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Predictors of In-Hospital Mortality in Children with Severe Traumatic Brain Injury in a Middle-Income Country: A 5-Year Retrospective Cohort Study

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Predictors of In-Hospital Mortality in Children with Severe Traumatic Brain Injury in a Middle-Income Country: A 5-Year Retrospective Cohort Study

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Predictors of In-Hospital Mortality in Children with Severe Traumatic Brain Injury in a Middle-Income Country: A 5-Year Retrospective Cohort Study

Abstract

Purpose: Severe traumatic brain injury (sTBI) is a leading cause of death and disability in children worldwide. Although guidelines emphasize the prevention of secondary insults, data on sTBI outcomes and prognostic factors remain scarce in low- and middle-income countries (LMICs). We aimed to identify the predictors of in-hospital mortality among children with sTBI admitted to the pediatric intensive care unit (PICU) of a tertiary referral center in an LMIC.

Methods: We conducted a 5-year retrospective single-center cohort study (January 2019–December 2023). Children aged <15 years admitted with sTBI (post-resuscitation Glasgow Coma Scale [GCS] ≤8) were included. Variables with p <0.20 in univariate analysis were entered into a multivariable logistic regression model to identify predictors of in-hospital mortality.

Results: Of 279 children admitted with traumatic brain injury, 188 had sTBI and 114 with complete records were analyzed. Mean age was 6.6 ± 3.9 years, 77% were boys, and road traffic accidents were the main mechanism (54.4%). In-hospital mortality was 17.5% (20/114). In the exploratory multivariable model, each one-point increase in admission GCS was associated with lower odds of death (odds ratio [OR] 0.82, 95% confidence interval [CI] 0.70–0.96). Other independent predictors were bilateral fixed mydriasis (OR 3.85, 95% CI 1.17–12.65), hemodynamic instability at admission (OR 5.40, 95% CI 2.50–11.60), vasoactive drug requirement (OR 2.10, 95% CI 1.03–4.32), prothrombin time <70% (OR 2.90, 95% CI 1.40–6.02), thrombocytopenia (OR 1.95, 95% CI 1.03–3.68) and hyponatremia (OR 2.30, 95% CI 1.10–4.84).

Conclusion: In this LMIC cohort, in-hospital mortality after pediatric sTBI was associated with both initial neurological severity and early systemic disturbances at admission, mainly hemodynamic instability, coagulation abnormalities and hyponatremia. These readily available parameters may help identify high-risk children early, and prospective multicenter studies are needed to confirm these findings.

Keywords: severe traumatic brain injury; children; in-hospital mortality; prognostic factors; pediatric intensive care; Glasgow Coma Scale; coagulopathy; low- and middle-income countries

What is Known:

• Severe traumatic brain injury is a leading cause of death and disability in children, and guidelines emphasize the prevention of secondary insults.

• Data on outcomes and prognostic factors remain scarce in low- and middle-income countries.

What is New:

• In this middle-income country cohort, in-hospital mortality after pediatric severe traumatic brain injury was 17.5%.

• Besides admission GCS and bilateral fixed mydriasis, hemodynamic instability, coagulation abnormalities and hyponatremia at admission were associated with mortality.

Abbreviations

CI, confidence interval; CT, computed tomography; GCS, Glasgow Coma Scale; ICP, intracranial pressure; IQR, interquartile range; LMICs, low- and middle-income countries; OR, odds ratio; PaO_2, arterial partial pressure of oxygen; PICU, pediatric intensive care unit; POCUS, point-of-care ultrasound; PT, prothrombin time; SAMU, Service d'Aide Médicale Urgente (Emergency Medical

Service); SD, standard deviation; SSBIs, secondary systemic brain insults; sTBI, severe traumatic brain injury; STROBE, Strengthening the Reporting of Observational Studies in Epidemiology; TBI, traumatic brain injury; TCD, transcranial Doppler

INTRODUCTION: Traumatic brain injury (TBI) is a major cause of mortality and long-term disability in children [1]. Severe TBI (sTBI) in children is associated with substantial morbidity and mortality, resulting from both the primary injury and secondary brain insults occurring during the prehospital and in-hospital phases. Pediatric patients have distinct anatomical and physiological characteristics**,** including incomplete myelination, greater cranial compliance, and age-related differences in cerebral autoregulation, which may influence the response to injury and clinical outcomes [2].

