ARTÍCULO ORIGINAL

IMPACT Score as a Predictor of Mortality in Patients with Severe Traumatic Brain Injury in The Intensive Care Unit

Puntaje IMPACT como Predictor de Mortalidad en Pacientes con Traumatismo Craneoencefálico Severo en la Unidad de Cuidados Intensivos

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Abstract

Objective: To assess whether the IMPACT score predicts mortality in patients with traumatic brain injury (TBI).

Design: Cohort study.

Setting: A total of 260 patients with moderate and severe TBI were enrolled from the Intensive Care Unit (ICU) of Hospital Regional Docente de Trujillo, a trauma referral hospital in northern Peru. The data collection period spanned from 2018 to 2023.

Patients: Patients with moderate and severe TBI admitted to the ICU.

Interventions: Patients with TBI in whom the predictive performance of the IMPACT score for mortality was assessed.

Main Outcome Measure: In-hospital mortality.

Results: A total of 260 patients with moderate and severe TBI were included. When evaluating the IMPACT score and its models (core, extended, and laboratory), we found adequate predictive values expressed by their respective ROC AUCs (0.74, 0.83, and 0.84). Additionally, upon constructing a predictive model utilizing the IMPACT score laboratory model, an increased risk of mortality was observed (adjusted RR 1.21; 95% CI 1.12–1.31). Furthermore, the variables: mechanism of TBI (adjusted RR 5.6; 95% CI 3.03–10.85), Glasgow Coma Scale (GCS) (adjusted RR 1.35; 95% CI 1.15–1.58), and use of vasopressors (adjusted RR 2.2; 95% CI 1.14–4.42) behaved as independent predictors of in-hospital mortality.

Keywords: severe traumatic brain injury, IMPACT score, mortality

Resumen

Objetivo: Evaluar si el puntaje IMPACT predice la mortalidad en pacientes con traumatismo craneoencefálico (TCE).

Diseño: Estudio de cohorte.

Ámbito: Se incluyeron 260 pacientes con TCE moderado y severo ingresados en la Unidad de Cuidados Intensivos (UCI) del Hospital Regional Docente de Trujillo, un hospital de referencia en trauma del norte del Perú. El periodo de recolección de datos abarcó desde 2018 hasta 2023.

Pacientes: Pacientes con TCE moderado y severo ingresados a la UCI.

Intervenciones: Se evaluó el desempeño predictivo del puntaje IMPACT para mortalidad en pacientes con TCE.

Medida de resultado principal: Mortalidad intrahospitalaria.

Resultados: Se incluyeron 260 pacientes con TCE moderado y severo. Al evaluar el puntaje IMPACT y sus modelos (básico, extendido y de laboratorio), se encontraron valores predictivos adecuados expresados por sus respectivas áreas bajo la curva ROC (0.74, 0.83 y 0.84). Además, al construir un modelo predictivo utilizando el modelo de laboratorio del puntaje IMPACT, se observó un mayor riesgo de mortalidad (RR ajustado 1.21; IC 95%: 1.12–1.31). Asimismo, las variables: mecanismo del TCE (RR ajustado 5.6; IC 95%: 3.03–10.85), escala de coma de Glasgow (ECG) (RR ajustado 1.35; IC 95%: 1.15–1.58) y uso de vasopresores (RR ajustado 2.2; IC 95%: 1.14–4.42) se comportaron como predictores independientes de mortalidad intrahospitalaria.

Palabras clave: traumatismo craneoencefálico severo, puntaje IMPACT, mortalidad

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Introduction

Traumatic brain injury (TBI) is defined as an alteration in brain function secondary to a traumatic event, requiring the presence of at least one of the following criteria: impaired level of consciousness, neurophysiological changes, or radiological evidence of skull fracture or intracranial lesions resulting from the trauma, including damage to brain tissue, blood vessels, or the brainstem.[1,2] Its clinical relevance lies in the substantial mortality it presents primarily in-hospital as well as in the significant burden among survivors, who often experience severe disability or a vegetative state. Both outcomes entail high costs for healthcare systems.[3]

