INTERNATIONAL JOURNAL OF HEALTH & MEDICAL RESEARCH

ISSN(print): 2833-213X, ISSN(online): 2833-2148

Volume 05 Issue 01 January 2026

DOI : 10.58806/ijhmr.2026.v5i1n01

Page No. 1 - 4

Predictors of Mortality in Hemorrhagic Shock among Polytraumatized Patients

Belkhou Youssef[1], Benzakour Maria[2], Afkir Amine[3], Chabbar Sara[4], Zerhouni Amine[5], Fz Faouji[6], Mounir Anas[7], Bouhouri Aziz[8]

ABSTRACT

Introduction : Hemorrhagic shock remains a leading cause of early mortality in polytraumatized patients. Early identification of high-risk patients is essential to optimize management and improve outcomes.

Materials and Methods : Retrospective review 200 polytraumatized patients admitted to a surgical ICU

Results : This retrospective study included 200 polytraumatized patients admitted with systolic blood pressure < 90 mmHg between 2020 and 2023. Patients were predominantly young and male. Abdominal and cranial injuries were the most frequent. Clinical severity markers included tachycardia, hypoxia, low Glasgow Coma Scale, urinary disturbances, and hypothermia. Severe anemia (hemoglobin < 5 g/dL), thrombocytopenia, and coagulation abnormalities were significantly associated with mortality. Overall mortality was 37.5%.

Discussion : Poor prognosis was associated with age, trauma severity, abdominal or cranial injury, shock index > 0.9, neurological impairment, hypothermia, and major biological abnormalities. These findings are consistent with existing literature and highlight the importance of early severity assessment.

Conclusion : Early recognition of prognostic factors and optimization of prehospital and hospital trauma care are crucial to reduce mortality from hemorrhagic shock in polytraumatized patients.

KEYWORDS : Hemorrhagic shock / Polytrauma / Prognostic factors / Mortality / Massive transfusion / Prehospital care

INTRODUCTION : Hemorrhagic shock remains a leading cause of early mortality in polytraumatized patients, particularly during the first hours following severe trauma [1,2]. It is characterized by acute circulatory failure secondary to massive blood loss, resulting in reduced tissue perfusion and potential progression to multiple organ dysfunction [3]. In polytrauma, the coexistence of multiple injuries, as well as the mechanism and severity of trauma, increases diagnostic and therapeutic complexity[4]. Hemorrhagic shock accounts for nearly 40% of early preventable deaths within the first 24 hours after trauma, emphasizing the importance of early identification of prognostic factors to guide timely and appropriate management [5].

MATERIALS AND METHODS

This retrospective study included 200 polytraumatized patients admitted to the intensive care unit of Ibn Rochd University Hospital in Casablanca between January 2020 and December 2023. Hemorrhagic shock was defined by a systolic blood pressure below 90 mmHg associated with clinical signs of peripheral hypoperfusion. Patients with non-hemorrhagic shock, those who died on admission, or those not immediately admitted to the intensive care unit were excluded. Collected variables included epidemiological data, comorbidities, injury mechanisms, clinical presentation, biological and radiological findings, therapeutic interventions, and outcomes. Patients were classified into survivors and non-survivors, and comparative statistical analyses were performed using Epi Info 7 and MedCalc software. A p-value < 0.05 was considered statistically significant.

RESULTS

A total of 200 polytraumatized patients admitted with hemorrhagic shock were included in the analysis. The cohort was predominantly composed of young adults, with 67% of patients aged under 40 years, and showed a marked male predominance (male-to-female ratio 4:1). Road traffic accidents represented the main mechanism of injury. Abdominal trauma was the most frequently involved anatomical region (60%), followed by cranial trauma (30%) and extremity injuries (36%), often occurring in combination and reflecting the severity of polytrauma.

Impact Points in the Context of Trauma.

impact point	number	%
Cranial	63	31.8
thoracic	30	15
abdominal	124	62
Pelvic	34	17
Limbs	72	36

At admission, hemodynamic instability was prominent. Tachycardia was observed in 83% of patients, and an elevated shock index (> 0.9) was significantly more frequent among non-survivors. Neurological impairment was common, with 15% of patients presenting a Glasgow Coma Scale score below 9, particularly in cases of associated cranial injury. Respiratory compromise, defined by oxygen saturation < 92%, was noted in 26% of cases. Clinical signs of peripheral hypoperfusion were frequent, including urinary disturbances in 31% and hypothermia in 12%, both of which were significantly associated with increased mortality.

Biological abnormalities were profound. Severe anemia was documented in the majority of patients, with hemoglobin levels between 5 and 10 g/dL in 77% of cases and below 5 g/dL in 8%. Thrombocytopenia was observed in 90% of patients, and coagulation disorders were identified in 56%, consistent with trauma-induced coagulopathy. These abnormalities were significantly more prevalent among non-survivors.