Although prognostic factors for pediatric sTBI have been extensively investigated in high-income countries, data from low- and middle-income countries (LMICs) remain limited [3]. Variations in prehospital care, intensive care resources, neuromonitoring, and access to pediatric neurosurgical expertise may contribute to differences in outcomes. Identifying locally relevant prognostic factors is therefore important for improving risk stratification and optimizing the management of children with sTBI in resource-limited settings.

This study aimed to identify the predictors of in-hospital mortality among children with sTBI admitted to the pediatric intensive care unit (PICU) of a tertiary referral center in an LMIC over a five-year period.

METHODS:

Study design and setting: We conducted a retrospective, single-center, observational cohort study in the PICU of a tertiary referral center from January 2019 to December 2023. The study was reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines.

Study population and eligibility:

Inclusion criteria: All children younger than 15 years admitted to the PICU with sTBI were eligible for inclusion. We defined sTBI as a post-resuscitation Glasgow Coma Scale (GCS) score of 8 or lower, assessed after airway stabilization and initial resuscitation. Children with sTBI were included regardless of the presence of associated extracranial injuries.

Exclusion criteria: Patients were excluded from the analysis if they met one or more of the following criteria:

- Death within the first 24 hours of admission due to causes unrelated to TBI.
- incomplete medical records precluding reliable statistical analysis.

Data collection and variables:

Data were systematically extracted from hospital records using a standardized case report form. Variables were collected in four domains:

- Baseline demographics and prehospital characteristics: age, sex, weight, relevant medical history, accident setting, mechanism of injury, transport mode and time to management.

- Clinical status at admission: post-resuscitation GCS score, pupillary status (normal, anisocoria or bilateral fixed mydriasis), post-traumatic seizures and baseline respiratory and hemodynamic status.
- Secondary systemic brain insults (SSBIs) and laboratory variables: SSBIs were defined using prespecified criteria: hemodynamic instability, hypoxemia (PaO_2 < 60 mmHg), hyperthermia (> 38.5°C), thrombocytopenia (platelet count < 150,000/mm^3), reduced prothrombin activity (PT < 70%) and hyponatremia (serum sodium < 135 mEq/L). Vasoactive drug requirement was defined as the need for a norepinephrine infusion at any time during the PICU stay, started for hypotension not responding to the initial fluid bolus or for pathological transcranial Doppler findings.
- Hospital course and outcomes: extracranial injuries, admission and follow-up computed tomography (CT) findings, vasoactive drug requirement, PICU complications, PICU length of stay and vital status at hospital discharge.

Management: Patient management followed an institutional pediatric neurocritical care protocol adapted from the Brain Trauma Foundation guidelines [3], with the primary objective of preventing and correcting secondary brain insults. Initial management included head-of-bed elevation at 30° with the head kept in a neutral midline position, maintenance fluids with normal saline, early enteral nutrition, and prevention or treatment of seizures when indicated. Hemodynamic support consisted of an initial fluid bolus. Norepinephrine was started if hypotension did not respond to the initial fluid bolus or if transcranial Doppler (TCD) findings were pathological, and was weaned progressively, each dose reduction being conditional on TCD findings remaining normal. Patients received analgesia and deep sedation using opioids and benzodiazepines, as appropriate. Hyperosmolar therapy with 3% hypertonic saline was administered in cases of cerebral edema, significant mass effect, midline shift, or clinical and radiological signs of impending cerebral herniation.

Standard monitoring included continuous electrocardiography, pulse oximetry, invasive arterial blood pressure, urine output, core temperature, point-of-care glucose and serial laboratory tests, with arterial blood gases and lactate measured systematically, serum electrolytes including sodium, complete blood count and coagulation tests, corresponding to the SSBIs defined above. Invasive intracranial pressure monitoring was not available in the unit; neuromonitoring relied on TCD performed several times daily, with repeat brain CT every 48–72 hours or in case of neurological deterioration. Hemodynamic assessment used point-of-care ultrasound (POCUS) when clinically indicated. Neurosurgical consultation was requested in case of CT findings suggestive of a surgically treatable intracranial lesion or neurological deterioration.

Outcomes: The primary outcome was all-cause in-hospital mortality. Secondary outcomes were PICU length of stay and the incidence of nosocomial infections (systemic infection, meningitis, septic shock, healthcare-associated pneumonia and catheter-related bloodstream infection).

