Scholars Journal of Applied Medical Sciences
Abbreviated Key Title: Sch J App Med Sci
ISSN 2347-954X (Print) | ISSN 2320-6691 (Online)
Journal homepage: https://saspublishers.com

OPEN ACCESS

Surgical Intensive Care

Factors Associated with In-Hospital Mortality in Patients with Mixed-Etiology Hemorrhagic Shock: A Retrospective Single-Center Study

Abdelhak Tissir[1]*, Kaoutar Zirhirhi[1], Othmane Tahri Joutey[1], Saara Lamghari[1], Lina Berrada[1], Sara Chabbar[1], Fatima Zahra Faouji[1], Anas Mounir[1], Bouhouri Aziz[1]

[1]Department of Surgical Intensive Care, Ibn Rochd University Hospital Center, Casablanca, Morocco

DOI: https://doi.org/10.36347/sjams.2026.v14i05.039 | **Received:** 25.03.2026 | **Accepted:** 05.05.2026 | **Published:** 27.05.2026

***Corresponding author:** Abdelhak Tissir
Department of Surgical Intensive Care, Ibn Rochd University Hospital Center, Casablanca, Morocco

Abstract **Original Research Article**

Introduction: Hemorrhagic shock carries high case-fatality rates in trauma and surgical populations, yet data on factors associated with mortality in North African intensive care settings remain limited. This study aimed to explore which clinical, biological, and therapeutic variables were associated with in-hospital mortality in a mixed-etiology hemorrhagic shock cohort admitted to a Moroccan surgical ICU. ***Methods:*** We performed a retrospective analysis of ICU admissions at Ibn Rochd University Hospital Center, Casablanca, from January 2022 to December 2024. Among 223 screened patients, 66 fulfilled the inclusion criterion of systolic blood pressure < 90 mmHg in the setting of polytrauma, penetrating trauma, or gastrointestinal hemorrhage. Twelve candidate variables were tested in univariate analysis; eight with $p < 0.20$ were considered for multivariate logistic regression. To avoid overfitting in the context of 21 events, a parsimonious three-variable model was constructed based on clinical relevance and collinearity assessment. ***Results:*** The cohort was predominantly young (67% aged < 40 years) and male (sex ratio 4:1). Polytrauma accounted for 56% of cases. Twenty-one patients died (32%), with 60% of deaths occurring within the first 24 hours. In multivariate analysis, three variables were independently associated with in-hospital mortality: Glasgow Coma Scale < 8 (OR 3.42; 95% CI 1.28-9.11; $p = 0.014$), Shock Index > 0.9 (OR 2.87; 95% CI 1.10-7.46; $p = 0.031$), and hemoglobin < 5 g/dL (OR 3.95; 95% CI 1.36-11.42; $p = 0.011$). The mean time to hemorrhage control was longer among non-survivors (112 ± 35 vs. 72 ± 25 minutes; $p < 0.001$). These findings should be considered exploratory given the limited sample size. ***Conclusion:*** In this mixed-etiology surgical ICU cohort, the Shock Index, Glasgow Coma Scale, and admission hemoglobin were independently associated with mortality and may serve as early bedside stratification tools. These findings should be interpreted with caution due to the retrospective design and limited sample size, and require validation in larger, prospective, multicenter studies.

Keywords: hemorrhagic shock, intensive care unit, morocco, mortality, polytrauma, shock index.

INTRODUCTION

Hemorrhagic shock—the state of circulatory collapse driven by rapid blood loss—accounts for an estimated 30-40% of trauma-related deaths worldwide and remains one of the most time-critical emergencies in surgical practice [1-3]. Over the past two decades, the treatment paradigm has shifted from aggressive crystalloid infusion toward damage control resuscitation (DCR), which prioritizes balanced blood product transfusion and early hemorrhage control [4-6]. While these principles have been validated in high-volume trauma centers, their implementation and outcomes in settings with constrained resources and prolonged pre-hospital intervals have been less extensively studied [7].

In Morocco, road traffic injuries and urban violence generate a substantial caseload of hemorrhagic shock admissions in university hospital ICUs [8]. The patient population is often heterogeneous, encompassing polytrauma, penetrating injuries, and non-traumatic hemorrhage, each with distinct bleeding kinetics and management challenges. Identifying which early clinical and biological parameters are most closely associated with mortality in this context could help clinicians prioritize interventions during the narrow initial resuscitation window.

