Critical Loss

Resuscitation of the Patient in Hemorrhagic Shock

LESSON 24



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OBJECTIVES

On completion of this lesson, you should be able to:

1. Identify the key clinical features of hemorrhagic shock.
2. Detail appropriate laboratory and radiology tests to aid in the diagnosis and management of hemorrhagic shock.
3. Explain the principles of damage control resuscitation.
4. Describe the initial management of hemorrhagic shock and the components of a massive transfusion.
5. Detail the appropriate disposition for patients in hemorrhagic shock.

FROM THE EM MODEL

1.0 Signs, Symptoms, and Presentations
- 1.3 General
  - 1.3.42 Shock

CRITICAL DECISIONS

- How is hemorrhagic shock diagnosed?
- What laboratory and radiology tests are useful when managing hemorrhagic shock?
- How should patients in hemorrhagic shock be resuscitated?
- What treatment should be initiated immediately?
- What are the components of a massive transfusion protocol?
- What is the appropriate disposition for patients in hemorrhagic shock?

Patients in hemorrhagic shock are faced with an immediately life-threatening condition. Emergency physicians must be able to recognize this critical presentation early in the course, be prepared to provide optimal resuscitation, and arrange for definitive and expeditious care.

CASE PRESENTATIONS

CASE ONE

A 24-year-old woman arrives via ambulance after a high-speed motor vehicle collision in which she was the unrestrained driver. Her vital signs are blood pressure 70/30, heart rate 140, respiratory rate 36, and temperature 35.8°C (96.4°F). She has a Glasgow Coma Scale (GCS) score of 7, a scalp laceration, and obvious trauma to her face. She has bilateral breath sounds, bruising and abrasions to her left abdomen and flank, and an obvious deformity of her left lower leg.

CASE TWO

A 63-year-old man with a medical history of hypertension and hepatitis C presents with epigastric pain, nausea, and three episodes of emesis. His vital signs are blood pressure 88/54, heart rate 106, respiratory rate 18, temperature 37.3°C (99.1°F), and oxygen saturation 98% on room air. He is ill-appearing, diaphoretic, and sitting upright while actively retching into a plastic bag. He produces about 100 mL of coffee-ground emesis that contains dark red streaks. His abdomen is moderately distended with epigastric tenderness, and a fluid wave is present. A rectal examination reveals melena without frank red blood.

CASE THREE

A 68-year-old man with a history of hypertension arrives via ambulance with a complaint of abdominal pain. He says the pain started suddenly 2 hours ago without any triggering events and describes it as diffuse, sharp, and severe. He denies fever, chills, chest pain, shortness of breath, nausea, vomiting, and diarrhea. The paramedics recorded a blood pressure of 75/45 and a heart rate of 130 en route.

The patient had a brief syncopal episode while being transferred onto the stretcher. His vital signs are now blood pressure 79/43 and pulse rate 136. He appears to be in significant distress and is moaning and diaphoretic. He is tachycardic and has palpable but diminished femoral and dorsalis pedis pulses. He is tachypneic with the use of supraclavicular and intercostal accessory muscles. The abdominal examination is notable for distention, dullness to percussion, and a pulsating mass palpable at the umbilicus.

Shock is a condition characterized by inadequate organ perfusion due to an imbalance between the delivery and consumption of oxygen and metabolic substrates.[1] The causes of shock are divided into four categories: *hypovolemic*, *cardiogenic* (eg, myocardial infarction), *distributive* (eg, sepsis, neurogenic, anaphylactic), and *obstructive* (eg, tension pneumothorax, cardiac tamponade).

Hemorrhagic shock, a type of hypovolemic shock that is caused by acute blood loss, most commonly occurs with trauma. Every year, more than 5 million people worldwide die from trauma, which is the fifth leading cause of death in the United States.[2,3] Hemorrhage is the leading cause of trauma-associated death, and the majority of these patients die within a few hours of presentation.[3]

When the body loses a significant amount of blood volume, oxygen delivery is impaired, and cellular metabolism proceeds with anaerobic glycolysis, ultimately leading to lactate production and metabolic acidosis.[3] The proinflammatory mediators that are released by cells in a shock state damage endothelial cells, inducing tissue swelling and cellular death. The body further compensates by releasing endogenous catecholamines that increase the heart rate and cardiac output, a cascade that leads to peripheral vasoconstriction. As inadequate organ perfusion persists, acidosis and hypoxemia worsen, impairing peripheral vasoconstriction and eventually leading to cardiovascular collapse.

