



REVIEW

Hemorrhagic shock: A mini review

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ABSTRACT

Hemorrhagic shock is a life-threatening condition caused by acute intravascular volume loss, leading to systemic tissue hypoperfusion, cellular hypoxia, and circulatory failure. It remains a leading cause of preventable death after injury worldwide. This review synthesizes current evidence on the pathophysiology, classification, diagnosis, and clinical management of hemorrhagic shock. Pathophysiologically, acute blood loss reduces cardiac preload and output, triggering neurohormonal compensation, anaerobic metabolism, and progression toward the lethal triad of trauma: hypothermia, metabolic acidosis, and coagulopathy. Diagnostic evaluation relies on the Advanced Trauma Life Support (ATLS) classification system, point-of-care ultrasound, and serial biomarker assessment. Contemporary clinical management emphasizes damage control resuscitation (DCR), including early definitive hemostasis, restrictive crystalloid administration, permissive hypotension, balanced transfusion of blood products, and damage control surgery. Prompt recognition and protocol-driven resuscitation significantly improve survival.

Keywords: hemorrhagic shock, damage control resuscitation, trauma, hemorrhage, ATLS

Introduction

Hemorrhagic shock is a medical emergency caused by acute, significant blood loss that leads to decreased intravascular volume, impaired tissue perfusion, and failure of the circulatory system to meet cellular oxygen requirements.^{1,2} This condition is the leading preventable cause of death in trauma patients and represents the most common form of hypovolemic shock encountered in clinical practice.³⁻⁵ Uncontrolled hemorrhage results in tissue hypoxia, cellular metabolic derangement, multiple organ failure, and death.^{6,7}

Globally, massive hemorrhage due to trauma contributes to nearly half of all trauma-related deaths. In addition to trauma, hemorrhagic shock may occur secondary to gastrointestinal bleeding, aneurysm rupture, obstetric complications, surgical procedures, or spontaneous bleeding in patients with coagulation disorders.^{8,9} Advances in modern resuscitation have transformed the management paradigm for hemorrhagic shock, shifting from exclusive crystalloid administration to damage control resuscitation. This strategy emphasizes early hemorrhage control, balanced blood product transfusion, and prevention of traumatic coagulopathy.^{10,11} This review summarizes current knowledge on the definition, epidemiology, etiology, risk factors, pathophysiology, classification, clinical manifestations, diagnosis, management, complications, and prognosis of hemorrhagic shock to inform clinical practice and education.

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Method

This manuscript was prepared as a narrative review. The literature search included surgical textbooks, clinical practice guidelines from trauma surgery professional organizations, and scientific articles addressing the physiology, diagnosis, and management of hemorrhagic shock. Sources comprised the *Advanced Trauma Life Support (ATLS) Student Course Manual*, 11th edition; *Schwartz's Principles of Surgery*; *Sabiston Textbook of Surgery*; *Bailey & Love's Short Practice of Surgery*; StatPearls reviews; and peer-reviewed journal articles on damage control resuscitation and the pathophysiology of hemorrhagic shock. Information from these sources was synthesized thematically to provide a comprehensive overview of hemorrhagic shock.

Discussion

Definition

Hemorrhagic shock is a form of hypovolemic shock caused by acute, significant blood loss that reduces intravascular volume, decreases ventricular preload, lowers cardiac output, and ultimately impairs tissue perfusion and oxygen delivery to vital organs.⁸ This mismatch between tissue oxygen demand and supply leads to cellular hypoxia that, if sustained, progresses to multiple organ dysfunction and death.¹² According to ATLS guidelines, any trauma patient presenting with hypotension should be presumed to have hemorrhage until another cause is identified.¹³

Epidemiology

Trauma remains a leading cause of death worldwide, particularly among individuals of productive age.⁸ The World Health Organization estimates that more than 5 million deaths annually are trauma-related, with massive hemorrhage contributing to approximately 30% to 40% of deaths from severe trauma.¹⁴ Most hemorrhage-related deaths occur within the first 6 hours after injury, a period known as the "golden hour" in trauma care.¹³ In low- and middle-income countries, mortality from hemorrhagic shock tends to be higher due to delays in patient transport, limited trauma center capacity, and constrained availability of blood products and trained personnel.⁸ Beyond trauma, hemorrhagic shock is an important cause of maternal mortality from postpartum hemorrhage, carries high mortality in ruptured abdominal aortic aneurysm, and contributes significantly to mortality from massive gastrointestinal bleeding in older adults.^{15–17}