Therefore, multiple predictive variables have been investigated to estimate both mortality risk and neurological deterioration among survivors. Mortality in patients with TBI has been associated with clinical variables such as initial Glasgow Coma Scale (GCS) score, pupillary abnormalities, hypoxia, and tracheostomy, as well as biochemical markers including neutrophil-to-lymphocyte ratio (NLR), hyperglycemia, hypernatremia, and hyperuricemia, all of which are also linked to poor neurological outcomes.[4,5]

Numerous predictive models have been developed to estimate both mortality and disability. These include the Abbreviated Injury Scale (AIS), the Marshall CT Classification, and, most notably, the Corticosteroid Randomisation After Significant Head Injury (CRASH) model[6-8] (CRASH) and, most notably, the Corticosteroid Randomisation After Significant Head Injury (CRASH) model and the International Mission for Prognosis and Analysis of Clinical Trials in TBI (IMPACT) model.[9,10] These models have demonstrated adequate prognostic performance in predicting mortality at 14 days (CRASH) and 6 months (IMPACT), as well as neurological disability at 6 months. Although these scales are widely validated in the literature, some reports have identified overestimations in mortality at 3 and 6 months—particularly in the case of CRASH, which originally assessed 14-day mortality but showed high variability in sensitivity. Considering that the datasets used to develop these models are over 20 years old—derived from 8,509 patients across eight randomized clinical trials and three observational studies conducted between 1984 and 1997—and acknowledging the advances in current TBI management, it is necessary to continue evaluating their predictive performance.[11,12] However, there is limited clarity regarding the performance of the IMPACT score in predicting in-hospital mortality within the first 30 days—a critical period during which most TBI-related deaths occur. Although the CRASH model is designed to predict early mortality, its sensitivity has shown considerable variability.[13]

The objective of the present study is to evaluate the IMPACT score as a predictor of in-hospital mortality in patients with moderate to severe traumatic brain injury (TBI) admitted to the Intensive Care Unit (ICU) of a trauma referral hospital in a city in northern Peru.

Materials and methods

Study Design and Population: A retrospective cohort study was conducted. Medical records of patients with moderate to severe TBI admitted to the Intensive Care Unit (ICU) of the Hospital Regional Docente de Trujillo (HRDT), located in Trujillo, Peru—a regional trauma referral center—were reviewed. The ICU is a polyvalent unit with 13 beds that manages multisystem trauma patients across the region. A total of 260 patients treated between January 2018 and August 2023 were included. Patients were admitted from the emergency department, where they initially received hospital care. If a neurosurgical intervention was indicated following evaluation by a neurosurgeon, it was performed before ICU admission; otherwise, patients were directly admitted. The unit handles approximately 180 patients per year, of whom about 30% are diagnosed with TBI.

Population and Sample: The study population included all patients with moderate and severe TBI admitted to the ICU of HRDT during the study period. Inclusion criteria were patients aged 18 years or older, of either sex, admitted with moderate or severe TBI. Exclusion criteria included patients who died within the first 24 hours of hospitalization and pregnant patients. The sample size calculation was based on the study by Camarano et al.,[14] which reported a sensitivity of 80.1% and a specificity of 77.9%, with a 5% precision and a 95% confidence level. Based on these parameters, a minimum of 246 patients with moderate to severe TBI was required; therefore, all eligible patients within the specified study period were included.

Data and Variables: A database was created in the ICU of HRDT including all patients admitted from January 2018 to August 2023. Data collected included clinical variables, biochemical laboratory parameters, ICU length of stay, and mortality outcomes. The primary outcome variable was in-hospital mortality, measured within 30 days of ICU admission. The exposure variable was the IMPACT score, categorized as high or low risk, based on its three models: core, extended, and laboratory. The IMPACT score comprises variables such as age, motor score, and pupillary reactivity (core model); hypoxia, hypotension, Marshall CT classification, subarachnoid hemorrhage, and epidural hematoma (core + CT); and laboratory tests such as glucose and hemoglobin measured within the first 48 to 72 hours of ICU admission (laboratory model).