Statistical analysis: Data distribution was assessed using the Shapiro–Wilk test. Continuous variables were presented as mean ± standard deviation (SD) or median and interquartile range (IQR), as appropriate. Categorical variables were expressed as frequencies and percentages. Comparisons between survivors and non-survivors were performed using Student's t-test or the Mann–Whitney U test for continuous variables and the chi-square or Fisher's exact test for categorical variables, as appropriate. Variables with a p-value <0.20 in univariate analysis were entered into an exploratory multivariable logistic regression model to identify independent predictors of in-hospital mortality. Results were reported as odds ratios (ORs) with 95% confidence intervals (CIs)**.** A two-sided p-value <0.05 was considered statistically significant. Statistical analyses were performed using IBM SPSS Statistics version 26.0 (IBM Corp., Armonk, NY, USA).

RESULTS

Study population and baseline characteristics: The patient screening, eligibility assessment and inclusion process are presented in Figure 1. Among 279 children admitted with TBI during the 5-year study period**,** 91 had mild-to-moderate TBI (post-resuscitation GCS >8) and were not eligible. Of the remaining 188 children with sTBI, 74 were excluded because of incomplete clinical or laboratory records, leaving 114 children in the final analysis.

Baseline characteristics are summarized in **Table 1**. Mean age was 6.6 ± 3.9 years (range, 1 month to 14 years). There were 88 boys (77.2%) and 26 girls (22.8%), a male-to-female ratio of 3.4:1. Road traffic accidents were the leading mechanism of injury (n = 62, 54.4%), followed by domestic accidents (n = 44, 38.6%). Most patients were transported by the Emergency Medical Service (SAMU).

Associated extracranial injuries were present in 54 children (47.4%).

Initial neurological severity and neuroimaging findings: Neurological severity at PICU admission was greater among non-survivors than survivors (Table 2). The mean ± SD admission GCS score was lower in non-survivors than in survivors (4.9 ± 1.9 vs. 6.1 ± 2.1; p = 0.018), and 32 children (28.1%) had a GCS score of 3–5. Bilateral fixed mydriasis was significantly more frequent among non-survivors than survivors (10/20 [50.0%] vs. 4/94 [4.3%]; p < 0.001). Post-traumatic seizures occurred in 41 children (36.0%), with no significant difference between groups (36.2% vs. 35.0%).

On brain CT, diffuse cerebral edema (70.0% vs. 23.4%) and signs of cerebral herniation (45.0% vs. 3.2%) were significantly more frequent among non-survivors than survivors (both p < 0.001). Focal

hematoma (55.0% vs. 37.2%) and extradural hematoma (5.0% vs. 22.3%) did not differ significantly between groups.

Secondary systemic brain insults at admission: Secondary systemic brain insults (SSBIs) were frequent at admission (Table 3). Hemodynamic instability was significantly more frequent among non-survivors than survivors (65.0% vs. 23.4%; p = 0.001). Coagulation abnormalities were also more frequent among non-survivors: a low prothrombin time (PT < 70%) was found in 70.0% vs. 28.7% of survivors (p < 0.001), and thrombocytopenia (< 150,000/mm³) in 40.0% vs. 11.7% (p = 0.003). Among metabolic disturbances, admission hyponatremia (< 135 mEq/L) was significantly more frequent among non-survivors than survivors (30.0% vs. 8.5%; p = 0.012). In contrast, hypoxemia (10.0% vs. 9.6%) and hyperthermia (15.0% vs. 10.6%) did not differ significantly between groups.

Outcomes: In-hospital mortality was 17.5% (20/114); 94 children (82.5%) survived to hospital discharge. The mean ± SD PICU length of stay was 11.3 ± 8.5 days (range, 2–53). Nosocomial infections included systemic infection (n = 15, 13.2%), meningitis (n = 12, 10.5%), septic shock (n = 10, 8.8%), healthcare-associated pneumonia (n = 9, 7.9%) and catheter-related bloodstream infection (n = 9, 7.9%). These events occurred after admission and were not included as predictors in the multivariable analysis.

