Section: Medicine

Systematic Review Article

EARLY RECOGNITION AND EMERGENCY MANAGEMENT OF HAEMORRHAGIC SHOCK IN TRAUMA: A SYSTEMATIC REVIEW

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ABSTRACT

Background: Haemorrhagic shock (HS) is a leading cause of preventable trauma-related deaths worldwide. Timely recognition and rapid emergency management significantly improve survival, but compensatory mechanisms often mask early deterioration. **Objective:** To systematically review evidence on early recognition strategies, biomarkers, diagnostic tools, and emergency management of haemorrhagic shock in trauma patients.

Materials and Methods: We searched PubMed, Scopus, Web of Science, and Embase (January 1993–September 2025) using keywords including "haemorrhagic shock," "trauma," "biomarkers," "shock index," and "damage control resuscitation." Inclusion criteria: Trauma-related HS addressing diagnostic markers, prediction scores, imaging, or management strategies (RCTs {randomized control trials}, cohort studies, meta-analysis, guidelines); English language. Exclusion criteria: Non-English articles, studies lacking extractable data, or those without clear outcome measures, editorials, commentaries, conference abstracts excluded. Titles/abstracts and full texts screened; data extracted on diagnostic accuracy, biomarker performance, and treatment outcomes. Qualitative synthesis performed due to heterogeneity. PRISMA 2020 guidelines followed.

Results: From 3,847 records found and twenty-four studies were included. Clinical signs alone unreliable for early HS diagnosis. Key findings: shock index >0.9, lactate >4 mmol/L, base deficit <-6, syndecan-1, and copeptin enhance occult shock detection. POCUS (Point of care ultrasound), TEG (thromboelastography), ABC score (assessment of blood consumption score). TIC (trauma induced coagulopathy), SI (shock index) and TIC improve triage. Management: permissive hypotension (SBP 80-90 mmHg), 1:1:1 transfusion (PROPPR {pragmatic, Randomized Optimal Platelet and Plasma Ratios} trial), whole blood, TXA (Tranexamic Acid) (CRASH-2), and haemorrhage control reduce mortality.

Conclusions: Multimodal integration of biomarkers (lactate, base deficit, syndecan-1), physiologic scores (SI, ABC), and point-of-care diagnostics (POCUS, TEG) enables accurate early HS recognition. Damage control resuscitation remains the evidence-based cornerstone.

Keywords: Haemorrhagic shock, trauma, biomarkers, damage control resuscitation.

INTRODUCTION

Haemorrhagic shock (HS) remains one of the most time-sensitive emergencies in trauma care and contributes substantially to early preventable deaths. Trauma accounts for nearly five million global deaths annually, with uncontrolled haemorrhage responsible for 30–40% of trauma-related mortality, making it one of the leading causes of potentially survivable deaths (Sauaia et al.).[1]

The early recognition of haemorrhagic shock is difficult due to compensatory physiological mechanisms that maintain blood pressure in spite of

substantial blood loss. Reliance solely on clinical signs, such as tachycardia and hypotension, leads to delayed diagnosis, particularly in pre-hospital settings. Patients with a GCS (Glasgow Coma Scale) =15 presented normotensive and with a HR of 88/min, whereas patients with a GCS<12 showed a slight reduced SBP of 117mmHg and HR was unaltered. (Mutschler M et al.).[2]

This systematic review evaluates the evidence on early recognition strategies—including biomarkers—and emergency management of haemorrhagic shock.

MATERIALS AND METHODS

Database Search

This systematic review was conducted and reported in accordance with the PRISMA 2020 guidelines. A comprehensive search of PubMed, EMBASE, Scopus, and Web of Science was carried using keywords including: "haemorrhagic shock," "trauma," "occult shock," "shock index," "lactate," "syndecan-1," "damage control resuscitation," "permissive hypotension," and "massive transfusion."

Inclusion Criteria

- Trauma-related haemorrhagic shock
- Diagnostic markers, prediction scores, imaging tools, or management strategies
- RCTs, cohort studies, meta-analysis, guidelines
- Articles published in English

Exclusion Criteria

- Non-English articles, studies lacking extractable data, or those without clear outcome measures, editorials, commentaries, conference abstracts.

Titles, abstracts, and full texts were independently screened by two reviewers, with disagreements resolved by consensus. Data were collected on diagnostic accuracy, biomarker performance, hemodynamic indices, imaging modalities, and treatment outcomes. Due to heterogeneity, a qualitative synthesis was prepared.