The main covariates considered potential confounders for in-hospital mortality in TBI patients included: age, sex, serum sodium, serum potassium, associated trauma (none if the patient had isolated TBI; closed trauma + fractures, referring to thoracic and/or abdominal closed trauma with extra-

cranial fractures; fractures alone; and closed trauma referring to isolated thoracoabdominal trauma), trauma mechanism (primarily direct mechanisms such as impact, including traffic accidents, falls, and assaults), GCS score, TBI severity, systolic blood pressure, use of vasopressors, emergency surgical intervention, need for and duration of mechanical ventilation, blood glucose, and hemoglobin. These values were recorded upon admission to the ICU.[4,15,16]

Statistical Analysis: Data were organized using Excel 2019 and analyzed with SPSS® version 28. For numerical variables, measures of central tendency such as median and interquartile range (p25–p75) were calculated, as the distribution was non-normal according to the Kolmogorov-Smirnov test. Categorical variables were summarized using frequencies and percentages. The association between the exposure variable and intervening variables with the outcome variable (in-hospital mortality) was assessed using the Chi-square test, with statistical significance defined as $p < 0.05$. Crude relative risks (cRR) were calculated in the bivariate analysis. Variables showing statistical significance ($p < 0.05$) were included in multivariate predictive models using generalized linear models with Poisson distribution and robust variance to estimate adjusted relative risks (aRR). The performance of the predictive models was assessed using the ROC curve. In addition, the IMPACT score and its models (core, extended, and laboratory) were evaluated for sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) in predicting in-hospital mortality.

Ethical Considerations: The research protocol was reviewed and approved by the Research Committee of the Faculty of Medicine at Universidad Privada Antenor Orrego (UPAO) (Resolution No. 0852-2023-UPAO) and by the Research Ethics Committee of Hospital Regional Docente de Trujillo.

Results

A total of 287 medical records were reviewed, of which 27 were excluded due to missing data. Ultimately, 260 patients were included. The cohort showed an in-hospital mortality rate of 16% (41 patients) within the first 30 days. The deceased patients had a slightly higher median age than the survivors, though the difference was not statistically significant (48 vs. 39.5 years; $p > 0.05$). Male patients predominated over females in both the deceased and surviving groups (80.5% and 81.3%, respectively), with no significant difference in mortality by sex.

Regarding associated trauma in patients with TBI, there were statistically significant differences between deceased and surviving patients in terms of the type of associated trauma and the mechanism of TBI ($p < 0.001$ for both). Importantly, the deceased group had a lower median Glasgow Coma Scale (GCS) score at admission compared to survivors (7 vs. 9 points; $p < 0.001$), which was consistent with the severity of TBI.

Among deceased patients, 80.5% required mechanical ventilation (MV), compared to 55.3% of survivors ($p < 0.05$). However, although the median duration of MV was longer in the deceased group, the difference was not statistically significant ($p > 0.05$). Additionally, 61% of the deceased required vasopressors, compared to only 12.3% of survivors—a statistically significant difference for mortality ($p < 0.001$).

Among the laboratory variables evaluated, serum sodium, admission glucose, and hemoglobin levels were associated with mortality ($p < 0.05$). Deceased patients had lower median systolic blood pressure (SBP) at admission compared to survivors ($p < 0.05$). No significant association was found between serum potassium and mortality ($p > 0.05$) (Table 1).

Table 1. General Characteristics and Bivariate Analysis of Clinical and Laboratory Factors Associated with Mortality in Patients with TBI.