CRITICAL DECISION

How is hemorrhagic shock diagnosed?

Hemorrhagic shock is a clinical diagnosis that incorporates key historical information, examination findings, and laboratory data. The hallmark red flag is an acute precipitating event in which a large amount of blood loss occurs. The diagnosis is most commonly the result of blunt or penetrating trauma; however, it can also arise from disorders that cause symptoms such as hematemesis, hemoptysis, rectal bleeding or melena, vaginal bleeding, epistaxis, or easy bruising (*Table 1*).

Other concerning presentations include generalized weakness, syncope, confusion, and lethargy. The patient's vital signs can signal severe shock or be an early harbinger of developing shock. It is also important to gather as much information as possible about the patient's medical and surgical history, pregnancy status, use of anticoagulants or antiplatelet agents, and any history of bleeding diathesis.

Vital signs are largely dependent on the quantity of blood loss, classically divided into four classes of hemorrhagic shock.[3] Although tachycardia typically develops after 15% to 30% of blood volume has been lost, blood pressure remains normal due to physiological compensation. Therefore, any tachycardic trauma patient is in shock until proven otherwise.[3] However, the heart rate may remain normal due to certain medications (eg, beta-blockers, calcium channel blockers), an excellent baseline physiological status, or vagal stimulation from significant intra-abdominal hemorrhage.

Hypotension is a late sign of significant hemorrhage that occurs after 30% to 40% of blood volume has been lost. A narrowed pulse pressure is a more sensitive finding, as early

sympathetic responses cause peripheral vasoconstriction to increase the diastolic pressure. Despite their widespread teaching, hemorrhage classifications are unreliable due to a variety of variables, such as the patient's age, comorbidities, medication use, and level of physiological compensation, all of which can differ based on the injuries sustained.

Data from a large trauma registry show that the interrelationship between vital sign derangements seen in the Advanced Trauma Life Support (ATLS) classifications occurs to a much lesser degree (*Table 2*).[4] Research suggests that the shock index (SI), defined as the ratio of heart rate to systolic blood pressure, may be a more reliable diagnostic variable. SI can be used to better risk-stratify patients for increased transfusion requirements and early mortality when using a cutoff score above 0.9.[5,6]

While the early recognition of hemorrhagic shock is important, emergency physicians must concurrently consider other etiologies that can cause or contribute to the patient's state of shock. For example, shock in a pedestrian struck by a motor vehicle is most likely due to hemorrhage caused by a high-grade splenic laceration. However, the patient may also have concomitant cardiac tamponade, a tension pneumothorax, a myocardial contusion, a spinal cord injury, or even sepsis (if presenting late).

Clinicians must remain vigilant for signs of shock when managing any patient with trauma or a history suggestive of hemorrhage. A physical examination and repeat vital sign measurements play crucial roles in diagnosing shock. In cases of trauma, the examination should be focused on any evidence of hemorrhage in the chest, abdomen, retroperitoneum, pelvis, long bones, or external compartments (*Table 3*).

The patient must be fully exposed when evaluating for areas of bleeding. Clinicians should be careful to assess patients for scalp lacerations, epistaxis, flank ecchymoses, scrotal enlargement, extremity deformities, a pulsatile abdominal mass, or an unstable pelvis. Decreased cerebral perfusion can present as weakness, syncope, or altered mental status. An examination of the skin may reveal diaphoresis, bruising, petechiae, cyanosis, delayed capillary refill, pallor, or a mottled appearance.

The complete clinical picture must be considered when evaluating for shock, the diagnosis of which depends on a combination of hemodynamic factors and evidence of vital organ hypoperfusion (eg, altered mental status or decreased urine output).