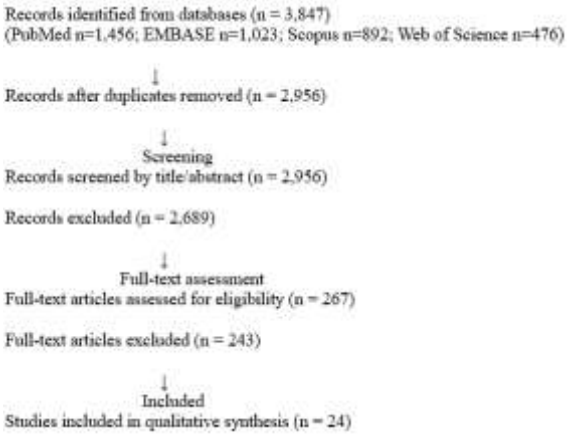

Figure 1: PRISMA Flow chart of Study Selection Process

RESULTS

1. Pathophysiology of Haemorrhagic Shock

Haemorrhagic shock results from acute blood volume loss leading to impaired oxygen delivery and cellular hypoxia. The progression occurs in three phases:

➢ **Compensated Shock**

- Activation of sympathetic nervous system
- Tachycardia, vasoconstriction
- Normal or near-normal blood pressure
- Reduced skin perfusion

➢ **Decompensated Shock**

- Hypotension
- Worsening tachycardia
- Metabolic acidosis
- Altered mental status
- Endothelial dysfunction and glycocalyx shedding

➢ **Irreversible Shock**

- Persistent hypoperfusion despite resuscitation
- Lactic acidosis, coagulopathy, organ failure

Understanding these stages supports targeted early intervention.

2. Early Recognition of Haemorrhagic Shock

Clinical Indicators

For hypotensive patients with penetrating torso injuries, delay of aggressive fluid resuscitation until operative intervention improves the outcome (Bickell et al.).[3]

3. Biomarkers in Early Recognition of Haemorrhagic Shock

Biomarkers have emerged as crucial components of early diagnosis, especially in detecting occult shock not apparent through vital signs.

Table 1: Biomarkers for Early Detection of Haemorrhagic Shock

Biomarker	Evidence / Significance	Clinical Utility
Lactate	Elevated lactate correlated with organ failure and mortality (Manikis et al.)[4]	Early recognition, prognostication
Base Deficit (BD)	Predicts transfusion needs and depth of shock (Davis et al.)[5]	Guides resuscitation
Copeptin	Rapid rise in acute illness and shock (Nickel et al.)[6]	Early indicator of circulatory stress
Syndecan-1		

	Reflects endothelial glycocalyx degradation, associated with severe shock (Johansson et al.) [7]	Predicts coagulopathy and massive transfusion
D-dimer	Elevated in trauma (Hazarika A et al.) [8]	good screening tool for coagulation activation
IL-6 (interleukin-6)	Correlates with injury severity and mortality (Frink et al.) [9]	Marker of systemic inflammatory response
Haemoglobin variability (HbV)	Early dynamic change predicts ongoing bleeding (Figueiredo S et al.) [10]	Useful in serial monitoring

3.1 Lactate

Lactate is a widely used early biomarker indicating tissue hypoxia. Elevated levels (>4 mmol/L) predict severe shock and mortality. Serial lactate clearance is associated with improved outcomes (Manikis et al.).[4]

3.2 Base Deficit

A base deficit worse than −6 correlates with increased transfusion needs, shock severity, and mortality (Davis et al.).[5]

3.3 Copeptin

As a surrogate for vasopressin, copeptin rises dramatically in early shock states and is considered a highly sensitive early physiological marker (Nickel et al.).[6]

3.4 Syndecan-1

Syndecan-1 indicates endothelial glycocalyx degradation, a key event in shock-induced coagulopathy. High levels are strongly predictive of massive transfusion and mortality (Johansson et al.).[7]

3.5 D-dimer and IL-6

These inflammatory and coagulation markers reflect trauma-induced hyper fibrinolysis (Hazarika A et al.),[8] and systemic inflammation (Frink et al.).[9]

4. Diagnostic Tools and Scoring Systems

4.1 Shock Index (SI)

With apparently stable vital signs, an abnormal elevation of the SI to more than 0.9 was associated with an illness that was treated immediately, admission to the hospital, and intensive therapy on admission (Rady et al.).[11]

4.2 Ultrasound

Ultrasound is rapid and reliable for detecting free fluid in thoracic and abdominal cavities (Rozycki et al.).[12]

