pre-approved Case Record Forms. Collected data included demographic details, mode of injury, brief general, and neurological examination findings and all available routine laboratory investigation reports obtained on admission until the end point of treatment, CT brain findings, daily neurosurgery opinion, and progressive evaluation of neurological status in the form of Glasgow Coma Scale (GCS) and pupillary reaction to light until the end point of indoor treatment (conversion from conservative to operative management/death/discharge). Signs of systemic inflammatory response syndrome (SIRS) were noted, and routine electrolytes charted and monitored for imbalance. All patients were evaluated for three risk factors of neurological deterioration after resuscitation and stabilization: Progressive hemorrhagic injury (PHI), dyselectrolytemia, and SIRS.

Statistical analysis

Medical records of 275 patients of acute TBI presenting to the emergency department were studied. The recorded data were analyzed to determine the predictors of neurological deterioration using univariate and binary logistic regression analysis in SPSS-16 software [Tables 1 and 2]. Continuous variables are presented as the mean or frequency (*n*) and percentage (%). Categorical variables were analyzed using Chi-square test. Logistic regression analysis was applied to determine the independent predictors of mortality. $P < 0.05$ was considered statistically significant.

RESULTS

General characteristics

The mean age of patients was 42.38 years with more prevalence in male gender (74.54%). About 28.36% of patients had alcohol consumption before the event. The

Table 1: Frequency of categorical variables and outcome.

Variables	*n* (%)
Age in years (mean)	42.38
Sex	
Male	205 (74.54)
Female	70 (25.45)
Alcohol	
Present	78 (28.36)
Absent	197 (71.63)
Mode of injury	
Assault	17 (6.18)
Fall	83 (30.18)
RTA	169 (61.45)
Railway accident	6 (2.18)
CT brain	
Contusion	63 (22.9)
Multiple contusion	21 (7.6)
Diffuse axonal injury	2 (0.72)
EDH	43 (15.63)
tICH	6 (2.18)
SAH	24 (8.72)
SDH	53 (19.27)
Pneumocephalus	5 (1.81)
Multiple injury	58 (21.09)
TBI	
Severe	33 (12)
Moderate	49 (17.81)
Mild	193 (70.18)
PLT	
<1.5 lakh/mm^3	32 (11.63)
>1.5 lakh/mm^3	243 (88.36)
INR	
>1.4	12 (4.36)
<1.4	263 (95.63)
CE	
Generalized	30 (10.9)
Perilesional	86 (31.27)
Absent	159 (57.81)
PHI	
Present	52 (18.9)
Absent	223 (81.09)
SIRS	
Present	31 (11.27)
Absent	244 (88.72)
Dyselectrolytemia	
Present	67 (24.36)
Absent	208 (75.63)
Deterioration	
Present	86 (31.27)
Absent	189 (68.72)
Outcome	
Discharge	205 (74.54)
Operation	17 (6.18)
Death	53 (19.27)

TBI: Traumatic brain injury, SIRS: Systemic inflammatory response syndrome, PLT: Platelet, INR: International normalized ratio, CE: Cerebral edema, PHI: Progressive hemorrhagic injury

Table 2: Analysis of predictors of deterioration.

Predictors	Deterioration		Univariate analysis		Logistic regression			Odd's ratio
	Present (%)	Absent (%)	Chi-square (χ^2)	Sig. *P* value	df	Sig. *P* value	C.I (95%)	
TBI								
Severe	27 (81.8)	6 (18.2)	44.576	0.000	1	0.008	1.510–15.97	4.913
Moderate	12 (24.5)	37 (75.5)			1	0.387	0.260–1.686	
Mild	47 (24.4)	146 (75.6)						
CE								
Generalized	20 (66.7)	10 (33.3)	20.093	0.000	2	0.172		
Perilesional	25 (29.1)	61 (70.9)						
PHI								
Present	45 (86.5)	7 (13.5)	91.605	0.000	1	0.000	6.470–42.535	16.59
Absent	41 (18.3)	183 (81.7)						
SIRS								
Present	29 (93.5)	2 (6.5)	63.37	0.000	1	0.000	3.693–98.023	19.025
Absent	57 (23.3)	188 (76.7)						
DELT								
Present	21 (30.9)	47 (69.1)	0.003	0.955	1	0.173		
Absent	65 (31.3)	143 (68.8)						
PLT								
<1.5 lakh/mm3	15 (46.9)	17 (53.1)	4.168	0.041	1	0.598		
>1.5 lakh/mm3	71 (29.1)	173 (70.9)						
INR								
<1.4	81 (30.7)	183 (69.3)	0.646	0.422	1	0.477		
>1.4	5 (41.7)	7 (58.3)						
Alcohol								
Present	27 (34.2)	52 (65.8)	0.47	0.493	1	0.611		
Absent	59 (29.9)	138 (70.1)						