Several bedside markers have been proposed for this purpose, including the Shock Index (heart rate divided by systolic blood pressure) [9], the Injury Severity Score [10], the Glasgow Coma Scale [11], and admission hemoglobin concentration [12]. Each captures

a different facet of the physiological insult, yet their independent association with mortality has not been specifically evaluated in a Moroccan surgical ICU population.

The present study was designed to address this gap by analyzing retrospective data from a three-year cohort of patients admitted with hemorrhagic shock to the surgical ICU of Ibn Rochd University Hospital Center in Casablanca. Its primary objective was to identify factors independently associated with in-hospital mortality. A secondary objective was to describe the temporal pattern of mortality and the therapeutic profile of this mixed-etiology cohort.

MATERIALS AND METHODS

Study Design

This was a retrospective, observational, single-center study conducted over a three-year period from January 2022 to December 2024 in the Surgical Intensive Care Unit of Ibn Rochd University Hospital Center, Casablanca, Morocco.

Ethical Considerations

The study protocol received approval from the Ethics Committee for Biomedical Research of Casablanca prior to data collection. Because the analysis relied exclusively on anonymized data extracted retrospectively from existing medical records, the committee granted a waiver of individual informed consent in accordance with Moroccan regulations governing non-interventional observational research. All patient identifiers were removed at the extraction stage, and the anonymized dataset was stored on a secure, password-protected institutional server accessible only to the study investigators. A declaration for retrospective data processing was filed in compliance with national data governance requirements.

Inclusion and Exclusion Criteria

Inclusion criteria required a systolic blood pressure (SBP) < 90 mmHg at admission in the context of polytrauma, penetrating trauma (stabbing), or gastrointestinal hemorrhage. Exclusion criteria included incomplete medical records, cardiac arrest on arrival, non-hemorrhagic shock (septic, cardiogenic, or mixed), and patients initially managed at another center with delayed secondary transfer.

Patient Selection

A total of 223 patients were initially screened from ICU admission records during the study period. Of these, 157 were excluded: 82 for SBP ≥ 90 mmHg (absence of hemorrhagic shock criteria), 34 for incomplete medical records, 18 for cardiac arrest on admission, 13 for non-hemorrhagic shock etiologies, and 10 for delayed secondary transfers with initial management outside the center. The final study population comprised 66 patients (Figure 1). Notably, the cohort included patients with heterogeneous etiologies (polytrauma, penetrating trauma, and gastrointestinal hemorrhage), reflecting the real-world case mix of a surgical ICU. This heterogeneity is acknowledged as a limitation and is addressed in the Discussion.

Data Collection

Data were extracted from medical records and included: demographic variables (age, sex), clinical parameters (GCS [11], SBP, heart rate, Shock Index), trauma severity scores (Injury Severity Score [ISS], Simplified Acute Physiology Score II [SAPS II]), biological markers (hemoglobin, lactate, platelet count, coagulation parameters, temperature), etiology, therapeutic interventions (fluid resuscitation volumes, blood product transfusions, vasopressor use, time to hemostasis), and clinical outcomes (mortality, timing of death).

Operational Definitions

The following operational definitions were used throughout the study. Hypothermia was defined as a core body temperature below 35°C measured at ICU admission. Coagulopathy was defined as the presence of at least one of the following: prothrombin time (PT) ratio below 70%, international normalized ratio (INR) above 1.5, activated partial thromboplastin time (aPTT) exceeding 1.5 times the control value, or fibrinogen level below 1.5 g/L. Time to hemostasis was defined as the interval in minutes between ICU admission and the achievement of definitive hemorrhage control, whether by surgical intervention, interventional radiology (embolization), or endoscopic procedure, as documented in the operative or procedural report.

Statistical Analysis

A total of 12 clinically relevant variables were tested in univariate analysis: age, sex, etiology, polytrauma, traumatic brain injury (TBI), abdominal injury, Glasgow Coma Scale (GCS) [11] score, Shock Index, hemoglobin level, platelet count, coagulation disorders, and hypothermia.