4.3 NIRS (Near-infrared spectroscopy)

Near-infrared spectroscopy (NIRS) can continuously and noninvasively monitor tissue oxygen saturation (StO2) in muscle and may be an indicator of shock severity (Cohn SM et al.).[13]

4.4 ABC Score (assessment of blood consumption score)

The ABC score, which uses nonlaboratory, nonweighted parameters, is a simple and accurate in identifying patients who will require MT as compared with those previously published scores. (Nunez TC et al.).[14]

4.5 Machine Learning Tools

(Lee HS et al.) highlighted role of machine learning tools to predict the risk of ICH (intracranial haemorrhage) progression using initial CT scans and identify clinical factors associated with this progression.[15]

5. Emergency Management of Haemorrhagic Shock

Emergency management is guided by Damage Control Resuscitation (DCR) principles.

5.1 Permissive Hypotension

It targets SBP 80–90 mmHg in patients without traumatic brain injury. Reduces bleeding and improves survival in penetrating trauma (Bickell et al.).[3]

5.2 Haemostatic Resuscitation

Balanced Transfusion Ratio (1:1:1)

In severely injured patients with major bleeding, a 1:1:1 transfusion strategy did not reduce 24-hour or 30-day mortality compared with a 1:1:2 ratio, but it improved early haemostasis and reduced deaths from exsanguination. Despite greater plasma and platelet use, no additional safety concerns were observed (Holcomb et al.).[16]

Whole Blood Resuscitation

Warm fresh whole blood improves survival by providing all clotting components simultaneously. It is proven effective in US Military combat casualty patients (Spinella et al.).[17]

Restricting Crystalloids

Elements of volume resuscitation from haemorrhagic shock, such as amount of blood product and crystalloid administration, have been shown to be associated with Multiple Organ Dysfunction (MOD) may be reflecting overall resuscitation requirements and burden of injury rather than independent causation (Brakenridge SC et al.).[18]

5.3 Tranexamic Acid (TXA)

Early administration of TXA safely reduced the risk of death in bleeding trauma patients and is highly cost-effective (Roberts I et al.).[19]

5.4 Calcium Supplementation

(Mehr et al.) underscore complex role of citrate in massive transfusion, and the contribution of hypocalcemia to mortality is becoming recognized.[20]

5.5 Coagulation Monitoring Tools

TEG (thromboelastography)

The use of TEG (or similar assays) aids the earlier identification of defect of coagulation and TEG-guided protocol appears to improve survival while reducing overall product use; trials are in progress, which will continue to inform the evidence base (Howley IW et al.).[21]

5.6 Pre-hospital Interventions

- Tourniquets

- Haemostatic dressings
- Pre-hospital blood transfusion

These interventions significantly improve survival (Berry et al.).[22]

5.7 Definitive Haemorrhage Control

Damage Control Surgery (DCS)

It involves rapid bleeding control, temporary closure, and staged repair.

REBOA (Resuscitative endovascular balloon occlusion of the aorta)

The use of REBOA provides for temporary haemorrhage control and improved hemodynamics until definitive surgical or endovascular haemorrhage control can be achieved (Osborn LA et al.).[23]

DISCUSSION

This review synthesizes evidence demonstrating that early recognition of haemorrhagic shock must go beyond traditional clinical markers. Biomarkers—particularly lactate, base deficit, copeptin, and syndecan-1—play a major role in detecting occult shock, especially during the compensated phase where vital signs may remain normal. The analysis confirms fibrinogen, thrombin, and APC as diagnostic blood markers that help identify and determine the outcomes of TIC (Rashid NUA et al.).[24]

Point-of-care modalities such as Ultrasound, NIRS, and viscoelastic testing enhance diagnostic performance. Scoring systems (ABC, SI, TIC) assist triage and predict transfusion requirements.

Damage control resuscitation principles—including permissive hypotension, balanced transfusion, whole blood, early TXA, and minimal crystalloid use—have shown consistent survival benefit.

Ongoing advancements such as AI-driven prediction models and next-generation biomarkers hold promise for further improvement.

CONCLUSION

Early recognition and emergency management of traumatic haemorrhagic shock require a multimodal approach integrating clinical assessment with biomarkers, imaging, and physiologic scoring. Damage control resuscitation is strongly supported by evidence and remains the cornerstone of therapy. Future research should focus on personalized resuscitation using real-time physiologic monitoring and biomarker-guided interventions.

