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Identifying Clinical and Demographic Predictors of Post-Traumatic Cerebral Infarction in Patients with Traumatic Brain Injury

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ABSTRACT

Background: Traumatic brain injury (TBI) is a major public health concern, often leading to severe morbidity and mortality. One of the critical secondary complications following TBI is post-traumatic cerebral infarction (PTCI), which significantly impacts patient outcomes. This study aims to identify clinical and demographic predictors of PTCI in TBI patients to enhance management strategies.

Methods: A retrospective analysis was conducted on 150 patients diagnosed with TBI over the last year. Patients were included based on the presence of hemorrhagic findings on initial computed tomography (CT) scans. Exclusion criteria eliminated individuals with pre-existing neurological conditions or incomplete medical records. Data collection focused on demographic characteristics, Glasgow Coma Scale (GCS) scores, and clinical conditions. Patients with brain herniation were identified through CT scans showing midline shift and clinical signs of altered consciousness. This comprehensive approach ensured thorough monitoring of patient conditions and the effectiveness of interventions following severe head injuries.

Results: Among the 150 patients, 21 developed PTCI, with a notable correlation between lower GCS scores and increased mortality rates. The incidence of PTCI was highest within the first two weeks post-injury, with significant predictors including low GCS scores, the presence of subarachnoid hemorrhage, and hypotensive shock. The mortality rate was significantly higher in patients with GCS scores below 8 compared to those with higher scores.

Conclusion: The study underscores the significant association between low GCS scores and the development of PTCI in patients with TBI. The findings highlight the need for vigilant monitoring and early intervention strategies to mitigate the risk of PTCI and improve patient outcomes.

Key-words: Traumatic brain injury (TBI), Glasgow Coma Scale (GCS) scores, Computed tomography (CT), Subarachnoid hemorrhage, Hypotensive shock

INTRODUCTION

Traumatic brain injury (TBI) continues to be a major public health problem due to its high global incidence of morbidity and mortality. The multiple nature of TBI, which can result in a variety of secondary insults, including PTCI, highlighting its complexity. PTCI has been linked to poor outcomes in patients with severe TBI and is characterized by the formation of brain infarcts after the initial traumatic event [1,2]. PTCI varies in prevalence in this population, but research suggests that a significant percentage of patients, especially those with serious injuries, may experience it [3,4].

Despite being relatively uncommon, PTCI is linked to significant secondary brain injury, which is frequently made worse by variables such as high neutrophil-to-lymphocyte ratios, which can act as biomarkers for early diagnosis [5]. In addition to systemic problems including hypercoagulability and microcirculatory failure that led

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to ischemic damage, the pathogenesis of PTCI includes mechanisms such as brain herniation, cervical artery dissection, and post-traumatic vasospasm [6]. Additionally, TBI is associated with a higher risk of vascular alterations and neurological diseases, which can result in long-term health issues such as myocardial infarctions and strokes [7,8]. A multidisciplinary strategy is necessary for the effective care of PTCI, with a focus on early detection and intervention to reduce negative outcomes [9].

Understanding the risk factors and mechanisms involved in PTCI is important for improving patient management and outcomes. Several studies have reported various risk factors, including the severity of the initial injury, the presence of coagulopathy, and the timing of intervention [10,11]. Moreover, the pathophysiological mechanisms leading to PTCI are multifactorial and may be a result of vascular, metabolic, and inflammatory processes [1].

Despite the significant increase in literature on PTCI, there remains a demand for extensive studies assessing the effect of PTCI on long-term outcomes among TBI patients. The National Traumatic Coma Data Bank has provided important data about the characteristics and outcomes of TBI patients; however, the role of PTCI in altering outcomes is not well documented [12]. This manuscript will attempt to explore the characteristics, risk factors, and possible mechanisms of PTCI in the context of severe TBI, with the ultimate aim of furthering our understanding of this critical secondary insult and its implications for patient care.

MATERIALS AND METHODS

This study was a retrospective analysis conducted at Sir Sayajirao Gaekwad Hospital, Vadodara which is a tertiary referral centre over the past 12 months (July 2023 to July 2024), focusing on patients diagnosed with TBI. A total of 150 patients were included in the study.

Inclusion criteria- Patients included in the study were those who suffered TBI and exhibited any hemorrhagic findings on their initial computed tomography scans. All the patients had their initial CT scan in the Emergency Department, while subsequent CT surveillance was carried out during their hospital admission. The inclusion criteria focused on capturing a wide range of TBI severity and its associated complications.