Given the limited number of events (n = 21 deaths), a parsimonious model was constructed in accordance with the rule of approximately 10 events per variable, restricting the final model to three covariates. Variable selection followed a combined strategy based on clinical relevance, avoidance of composite scores, and assessment of collinearity between variables reflecting overlapping physiological pathways. Specifically: GCS was retained over TBI because it provides a direct, quantitative measure of neurological status whereas TBI overlaps conceptually and statistically with GCS; Shock Index was preferred over SAPS II because the latter is a composite score incorporating multiple physiological parameters, introducing redundancy; and hemoglobin was selected over lactate because it directly reflects the volume of blood loss and is immediately available at bedside, whereas lactate is an indirect downstream

marker of tissue hypoperfusion. Collinearity between candidate variables was assessed using Spearman correlation coefficients for continuous variable pairs and by examining variance inflation factors (VIF), with a VIF above 5 considered indicative of problematic multicollinearity. Variables sharing the same physiological construct (e.g., GCS and TBI, r = 0.74; Shock Index and SAPS II, VIF > 5 when both included) were not entered simultaneously into the model, and the more clinically actionable variable of each pair was retained.

Results were expressed as Odds Ratios (OR) with 95% confidence intervals (CI). A p-value < 0.05 was considered statistically significant. All analyses were performed using SPSS software (IBM Corp., Armonk, NY, USA). Given the exploratory nature of the analysis and the limited sample size, the results of the multivariate model should be interpreted with caution and require external validation.

RESULTS

Patient Characteristics

The study included 66 patients with a mean age of 36 ± 13 years. The cohort was predominantly young (67% aged under 40 years) and male (sex ratio 4:1, 80% male). The etiological distribution was as follows: polytrauma in 37 patients (56%), penetrating trauma by stabbing in 17 patients (26%), and gastrointestinal hemorrhage in 12 patients (18%). Traumatic brain injury (TBI) was present in 30% of the cohort (20/66 patients). Abdominal involvement was noted in 60% of trauma cases. The mean ISS was 25 ± 8, and the mean SAPS II was 40 ± 10. Metabolic acidosis was prevalent, with mean lactate levels of 4.6 ± 2.0 mmol/L. Hypothermia at admission was observed in 35% of patients (23/66).

Therapeutic Interventions

Aggressive fluid resuscitation was required in 68% of patients, who received more than 2 liters of crystalloids during initial resuscitation, in addition to catecholamines. Regarding transfusion requirements, 64% of patients received more than 6 units of packed red blood cells (PRBC), 55% received more than 6 units of platelets, and 51% received more than 6 units of fresh frozen plasma (FFP). The mean transfusion ratio approached 1:1:1 (PRBC: Platelets:FFP). The mean time to hemorrhage control was 90 ± 30 minutes.

Comparison of Survivors and Non-Survivors

The comparative analysis between survivors (n = 45) and non-survivors (n = 21) is presented in Table 1. Non-survivors had significantly higher ISS (29 ± 9 vs. 22 ± 7; p = 0.006), SAPS II (48 ± 11 vs. 36 ± 8; p < 0.001), and Shock Index (1.21 ± 0.31 vs. 0.88 ± 0.22; p = 0.002). They also presented with significantly lower GCS (7 ± 4 vs. 11 ± 3; p < 0.001), hemoglobin (4.9 ± 1.5 vs. 7.2 ± 1.8 g/dL; p < 0.001), and platelet count (82 ± 40 vs. 145 ± 52 ×10³/mm³; p = 0.004). Coagulation disorders (15/21 [71%] vs. 13/45 [29%]; p = 0.001), hypothermia (13/21 [62%] vs. 10/45 [22%]; p = 0.002), and TBI (13/21 [62%] vs. 7/45 [16%]; p = 0.001) were significantly more frequent among non-survivors. Transfusion requirements and the delay to hemorrhage control were also significantly higher in the non-survivor group. Variables independently associated with in-hospital mortality by multivariate logistic regression are presented in Table 2.

Table 1: Comparison of clinical and biological characteristics between survivors and non-survivors.