	Mortality			
	Yes	No		
	41 (%)	219 (%)	RR (IC95%)	p-value
Age (years)	48 [28 – 69]	39.5 [28 – 54]	–	0.361*
18-39	16 (39%)	98 (44.7%)		
40-60	10 (24.4%)	71 (32.4%)	–	0.166**
>60	15 (36.6%)	50 (22.8%)		
Sex				
Male	33 (80.5%)	178 (81.3%)	0.96 (0.41-2.21)	0.905
Female	8 (19.5%)	41 (18.7%)		
Associated injuries				
None	8 (19.5%)	131 (59.8%)		
Closed + fractures	13 (31.7%)	9 (4.1%)	–	
Fractures	17 (41.5%)	60 (27.4%)		<0.001**
Closed	3 (7.3%)	19 (8.7%)		
TBI mechanism				
Traffic accident	32 (78%)	35 (16%)	–	
Assault	4 (9.8%)	43 (19.6%)		<0.001**
Fall	5 (12.2%)	141 (64.4%)		
GCS (puntos)	7 [6 – 9]	9 [6.8 – 11]	–	<0.001*
TBI severity				
Severe	29 (70.7%)	76 (34.7%)	3.57 (1.91-6.67)	<0.001
Moderate	12 (29.3%)	143 (65.3%)		
Need of MV				
Yes	33 (80.5%)	121 (55.3%)	2.84 (1.37-5.9)	0.004
No	8 (19.5%)	98 (44.7%)		
VM days	7.5 [5 – 15]	6 [5 – 8]	–	0.130*
Vasopressors				
Yes	25 (61%)	27 (12.3%)	6.25 (3.61-10.8)	<0.001
No	16 (39%)	192 (87.7%)		
Emergency surgery				
Yes	17 (41.5%)	84 (38.4%)	1.15 (0.63-1.97)	0.708
No	24 (58.5%)	135 (61.6%)		
Sodium (mEq/L)	144 [137 – 150]	140 [136 – 146]	Not applicable	0.002*
SBP (mmHg)	100 [90 – 120]	110 [94 – 130]	Not applicable	0.027*
Glucose (mg/dl)	164 [128 – 182]	126 [104 – 148]	Not applicable	<0.001*
Hemoglobin (g/dl)	11.3 [9.3 – 13.3]	12.2 [10 – 13.5]	Not applicable	0.025*
Potassium (mEq/L)	3.6 [3.3 – 3.9]	3.6 [3.3 – 4]	No aplica	0.205*

GCS, Glasgow coma score; SBP, systolic blood pressure; MV, mechanical ventilation.
*U de Mann-Whitney, median [P_{25}-P_{75}].

When predictive models were constructed using the significant variables from Table 1 (Models 1 and 2), incorporating the laboratory IMPACT model, we found that the variables independently predicting in-hospital mortality were: the laboratory IMPACT score (aRR 1.21; 95% CI: 3.03–10.85; $p < 0.001$), trauma mechanism due to traffic accident (aRR 5.6; 95% CI: 1.15–1.58; $p < 0.001$), admission GCS score (aRR 1.35; 95% CI: 1.15–1.58; $p < 0.001$), and the need for vasopressors (aRR 2.2; 95% CI: 1.14–4.42; $p < 0.05$) (Table 2).

The total IMPACT score showed higher median values in the deceased group compared to survivors across all three models ($p < 0.001$) (Table 3). Furthermore, when evaluating the IMPACT score and its models (core, extended, and laboratory), all demonstrated adequate predictive performance for in-hospital mortality. However, the laboratory model showed the highest performance (sensitivity: 85%; AUC ROC: 0.844; $p < 0.001$) (Table 4 and Figure 1).

Discussion

In our study, when evaluating the IMPACT score as a predictor of in-hospital mortality, we found that the core, extended, and laboratory models all demonstrated adequate predictive performance in patients with TBI. The laboratory model, in particular, was associated with a 21% increased risk of in-hospital mortality (aRR 1.21; 95% CI: 1.12–1.31), with an AUC ROC of 0.84. Additionally, the variables trauma mechanism (aRR 5.6; 95% CI: 3.03–

Table 2. Predictive Models of In-Hospital Mortality from Factors Associated with Mortality in Patients with TBI.