Exclusion criteria- Patients with pre-existing neurological conditions that could confound the results, such as prior strokes or significant neurodegenerative diseases, were excluded from the study. Moreover, patients who did not have initial CT imaging or whose medical records were incomplete were excluded to ensure the integrity of the data.

Methodology- All patients underwent evaluation and treatment according to the protocols for managing severe head injuries. Neurological evaluations included assessing GCS scores, as well as pupil size and responsiveness. To reduce intracranial pressure (ICP), patients were administered intravenous mannitol in a bolus infusion, starting with a dose of 0.5 g/kg, which could be repeated every 4 to 8 hours. This treatment was monitored to maintain serum osmolality within a target range of 300-320 mOsm/L.

Patients diagnosed with brain herniation syndrome and exhibiting clear radiological evidence of midline shift on CT scans underwent surgical decompression. Brain herniation syndromes were characterized by both imaging results and neurological symptoms or signs. The definitive imaging characteristics included displaced or compressed ventricles and cisterns, while the neurological symptoms or signs encompassed a progressive decline in consciousness, limb weakness, dilated pupils (greater than 5 mm), absence of pupillary response to light, and irregularities in breathing and pulse.

Statistical Analysis- The analysis was conducted using the SPSS software. The independent contributions of predictive factors to the outcome were assessed using the Chi-square test and Fisher's exact test. A probability value of less than 0.05 was considered statistically significant.

RESULTS

The total number of patients included in the study is 150, with 21 of these patients experiencing PTCI. Table 1 provides a comprehensive overview of the demographic and clinical characteristics of patients diagnosed with TBI, specifically focusing on those who developed PTCI. Starting with age, the data indicates that out of the 150 patients, 115 were under the age of 65, and only 9 of these patients developed PTCI.

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In contrast, among the 35 patients aged 65 and older, 12 experienced PTCI. This significant difference is highlighted by a p<0.001, suggesting that younger patients are less likely to develop PTCI compared to older patients, who show a higher incidence of this complication.

When examining sex distribution, the table shows that out of 90 male patients, 10 developed PTCI, while among 60 female patients, 11 experienced PTCI. The p-value of 0.501 indicates that there is no statistically significant difference in the incidence of PTCI between male and female patients, suggesting that gender does not play a critical role in the development of PTCI in this cohort.

The initial GCS scores, which assess the level of consciousness in TBI patients, reveal a concerning trend. Among the 50 patients with a GCS score of less than 8, a significant 15 developed PTCI, indicating a strong association between low GCS scores and the occurrence of PTCI (p<0.001). In contrast, only 5 out of 70 patients with GCS scores between 8 and 11 experienced PTCI, and just 1 patient with a GCS score of 12 or higher developed the condition. This data underscores the critical nature of GCS scores as a predictor for PTCI, with lower scores correlating with a higher risk.

The presence of brain herniation was also assessed, revealing that out of 30 patients with brain herniation, a striking 18 developed PTCI. This association is statistically significant, with a p-value of less than 0.001, indicating that brain herniation is a critical predictor of PTCI in TBI patients. In contrast, only 3 out of 120 patients without brain herniation experienced PTCI, further emphasizing the risk associated with this condition.

Finally, the relationship between decompressive craniectomy and PTCI was examined. Among the 20 patients who underwent this surgical procedure, 10 developed PTCI, resulting in a p-value of 0.033, which suggests a significant association between the need for decompressive craniectomy and the occurrence of PTCI. In comparison, 11 out of 130 patients who did not undergo the procedure developed PTCI.

