BMC Pediatrics

RESEARCH

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Incidence, risk factors, and clinical impact of hyponatremia in pediatric trauma: a 9-year retrospective cohort study

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Abstract

Background Hyponatremia is common in critically ill children, and may be triggered by trauma-related stress responses. However, its clinical impact in pediatric trauma remains poorly defined. In this study, we investigated the incidence, risk factors, and outcomes of hyponatremia in pediatric patients with trauma.

Methods This retrospective observational study investigated patients younger than 19 years admitted to a level I trauma center between 2016 and 2024 who had at least two serum sodium measurements during hospitalization. Patient demographic/anthropometric characteristics and trauma-related data were retrieved. Subsequently, demographic and admission/resuscitation characteristics were compared between patients with and without hyponatremia to investigate the epidemiology and risk factors of hyponatremia.

Results Of 469 patients, 166 (35.4%) developed hyponatremia. In multivariable logistic regression analysis, very severe injury (ISS ≥ 25), surgical intervention, and early transfusion were identified as independent risk factors for hyponatremia. After adjustment for confounding variables, hyponatremia was independently associated with longer hospital stay, prolonged ICU stay, and increased duration of mechanical ventilation.

Conclusions Hyponatremia affects over one-third of pediatric patients with trauma and is strongly associated with injury severity and increased resource utilization. Early recognition of hyponatremia may help identify high-risk pediatric trauma patients and support optimized supportive care.

Keywords Hyponatremia, Pediatric, Trauma, Critical illness, Risk factors

Background

Hyponatremia, defined by a serum sodium concentration < 135 mEq/L, is the most common electrolyte disturbance in hospitalized patients, affecting up to 30% of critically ill adults [1–4]. This condition is associated with significant clinical complications, including cerebral edema, seizures, altered consciousness, and increased mortality risk [5]. In adult intensive care populations, hyponatremia independently predicts prolonged mechanical ventilation, longer hospital stays, and worse clinical outcomes, serving as both a marker of disease severity and a potentially modifiable risk factor [6–9].

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Pediatric populations are particularly vulnerable to hyponatremia-associated complications, although pediatric-specific literature remains relatively limited. Hospital-acquired hyponatremia has been reported in approximately 20–50% of pediatric intensive care unit (PICU) admissions. Recent large-scale studies have indicated that even mild hyponatremia (130–134 mEq/L) is associated with a nearly 5.6-fold higher risk of mortality and approximately 2.6-fold higher risk of PICU admission [10–12]. This heightened susceptibility arises from rapid electrolyte shifts related to immature renal solute handling, limited physiological reserves, and a relatively large brain-to-skull volume ratio, underscoring the importance of early recognition and management [13].

Although pathophysiology mechanisms suggest that trauma may trigger hyponatremia through stress-mediated vasopressin release, pain responses, and inflammatory cytokine activation, no studies have comprehensively examined this relationship [14]. Focusing on the initial evaluation, recent clinical evidence shows that pediatric patients with trauma, including those with head injuries or fractures, rarely exhibit significant sodium disturbances [15]. Large-scale pediatric emergency department analyses report an overall hyponatremia prevalence of only 3.7%, and trauma cohorts infrequently demonstrate sodium derangements despite mechanistic expectations [12]. However, a few other studies have shown that in pediatric polytrauma, traumatic brain injury cases with cerebral salt wasting (0.8%–34.6%) and isolated crush injuries associated with hyponatremia suggest a potentially higher incidence, yet comprehensive investigations beyond these remain limited [16–18]. This gap becomes particularly concerning given that when hyponatremia occurs in trauma contexts, it is associated with substantially worse outcomes.

Therefore, the present study aimed to determine the incidence of hyponatremia in pediatric patients with trauma admitted to our trauma center, identify associated clinical and trauma-related risk factors, and evaluate its impact on clinical outcomes. By addressing these objectives, we sought to clarify the clinical significance of hyponatremia in pediatric patients with trauma and provide evidence to guide improved patient management.

Methods

Participants and ethics

We retrospectively reviewed children and adolescents (< 19 years), consistent with the World Health Organization definition of adolescence and our institutional pediatric trauma admission policy, who were urgently admitted to our trauma center between January 2016 and December 2024 and underwent serum sodium (sNa) level measurements on at least 2 consecutive hospital days to allow reliable identification of nadir sodium values. Patients were excluded if they had pre-existing chronic renal disease or endocrine disorders known to affect sodium homeostasis, were transferred from another hospital, were re-admitted during the study period, or had missing key clinical or laboratory data required for analysis. The study period was selected to correspond to the interval following the designation of our institution as a regional trauma center, during which standardized trauma registry data collection and consistent laboratory measurements, including serum sodium levels, were available. A detailed flow diagram describing patient identification, inclusion, and exclusion is provided in Figure S1 in Additional file 1.