Predictors of in-hospital mortality: In the exploratory multivariable logistic regression model (Table 4), hemodynamic instability at admission had the highest odds ratio (OR 5.40, 95% CI 2.50–11.60; p < 0.001). Lower admission GCS (OR 0.82 per one-point increase, 95% CI 0.70–0.96; p = 0.015) and bilateral fixed mydriasis (OR 3.85, 95% CI 1.17–12.65; p = 0.027) remained independently associated with mortality. Among the other secondary systemic brain insults, a reduced PT (< 70%) (OR 2.90, 95% CI 1.40–6.02; p = 0.004), thrombocytopenia (OR 1.95, 95% CI

1.03–3.68; p = 0.040) and hyponatremia (OR 2.30, 95% CI 1.10–4.84; p = 0.027) were independently associated with mortality, as was vasoactive drug requirement (OR 2.10, 95% CI 1.03–4.32; p = 0.041). Each additional day of PICU stay was associated with lower odds of death (OR 0.84, 95% CI 0.75–0.95; p = 0.005).

DISCUSSION:

In this five-year retrospective cohort of 114 children with severe traumatic brain injury (sTBI) admitted to a tertiary pediatric intensive care unit (PICU), in-hospital mortality was 17.5% (20/114). In an exploratory multivariable analysis, lower admission Glasgow Coma Scale (GCS) score, bilateral fixed mydriasis, hemodynamic instability, reduced prothrombin activity, thrombocytopenia, and hyponatremia at admission were associated with mortality. Vasoactive drug requirement during the PICU stay was also associated with death, but was not a variable available at admission. These findings emphasize the prognostic relevance of early neurological assessment and systemic disturbances, although the adjusted estimates require cautious interpretation.

Mortality in low- and middle-income and high-income countries

Our mortality rate was close to the 17% and 15% reported in Colombian and US centers treating pediatric sTBI, respectively [4], and lower than the 22% reported in a French pediatric trauma center [5]. In a multicenter PICU study of moderate-to-severe pediatric TBI in Asia and Latin America, mortality did not differ significantly between low- and middle-income country (LMIC) and non-LMIC sites (9.6% vs. 12.9%), although functional outcomes were poorer in LMICs [6]. A systematic review including pediatric TBI of all severities in LMICs reported a median case-fatality rate of 7.3%, with high-quality data available from fewer than one-quarter of LMICs [7]. These results are

not directly comparable because of differences in injury severity, case mix, inclusion criteria, and timing of outcome assessment.

Prognostic factors identified

Admission GCS and pupillary status were the principal neurological markers associated with mortality in our cohort. This accords with previous TBI prognostic studies, including the model developed by Signorini et al. and the IMPACT analysis, although these largely involved adult patients [8,9]. Bilateral fixed mydriasis may reflect severe intracranial injury and brainstem dysfunction. In univariate analysis, diffuse cerebral edema and signs of herniation were more frequent among non-survivors, whereas focal and extradural hematomas did not differ significantly. In our unit, transcranial Doppler (TCD) was used for bedside neuromonitoring because invasive intracranial pressure (ICP) monitoring was unavailable; however, TCD cannot be considered a reliable substitute for direct ICP measurement [10,11].

Hemodynamic instability at admission had the highest estimated odds ratio in our exploratory model (OR 5.40, 95% CI 2.50–11.60). Early circulatory collapse and hypotension have similarly been linked to adverse outcomes after pediatric sTBI [5,12], potentially through reduced cerebral perfusion and secondary ischemic injury. Vasoactive drug requirement should be interpreted as a marker of clinical severity rather than evidence of a harmful effect of norepinephrine. In our unit, norepinephrine was initiated for hypotension refractory to fluid resuscitation or abnormal TCD findings and was weaned according to TCD assessment. TCD-guided hemodynamic approaches have been described in adults [13,14], but the impact of this practice, particularly TCD-guided norepinephrine weaning, was not assessed in our study and requires prospective evaluation.

Reduced prothrombin activity (<70%) and thrombocytopenia at admission were associated with mortality, consistent with pediatric data linking early trauma-related coagulopathy to poor outcomes [15,16]. Different definitions of coagulopathy across studies limit direct comparisons, and our observational findings do not establish that correcting these abnormalities improves survival. Hyponatremia was also associated with death, in agreement with a multicenter pediatric TBI study reporting an adjusted OR of 2.47 (95% CI 1.31–4.66) [17]. Hypernatremia was not included among our prespecified systemic insults, although it has also been linked to adverse outcomes; treatment with hypertonic saline may complicate its interpretation [17,18]. Syndrome of inappropriate antidiuretic hormone secretion and cerebral salt wasting are possible causes of hyponatremia [19], but their contributions could not be determined in this retrospective cohort.