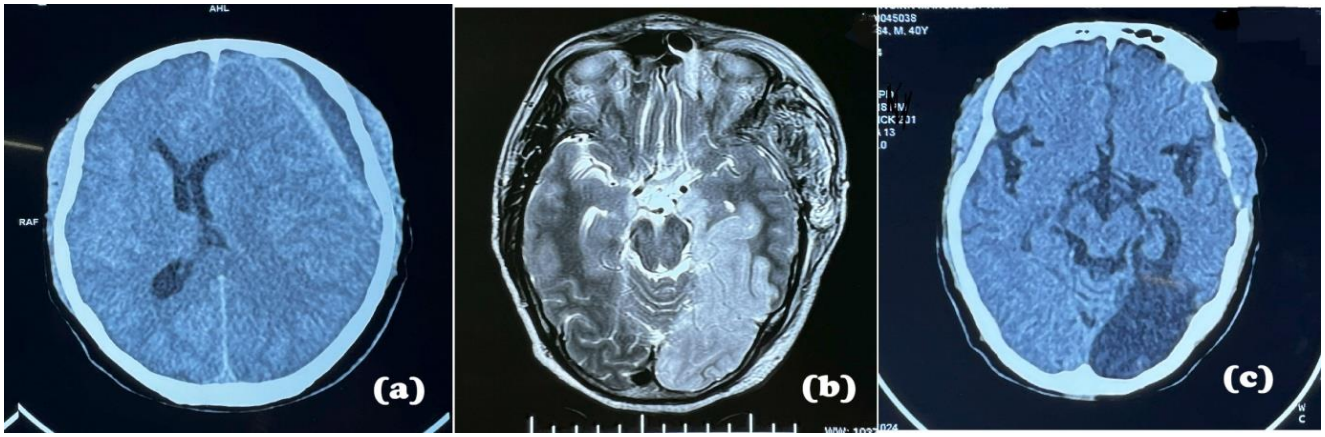

Fig. 1: a) Non-Contrast Computer tomography (NCCT) of a patient with left frontoparietotemporal acute subdural hematoma with midline shift **b)** Magnetic Resonance Imaging of the same patient's immediate postoperative state showing left posterior cerebral artery infarct and postoperative changes after undergoing decompressive craniectomy **c)** NCCT at 3 months follow up with the good recovery of patient and posterior cerebral artery territory infarct.

Table 1: Demographic and clinical data of patients with PTCI.

Characteristic	Total Patients (N=150)	PTCI Patients (N=21)	p-value
Age			
< 65 years	115	9	<0.001
≥ 65 years	35	12	
Sex			
Male	90	10	0.501
Female	60	11	
Initial GCS Score			
< 8	50	15	<0.001
8-11	70	5	

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≥ 12	30	1	
Brain Herniation			
Present	30	18	<0.001
Absent	120	3	
Decompressive Craniectomy			
Yes	20	10	0.033
No	130	11	

PTCI: Post-traumatic cerebral infarction, GCS: Glasgow coma scale

Table 2 presents the distribution of PTCI among various vascular territories. For the anterior circulation, it shows that the Anterior Cerebral Artery (ACA) is affected in five patients, accounting for 23.8% of the cases. In contrast, the Middle Cerebral Artery (MCA) is affected in 10 patients, making up 47.6% of the cases. For the posterior circulation, the Posterior Cerebral Artery (PCA) accounts for 14.3% of cases, with three patients affected. The Basilar Artery is involved in two patients, representing 9.5% of the cases. Additionally, there is one patient with infarctions in multiple territories, making up 4.8% of the total cases.

Table 2: Distribution of PTCI by vascular territory.

Vascular Territory	Number of PTCI Patients (N=21)	Percentage of Total PTCI Patients (%)
Anterior Circulation		
Anterior Cerebral Artery (ACA)	5	23.8
Middle Cerebral Artery (MCA)	10	47.6
Posterior Circulation		
Posterior Cerebral Artery (PCA)	3	14.3
Basilar Artery	2	9.5
Multiple Territories	1	4.8

PTCI: Post-traumatic cerebral infarction, GCS: Glasgow coma scale

The distribution of different types of hemorrhagic patterns is shown in Fig. 2. The most common type is Acute Subdural Hematoma (SDH), accounting for 71 patients, which is 47.3% of the cases. Acute Epidural Hematoma affects 6% (9 patients), and Traumatic Subarachnoid Hemorrhage is present in 18% (27 patients). Hemorrhagic Parenchymal Contusion is noted in 4% (6 patients), while isolated skull fractures are relatively rare at 2.7% (4 patients). The percentages provide a clear overview of the prevalence of each condition, highlighting the significance of Acute Subdural Hematoma.