Our center, designated in 2015 as equivalent to a Level I trauma center in the United States, is a tertiary hospital serving approximately 7 million residents in the southern region of Gyeonggi Province, South Korea [19]. Each year, more than 500 patients with major trauma (Injury Severity Score [ISS] > 15) are admitted, of whom approximately 20% are pediatric patients. At our center, balanced isotonic solutions are routinely administered as maintenance fluids in pediatric patients after hemodynamic stabilization, except in cases requiring specialized fluid management for severe electrolyte abnormalities or other specific metabolic indications [20].

This study was approved by the Institutional Review Board of Ajou University Hospital (IRB No. AJOUIRB-DB-2025-265, approval date: 26 March 2025), and the requirement for informed consent was waived owing to its retrospective design. The study was conducted in accordance with the principles of the Declaration of Helsinki and adhered to the STROBE guidelines for retrospective observational studies. All statistical analyses were performed using R software (R Foundation for Statistical Computing, Vienna, Austria).

Data collection

Patient demographic and anthropometric characteristics, along with trauma-related data, were retrieved. Information on the injury mechanism, ISS, and organ-specific Abbreviated Injury Scale (AIS; head, face, chest, abdomen, extremity, and external) was obtained from the Ajou Trauma Data Bank, which is prospectively maintained and periodically updated by trained coordinators. Given the epidemiology of pediatric trauma in our region, the mechanism of injury was predominantly blunt trauma (e.g., traffic accidents, falls). Multi-system injuries were characterized by calculating the AIS score for each anatomical region, and the overall injury burden was summarized using the ISS. Approximately 5% of missing or implausible entries were corrected through direct chart review. The Glasgow Coma Scale (GCS) score and serum lactate and C-reactive protein (CRP) levels at presentation were recorded, and details of surgical interventions

and transfusions during resuscitation were collected. Craniotomy was also documented as an indicator of severe traumatic brain injury, a condition frequently associated with hyponatremia [21]. However, due to the retrospective nature of the study and inconsistent availability of urine electrolytes and paired osmolality data, a definitive etiologic classification (e.g., distinguishing syndrome of inappropriate antidiuretic hormone secretion [SIADH], cerebral salt wasting, or dilutional hyponatremia) was not performed for the entire cohort.

Subsequently, we reviewed all serial sNa measurements. The lowest sNa value and its timing were documented; hyponatremia was defined as a nadir sNa value < 135 mEq/L [22]. We also evaluated early-onset hyponatremia, defined as a nadir sNa < 135 mEq/L within the first 7 days of hospitalization. Patient outcomes were defined as hospital length of stay, ICU length of stay, and duration of mechanical ventilation; patients not requiring ICU admission or mechanical ventilation were assigned a value of zero.

The primary outcome of this study was the occurrence of hyponatremia (defined as a nadir serum sodium level < 135 mEq/L) during hospitalization. Secondary outcomes included clinical outcomes reflecting disease severity and resource utilization: hospital length of stay, ICU length of stay, duration of mechanical ventilation, and in-hospital mortality.

Data analysis

To investigate the epidemiology and risk factors of hyponatremia, demographic and admission/resuscitation characteristics were compared between patients with and without hyponatremia. ISS was categorized as mild to moderate (1–15), severe (16–24), and very severe (≥ 25), while AIS scores were dichotomized at the severe injury threshold (AIS ≥ 3) [23, 24].

Categorical variables were analyzed using the chi-square test. Continuous variables were assessed for normality and are presented as mean ± standard deviation or median with interquartile range, as appropriate. Group comparisons were performed using Student's t-test for normally distributed variables and the Wilcoxon rank-sum test for non-normally distributed variables. Time-to-event analyses were performed using Kaplan–Meier survival curves, and differences between groups were assessed using the log-rank test.