TABLE 1. Causes of Atraumatic Hemorrhagic Shock

Abdominal aortic aneurysm rupture
Pulmonary hemorrhage (eg, malignancy, bronchiectasis, tuberculosis)
Upper GI bleed (eg, esophageal or gastric varices, peptic ulcer disease, Mallory-Weiss syndrome)
Lower GI bleed (eg, diverticula, arteriovenous malformation, mass, aortoenteric fistula)
Pregnancy complications (eg, ruptured ectopic pregnancy, placental abruption/previa)

TABLE 2. ATLS Classes of Hemorrhagic Shock (for a 70-kg man)[2]

	Class I	Class II	Class III	Class IV
Blood loss (mL)	<750	750-1,500	1,500-2,000	>2,000
Blood loss (% blood volume)	<15%	15-30%	30-40%	>40%
Heart rate	<100	100-120	120-140	>140
Systolic blood pressure	Normal	Normal	Decreased	Decreased
Pulse pressure	Normal or increased	Decreased	Decreased	Decreased
Respiratory rate	14-20	20-30	30-40	>35
Urine output (mL/hr)	>30	20-30	5-15	Negligible
Mental status	Slightly anxious	Mildly anxious	Anxious, confused	Confused, lethargic

CRITICAL DECISION

What laboratory and radiology tests are useful when managing hemorrhagic shock?

Laboratory testing should include a complete blood count, arterial or venous blood gas measurements, chemistries, coagulation studies, type and crossmatching, and a urine or serum pregnancy test. While a normal hemoglobin level is unreassuring in patients with ongoing bleeding, it provides a baseline level to trend against throughout the evaluation. Acidosis seen on blood gas measurements can indicate the severity of the shock state. When available, thromboelastography can guide the transfusion strategy for a bleeding patient by measuring the viscoelastic properties of blood.[7] An electrocardiogram (ECG) also should be obtained in patients with tachycardia or when there is concern for cardiac contusions or myocardial ischemia. Patients who are in a prolonged shock state may show ischemic signs on an ECG, a finding commonly described as *demand* ischemia.

Radiology testing, when the patient's condition allows, can help pinpoint the location of the hemorrhage; however, it is crucial to exclude other etiologies of shock. Initial images include chest and pelvis x-rays to evaluate for fractures and hemopneumothoraces. An immediate extended Focused Assessment with Sonography for Trauma (eFAST) should be performed to identify intra-abdominal or pelvic free fluid, pericardial effusion, pleural effusion, or a pneumothorax. A Rapid Ultrasound in Shock (RUSH) examination can also aid in diagnosing other causes of shock by adding views of the inferior vena cava (to assess volume status) and the abdominal aorta (to assess for the presence of an aneurysm).[8,9] Diagnostic peritoneal lavage (DPL) has fallen out of favor due to the utility of ultrasound; however, it remains an option when evaluating for the presence of intraperitoneal hemorrhage.[3]

TABLE 3. Blood Loss Compartments in Trauma

External/Scalp
Chest
Abdomen
Retroperitoneum
Pelvis
Long bones

If the patient is stable enough for imaging, computed tomography (CT) is the most practical modality for evaluating hemorrhage in the setting of trauma.[10] CT of the spine, when indicated, is useful to evaluate for injuries that may cause spinal cord injury and neurogenic shock. In addition, CT angiography has diagnostic value for identifying vascular injuries, which may warrant surgical or interventional endovascular hemorrhage control. Plain films of the extremities should be obtained to evaluate for fractures, particularly of the femur, where a significant volume of blood can accumulate.

CRITICAL DECISION

How should patients in hemorrhagic shock be resuscitated?

While resuscitation protocols for patients in hemorrhagic shock continue to evolve, the process has yet to be fully established or standardized across hospitals or even countries.[10,11] The former practice of early aggressive fluid resuscitation with crystalloid solutions (eg, normal saline or lactated Ringer's solution) is believed to worsen hemorrhage by dislodging potential clots and causing dilutional coagulopathy, hypothermia, and metabolic acidosis. Perioperative aggressive hydration has been shown to increase the risk of death associated with certain conditions, including ruptured abdominal aortic aneurysms.[12]

The current practice of "damage control resuscitation," which developed as the result of military advancements, is now widely accepted.[13] This management paradigm incorporates permissive hypotension, hemostatic resuscitation with a fixed-ratio blood transfusion, and damage control surgery.[14] The goal is to prevent and reverse hemorrhagic shock and to avoid the lethal triad of trauma: coagulopathy, hypothermia, and acidosis.