TBI: Traumatic brain injury, CE: Cerebral edema, PHI: Progressive hemorrhagic injury, SIRS: Systemic inflammatory response syndrome, DELT: Dyselectrolytemia, PLT: Platelet, INR: International normalized ratio

most common mode of injury in study was road traffic accident (RTA) (61.45%) followed by fall (30.18%) [Table 1]. The mean time between injury and presentation to the emergency department was approximately 8.56 h. All patients underwent a CT scan of the brain within 24 h of injury. Majority of the patients had a normal coagulation profile. About 11.63% of patients had a reduced platelet count and 4.36% of the patients had a raised international normalized ratio (INR) [Table 1].

Injury characteristics

The most common finding on CT brain was hemorrhagic contusion (22.9%) followed by multiple injuries (21.09%). This was followed by SDH (19.27%), extradural hematoma (15.63%), and SAH (8.72%) [Table 1]. According to the GCS score, 70.18%, 17.81%, and 12% of patients suffered from mild, moderate, and severe TBI, respectively [Table 1].

Risk factors

In this study population, 18.9% (n = 52) of the study sample suffered progressive hemorrhagic injury and 11.27% (n = 31) of patients displayed SIRS. The proportion of patients with dyselectrolytemia was 24.36% (n = 67). Neurological deterioration was found in 31.27% (n = 86) of patients during their course of stay in the hospital. Among 275 patients, 205 were discharged, while the treatment plan of 6.18% (n = 17) of patients was converted to surgical treatment and 19.27% (n = 53) of the study sample died indoor [Table 1]. When comparing patients who displayed neurological deterioration with patients who did not display deterioration, the average age of patients in the two groups does not seem to differ significantly. Contrary to the popular belief, alcohol consumption (28.36%) of the studied patients of acute TBI did not contribute to neurological deterioration significantly in (Chi-square value – 0.47, P = 0.611) [Table 2]. Out of a sample size of 275 patients, 193 patients were afflicted with mild TBI (70.18%), 49 patients were afflicted with moderate TBI (17.81%), and 33 with severe TBI (12%) [Table 2]. Among severe, moderate, and mild TBI patients, severe is the independent predictors of neurological deterioration during their course of admission (P = 0.008, confidence interval [CI] = 1.510–15.97). Moderate TBI showed statistical significance along with severe TBI (Chi-square value – 44.576, P = 0.000) in univariate analysis; however,

in logistic regression analysis, only severe TBI emerged as a strong predictor of deterioration (odds ratio – 4.9) [Table 2]. Platelet count of <1.5 lakh/mm_3 and INR more than 1.4 did not show any statistical significance in logistic regression test, respectively (P = 0.598, P = 0.477) [Table 2]. PHI was found in 86.5% of patients in the neurological deterioration group. PHI emerged as an independent predictor of neurological deterioration (P = 0.000) and patient with moderate PHI had 16.59 times risk of deterioration (CI = 6.470–42.535). SIRS was present in 93.5% of patients who had deteriorated neurologically and was statistically significant (P = 0.000). The risk of deterioration in patients with SIRS was 19.03 times more than the patients without SIRS (CI = 3.693–98.023) [Table 2]. Dyselectrolytemia, that is, hyponatremia or hypernatremia was the biochemical derangements seen in 24.36% (n = 67) of cases; however, it was not statistically significant contributor of neurological deterioration in our study (P = 0.173). Perilesional edema (31.21%) was more common than generalized cerebral edema (10.9%). A larger proportion of patients with generalized cerebral edema (66.7%) as compared with perilesional edema (29.1%) showed neurological deterioration which was statistically significant in univariate analysis (Chi-square value – 20.093, P = 0.000) but not in logistic regression analysis (P = 0.172) [Table 2]. This suggests that there must be other confounding factors, leading to deterioration. In the outcome, 74.54% (n = 205) of patients were discharged, and operative decision was made on 6.18% (n = 17) of patients and 19.27% (n = 53) died. This study revealed severe TBI, PHI, and SIRS to be strong and independent risk factors of neurological deterioration [Table 2].