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REFERENCES

1. Sauaia A, Moore FA, Moore EE, Moser KS, Brennan R, Read RA, Pons PT. Epidemiology of trauma deaths: a reassessment. J Trauma. 1995 Feb;38(2):185-93. doi: 10.1097/00005373-199502000-00006. PMID: 7869433.
2. Mutschler M, Nienaber U, Brockamp T, Wafaisade A, Wyen H, Peiniger S, Paffrath T, Bouillon B, Maegele M; TraumaRegister DGU. A critical reappraisal of the ATLS classification of hypovolaemic shock: does it really reflect clinical reality? Resuscitation. 2013 Mar;84(3):309-13. doi: 10.1016/j.resuscitation.2012.07.012. Epub 2012 Jul 24. PMID: 22835498.
3. Bickell WH, Wall MJ Jr, Pepe PE, Martin RR, Ginger VF, Allen MK, Mattox KL. Immediate versus delayed fluid resuscitation for hypotensive patients with penetrating torso injuries. N Engl J Med. 1994 Oct 27;331(17):1105-9. doi: 10.1056/NEJM199410273311701. PMID: 7935634.
4. Manikis P, Jankowski S, Zhang H, Kahn RJ, Vincent JL. Correlation of serial blood lactate levels to organ failure and mortality after trauma. Am J Emerg Med. 1995 Nov;13(6):619-22. doi: 10.1016/0735-6757(95)90043-8. PMID: 7575797.
5. Davis JW, Parks SN, Kaups KL, Gladen HE, O'Donnell-Nicol S. Admission base deficit predicts transfusion requirements and risk of complications. J Trauma. 1996 Nov;41(5):769-74. doi: 10.1097/00005373-199611000-00001. PMID: 8913202.
6. Nickel CH, Bingisser R, Morgenthaler NG. The role of copeptin as a diagnostic and prognostic biomarker for risk stratification in the emergency department. BMC Med. 2012 Jan 20;10:7. doi: 10.1186/1741-7015-10-7. PMID: 22264220; PMCID: PMC3275505.
7. Johansson PI, Stensballe J, Rasmussen LS, Ostrowski SR. A high admission syndecan-1 level, a marker of endothelial glycocalyx degradation, is associated with inflammation, protein C depletion, fibrinolysis, and increased mortality in trauma patients. Ann Surg. 2011 Aug;254(2):194-200. doi: 10.1097/SLA.0b013e318226113d. PMID: 21772125.
8. Hazarika A, Kumar M, Ahluwalia J, Khurana BJK, Mahajan V, Bhatia N, Naik N, Kumar D. D-dimer levels at the time of admission to hospital as a predictor of outcome in trauma patients: A prospective observational study. Saudi J Anaesth. 2025 Jul-Sep;19(3):303-308. doi: 10.4103/sja.sja_601_24. Epub 2025 Jun 16. PMID: 40642615; PMCID: PMC12240523.
9. Frink M, van Griensven M, Kobbe P, Brin T, Zeckey C, Vaske B, Krettek C, Hildebrand F. IL-6 predicts organ dysfunction and mortality in patients with multiple injuries. Scand J Trauma Resusc Emerg Med. 2009 Sep 27;17:49. doi: 10.1186/1757-7241-17-49. PMID: 19781105; PMCID: PMC2763001.
10. Figueiredo S, Taconet C, Harrois A, Hamada S, Gauss T, Raux M, Duranteau J; Traumabase Group. 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 Jul 6;8(1):76. doi: 10.1186/s13613-018-0420-8. PMID: 29980953; PMCID: PMC6035120.
11. Rady MY, Smithline HA, Blake H, Nowak R, Rivers E. A comparison of the shock index and conventional vital signs to identify acute, critical illness in the emergency department. Ann Emerg Med. 1994 Oct;24(4):685-90. doi: 10.1016/s0196-0644(94)70279-9. Erratum in: Ann Emerg Med 1994 Dec;24(6):1208. PMID: 8092595.
12. Rozycki GS, Ochsner MG, Jaffin JH, Champion HR. Prospective evaluation of surgeons' use of ultrasound in the evaluation of trauma patients. J Trauma. 1993 Apr;34(4):516-