Multivariable logistic regression analysis was performed to identify independent risk factors for hyponatremia after adjustment for potential confounders. Variables were considered eligible for inclusion in the multivariable models based on clinical relevance supported by prior literature and/or an association with the outcome in univariable analyses ($p < 0.20$). Model construction followed a purposeful selection strategy rather than automated forward or backward stepwise procedures. Clinically important indicators of injury severity and physiological stress, including GCS score and serum lactate level, were retained in the final models regardless of statistical significance. The associations between hyponatremia and clinical outcomes, including hospital length of stay, ICU length of stay, and duration of mechanical ventilation, were assessed using multivariable linear regression models with adjustment for the same set of covariates. Length-of-stay variables were treated as continuous outcomes, acknowledging their typically right-skewed distributions. Model assumptions, including linearity, normality of residuals, and homoscedasticity, were evaluated using standard diagnostic approaches. Multicollinearity among covariates in the multivariable regression models was assessed using variance inflation factors (VIF). The discriminative performance of the final multivariable logistic regression model was evaluated using receiver operating characteristic (ROC) curve analysis. The area under the curve (AUC) with 95% confidence intervals (CI) was calculated to assess the model's predictive ability.

For patients with moderate-to-severe hyponatremia (serum sodium < 130 mEq/L), detailed chart review was conducted to document the timing of onset, presumed etiologies, therapeutic interventions, and subsequent clinical course. Statistical significance was defined as a two-sided p value < 0.05. All analyses were performed using R version 4.5.1 (R Foundation for Statistical Computing, Vienna, Austria).

Results

From January 2016 to December 2024, 1,027 pediatric patients (< 19 years) were admitted to our trauma center. Of these, 469 patients remained hospitalized for ≥ 2 days and had sufficient sNa measurements for analysis. The median age was 16 years (interquartile range [IQR], 14–17 years), and 312 (66.5%) were male.

We first evaluated the incidence and timing of hyponatremia as the primary outcome, followed by analyses of secondary clinical outcomes. Among the cohort, 166 patients (35.4%) developed at least 1 episode of hyponatremia (sNa < 135 mEq/L) during hospitalization (Table 1). Hyponatremia first occurred at a median of 2 days after admission (IQR, 1–7 days), with the nadir level observed at a median of 3 days (IQR, 1–8 days). The mean nadir sodium level was 132.4 ± 2.4 mEq/L in the hyponatremia group and 137.0 ± 1.7 mEq/L in the non-hyponatremia group. Significant differences were observed across ISS categories, with lower nadir sodium levels associated with increasing injury severity (Table 1).

We examined the association between demographic, injury-related, and resuscitation characteristics and the occurrence of hyponatremia. Patients who developed

Table 1 Comparison of lowest serum sodium levels according to injury severity score categories in pediatric trauma patients with and without hyponatremia

Variables	Normonatremia ($n=303$)	Hyponatremia ($n=166$)	p - value
Lowest serum sodium level during admission (mEq/L), mean ± SD			
Overall	137.0 ± 1.7	132.4 ± 2.4	< 0.001
ISS severity			
Mild to moderate (1–15)	137.0 ± 1.6 ($n=156$)	133.0 ± 1.4 ($n=39$)	< 0.001
Severe (16–24)	137.0 ± 1.9 ($n=94$)	132.0 ± 2.6 ($n=53$)	< 0.001
Very severe (≥ 25)	136.0 ± 1.5 ($n=53$)	132.0 ± 2.7 ($n=74$)	< 0.001

Abbreviations: *SD* Standard Deviation, *ISS* Injury severity score

hyponatremia had greater overall injury severity and more frequently sustained severe truncal and extremity injuries compared with those without hyponatremia (Table 2). In contrast, the prevalence of severe head injury did not differ significantly between groups, contrary to the conventional assumption that traumatic brain injury, often reflected by lower GCS scores, predisposes patients to hyponatremia [16]. Patients with hyponatremia more frequently presented with abnormal GCS scores and required surgery, and transfusion. In multivariable logistic regression evaluating risk factors for hyponatremia, higher ISS, surgery, and transfusion were identified as independent predictors (adjusted OR for severe injury, 1.85; 95% CI, 1.11–3.11; adjusted OR for very severe injury, 2.45; 95% CI, 1.32–4.58; Fig. 1 and Table S3 in Additional file 1). The discriminative performance of the final multivariable logistic regression model was further evaluated using ROC analysis. The area under the curve (AUC) was 0.74 (95% CI, 0.69–0.78), indicating acceptable discrimination for predicting hyponatremia (Figure S4 in Additional file 1) (Table 3, Fig. 2).

After adjusting for confounders in multivariable linear regression (Table 4), hyponatremia was independently associated with increases in hospital stay (+ 10.7 days), ICU stay (+ 3.4 days), and mechanical ventilation duration (+ 1.8 days). No significant association with mortality was observed in the multivariable model (Figure S3 in Additional file 1).