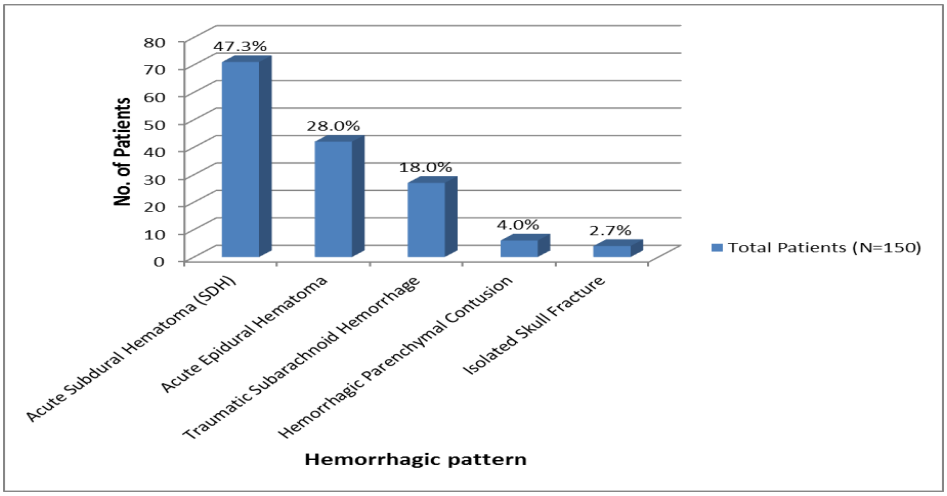

Fig. 2: Types of hemorrhagic patterns observed.

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Fig. 3 shows the timing of PTCI onset after an injury, categorizing patients based on how many weeks post-injury the onset occurred. Out of 21 patients, 33.3% experienced PTCI within the first week after injury. The majority, 42.8%, developed PTCI between 8 days to 2 weeks post-injury. This suggests a significant occurrence in the first couple of weeks following an injury. Additionally, fewer patients experienced PTCI onset between 2 to 3 weeks (9.5%), 3 weeks to 1 month (9.5%), and 1 to 3 months (4.8%) post-injury. This indicates a tapering of cases as time progresses from the point of injury.

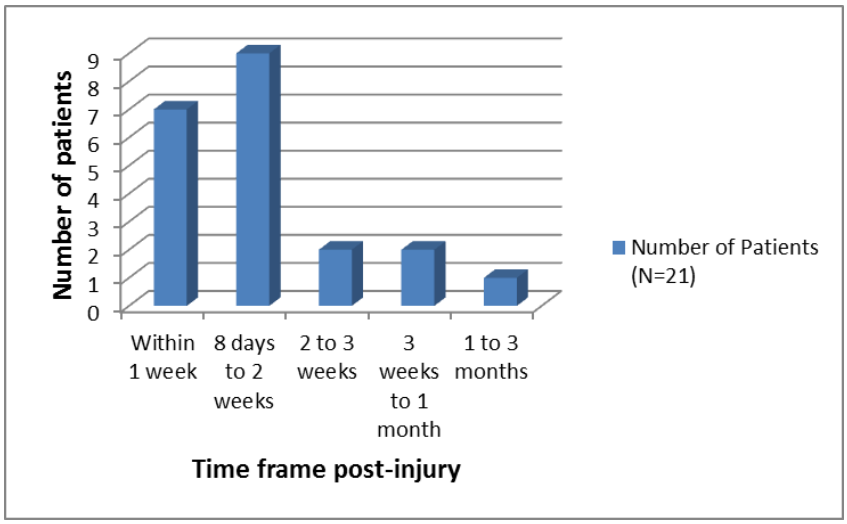

Fig. 3: Timing of PTCI onset post-injury

Table 3 presents mortality rates in patients with and without PTCI, categorized by their admission GCS scores. For patients with GCS scores less than 8, the mortality rate was 80% with PTCI and 57.1% without PTCI, showing statistically significant differences (p=0.04). In the 8-11 GCS group, the mortality rate was 20% with PTCI and 3.1% without, with a p-value of 0.001 indicating significance. However, for patients with GCS scores of 12 or higher, no deaths in either group were observed (0%). These results suggest that PTCI may improve survival in patients with lower GCS scores, but the advantage lessens as the GCS score increases.

Table 3: Mortality rates in patients with and without PTCI according to admission GCS score.

Admission GCS Score	Total patients (N=150)	Patients with PTCI (N)	Mortality rate with PTCI (%)	Patients without PTCI (N)	Mortality rate without PTCI (%)	p-value
< 8	50	15	12 (80%)	35	20 (57.1%)	0.04
8 - 11	70	5	1 (20%)	65	2 (3.1%)	0.001
≥ 12	30	1	0 (0%)	29	0 (0%)	-

PTCI: Post-traumatic cerebral infarction, GCS: Glasgow coma scale

DISCUSSION

For TBI, PTCI is a serious secondary injury that directly contributes to high rates of death and disability [13-16]. According to Tawil and colleagues' study [17], which only included patients with TBI, the death rate for all PTCI patients was as high as 45%, while the incidence of PTCI was 8%. Recent years have shown an increase in the number of cases found, even in individuals with TBI [18].