DISCUSSION

As per the World Health Organization (WHO), RTA is the sixth most common cause of death and will be the third leading cause of mortality and morbidity by 2030 in the developing countries.[9,10] According to age groups, adolescents and adults showed high rate of traffic accidents while falls were the most common mechanism in the infants, preschoolers, and schoolchildren. The most common mode of injury was RTA (61.45%) in our study. Skull fracture was the most common injury in patients with mild and moderate TBI followed by extradural hematoma. In patients with severe head injury, skull fractures and SDH accounted for the highest proportions followed by sub-arachnoid hemorrhage and intraventricular hemorrhages.[9] TBI is more prevalent in male population.[10] However, there was no significant association of neurological deterioration with either age or gender of the patient in our study.

The management of TBI encompasses pre-hospital evaluation and care, in-hospital management, and continued follow up. The initial management of patients with TBI is identical to that of all trauma patients, focusing on the Advanced Trauma Life Support principles of the management of airway, breathing, and circulation. The primary goal of pre-hospital and in-hospital airway assessment and hemodynamic stabilization is to prevent hypoxia and hypotension which are known to be major causes of secondary brain insult. Hypoxia is defined as PaO_2<60 mmHg and hypotension as systolic blood pressure <90 mmHg. Endotracheal intubation in patients with TBI is indicated in patients with a compromised airway, SpO_2<90 despite supplemental oxygen, and a Glasgow Coma Scale (GCS) score of <9.[7,11-13] Patients with TBI should be presumed to have spine injuries and immobilized during transport. A detailed history and examination of injuries and neurological assessment are followed in the emergency department.

Alcohol abuse has been known to be associated with increased morbidity and mortality among patients of TBI due to a blunting of sympathetic response.[11,14] Around 28.36% of our patients had a history of alcohol consumption before the traumatic event. However, no significant association between the consumption of alcohol and neurological deterioration was seen in our study. The effect of alcohol on trauma outcome has been studied with mixed results.[14-16] The mechanisms whereby alcohol intoxication reduces the mortality of head injury are still unknown. The diuretic effect of alcohol might reduce the intracranial pressure after trauma. In addition, brain atrophy in some chronic alcoholic cases may have more space for hematoma to prevent increased intracranial pressure.[15,16] In addition, most of our patients had motorcycle-related injury, which might result in the lower injury severity score compared to other studies.[15,16] Further study on the relationship between the level of blood alcohol and mortality is needed to elucidate the mechanism.

The brain, through autoregulatory mechanisms, maintains the cerebral blood flow over a wide range of blood pressures. This capacity is, however, deranged in approximately one third of the patient of acute TBI. PHI has been shown to be closely associated with neurological deterioration in patients of TBI worldwide with fivefold worsening of prognosis. PHI has been defined as worsening picture on serial CT brain due to the development of a new lesion or increase in volume of a previous lesion by more than 25%.[17] Few authors also stated that incidence of PHI in head trauma patients was 42.3%.[17] PHI has been found to be associated with deranged coagulation profile, older age, male gender, hyperglycemia, raised D-dimer, and increased time from injury to first CT scan.[18] In this study, PHI was seen in 18.9% of the patients and among them 86.5% of patients showed neurological deterioration due to progression of lesion in serial scans without any association of other factors. There have been various prognostic models to predict the occurrence of PHI in patients of acute TBI based on admission variables. These include increasing age, prothrombin time more than 14 s,