To determine whether our findings reflected the direct effects of trauma or the consequences of prolonged hospitalization, we performed a secondary analysis of early-onset hyponatremia (nadir sNa < 135 mEq/L within the first 7 days of admission) and observed no significant differences in trauma-related characteristics (Tables S2 and S4 in Additional file 1).

Moderate-to-severe hyponatremia (sNa < 130 mEq/L) developed in 13 patients (2.8%) after a median of 11 days of hospitalization (IQR, 9–21), with a nadir sNa

Table 2 Baseline clinical characteristics and demographics of pediatric trauma patients with and without hyponatremia

Variables	Normonatremia ($n=303$)	Hyponatremia ($n=166$)	p - value
Demographics			
Age (years), median [IQR]	16.0 [13.0, 17.0]	16.0 [14.0, 17.0]	0.47
Sex, male, n (%)	210 (69.3)	102 (61.4)	0.11
Height (cm), median [IQR]	169.0 [159.0, 175.0]	168.0 [160.0, 174.0]	0.38
Weight (kg), median [IQR]	59.8 [52.0, 70.1]	60.0 [50.0, 68.5]	0.68
BMI (kg/m^2), median [IQR]	21.6 [18.9, 24.4]	21.4 [18.7, 24.2]	0.81
Injury characteristics			
Mechanism: Blunt trauma, n (%)	295 (97.4)	163 (98.2)	0.80
GCS, median [IQR]	15.0 [14.0, 15.0]	14.0 [9.0, 15.0]	< 0.001
GCS < 15	108 (35.6)	101 (60.8)	< 0.001
GCS < 9	29 (9.6)	35 (21.1)	0.001
ISS, median [IQR]	14.0 [9.0, 22.0]	22.0 [16.0, 33.8]	< 0.001
ISS severity, n (%)			< 0.001
Mild to moderate (1–15)	156 (51.5)	39 (23.5)	
Severe (16–24)	94 (31.0)	53 (31.9)	
Very severe (≥ 25)	53 (17.5)	74 (44.6)	
AIS ≥ 3, n (%)			
Head	95 (31.4)	58 (34.9)	0.49
Face	0 (0.0)	3 (1.8)	0.08
Chest	100 (33.0)	89 (53.6)	< 0.001
Abdomen	60 (19.8)	62 (37.3)	< 0.001
Extremity	62 (20.5)	76 (45.8)	< 0.001
External	0 (0.0)	1 (0.6)	0.76
Resuscitation & Interventions, n (%)			
Requiring surgery, n (%)	205 (67.7)	145 (87.3)	< 0.001
Craniotomy	6 (2.0)	7 (4.2)	0.26
Transfusion within 4 h	37 (12.2)	65 (39.2)	< 0.001
Transfusion within 24 h	20 (6.6)	43 (25.9)	< 0.001
CRP, median [IQR]	1.9 [0.5, 5.5]	6.0 [1.6, 12.3]	< 0.001
Lactate, median [IQR]	2.4 [1.7, 3.3]	2.9 [1.7, 4.5]	0.009

Abbreviations: *IQR* Interquartile Range, *BMI* Body mass index, *GCS* Glasgow Coma Scale, *ISS* Injury severity score, *AIS* abbreviated injury scale, *CRP* C-Reactive Protein

concentration of 127 mEq/L (IQR, 124–128). Regarding clinical characteristics and presumed etiologies, 7 patients had severe head injuries (AIS ≥ 3). Based on clinical assessment, SIADH was suspected in 2 patients, and cerebral salt wasting (CSW) in 3 patients. Medication-related effects were considered contributory in 3 cases, while the underlying cause remained undetermined in the remaining patients. Management strategies were individualized according to the severity of hyponatremia and the overall clinical context. One patient with concomitant acute kidney injury required renal replacement