Prognostic factors not identified

Age, sex, mechanism of injury, time to management, associated extracranial injuries, post-traumatic seizures, hypoxemia, and hyperthermia were not significantly associated with mortality in our cohort. This does not exclude clinically important effects, given the small number of deaths and limited statistical power. Hypoxemia and hyperthermia were assessed at PICU admission, after initial resuscitation, and may not capture transient prehospital insults. The cohort also did not include children who died before PICU admission. Extradural hematoma was numerically more frequent among survivors than non-survivors (22.3% vs. 5.0%), possibly reflecting the potential for surgical treatment, but this difference was not statistically significant and remains hypothesis-generating.

Limitations

Several limitations should be acknowledged. First, the retrospective, single-center design limits causal inference and generalizability; the exclusion of 74 of 188 potentially eligible children (39.4%)

because of incomplete records may have introduced selection bias. Second, the eligibility criterion of post-resuscitation GCS ≤8 excluded 91 children with a higher initial GCS, some of whom might have had severe intracranial lesions or deteriorated subsequently. Such patients would not be captured by our admission-based definition, whereas other pediatric sTBI cohorts have included children with initially moderate GCS who deteriorated rapidly [4]. Third, only 20 deaths occurred for eight variables in the reported multivariable model (approximately 2.5 events per variable), creating a substantial risk of overfitting and unstable adjusted estimates. Fourth, vasoactive drug requirement and PICU length of stay were measured after admission. The former is influenced by treatment decisions and may overlap with hemodynamic instability; the inverse association between length of stay and mortality is likely influenced by survival-time bias and should not be viewed as protective [20]. Finally, invasive ICP monitoring was unavailable, TCD measurements were not documented for prognostic analysis, post-admission complications were not compared by survival status, and long-term neurological outcomes were not assessed.

Future prospective multicenter studies should validate early neurological, hemodynamic, coagulation, and metabolic markers; include children who deteriorate after an initially higher GCS; and document TCD findings and long-term functional outcomes. A national pediatric neurotrauma registry could support these objectives.

CONCLUSION:

In this five-year cohort of children with severe TBI admitted to a PICU in a middle-income country, in-hospital mortality was 17.5%. Lower admission GCS, bilateral fixed mydriasis, hemodynamic instability, reduced prothrombin activity, thrombocytopenia, and hyponatremia at admission were associated with in-hospital mortality in an exploratory multivariable analysis. These readily available

parameters may help identify high-risk children early, particularly where advanced neuromonitoring is limited. However, given the retrospective design and the small number of deaths (n = 20), these findings require validation in larger prospective multicenter studies.

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Statements and Declarations

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Author contributions: Salma Aouadi and Mehdi Trifa contributed to the study conception and design. Data collection was performed by Salma Aouadi, Donia Belhadj Amor, Ahmed Ben Slimene, Rym Karaborni, Salmen Mannai and Hajer Blaiti. Data analysis was performed by Salma Aouadi. The first draft of the manuscript was written by Salma Aouadi, and all authors commented on

previous versions of the manuscript. Mehdi Trifa supervised the study. All authors read and approved the final manuscript.

Ethics approval: In accordance with the policy of Bechir Hamza Children's Hospital, approval by an ethics committee was not required for this retrospective, non-interventional study based on anonymized data collected during routine care. The study was conducted in accordance with the 1964 Declaration of Helsinki and its later amendments.

Consent to participate: Owing to the retrospective design and the use of anonymized data, individual informed consent from parents or legal guardians was not required.

Consent to publish: Not applicable.

Data availability: The datasets generated and analysed during the current study are available from the corresponding author on reasonable request.