Etiology and Risk Factors

Causes of hemorrhagic shock are categorized as traumatic or nontraumatic. Traumatic causes include solid organ injury (liver and spleen) from blunt abdominal trauma, penetrating injury to major blood vessels, pelvic fractures with retroperitoneal hemorrhage, bilateral femur fractures, and massive hemothorax from thoracic trauma.^{13,16,17} Nontraumatic causes include upper and lower gastrointestinal bleeding, postpartum hemorrhage, ruptured ectopic pregnancy, placental abruption, ruptured abdominal aortic aneurysm, and congenital or acquired coagulation disorders, including anticoagulant or antiplatelet use.^{8,15,17}

Risk factors that increase the likelihood of hemorrhagic shock include high-energy trauma, advanced age, anticoagulant use, hepatic dysfunction, coagulopathy, uncontrolled hypertension, aortic aneurysm, obstetric complications, and a history of major surgery.¹⁷ Older adults have reduced physiologic reserve and are more susceptible to hemodynamic decompensation even with relatively smaller blood losses; beta-blocker use may also mask early signs of shock.⁸

Pathophysiology

Acute blood loss decreases intravascular volume and venous return to the heart, reducing ventricular preload. By the Frank-Starling mechanism, decreased preload lowers stroke volume and cardiac output, thereby reducing oxygen delivery to tissues.¹² In compensation, the sympathetic nervous system activates, releasing epinephrine and norepinephrine, which produce tachycardia, increased myocardial contractility, and peripheral vasoconstriction. Concurrent activation of the renin-angiotensin-aldosterone system causes systemic vasoconstriction and sodium and water retention.¹⁸

Prolonged tissue hypoperfusion forces cells to shift from aerobic to anaerobic metabolism, generating lactate and metabolic acidosis, which serve as key indicators of shock severity.¹² In later stages, tissue hypoxia triggers release of inflammatory mediators such as tumor necrosis factor-alpha, interleukin-1, and interleukin-6, which increase capillary permeability and worsen endothelial dysfunction.¹⁸ This process is accompanied by trauma-induced coagulopathy, which, together with hypothermia and acidosis, forms the

lethal triad of trauma—three interrelated conditions that amplify one another and represent a major cause of death in severe hemorrhagic shock.^{14,19}

Classification

The ATLS classification divides hemorrhagic shock into four classes based on percentage of blood loss, vital sign changes, mental status, and urine output, assuming a total blood volume of approximately 7% of body weight in adults.^{13,17} Class I involves blood loss of less than 15% (<750 mL) with generally normal vital signs. Class II occurs with 15% to 30% blood loss (750–1500 mL), characterized by tachycardia (>100 beats/min), narrowed pulse pressure, and urine output of 20 to 30 mL/h. Class III involves 30% to 40% blood loss (1500–2000 mL) with overt hypotension, heart rate >120 beats/min, respiratory rate of 30 to 40 breaths/min, urine output of 5 to 15 mL/h, and altered mental status. Class IV, with blood loss exceeding 40% (>2000 mL), presents with severe hypotension, extreme tachycardia, oliguria to anuria, and marked depression of consciousness, accompanied by a high risk of multiple organ failure.¹³ Physiologic responses to blood loss may vary according to age, medication use, and comorbidities; therefore, comprehensive clinical assessment remains essential.⁸

Clinical Manifestations

Clinical manifestations depend on the volume and rate of blood loss, patient age, and physiologic compensatory capacity.⁸ Early symptoms include thirst, weakness, dizziness, palpitations, and anxiety; with progressive blood loss, patients may develop dyspnea, blurred vision, and decreased consciousness.¹⁷ Physical examination reveals tachycardia, peripheral vasoconstriction, cool and pale skin, and prolonged capillary refill; hypotension typically appears only in later stages due to compensatory mechanisms.¹³ Oliguria from reduced renal perfusion is an important indicator of organ hypoperfusion.¹² ATLS identifies five primary locations of hemorrhage in trauma patients that must be systematically evaluated: the thoracic cavity, abdominal cavity, retroperitoneum, pelvis, and long bones.¹³