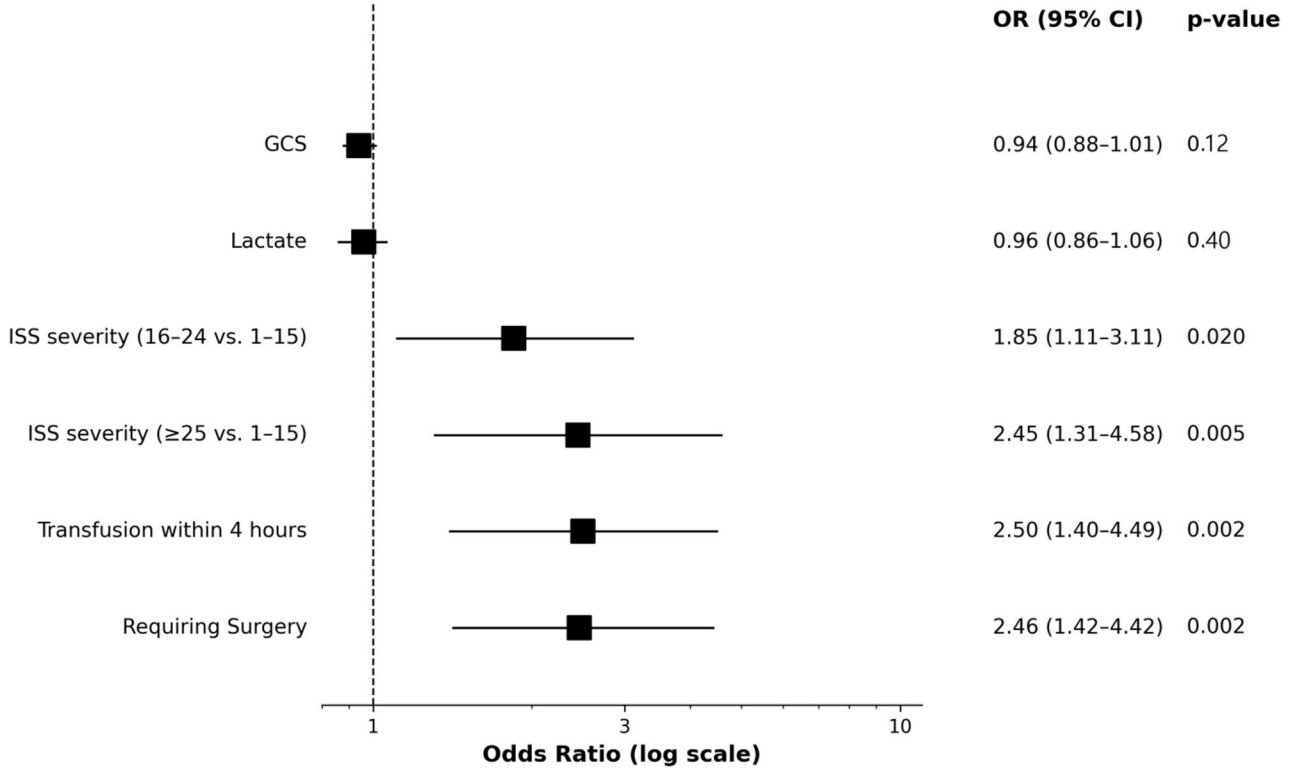

Fig. 1 Forest plot of risk factors associated with hyponatremia in pediatric trauma patients

Table 3 Comparison of clinical outcomes between pediatric trauma patients with and without hyponatremia

Variables	Normonatremia ($n=303$)	Hyponatremia ($n=166$)	p - value
Length of stay, median [IQR]			
Hospital length of stay	13 [9, 19]	20.5 [15, 39]	< 0.001
ICU length of stay	2 [1, 4]	6.0 [3, 10]	< 0.001
Mechanical ventilation duration, median [IQR]	0.0 [0, 0]	2.0 [0, 6]	< 0.001
Mortality, n (%)	6.0 (2.0)	7.0 (4.2)	0.24

Abbreviations: *IQR* Interquartile Range, *ICU* Intensive Care Unit

therapy. Among the remaining patients, oral salt supplementation was used primarily in cases with moderate hyponatremia, whereas hypertonic saline was administered in patients with more severe sodium derangements. No standardized treatment protocol for hyponatremia was applied. Except for two non-survivors, all patients were discharged with normalized serum sodium levels and required no further sodium-specific therapy.

Discussion

In this retrospective cohort of pediatric trauma patients, hyponatremia was observed in more than one-third of cases, a prevalence substantially higher than that reported in general pediatric emergency department populations and within the upper range described in PICU cohorts [10–12, 25, 26]. Our findings demonstrate that hyponatremia in pediatric trauma is strongly associated with injury severity and independently associated with prolonged hospital stay, ICU stay, and longer duration of mechanical ventilation.

Our analysis revealed a strong association between injury severity and the development of hyponatremia. Patients with hyponatremia had significantly higher ISS (median 22.0 vs. 14.0, $p < 0.001$), and 44.6% sustained very severe injuries (ISS ≥ 25) compared with 17.5% of normonatremic patients. This pattern aligns with prior literature suggesting that electrolyte disturbances reflect cumulative physiological stress and overall injury burden in trauma patients [27, 28]. In multivariable analysis, higher ISS severity, surgical intervention, and early transfusion remained significantly associated explanatory factors—features that collectively represent severe trauma requiring aggressive resuscitation and invasive management. Taken together, these findings support the interpretation that trauma-associated hyponatremia is a marker of global physiological stress rather than an organ-specific phenomenon, consistent with studies linking dysnatremia to adverse clinical outcomes in critically injured patients [29–31].