Table 1. Baseline demographic and injury characteristics according to survival status

Variable	Overall Cohort (n=114)	Survivors (n=94)	Non-survivors (n=20)	p-value
Age, years, mean ±SD	6.6 ± 3.9	6.9± 3.9	5.5 ±3.7	NS
Male sex, n (%)	88 (77.2)	73 (77.7)	15 (75.0)	NS
Mechanism of injury, n (%)				NS
Road traffic accident	62 (54.4)	49 (52.1)	13 (65.0)	
Domestic accident	44 (38.6)	38 (40.4)	6 (30.0)	
Other mechanisms	8 (7.0)	7 (7.4)	1 (5.0)	
Time to management (hours), median (IQR)	3.0 (2.0–9.0)	3.0 (1.0–5.0)	4.0 (2.0–9.2)	NS
Associated extracranial injury, n (%)	54 (47.4)	44 (46.8)	10 (50.0)	NS

Data are presented as mean ± standard deviation (SD), median (interquartile range) or number (percentage). Continuous variables were compared using Student's t-test or the Mann–Whitney U test, and categorical variables using the chi-square or Fisher's exact test, as appropriate. NS: not significant ($p \geq 0.05$). SD: standard deviation.

Table 2. Admission neurological severity and key neuroradiological findings.

Clinical and Radiological Marker	Survivors (n=94)	Non-survivors (n=20)	p-value
Admission GCS score, mean ± SD	6.1± 2.1	4.9 ± 1.9	**0.018**
Bilateral fixed mydriasis, n (%)	4 (4.3)	10 (50.0)	**<0.001**
Post-traumatic seizures, n (%)	34 (36.2)	7 (35.0)	NS
Brain CT Findings, n (%)			
Diffuse cerebral edema	22 (23.4)	14 (70.0)	**<0.001**
Cerebral herniation signs	3 (3.2)	9 (45.0)	**<0.001**
Focal hematoma	35 (37.2)	11 (55.0)	NS
Extradural hematoma	21 (22.3)	1 (5.0)	NS

Data are presented as mean ± standard deviation (SD) or number (percentage). Continuous variables were compared using Student's t-test or the Mann–Whitney U test, and categorical variables using the chi-square or Fisher's exact test, as appropriate. Bold values indicate $p < 0.05$. NS: not significant ($p \geq 0.05$). CT: computed tomography; GCS: Glasgow Coma Scale; SD: standard deviation.

Table 3. Secondary systemic brain insults (SSBIs) at PICU admission.

Systemic Insult	Survivors (n=94)	Non-survivors (n=20)	p-value
Hemodynamic instability, n (%)	22 (23.4)	13 (65.0)	**0.001**
Prothrombin time (PT) < 70%, n (%)	27 (28.7)	14 (70.0)	**<0.001**
Thrombocytopenia (< 150,000/mm³), n (%)	11 (11.7)	8 (40.0)	**0.003**
Hyponatremia (<135 mEq/L), n (%)	8 (8.5)	6 (30.0)	**0.012**
Hypoxemia (PaO_2 < 60 mmHg), n (%)	9 (9.6)	2 (10.0)	NS
Hyperthermia (> 38.5°C), n (%)	10 (10.6)	3 (15.0)	NS

Data are presented as number (percentage). Survivors and non-survivors were compared using the chi-square or Fisher's exact test, as appropriate. Bold values indicate $p < 0.05$. NS: not significant ($p \geq 0.05$). PICU: pediatric intensive care unit; PT: prothrombin time; SSBIs: secondary systemic brain insults