Distribution of patients according to admission hemoglobin level.

hemoglobin	number	%
<5g	15	7.58
>10g	30	15.15%
5-10g	153	77.27%

Radiological investigations revealed cerebral lesions in 47% of patients, hepatic injuries in 35%, and splenic injuries in 32%, with cranial and abdominal lesions being strongly associated with poor outcomes.

Resuscitation management required aggressive fluid therapy and blood product transfusion. Crystalloid volumes exceeding 2 L/day were administered to 68% of patients. Massive transfusion was frequent, with more than 4 units of packed red blood cells given in 64% of cases, more than 6 units of platelet concentrates in 55%, and more than 6 units of fresh frozen plasma in 51%. Vasopressor support was required in 65% of patients. Overall mortality was 37.5%, and non-survivors had a significantly longer intensive care unit length of stay compared to survivors.

DISCUSSION

Hemorrhagic shock remains one of the leading causes of early mortality in polytraumatized patients and constitutes a major challenge for anesthesiologists involved in prehospital care, emergency resuscitation, and perioperative management[6]. Early deaths are primarily related to uncontrolled hemorrhage, whereas delayed mortality results from multiple organ dysfunction driven by prolonged hypoperfusion, ischemia–reperfusion injury, systemic inflammation, and trauma-induced coagulopathy[7].

The physiological response to hemorrhage is biphasic. The initial sympatho-excitatory phase preserves cerebral and coronary perfusion through tachycardia and peripheral vasoconstriction, followed by a sympatho-inhibitory phase characterized by vasodilation, hypotension, and paradoxical bradycardia[8]. Anesthetic induction during this period may precipitate abrupt hemodynamic collapse by blunting compensatory sympathetic mechanisms. Moreover, hemorrhagic shock profoundly alters the pharmacokinetics and pharmacodynamics of anesthetic agents, leading to increased plasma concentrations, enhanced drug sensitivity, and prolonged effects[9]. These changes mandate reduced dosing, careful titration, and close hemodynamic and neurological monitoring.

Several prognostic factors identified in this study—advanced age, severity of polytrauma, traumatic brain injury, abdominal injuries, shock index > 0.9, Glasgow Coma Scale < 9, hypothermia, oliguria, severe anemia, thrombocytopenia, and coagulation abnormalities—are directly relevant to anesthetic risk stratification[10]. Biological derangements such as elevated lactate levels and base deficit further reflect the severity of tissue hypoxia and predict adverse outcomes[11]. Radiological evidence of cerebral or solid organ injury indicates increased anesthetic vulnerability and the need for meticulous control of cerebral perfusion pressure and oxygen delivery.