In contrast to conventional literature emphasizing traumatic brain injury as a major contributor to hyponatremia [16, 21, 32, 33], we did not observe a significant

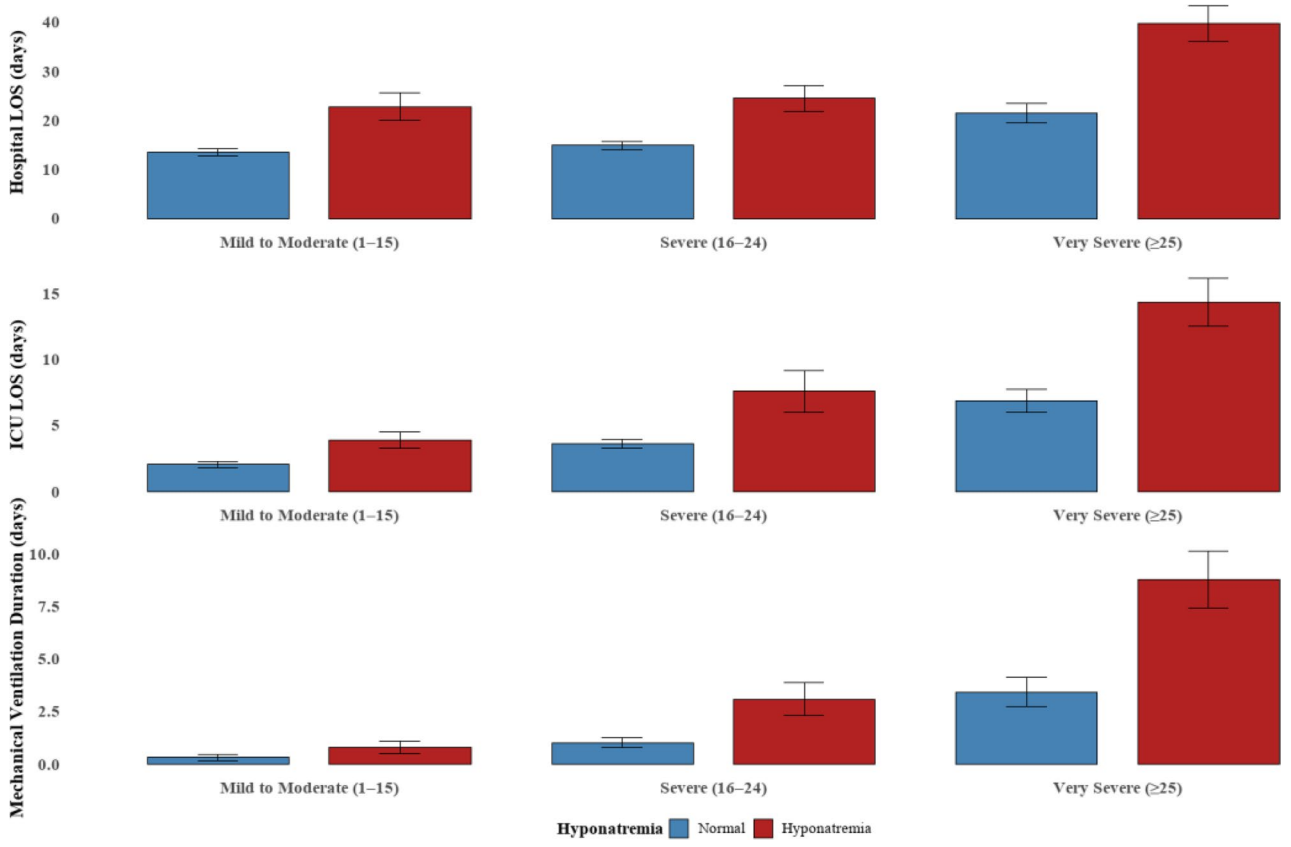

Fig. 2 Clinical outcomes stratified by injury severity score category and hyponatremia status in pediatric trauma patients

Table 4 Multivariable linear regression analysis of factors influencing clinical outcomes in pediatric trauma patients