	Model 1		Model 2	
Variables	**aRR (IC 95%)**	**p-value**	**aRR (IC 95%)**	**p-value**
IMPACT Laboratory Model	1.27 (1.15 a 1.40)	< 0.001	1.21 (1.12 a 1.31)	< 0.001
Age	0.99 (0.97 a 1.003)	> 0.05	-	-
Sex				
Female	Referencia		-	-
Male	0.77 (0.36 a 1.52)	> 0.05	-	-
Associated injuries				
None	Referencia		-	-
1 or more	1.12 (0.85 a 1.47)	> 0.05	1.11 (0.86 a 1.44)	> 0.05
TBI mechanism				
None traffic accident	Referencia	-	Referencia	-
Traffic accident	5.17 (2.7 a 10.4)	< 0.001	5.6 (3.03 a 10.85)	< 0.001
GCS	1.51 (1.18 a 1.97)	< 0.01	1.35 (1.15 a 1.58)	< 0.001
TBI severity	1.4 (0.38 a 5.25)	> 0.05	-	-
Need of MV	0.79 (0.32 a 1.91)	> 0.05	0.91 (0.4 a 2.05)	> 0.05
Vasopressors	2.52 (1.25 a 5.35)	< 0.05	2.2 (1.14 a 4.42)	< 0.05
Sodium (mmol/l)	1.004 (0.98 a 1.03)	> 0.05	1.01 (0.99 a 1.03)	> 0.05
SBP (mmHg)	1.006 (0.99 a 1.014)	> 0.05	1.003 (0.99 a 1.01)	> 0.05
Glucose	1.000 (0.99 a 1.004)	> 0.05	1.001 (0.99 a 1.004)	> 0.05
Hemoglobin	1.03 (0.92 a 1.16)	> 0.05	1.02 (0.92 a 1.13)	> 0.05

Adjusted Relative Risk (aRR): including the variables IMPACT score, associated trauma, TBI mechanism, TBI severity, use of vasopressors, need for mechanical ventilation, Sodium (mEq/L), GCS (points), Systolic Blood Pressure (mmHg), Hemoglobin (g/dL), and Glucose (mg/dL).
Model 1: AUC value: 0.93; 95% CI of AUC: 0.89 to 0.97.
Model 2: AUC value: 0.92; 95% CI of AUC: 0.87 to 0.97.

Table 3. Comparison of IMPACT Score Model Values According to In-Hospital Mortality Outcome.

	Mortality		
	Yes 41 (%)	**No 219 (%)**	**p-value**
IMPACT Score			
Central	8 [4 – 11]	4 [2 – 7]	<0.001*
Extended	11 [6 – 16]	5 [3 – 9]	<0.001*
Laboratorial	14 [10 – 19]	8 [6 – 11.3]	<0.001*

*U de Mann-Whitney, median [P_{25} – P_{75}].

Table 4. Predictive performance of the IMPACT score for TBI mortality.

IMPACT	**Cut-off point**	**AUC**	**p-value**	**Se**	**Sp**	**PPV**	**NPV**
Central	5.5	0.741	<0.001	71%	73%	33%	93%
Extended	9.5	0.832	<0.001	66%	86%	47%	93%
Laboratorial	11.5	0.844	<0.001	85%	70%	35%	96%

Se: Sensitivity, Sp: Specificity, PPV: Positive Predictive Value, NPV: Negative Predictive Value.

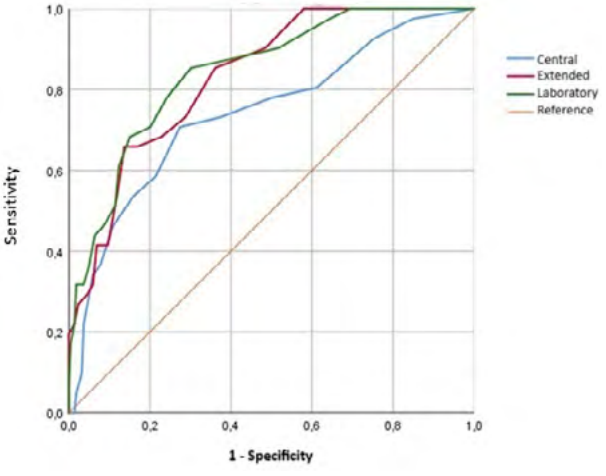

Figure 1. ROC Curves of the IMPACT Score and Its Models.

10.85), need for vasopressors (aRR 2.2; 95% CI: 1.14–4.42), and Glasgow Coma Scale score (aRR 1.35; 95% CI: 1.15–1.58) were identified as independent predictors of in-hospital mortality in patients with TBI.