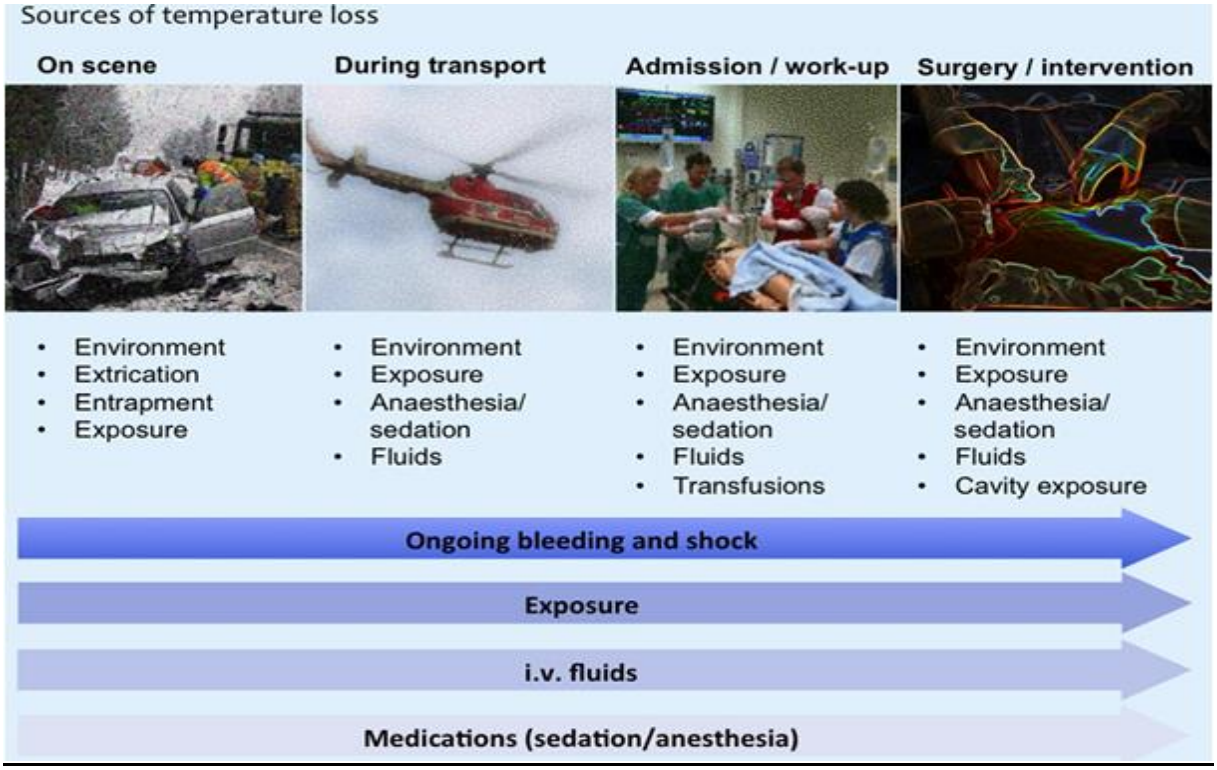

Figure : Sources of body Heat loss in trauma patients.

From an anesthetic perspective, optimal management relie on damage control resuscitation, early hemorrhage control, balanced blood product transfusion ratios, prevention of hypothermia and acidosis, and judicious use of vasopressors, particularly in patients with traumatic brain injury where maintenance of cerebral perfusion pressure is critical[12]. Agent selection should favor drugs with minimal hemodynamic impact and predictable pharmacological profiles. Early integration of anesthetic management within a multidisciplinary trauma strategy is essential to improve survival and functional outcomes in hemorrhagic shock.

CONCLUSION

Hemorrhagic shock in polytraumatized patients remains a life-threatening emergency requiring rapid, coordinated management to reduce mortality and severe complications. Diagnosis should begin in the prehospital phase through prompt clinical assessment, supported by adapted tools such as FAST ultrasound to detect internal bleeding and guide early therapeutic decisions. In the hospital setting, whole-body computed tomography and biological markers, particularly lactate levels, further assess shock severity. Management based on damage control resuscitation principles must start at the scene with early hemorrhage control, timely administration of blood products and pro-coagulant agents, and continue in hospital through close collaboration between anesthesia-critical care and surgical teams. Identification of prognostic factors highlights modifiable gaps in care and underscores the importance of both optimized trauma systems and preventive strategies, particularly targeting road traffic accidents.

REFERENCES

1) Sauaia A, Moore FA, Moore EE, et al. Epidemiology of trauma deaths: a reassessment. J Trauma. 1995;38(2):185–193.
2) Kauvar DS, Lefering R, Wade CE. Impact of hemorrhage on trauma outcome: an overview of epidemiology, clinical presentations, and therapeutic considerations.
3) J Trauma. 2006;60(6 Suppl):S3–S11.
4) Cannon JW. Hemorrhagic shock. N Engl J Med. 2018;378(4):370–379.
5) Trunkey DD.
6) Trauma. Accidental and intentional injuries account for more years of life lost in the U.S. than cancer and heart disease.
7) Sci Am. 1983;249(2):28–35.
8) Dutton RP, Stansbury LG, Leone S, et al.
9) Trauma mortality in mature trauma systems: are we doing better?
10) Anesthesiology. 2010;113(2):293–299.
11) 6.Spahn DR, Bouillon B, Cerny V, et al.
12) The European guideline on management of major bleeding and coagulopathy following trauma: fifth edition.
13) Crit Care. 2019;23:98.
14) Holcomb JB, Jenkins D, Rhee P, et al.

15) Damage control resuscitation: directly addressing the early coagulopathy of trauma.
16) J Trauma. 2007;62(2):307–310.
17) Guyton AC, Hall JE.
18) Textbook of Medical Physiology. 13th ed.
19) Philadelphia: Elsevier Saunders; 2016.
20) (Chapitre : circulatory shock)
21) 9.Egan TD, Shafer SL.
22) Target-controlled infusions for intravenous anesthetics: pharmacokinetic and pharmacodynamic considerations.
23) Anesthesiology. 2003;99(5):1211–1219.
24) 10.Charbit J, Tessler MJ, et al.
25) Prognostic value of shock index in the initial assessment of trauma patients.
26) Br J Anaesth. 2010;104(6):734–739.
27) 11.Jansen TC, van Bommel J, Schoonderbeek FJ, et al.
28) Early lactate-guided therapy in intensive care unit patients: a multicenter, open-label, randomized controlled trial.
29) Am J Respir Crit Care Med. 2010;182(6):752–761.
30) 12. CRASH-2 trial collaborators.
31) 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.
32) Lancet. 2010;376(9734):23–32.