Variables	B (95% CI)	Standardized β	*p*-value	VIF
Length of Hospital Stays				
Hyponatremia	10.67 (7.36–13.99)	0.56	< 0.001	1.15
Serum Lactate	2.06 (1.33–2.78)	0.26	< 0.001	1.38
Requiring surgery	7.50 (3.84–11.15)	0.39	< 0.001	1.12
Length of ICU Stays				
GCS	−0.55 (−0.79 - −0.31)	−0.21	< 0.001	1.52
Hyponatremia	3.39 (1.89–4.90)	0.38	< 0.001	1.18
Serum Lactate	1.12 (0.78–1.45)	0.30	< 0.001	1.47
Mechanical Ventilation duration				
ISS	0.06 (0.00–0.11)	0.11	0.03	1.92
GCS	−0.53 (−0.69 - −0.38)	−0.30	< 0.001	1.57
Hyponatremia	1.75 (0.77–2.74)	0.28	< 0.001	1.18
Serum Lactate	0.69 (0.47–0.91)	0.27	< 0.001	1.47
Transfusion within 4 hours	1.36 (0.03–2.68)	0.22	0.05	1.62

CI Confidence interval, *SE* Standard error, *VIF* Variance Inflation Factor, *GCS* Glasgow Coma Scale, *ISS* Injury severity score

association between severe head injury and hyponatremia development (34.9% vs. 31.4%, p = 0.49). Instead, severe truncal and extremity injuries appeared more prominent, suggesting that in pediatric polytrauma, hyponatremia may arise from multisystem pathophysiology rather than isolated neurogenic mechanisms [14, 27]. Potential pathways include substantial third-space fluid shifts from tissue disruption and capillary leakage, which often necessitate substantial crystalloid and blood product resuscitation and may contribute to dilutional hyponatremia. In parallel, systemic inflammation and stress responses can promote nonosmotic vasopressin release through cytokine-mediated hypothalamic stimulation [14, 34, 35]. Finally, perioperative stress, pain control, and certain medications may further predispose patients to SIADH-like physiology. These mechanisms provide a plausible explanation for why early transfusion and surgical intervention emerged as independent predictors in our cohort [35, 36]. Overall, our findings suggest that hyponatremia in pediatric trauma extends beyond brain injury alone and may serve as a useful clinical marker of injury severity in patients with moderate-to-severe trauma. This interpretation is further supported by prior pediatric studies in non-traumatic acute illnesses. Hyponatremia has been shown to correlate with disease

severity in conditions such as appendicitis, sepsis, pneumonia, and meningitis, where systemic inflammation and non-osmotic vasopressin release play central roles [37–39]. Viewed in this broader pediatric context, the association between hyponatremia and injury severity observed in our trauma cohort appears to reflect shared stress-response pathways rather than a trauma-specific mechanism.

Hyponatremia in our cohort was associated with substantial clinical impact across multiple endpoints. Hyponatremic patients experienced significantly longer hospital stays (median 20.5 vs. 13 days; $p < 0.001$), ICU stays (median 6.0 vs. 2.0 days; $p < 0.001$), and durations of mechanical ventilation (median 2.0 vs. 0.0 days; $p < 0.001$). These findings are consistent with prior pediatric and adult studies demonstrating that dysnatremia is associated with increased ICU utilization, delayed recovery, and higher healthcare resource consumption [28, 40, 41]. Despite these associations, mortality did not differ significantly between groups in our study (4.2% vs. 2.0%; $p = 0.24$), a finding that aligns with systematic reviews suggesting that hyponatremia primarily reflects illness severity rather than directly driving mortality, particularly in well-resourced trauma systems with early recognition and supportive care [28, 42]. Importantly, the observed associations with resource utilization should not be interpreted as causal. Hyponatremia may develop as a consequence of prolonged ICU stay, mechanical ventilation, infection, or other in-hospital complications, raising the possibility of reverse causation. Accordingly, our findings support interpreting hyponatremia as a downstream marker of cumulative physiological stress and critical illness rather than a direct determinant of adverse outcomes.

In our pediatric trauma cohort, the prevalence of hyponatremia was intermediate between that reported in the general PICU population and the most critically ill subgroups; nevertheless, the absolute incidence (35.4%) was clinically meaningful. Children's immature renal concentrating capacity and limited physiological reserves increase susceptibility to sodium disturbances, potentially amplifying their clinical impact compared with adults [5, 10]. In our study, this vulnerability was reflected in substantially greater healthcare utilization, including a median increase of 7.5 hospital days and 4 ICU days among hyponatremic patients, with important implications for bed capacity and resource use. Taken together, these findings suggest that clinicians should maintain heightened vigilance regarding serum sodium abnormalities in pediatric patients with moderate-to-severe trauma, particularly those requiring emergent surgery or early transfusion, rather than limiting attention to neurotrauma alone.