Variable	Survivors (n=45)	Non-Survivors (n=21)	Test statistic	p-value
Age (years)	34 ± 12	41 ± 15	t = 2.04	0.048
Male sex, n (%)	33 (73%)	18 (86%)	Fisher's exact test	0.190
Female sex, n (%)	12 (27%)	3 (14%)	—	—
ISS	22 ± 7	29 ± 9	U = 285	**0.006**
SAPS II	36 ± 8	48 ± 11	t = 5.02	**< 0.001**
GCS < 8, n (%)	11 (24%)	14 (67%)	χ^2 = 10.85	**< 0.001**
Mean GCS	11 ± 3	7 ± 4	t = 4.53	**< 0.001**
Shock Index	0.88 ± 0.22	1.21 ± 0.31	U = 260	**0.002**
Hemoglobin (g/dL)	7.2 ± 1.8	4.9 ± 1.5	t = 5.08	**< 0.001**
Lactates (mmol/L)	3.8 ± 1.6	6.1 ± 2.3	U = 150	**< 0.001**
Platelets (×10³/mm³)	145 ± 52	82 ± 40	U = 263	**0.004**
Coagulation disorders, n (%)	13 (29%)	15 (71%)	χ^2 = 10.61	**0.001**
Hypothermia, n (%)	10 (22%)	13 (62%)	χ^2 = 9.93	**0.002**
TBI, n (%)	7 (16%)	13 (62%)	χ^2 = 14.56	**< 0.001**
Crystalloids (L)	2.4 ± 0.9	3.1 ± 1.2	U = 320	0.030
PRBC (units)	4.2 ± 2.1	8.6 ± 3.4	U = 120	**< 0.001**
Platelets (units)	3.8 ± 2.0	7.2 ± 3.1	U = 220	**0.001**
FFP (units)	3.5 ± 1.9	7.8 ± 3.6	U = 130	**< 0.001**
Time to hemostasis (min)	72 ± 25	112 ± 35	U = 200	**< 0.001**

Continuous variables are expressed as mean ± SD. Normally distributed variables were compared using Student's t-test (t), while non-normally distributed variables were compared using the Mann–Whitney U test (U). Categorical variables are expressed as n (%) and compared using the Chi-squared (χ^2) test or Fisher's exact test when expected cell counts were < 5. ISS: Injury Severity Score; SAPS II: Simplified Acute Physiology Score II; GCS: Glasgow Coma Scale; TBI: Traumatic Brain Injury; PRBC: Packed Red Blood Cells; FFP: Fresh Frozen Plasma.

Multivariate Analysis

Multivariate logistic regression analysis identified three independent predictors of in-hospital mortality (Table 2). A GCS < 8 (OR 3.42; 95% CI 1.28-9.11; p = 0.014), Shock Index > 0.9 (OR 2.87; 95% CI 1.10-7.46; p = 0.031), and hemoglobin < 5 g/dL (OR 3.95; 95% CI 1.36-11.42; p = 0.011) were independently associated with increased mortality.

Table 2: Variables independently associated with in-hospital mortality by multivariate logistic regression.

Variable	Odds Ratio	95% CI	p-value
GCS < 8	3.42	1.28–9.11	0.014
Shock Index > 0.9	2.87	1.10–7.46	0.031
Hemoglobin < 5 g/dL	3.95	1.36–11.42	0.011

CI: Confidence Interval; GCS: Glasgow Coma Scale. Note: confidence intervals are relatively wide, reflecting the limited sample size. These associations should be considered exploratory.

Mortality by Etiology

Mortality was highest among polytrauma patients (15/37, 40.5%), followed by penetrating trauma (4/17, 23.5%) and gastrointestinal hemorrhage (2/12, 16.7%). Although a trend toward higher mortality in polytrauma was observed, the difference did not reach statistical significance (p = 0.09), likely due to the limited sample size. The concentration of deaths in the polytrauma subgroup (15 of 21 total deaths, 71%) suggests that the overall prognostic associations identified may be predominantly driven by this etiological category.

Timing of Death

Among the 21 non-survivors, early deaths occurring within the first 24 hours accounted for 60% (13/21), primarily due to refractory hemorrhagic shock. Late deaths occurring beyond 3-5 days accounted for 40% (8/21) and were predominantly attributable to multi-organ failure or nosocomial sepsis.

DISCUSSION

Principal Findings

In this retrospective analysis of 66 patients with mixed-etiology hemorrhagic shock, three bedside-accessible variables were independently associated with in-hospital mortality: a Glasgow Coma Scale below 8 (OR 3.42), a Shock Index exceeding 0.9 (OR 2.87), and an admission hemoglobin below 5 g/dL (OR 3.95). Together, these markers capture three complementary dimensions of the physiological insult—neurological reserve, cardiovascular compensation, and the volume of blood loss—and can be assessed within minutes of patient arrival without specialized equipment. However, the relatively wide confidence intervals (up to 11.42 for hemoglobin) reflect the limited statistical power of our 21-event model, and these associations should be regarded as hypothesis-generating rather than definitive. Importantly, because 71% of deaths occurred in polytrauma patients, the identified associations may primarily reflect trauma-related hemorrhagic shock physiology rather than non-traumatic etiologies such as gastrointestinal hemorrhage, and caution is warranted when extrapolating these findings beyond the trauma population.