low level of initial consciousness, platelet count between 1 and 1.5 lakhs/mm_3, raised D-dimer, intra-axial bleeding, and midline shift on CT brain of 5 mm or more. These have been studied by Yuan *et al*. to be independent risk factors of progressive hemorrhagic injury.[19] In our study, we have taken only platelet count and INR as a coagulation markers. However, we did not find a statistically significant association between low platelet count, deranged INR, and neurological deterioration. Majority of patients of our study had mildly deranged INR (1.5–2.0). Further, detailed study may analyze the effect of severe thrombocytopenia, high INR, and other coagulation parameters on head injury patients in our scenario. Moreover, it is noteworthy that prognostic models that predict the occurrence of PHI are not part of the initial assessment of patients of TBI in many trauma centers in India. Several factors including logistics and resource allocation limit the widespread use of these models. As the reported rate of PHI varies from 20 to 60%[19] and since we report a high proportion of this group requires operative intervention, it is imperative for intensivists and neurosurgeons around the world to develop and validate prognostic models to determine the likelihood of PHI.

Furthermore, in a tertiary care center in an American state, SIRS was found to be a predictor of unfavorable outcome in patients with hemorrhagic brain injury presenting as sepsis or pneumonia in 50–75% of patients with isolated head trauma.[20] Inflammatory mediators release after TBI is responsible for initiating and propagating early, delayed, and systemic effects of brain injury. Damage to the blood–brain barrier exposes the normally immune privileged brain to peripheral inflammatory mediators. Once systemic inflammation is triggered, it is subsequently followed by coagulopathy and complement deficit which may further exacerbate the bleeding associated with TBI.[21] Secondary brain insults such as hypoxia and hypotension have been shown to aggravate the SIRS. Alternatively, the counter anti-inflammatory response of the body may result in immune suppression and make the patient prone to superadded infections such as pneumonia and urinary tract infections, especially in patients with known immune-compromised states.[21,22] Musculoskeletal, chest, and abdominal injuries giving rise to significant systemic responses may also aggravate the neuroinflammatory responses to trauma by the aforementioned pathways.[23] Trauma triggers a complex cascade of events that can be divided into hemodynamic, metabolic, neuroendocrine, and immune responses, leading to a multifocal pathophysiological process. However, inflammation is not in itself detrimental. Inflammation is necessary for the removal of challenges to the organism and subsequent restoration of homeostasis. However, hemorrhage and trauma can induce a dysregulated acute inflammatory response that affects several organ systems and sets in motion a vicious cycle of inflammation and damage driven by chemokines, cytokines, and products of damaged, dysfunctional, or stressed tissue. Activation of early phase inflammatory mediators and complement system gives rise to a generalized inflammatory state – SIRS.[22] We found significant correlation between SIRS and neurological deterioration in our study.

Infective causes of SIRS, in our study, included pneumonia, urinary tract infection, central line-associated bloodstream infection, and wound infection. Pneumonia is a common non-neurological complication of TBI resulting from prolonged mechanical ventilation and pulmonary inflammation. It results in hypoxemia, fever, and hypotension which further aggravate secondary brain injury. Strict antisepsis, adequate pulmonary toilet, avoidance of urosepsis with timely change of urinary catheters, proper handling of central and peripheral venous catheters, and training of health-care officials for the prevention of cross infection are utmost essential for the avoidance of infective causes of SIRS in patients of TBI, which, as we report, is responsible for neurological deterioration in a significantly large proportion of our study population. Proper waste handling and administration of antimicrobials as per drug sensitivity reports will help overcome infective complications.