This study has several limitations. First, it was a single-center retrospective analysis, and external validation in multicenter, diverse trauma populations is warranted. Second, given the observational design, we cannot determine whether hyponatremia is a causal factor for adverse outcomes or merely a marker of injury severity, as shared pathophysiological pathways may underlie both conditions. Third, our focus on hospital-acquired hyponatremia rather than admission sodium levels may have influenced the observed associations with injury characteristics. Selection bias may also have been introduced by requiring at least two serum sodium measurements, potentially favoring the inclusion of patients with greater injury severity or longer hospitalization and inflating the observed incidence of hyponatremia. In addition, detailed data on cumulative fluid balance, temporal changes in fluid composition, and medication exposure (e.g., diuretics or antiepileptic drugs) were not consistently available, limiting our ability to fully distinguish trauma-related mechanisms from iatrogenic contributors. Furthermore, traumatic brain injury was classified using a binary AIS ≥ 3 threshold, which may have obscured important neuroanatomical subtypes—such as diffuse axonal injury, skull-base fractures, or hypothalamic–pituitary involvement—that are known to influence sodium homeostasis. Accordingly, the absence of a significant association between severe head injury and hyponatremia in our analysis should not be interpreted as contradicting established pathophysiological mechanisms. Finally, our cohort had a median age of 16 years, with a predominance of adolescent patients and a relatively small proportion of infants and young children; therefore, caution is warranted when extrapolating these findings to younger pediatric populations. Mechanistic investigations incorporating hormonal profiling, inflammatory marker quantification, and detailed fluid balance assessment were beyond the scope of this study but may help elucidate the biological pathways underlying hyponatremia in pediatric trauma.

Conclusion

Hyponatremia is a frequent finding in pediatric trauma, affecting more than one-third of patients and associated with prolonged hospital and ICU stays, as well as longer durations of mechanical ventilation. Although it is not a prognostic or actionable clinical marker and its direct impact on mortality remains uncertain, heightened awareness and careful monitoring of serum sodium levels may help support optimized supportive care in children with moderate-to-severe traumatic injuries.

Abbreviations

AIS	Abbreviated Injury Scale
CRP	C-reactive protein
GCS	Glasgow Coma Scale

ISS	Injury severity score
PICU	Pediatric intensive care unit
sNa	Serum sodium

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12887-026-06560-9.

Supplementary Material 1. This supplementary document provides additional analyses supporting the main manuscript. Supplementary Table 1: Baseline clinical characteristics and demographics of pediatric trauma patients with and without early hyponatremia. Supplementary Table 2: Comparison of clinical outcomes between patients with and without early hyponatremia. Supplementary Table 3: Multivariable logistic regression analysis of risk factors associated with hyponatremia in pediatric trauma patients. Supplementary Figure 1: PRISMA-style flow diagram of patient selection. Supplementary Figure 2: Kaplan–Meier survival curves comparing survival outcomes between pediatric trauma patients with and without hyponatremia. Supplementary Figure 3 Forest plot of multivariable Cox regression (Firth's penalized likelihood method) for mortality among pediatric trauma patients. Supplementary Figure 4: Receiver operating characteristic (ROC) curve of the multivariable logistic regression model for predicting hyponatremia in pediatric trauma patients. These tables and figures present extended statistical results and visualizations referenced in the main text.

Acknowledgements
Not applicable.

Authors' contributions
Hye Young Woo and Peong Gang Park conceived and designed the study. Hye Young Woo and Peong Gang Park collected and analysed the data and drafted the initial manuscript. Hye Young Woo, Kyoungwon Jung and Peong Gang Park contributed to data interpretation and critical revision of the manuscript. Keum Hwa Lee and Peong Gang Park contributed to data acquisition and manuscript editing. All authors read and approved the final version of the manuscript.

Funding
This work was supported by the Global Physician-Scientist Development Program in Korea Health Industry Development Institute (KHIDI), funded by the Ministry of Health & Welfare, Republic of Korea (R-2025-25468050 to Hye Young Woo and RS-2025-25467712 to Peong Gang Park).

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

Declarations

Ethics approval and consent to participate
This study was conducted in accordance with the principles of the Declaration of Helsinki. Ethical approval was obtained from the Institutional Review Board of Ajou University School of Medicine (approval No. AJOUIRB-DB-2025-265; 26 March 2025). The requirement for informed consent was waived owing to the retrospective design of the study.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

Received: 9 November 2025 / Accepted: 19 January 2026
Published online: 03 February 2026

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