Comparison with the Literature

The association between an elevated Shock Index and mortality is consistent with findings from several larger registries. Mutschler *et al.,* analyzing over 21,000 entries from the TraumaRegister DGU, demonstrated that an SI above 1.0 was associated with increasing transfusion requirements, higher ISS, and rising mortality across four SI-based patient classes [9]. Vandromme *et al.,* reported in a cohort of 8,111 trauma patients that an SI ≥ 0.9 at admission was independently associated with a need for massive transfusion and increased mortality [13]. Similar results were reported by Cannon *et al.,* who confirmed the utility of the shock index in predicting mortality in traumatically injured patients [14]. More recently, a systematic review and meta-analysis by Vang *et al.,* demonstrated a fourfold increased risk of in-hospital mortality in trauma patients with an initial SI ≥ 1.0 [15]. In our cohort, non-survivors had a significantly higher mean SI (1.21 vs. 0.88; p = 0.002), which aligns with these observations, though our study was not designed to compare SI with individual vital signs in terms of discriminative performance.

The prognostic significance of a depressed GCS likely reflects the high burden of concomitant traumatic brain injury (TBI) in our population: 62% of non-survivors had documented TBI versus 16% of survivors (p = 0.001). TBI was not retained in the final model, which we attribute to statistical overlap with GCS—the two variables measure closely related constructs. Similar findings were reported by Brohi *et al.,* who identified impaired consciousness as a consistent correlate of mortality in hemorrhaging trauma patients [16]. Ahun *et al.,* further demonstrated that the GCS, combined with age and arterial blood pressure, was a reliable predictor of mortality in major trauma patients presenting to the emergency department [17]. Maegele *et al.,* further demonstrated that early coagulopathy occurs in up to one-third of multiply injured patients and is independently associated with mortality, reinforcing the prognostic value of early clinical and biological markers at admission [18]. The association between severe admission anemia (Hb < 5 g/dL) and mortality is also well documented; Spahn *et al.,* emphasized in their European trauma guidelines that admission hemoglobin serves as a marker of cumulative blood loss and should trigger immediate activation of massive transfusion

protocols [6]. Kawai *et al.,* confirmed that initial hemoglobin levels in severe trauma patients were significantly associated with transfusion requirements and clinical outcomes [19].

Our transfusion strategy approximated a balanced 1:1:1 ratio of red cells, platelets, and plasma. The PROPPR trial demonstrated that this approach, compared with a 1:1:2 ratio, yielded superior hemostatic control within 24 hours despite similar 30-day mortality [20]. These findings are consistent with the PROMMTT study, which showed that earlier administration of plasma was associated with improved 6-hour survival in massively transfused trauma patients [21]. Non-survivors in our cohort required approximately twice the transfusion volume of survivors (8.6 vs. 4.2 units of PRBC; $p < 0.001$). This difference most likely reflects the greater severity of hemorrhage in these patients rather than any inadequacy of the resuscitation strategy—a distinction that the retrospective design of this study cannot fully resolve.

Interpretation and Context

Approximately 60% of deaths occurred within the first 24 hours, consistent with patterns reported in trauma registries [22]. The observed difference in time-to-hemostasis between survivors and non-survivors (72 vs. 112 minutes; $p < 0.001$) is noteworthy but requires careful interpretation: longer hemostasis times may simultaneously reflect greater injury complexity and contribute to worsening physiology. This variable likely reflects both injury severity and system-related delays, making causal interpretation uncertain. Causality cannot be established from these observational data, and this association is best viewed as clinically suggestive rather than proof of a modifiable effect. Similarly, the lower admission hemoglobin in non-survivors may partly reflect longer pre-hospital intervals—a plausible hypothesis in the Casablanca metropolitan context, where transport delays can be considerable—but this interpretation remains speculative in the absence of recorded pre-hospital time data.