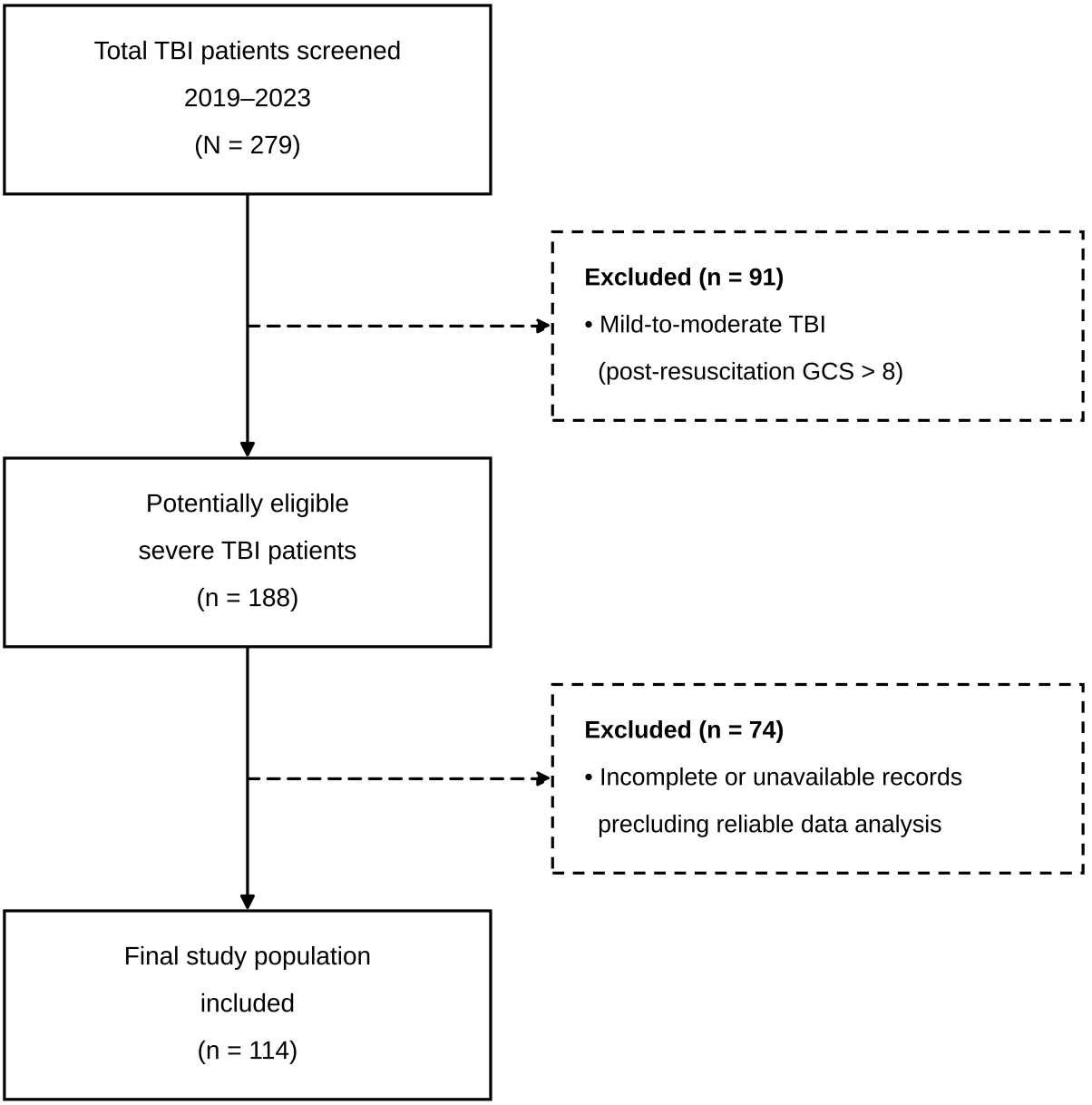
Table 4. Exploratory multivariable logistic regression model for in-hospital mortality.

Variable	OR	95% CI	p-value
Admission GCS, per point increase	0.82	0.70–0.96	**0.015**
Bilateral fixed mydriasis	3.85	1.17–12.65	**0.027**
PT < 70% at admission	2.90	1.40–6.02	**0.004**
Hyponatremia at admission	2.30	1.10–4.84	**0.027**
Hemodynamic instability at admission	5.40	2.50–11.60	**<0.001**
Vasoactive drug requirement (during PICU stay)	2.10	1.03–4.32	**0.041**
Thrombocytopenia (< 150,000/mm³) **at admission**	1.95	1.03–3.68	**0.040**
PICU length of stay, *per day*	0.84	0.75–0.95	**0.005**

Exploratory multivariable logistic regression including variables with $p < 0.20$ in univariate analysis. Odds ratios (OR) are given with 95% confidence intervals (CI). For binary variables, the reference category is the absence of the factor; for GCS and PICU length of stay, ORs are expressed per one-unit increase (one point and one day, respectively). Admission values refer to measurements obtained at PICU admission. Bold values indicate $p < 0.05$. CI: confidence interval; GCS: Glasgow Coma Scale; OR: odds ratio; PICU: pediatric intensive care unit; PT: prothrombin time. Only variables retained in the final model are shown.

Figure legend

Fig. 1 Study flow diagram showing patient selection. GCS, Glasgow Coma Scale; TBI, traumatic brain injury


Total TBI patients screened
2019–2023
(N = 279)
Excluded (n = 91)
• Mild-to-moderate TBI
(post-resuscitation GCS > 8)
Potentially eligible
severe TBI patients
(n = 188)
Excluded (n = 74)
• Incomplete or unavailable records
precluding reliable data analysis
Final study population
included
(n = 114)