Diagnosis

Diagnosis is established through a combination of history, physical examination, and adjunctive investigations to assess the degree of blood loss and identify the bleeding source.¹⁶ History should include the mechanism of injury, time of occurrence, anticoagulant use, and, in nontrauma cases, hematemesis, melena, or vaginal bleeding.¹⁵ Physical examination follows the ABCDE approach, with circulatory assessment including pulse rate, blood pressure, skin color and temperature, capillary refill, mental status, and urine output.¹³

Laboratory tests include hemoglobin and hematocrit, which may remain normal in early phases, as well as blood typing and crossmatching in preparation for transfusion.¹⁷ Arterial blood gas analysis and serum lactate levels assess the degree of metabolic acidosis and tissue hypoperfusion, whereas base deficit serves as an objective marker of resuscitation adequacy.¹⁸ Coagulation studies (prothrombin time, activated partial thromboplastin time, fibrinogen, and platelet count) are essential because coagulopathy frequently accompanies severe hemorrhagic shock.¹⁴ Imaging includes FAST to detect free fluid in the abdomen and pericardium in unstable patients, and contrast-enhanced computed tomography in hemodynamically stable patients.^{13,16}

Management

Management of hemorrhagic shock aims to maintain tissue oxygenation, control the bleeding source, and rapidly and safely restore circulating volume. The airway must be secured with high-concentration oxygen administration, and endotracheal intubation performed if ventilation is impaired or consciousness is depressed.¹³ Hemorrhage control is the highest priority, achieved through direct pressure and tourniquets for external bleeding, and surgery or embolization for internal bleeding.¹⁷

Fluid resuscitation should use limited volumes of warmed crystalloids, as excessive administration can cause hemodilution, tissue edema, hypothermia, and worsening coagulopathy.¹⁹ In massive hemorrhage, early blood transfusion is required with balanced ratios of packed red blood cells, fresh frozen plasma, and platelets approaching 1:1:1.¹⁴

Damage control resuscitation is the principal strategy to prevent the lethal triad of trauma. It includes the earliest possible hemorrhage control, crystalloid restriction, permissive hypotension, balanced blood product transfusion, hypothermia prevention, and coagulopathy correction, as outlined in current clinical

practice guidelines.^{10,14,19} Permissive hypotension targets a systolic blood pressure of approximately 80 to 90 mmHg until hemorrhage is controlled to avoid increasing intravascular pressure that could exacerbate bleeding.¹⁹ In patients with massive hemorrhage and severe physiologic instability, damage control surgery is performed as an initial abbreviated operation to stop bleeding and control contamination, followed by definitive surgery after stabilization in the intensive care unit.¹⁰

Complications

Prolonged tissue hypoperfusion can lead to acute kidney injury, acute respiratory distress syndrome from systemic inflammatory response, and disseminated intravascular coagulation that worsens hemorrhage.¹⁸ Late complications include bowel necrosis, hepatic dysfunction, neurologic impairment, and multiple organ dysfunction syndrome, which is a leading cause of death in patients who survive the initial trauma phase.⁸

Prognosis

Prognosis depends on the volume of blood loss, bleeding site and rate, patient age, comorbidities, and speed of medical intervention; early diagnosis and rapid hemorrhage control are the most critical determinants of treatment success.¹⁷ Patients with mild to moderate blood loss who receive prompt therapy generally have favorable outcomes, whereas massive hemorrhage accompanied by hypothermia, acidosis, and coagulopathy is associated with high mortality.¹⁷ Implementation of damage control resuscitation, massive transfusion protocols, and damage control surgery has been shown to reduce mortality in patients with hemorrhagic shock.¹⁰

Conclusion

Hemorrhagic shock is a medical emergency caused by acute blood loss that results in decreased intravascular volume, impaired tissue perfusion, and reduced oxygen delivery to vital organs, from both traumatic and nontraumatic causes. Its pathophysiology involves decreased cardiac output, neurohormonal compensation, anaerobic metabolism, and progression to the lethal triad of trauma if not promptly corrected. The ATLS classification remains the primary reference for assessing shock severity, whereas diagnosis is established through history, ABCDE physical examination, and adjunctive laboratory and imaging studies. Contemporary management emphasizes damage control resuscitation and damage control surgery as strategies to control hemorrhage, prevent coagulopathy, and reduce mortality. Early recognition and timely, appropriate intervention remain the keys to successful management of hemorrhagic shock.

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