The IMPACT score was developed primarily from data collected in eight randomized controlled trials (RCTs), and it was originally designed and extensively validated as a predictor of 6-month mortality and poor neurological outcomes. However, few studies have attempted to assess its performance specifically in predicting in-hospital mortality in TBI patients—a domain in which the CRASH model has traditionally been used, particularly for evaluating 14-day mortality.[17-19]

Roozenbeek et al.[20] evaluated the predictive role of the IMPACT score for in-hospital mortality within the first 14 days in patients with severe TBI. They used data from the Trauma Brain Foundation (TBI-trac) in New York, which included 3,125 patients admitted between 2000 and 2009. The reported 14-day TBI-related mortality rate was 23%. The study found that the IMPACT score demonstrated good discriminatory ability for mortality, with AUC ROC values of 0.79 and 0.83 for the core and extended models, respectively.

Similar results were obtained in our study with the IMPACT score and its models. The laboratory model, in particular, demonstrated a sensitivity and specificity of 85% and 70%, respectively, and had the highest AUC compared to the other models (AUC ROC: 0.84). While we used the original version of the IMPACT score, in contrast to Roozenbeek et al.[20] who made modifications to the laboratory model due to the unavailability of cranial CT data necessary for the Marshall classification. Instead, they used midline shift distance and the status of the basal cisterns as substitutes, thereby reformulating the original values of the score. Other studies have also confirmed the predictive value of the IMPACT score for mortality, though measured at 6 months, which aligns with the original purpose of the score—to predict adverse outcomes (mortality and neurological impairment) at 6 months. The contribution of our study lies in demonstrating that this same version of the IMPACT score is also useful for predicting in-hospital mortality, as evidenced by our results.

Some studies on the IMPACT score and its ability to predict 6-month mortality include, for example, the work by Rostami et al.[21] who found that the AUC ROC for mortality using the basic IMPACT model was 0.85, with no significant improvement observed in the extended model, but a modest increase in the laboratory model (AUC ROC: 0.89). On the other hand, Wongchareon et al.[10] conducted a cohort study enrolling 127 patients and found that the AUC ROC values for the IMPACT score's basic, extended, and laboratory models in predicting 6-month mortality were 0.683, 0.726, and 0.731, respectively. These results demonstrated improved outcome discrimination as the complexity of the model increased. Similarly, Moorthy et al.[22] conducted a prospective study involving 104 patients with TBI, in which they evaluated the IMPACT, CRASH, and TRISS scores. They found that the best cutoff points and discriminatory performance for 6-month mortality using the IMPACT score were: core model > 38 points (AUC ROC: 0.976), extended model > 27 points (AUC ROC: 0.968), and laboratory model > 41 points (AUC ROC: 0.954).

The strong performance and discriminatory capacity of the IMPACT score, particularly its laboratory model, in predicting mortality in TBI is largely determined by the parameters included in the model, such as age, motor and pupillary scores, hemodynamic status, severity of brain injury, and laboratory values like glucose and hemoglobin. All of these variables have been independently associated with the prognosis of severe TBI.[23] EIt is important to highlight that these variables are often interrelated. As the severity of TBI increases, clinical findings such as altered consciousness and the extent of brain damage tend to worsen, with lesions such as subarachnoid hemorrhage (SAH) and epidural hematoma (EH) frequently observed. Moreover, severe TBI typically results from high-impact mechanisms, which are often associated with additional traumatic injuries or polytrauma, leading to hemodynamic compromise. This can manifest as arterial hypotension and decreased hemoglobin due to active bleeding, stress-induced hyperglycemia, and hypoxic states.[4,24]