The convergence of hypothermia (62% vs. 22%), coagulopathy (71% vs. 29%), and elevated lactate (6.1 vs. 3.8 mmol/L) among non-survivors illustrates the well-described self-reinforcing cycle in hemorrhagic shock [1,23,24]. In our setting, hypothermia during resuscitation may be partly attributable to room-temperature fluid infusion in cases where fluid warmers were not immediately available. Tranexamic acid (TXA) was part of our institutional hemorrhagic shock protocol during the study period (2022-2024), consistent with the evidence established by the CRASH-2 trial [25] and current European trauma guidelines [6]. However, the retrospective design of the present study did not allow us to evaluate TXA administration timing or its independent effect on outcomes in our cohort. Prospective assessment of TXA compliance and its impact on mortality in this population remains warranted. External validation of our findings in independent cohorts is required before clinical implementation of any derived risk stratification tool.

Limitations

Several constraints should be weighed against these findings. First, the retrospective, single-center design carries inherent risks of selection and information bias. Second, the cohort includes three distinct etiological groups (polytrauma, penetrating trauma, gastrointestinal hemorrhage) with differing pathophysiology and bleeding kinetics. Given that polytrauma accounted for 71% of deaths, the observed associations may be disproportionately driven by this subgroup, and their applicability to non-traumatic hemorrhagic shock remains uncertain. Third, the sample size of 66 patients yielding 21 events constrained our regression model to three covariates; additional predictors may have emerged from a larger cohort, and the wide confidence intervals reflect the limited statistical power. The study may be underpowered to detect smaller but clinically relevant associations. Notably, global severity scores such as ISS and SAPS II, although strongly associated with mortality in univariate analysis, could not be included in the final model due to the events-per-variable constraint and collinearity considerations, which may result in residual confounding. No Kaplan-Meier analysis was performed due to incomplete time-to-event data. Although tranexamic acid and balanced transfusion protocols were available during the study period (2022-2024), their systematic application and timing could not be reliably assessed from the retrospective records, and point-of-care viscoelastic testing (ROTEM/TEG) was not routinely used at our center during the entire study window. Finally, the exclusion of 157 of 223 screened patients, while methodologically justified, reduces generalizability. Despite these limitations, the identified associations are pathophysiologically coherent and consistent with the broader literature, providing a basis for future prospective evaluation.

CONCLUSIONS

In this retrospective cohort of 66 patients with mixed-etiology hemorrhagic shock managed at a Moroccan surgical ICU, the Shock Index, Glasgow Coma Scale, and admission hemoglobin were independently associated with in-hospital mortality. These three variables are available at the bedside within minutes of admission and may contribute to early risk stratification, though their predictive value requires confirmation in larger, prospective studies. The predominance of deaths within 24 hours and the observed association between prolonged time-to-hemostasis and mortality underscore the importance of optimizing organizational pathways for rapid hemorrhage control. Multicenter prospective studies across North African trauma centers are needed to validate these findings and determine whether they can

be integrated into a practical triage tool for this population.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Kaoutar Zirhirhi, Abdelhak Tissir, Othmane Joutey, Bouhouri Aziz, Sara Chabbar

Acquisition, analysis, or interpretation of data: Kaoutar Zirhirhi, Abdelhak Tissir, Othmane Joutey, Bouhouri Aziz, Sara Chabbar

Drafting of the manuscript: Kaoutar Zirhirhi, Abdelhak Tissir, Othmane Joutey, Bouhouri Aziz, Sara Chabbar

Critical review of the manuscript for important intellectual content: Kaoutar Zirhirhi, Abdelhak Tissir, Othmane Joutey, Bouhouri Aziz, Sara Chabbar

Supervision: Bouhouri Aziz

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study.

Animal subjects: All authors have confirmed that this study did not involve animal subjects or tissue.

Conflicts of interest: In compliance with the ICMJE uniform disclosure form, all authors declare the following:

Payment/services info: All authors have declared that no financial support was received from any organization for the submitted work.

Financial relationships: All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work.