Decades of research have been conducted on the processes and risk factors of PTCI [19]. PTCI is mostly caused by mechanical compression or displacement of major intracranial arteries [20]. Direct mechanical force applied to the major cerebral arteries can be caused by tentorium, bone fractures, or intracranial hematomas. In the PTCI, this force primarily affects the PCA area, and then the ACA territories [17]. According to some theories,

the arteries were affected by the edge as they passed by the tentorium or falx after an elevated ICP caused a herniation. Depending on whatever area of the artery was affected, the infarction's extent varied [14].

A low GCS score, the presence of subarachnoid hemorrhage (SAH), and certain clinical conditions like hypotensive shock and brain herniation are the most important clinical predictors of PTCI in patients with TBI. Research shows that PTCI rates are higher among those with a GCS score of 3–8 (15.87%) than among those with a score of 9–12 (1.85%) [21]. Furthermore, hematoma features (location and volume), aberrant prothrombin time (PT), pupillary dilatation, and other parameters have been linked to an elevated risk [22]. Both the shock index and mean arterial pressure were found to be important predictors; the shock index was identified as the main predictor of PTCI [23]. When it comes to determining the risk of PTCI and directing clinical treatment for TBI patients, these indicators are crucial.

TBI's secondary effects may be more serious than its primary ones. Following TBI, numerous research has been carried out to investigate the development of relatively common secondary brain ailments, including brain edema, intracranial bleeding, vasospasm, and posttraumatic hydrocephalus [24-28]. Although cerebral infarction following brain trauma has been identified as a possible subsequent damage, there is limited evidence linking cerebral infarction to TBI. We attempted in this investigation to confirm a few of the elements that contribute to the development of PTCI.

PTCI may develop because of thrombo-embolic episodes brought on by damaged vertebral or carotid arteries. Patients who have experienced head and neck trauma may get vertebral artery dissection. One series found that 64% of vertebrobasilar ischemia symptoms and 70% of vertebral artery dissections were accompanied by headaches [29]. Arterial vasospasm is another condition that can cause PTCI. In one investigation, Pasqualin *et al.* [30] found that between 2% and 41% of people had posttraumatic angiographic vasospasm. However, our study's ability to identify the variables influencing vertebral artery dissection about arterial vasospasm is limited by the absence of normal angiographic workup. Our research revealed that among patients with TBI, decompressive craniectomy, brain herniation, lower GCS at admission, and growing age were risk factors for PTCI.

In patients who have experienced a cerebral infarction or trauma, decompressive craniectomy has been commonly used to reduce malignant brain edema by lowering intracranial pressure (ICP) and resistance to cerebrospinal fluid outflow with less constriction of cerebral vessels [31,32]. When compared to medical treatment alone, decompressive craniectomy has long been thought to increase survival rates, enhance functional outcomes, and reduce the risk of infarction in patients with severe cerebral edema [33,34].

CONCLUSIONS

In conclusion, this study highlights the critical predictors of PTCI in patients with TBI, emphasizing the strong correlation between low GCS scores and the incidence of PTCI. The findings reveal that older age, the presence of brain herniation, and the need for decompressive craniectomy are significant risk factors associated with PTCI, which in turn contributes to higher mortality rates. These insights underscore the importance of early assessment and intervention in TBI patients to mitigate the risk of PTCI and improve overall patient outcomes, thereby enhancing clinical management strategies in this vulnerable population. Future research should focus on developing targeted therapeutic interventions and refining predictive models to better identify at-risk patients, ultimately aiming to reduce the incidence of PTCI and improve long-term recovery outcomes.

CONTRIBUTION OF AUTHORS

Research concept- Ankit S. Shah, Nilay Langhnoja
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Supervision- Ankit S. Shah
Materials- Ankit S. Shah, Nilay Langhnoja
Data collection- Ankit S. Shah, Nilay Langhnoja
Data analysis and Interpretation- Ankit S. Shah, Nilay Langhnoja
Literature search- Ankit S. Shah, Nilay Langhnoja
Writing article- Ankit S. Shah, Nilay Langhnoja
Critical review- Ankit S. Shah
Article editing- Ankit S. Shah
Final approval- Ankit S. Shah

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