On the other hand, we found that certain variables—such as the mechanism of TBI, GCS score, and the use of vasopressors—were independent predictors of in-hospital mortality. Specifically, the presence of closed trauma and fractures associated with TBI was significantly related to in-hospital mortality, with a considerable increase in risk (aRR 3.3; $p < 0.01$). Traffic accidents were the most frequent trauma mechanism and were associated with a higher risk of mortality compared to patients without this mechanism (aRR 7.22; $p < 0.001$). Tsai et al. found that TBI associated with skull fracture increased the risk of in-hospital death by 80% (aOR 1.8; $p < 0.001$), although they did not observe significant differences in mortality based on the trauma mechanism.[25] In other study by Paul et al.[26] conducted in a retrospective cohort of 4,508 patients with moderate to severe TBI, it was observed that patients with associated injuries, such as femoral fractures, had higher in-hospital mortality ($p = 0.037$). Additionally, it was noted that traffic accidents were the primary mechanism of injury among patients with associated fractures ($p < 0.001$).

Additionally, the GCS level at admission has been shown to be an important predictor of mortality. Vásquez-Tirado et al.[4] In a study on factors associated with in-hospital mortality in moderate to severe TBI, a cohort of 238 patients with a mortality prevalence of 10% was analyzed.

The study found that GCS was associated with mortality, with each point above a GCS score of 8 at admission decreasing the risk of death by 18% (aHR 0.81; 95% CI: 0.69–0.94; $p < 0.01$). Additionally, abnormal pupillary responses such as anisocoria (aHR 2.9; 95% CI: 1.2–7.6; $p < 0.05$), and the presence of shock on admission—reflected by the need for vasopressors (aHR 6.6; 95% CI: 2.2–19.9; $p < 0.001$)—were also identified as independent predictors of in-hospital mortality. The same group, in another study involving 100 patients with severe TBI[15] imed to evaluate factors associated with mortality, found that hypernatremia was associated with a significantly increased risk of death (aOR 16.7; 95% CI: 1.96–142.8), along with the SOFA score (aOR 2.8; 95% CI: 1.79–4.6). Similar to our findings, admission GCS proved to be an important prognostic marker. However, although we observed that the mortality group had a higher mean sodium level compared to survivors, the difference was not statistically significant.

When evaluating an important variable such as blood glucose, we found that the median value among deceased patients was significantly higher than that of survivors. However, this association lost statistical significance in the regression models. In contrast, Demlie et al.[27] in a retrospective cohort of patients with moderate to severe TBI, hyperglycemia was identified as an independent predictor of in-hospital mortality (aHR: 2.28, $p = 0.020$). Similarly, Tsai et al.[28,29] observed that patients who developed stress-induced hyperglycemia had a significantly higher risk of mortality following TBI (OR: 3.25, $p = 0.039$).

Our study presents several strengths. First, although the IMPACT score has an undisputed prognostic value for predicting mortality and disability, it was originally designed to assess outcomes at 6 months. There are very few reports evaluating its utility for in-hospital mortality, a role typically attributed to the CRASH score. However, the broader and more comprehensive performance of the IMPACT model compared to CRASH led us to evaluate its ability to predict in-hospital mortality. Second, we analyzed a cohort of patients who received protocolized treatment for TBI, incorporating multiple neuroprotection strategies. Third, we applied appropriate statistical methods, including the construction of predictive models in which the IMPACT score and its components were integrated with new significant variables to enhance prognostic performance, as reflected by the AUC ROC and AIC values of the models.

Our study has several limitations. First, a considerable number of patients were excluded due to missing data in their medical records. Second, we evaluated patients with both moderate and severe TBI; however, since some individuals were initially admitted with moderate TBI and later progressed to severe forms, we were unable to assess the IMPACT score and its models according to TBI severity categories. Third, prehospital care—particularly for patients referred from remote healthcare centers—was not uniform across the cohort, potentially delaying the initiation of neuroprotective measures. Similarly, limited availability of timely neurosurgical intervention may have led to delays in surgical management for cases requiring it, thereby worsening outcomes. Fourth, approximately one-fourth of the cohort was treated during the COVID-19 pandemic period, during which our ICU was primarily dedicated to the care of patients with that condition, potentially leading to delays in management.

Conclusion

The IMPACT score and its models serve as predictors of in-hospital mortality in patients with traumatic brain injury. Additionally, trauma mechanism, Glasgow Coma Scale score, and the need for vasopressor support act as independent predictive variables.

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