26; discussion 526-7. doi: 10.1097/00005373-199304000-00008. PMID: 8487337.
13. Cohn SM, Nathens AB, Moore FA, Rhee P, Puyana JC, Moore EE, Beilman GJ; StO2 in Trauma Patients Trial Investigators. Tissue oxygen saturation predicts the development of organ dysfunction during traumatic shock resuscitation. J Trauma. 2007 Jan;62(1):44-54; discussion 54-5. doi: 10.1097/TA.0b013e31802eb817. PMID: 17215732.
14. Nunez TC, Voskresensky IV, Dossett LA, Shinall R, Dutton WD, Cotton BA. Early prediction of massive transfusion in trauma: simple as ABC (assessment of blood consumption)? J Trauma. 2009 Feb;66(2):346-52. doi: 10.1097/TA.0b013e3181961c35. PMID: 19204506.
15. Lee HS, Kim JH, Son J, Park H, Choi J. Machine learning models for predicting early hemorrhage progression in traumatic brain injury. Sci Rep. 2024 May 22;14(1):11690. doi: 10.1038/s41598-024-61739-3. PMID: 38778144; PMCID: PMC11111696.
16. Holcomb JB, Tilley BC, Baraniuk S, Fox EE, Wade CE, Podbielski JM, del Junco DJ, Brasel KJ, Bulger EM, Callcut RA, Cohen MJ, Cotton BA, Fabian TC, Inaba K, Kerby JD, Muskat P, O'Keeffe T, Rizoli S, Robinson BR, Scalea TM, Schreiber MA, Stein DM, Weinberg JA, Callum JL, Hess JR, Matijevic N, Miller CN, Pittet JF, Hoyt DB, Pearson GD, Leroux B, van Belle G; PROPPR Study Group. 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 Feb 3;313(5):471-82. doi: 10.1001/jama.2015.12. PMID: 25647203; PMCID: PMC4374744.
17. Spinella PC, Perkins JG, Grathwohl KW, Beekley AC, Holcomb JB. Warm fresh whole blood is independently associated with improved survival for patients with combat-related traumatic injuries. J Trauma. 2009 Apr;66(4 Suppl):S69-76. doi: 10.1097/TA.0b013e31819d85fb. PMID: 19359973; PMCID: PMC3126655.
18. Brakenridge SC, Phelan HA, Henley SS, Golden RM, Kashner TM, Eastman AE, Sperry JL, Harbrecht BG, Moore EE, Cuschieri J, Maier RV, Minei JP; Inflammation and the Host Response to Injury Investigators. Early blood product and crystalloid volume resuscitation: risk association with multiple organ dysfunction after severe blunt traumatic injury. J Trauma. 2011 Aug;71(2):299-305. doi: 10.1097/TA.0b013e318224d328. PMID: 21825930; PMCID: PMC3716363.
19. Roberts I, Shakur H, Coats T, Hunt B, Balogun E, Barnetson L, Cook L, Kawahara T, Perel P, Prieto-Merino D, Ramos M, Cairns J, Guerriero C. The CRASH-2 trial: a randomised controlled trial and economic evaluation of the effects of tranexamic acid on death, vascular occlusive events and transfusion requirement in bleeding trauma patients. Health Technol Assess. 2013 Mar;17(10):1-79. doi: 10.3310/hta17100. PMID: 23477634; PMCID: PMC4780956.
20. MEHR, Noah; FONG, Erik; CARLL, Timothy 2025. Citrate & Hypocalcemia in Massive Transfusion. Medical Research Archives, [S.l.], v. 13, n. 8, aug. 2025. ISSN 2375-1924.
21. Howley IW, Haut ER, Jacobs L, Morrison JJ, Scalea TM. Is thromboelastography (TEG)-based resuscitation better than empirical 1:1 transfusion? Trauma Surg Acute Care Open. 2018 Jan 8;3(1):e000140. doi: 10.1136/tsaco-2017-000140. PMID: 29766129; PMCID: PMC5887764.
22. Berry, C., Gallagher, J. M., Goodloe, J. M., Dorlac, W. C., Dodd, J., & Fischer, P. E. (2023). Prehospital Hemorrhage Control and Treatment by Clinicians: A Joint Position Statement. Prehospital Emergency Care, 27(5), 544–551.
23. Osborn LA, Brenner ML, Prater SJ, Moore LJ. Resuscitative endovascular balloon occlusion of the aorta: current evidence. Open Access Emerg Med. 2019 Jan 14; 11:29-38. doi: 10.2147/OAEM.S166087. PMID: 30666171; PMCID: PMC6336022.
24. Rashid NUA, Nasir AA, Fattani B, Minhas M, Tanvir S, Pathan S, Barkha F, Nangdev P, Khalid A. Evaluation of Trauma-Induced Coagulopathy by Systematic Insights Into Pathophysiology and Advances in Emergency Resuscitation. Cureus. 2025 May 10;17(5):e83839. doi: 10.7759/cureus.83839. PMID: 40491626; PMCID: PMC12147678.