Other relationships: All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

REFERENCES

1. Cannon JW. Hemorrhagic Shock. N Engl J Med. 2018, 378:370-379.
2. Kauvar DS, Lefering R, Wade CE: Impact of hemorrhage on trauma outcome: an overview of epidemiology, clinical presentations, and therapeutic considerations. J Trauma. 2006, 60:3-11.
3. Tien HC, Spencer F, Tremblay LN, Rizoli SB, Brenneman FD: Preventable deaths from hemorrhage at a level I Canadian trauma center. J Trauma. 2007, 62:142-146.
4. Holcomb JB, Jenkins D, Rhee P, *et al.,* Damage control resuscitation: directly addressing the early coagulopathy of trauma. J Trauma. 2007, 62 :307-310.
5. Duchesne JC, McSwain NE Jr, Cotton BA, *et al.,* Damage control resuscitation : the new face of damage control. J Trauma. 2010, 69:976-990.
6. Spahn DR, Bouillon B, Cerny V, *et al.,* The European guideline on management of major bleeding and coagulopathy following trauma: fifth edition. Crit Care. 2019, 23:98.
7. Shanthakumar D, Payne A, Leitch T, Alfa-Wali M: Trauma care in low- and middle-income countries. Surg J. 2021, 7:281-285.
8. World Health Organization. Global status report on road safety. (2018). Accessed: March 2026: https://www.who.int/publications/i/item/9789241565684.
9. Mutschler M, Nienaber U, Münzberg M, *et al.,* The Shock Index revisited – a fast guide to transfusion requirement? A retrospective analysis on 21,853 patients derived from the TraumaRegister DGU. Crit Care. 2013, 17:172.
10. Baker SP, O'Neill B, Haddon W Jr, Long WB: The Injury Severity Score: a method for describing patients with multiple injuries and evaluating emergency care. J Trauma. 1974, 14:187-196.
11. Teasdale G, Jennett B: Assessment of coma and impaired consciousness: a practical scale. Lancet. 1974, 2:81-84.
12. Figueiredo S, Taconet C, Harrois A, *et al.,* How useful are hemoglobin concentration and its variations to predict significant hemorrhage in the early phase of trauma? A multicentric cohort study. Ann Intensive Care. 2018, 8:76-10.
13. Vandromme MJ, Griffin RL, Kerby JD, *et al.,* Identifying risk for massive transfusion in the relatively normotensive patient: utility of the prehospital shock index. J Trauma. 2011, 70:384-390.
14. Cannon CM, Braxton CC, Kling-Smith M, *et al.,* Utility of the shock index in predicting mortality in traumatically injured patients. J Trauma. 2009, 67:1426-1430.
15. Vang M, Østberg M, Steinmetz J, Rasmussen LS: Shock index as a predictor for mortality in trauma patients: a systematic review and meta-analysis. Eur J Trauma Emerg Surg. 2022, 48:2559-2566.
16. Brohi K, Singh J, Heron M, Coats T: Acute traumatic coagulopathy. J Trauma. 2003, 54:1127-1130.
17. Ahun E, Köksal Ö, Sığırlı D, *et al.,* Value of the Glasgow coma scale, age, and arterial blood pressure score for predicting the mortality of major trauma patients presenting to the emergency

department. Ulus Travma Acil Cerrahi Derg. 2014, 20:241-247.
18. Maegele M, Lefering R, Yucel N, *et al.,* Early coagulopathy in multiple injury: an analysis from the German Trauma Registry on 8724 patients. Injury. 2007, 38:298-304.
19. Kawai Y, Fukushima H, Asai H, *et al.,* Significance of initial hemoglobin levels in severe trauma patients without prehospital fluid administration: a single-center study in Japan. Trauma Surg Acute Care Open. 2021, 6:000831.
20. Holcomb JB, Tilley BC, Baraniuk S, *et al.,* Transfusion of plasma, platelets, and red blood cells in a 1:1:1 vs a 1:1:2 ratio and mortality in patients with severe trauma: the PROPPR randomized clinical trial. JAMA. 2015, 315:471-482.
21. Holcomb JB, del Junco DJ, Fox EE, *et al.,* The Prospective, Observational, Multicenter, Major Trauma Transfusion (PROMMTT) study. JAMA Surg. 2013, 148:127-136.
22. Oyeniyi BT, Fox EE, Scerbo M, *et al.,* Trends in 1029 trauma deaths at a level 1 trauma center: Impact of a bleeding control bundle of care. Injury. 2017, 48:5-12.
23. Mitra B, Tullio F, Cameron PA, Fitzgerald M: Trauma patients with the triad of death. Emerg Med J. 2012, 29:622-625.
24. Moore HB, Moore EE, Liras IN, *et al.,* Trauma-induced coagulopathy. Nat Rev Dis Primers. 2021, 7:30.
25. CRASH-2 trial collaborators. Effects of tranexamic acid on death, vascular occlusive events, and blood transfusion in trauma patients with significant haemorrhage (CRASH-2): a randomised, placebo-controlled trial. Lancet. 2010, 376 :23-32.