Serum sodium abnormalities have been found as a sequel of brain injury due to the pivotal role played by the brain in sodium balance. The use of diuretics, intravenous fluids, syndrome of inappropriate secretion of antidiuretic hormone (SIADH), and cerebral salt wasting syndrome (CSWS) have also been found to be associated with dyselectrolytemia. Hypernatremia (>150 mEq/L) may occur either through free water loss (lack of access to free water, diabetes insipidus, or mannitol) or sodium gain due to administration of hypertonic saline. As such, control of intracranial pressure remains an important factor in the management of patients with TBI. Mannitol is the pharmacological agent of choice for the management of increased intracranial pressure. However, concerns over intravascular volume depletion and renal failure have led to the emergence of hypertonic saline as a therapeutic option. Multiple studies have demonstrated an increased mortality in patients of TBI with hypernatremia. In our study, electrolyte imbalance was seen in 24.36% of patients of TBI. After adjusting the effect of SIRS, cerebral edema, age, sex, alcohol, platelet, and INR by applying logistic regression on neurological deterioration, we did not find significant correlation ($P = 0.173$). It is also worth noting that a higher proportion of patients with dyselectrolytemia had hyponatremia rather than hypernatremia. Patients with neurotrauma have hyponatremia resulting from SIADH and CSWS. Hyponatremia is detrimental in patients with TBI as it causes exacerbation of cerebral edema and intracranial pressure. On the contrary, hypernatremia in patients of TBI is said to occur most likely secondary to diabetes insipidus,

and interestingly, post-administration of hypertonic saline. Mortality associated with hypernatremia is reported to be 40–60%.[24] Hence, aggressive efforts need to be invested to maintain electrolyte balance to prevent neurological deterioration. However, in our study, we could not find significant correlation which may be due to early detection of dyselectrolytemia, serial monitoring, and its aggressive correction. We use 3% NaCl as a decongestant in raised intracranial pressure which is also beneficial in correcting existing hyponatremia. On the other hand, only few patients in our study had hypernatremia which allowed us to use 3% NaCl as a primary cerebral decongestant.

According to the results of our study, cerebral edema, both perilesional and generalized, has a positive correlation with neurological deterioration in univariate analysis but not in logistic regression test. Ten times higher mortality among patients of non-penetrating TBI at a Level 1 trauma center was documented with cerebral edema.[25] As the severity of TBI increased from moderate to severe, the proportion of patients suffering from PHI, SIRS, and electrolyte imbalance increased in our study. Early identification and correction of risk factors associated with poor outcome and neurological deterioration in patients with TBI can lead to reduction in mortality among this group of patients.

About 6.18% of patients required decompressive craniotomy in our study. A large proportion of patients who deteriorated neurologically during their course of admission recovered with diligent monitoring, early correction of electrolyte imbalance, treatment of simultaneous infections, avoidance of hypothermia, and treatment of coagulopathy and acidosis. However, 19.27% of patients died whose etiology could not be depicted due to hemodynamic instability. Our study tries to highlight the management difficulties of TBI in the developing countries.

There are some limitations of our study. We could not assess the true effect of hypoxia and hypotension on SIRS in our study due to the confounding impact of initial resuscitation and hemodynamic stabilization. Non-infective causes of SIRS like neurogenic pulmonary edema were also not assessed in our study. Other coagulation factors like thrombin time have not been studied in our study to establish a definite relation with PHI.

Our center is a classical representative of trauma care ICU of developing world which caters services to the majority of population. Intracranial pressure monitoring, jugular venous oximetry, cerebral microdialysis, brain tissue oxygen tension monitoring, pressure reactivity index, and thermal diffusion flowmetry for the measurement of parameters such as cerebral blood flow, metabolism, and oxygen delivery are yet to be introduced in our center. These parameters are being studied in very advanced trauma ICUs of developed world. However, these techniques are yet to withstand the financial and logistic realities of the world. Authentic evidence is required before universal introduction of these parameters into health-care systems worldwide in the management of patients of TBI. Our findings may be extrapolated to the realities of vast majority of centers in India and the developing world.

CONCLUSION

We report severe TBI, PHI, and SIRS as strong independent predictors of neurological deterioration in patients of acute TBI. It is, therefore, imperative to keenly observe patients for signs of electrolyte imbalance and SIRS and correct the same in a timely manner to avoid a point of no reversal in these patients.

Declaration of patient consent

The authors certify that they have obtained all appropriate consent.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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