Permissive Hypotension

In most patients, permissive hypotension is recommended with a lower systolic blood pressure goal between 80 and 100 mm Hg. Although vital signs are readily accessible, other end points that can guide ongoing resuscitation include changes in mental status, urine output, vital signs, lactate levels, and base excess. Research is mixed regarding the mortality benefits of hypotensive resuscitation, but in one specific subset of penetrating torso injuries, improved mortality was found when aggressive fluid resuscitation was delayed until hemostasis had been achieved definitively.[15-17]

Which subset of patients would benefit most from permissive hypotension has not been clearly established. However, hypotension is associated with worse clinical outcomes in those with severe traumatic brain injury (TBI). Therefore, early control of any bleeding allows the clinician to maintain a blood pressure that promotes cerebral perfusion and prevents a secondary brain insult.[18]

In the hypotensive patient, ATLS recommends initiating resuscitation with 1 to 2 L of fluids.[2] While numerous studies have compared the various types of fluids, crystalloid solutions are recommended when beginning any resuscitation. No clear benefits are seen with the administration of colloid solutions.[19,20] Nevertheless, crystalloid solutions are not ideal for providing intravascular volume during continued resuscitation and can contribute to fluid accumulation and metabolic acidosis.

Hemostatic Resuscitation

Patients in hemorrhagic shock are at increased risk for developing trauma-associated coagulopathy; resuscitation must aim to reverse coagulopathy, hypothermia, and acidosis.[21] In addition to packed red blood cells, emergency physicians must provide fresh frozen plasma and platelets to mitigate coagulopathy and consider other products (eg, prothrombin complex concentrates, factor replacement, protamine) to reverse the use of any anticoagulant or antiplatelet agents. It may be necessary to initiate a massive transfusion protocol for patients with ongoing hemorrhage who require a significant blood product transfusion. By activating the massive transfusion protocol, the clinician is notifying the blood bank and other care providers that large amounts of blood products will be

TABLE 4. Four Stages of Damage Control[22,23]

Stage 0 – "ground zero"	Initiate a rapid transfer to a trauma center. Priority one: Stop the hemorrhage before beginning resuscitation. Prevent hypothermia. Measure blood gas. Initiate a rapid transfer to the operating room.
Stage 1 – Initial operation	Control hemorrhage. Control contamination. Use abdominal packing judiciously. Perform a rapid temporary abdominal closure. Prevent hypothermia.
Stage 2 – ICU resuscitation	Monitor perfusion and resuscitate in the ICU. Correct acidosis and coagulopathy. Rewarm the patient. Optimize oxygenation and ventilation. Measure intra-abdominal pressure.
Stage 3 – Definitive repair	Consider early reoperation if bleeding continues. Plan for reoperation once physiology has been restored. Perform definitive surgery. Consider an abdominal closure or staged closure.

needed emergently. In addition, fluids and blood products should be warmed prior to administration to prevent hypothermia.

Damage Control Surgery

The main goal of early damage control is patient survival. Damage control surgery aims to control hemorrhage and to minimize contamination and complications such as ongoing shock or abdominal compartment syndrome. Damage control surgery consists of four stages (*Table 4*).[22-24] It begins at "ground-zero," the prehospital phase, in which injured patients who require damage control are identified. In such cases, resuscitation is continued in the emergency department. *Stage 1* entails the immediate control of life-threatening hemorrhage and gross contamination, the use of intraluminal shunts, and the packing and temporary closure of any wounds.

In *Stage 2*, the focus is on correcting the patient's metabolic disorders, including acidosis, coagulopathy, and hypothermia. *Stage 3* involves early reoperation, in cases of ongoing bleeding. In all four phases, the patient continually receives timely and aggressive resuscitation to correct the altered physiological state. Damage control remains a vital tool in the management of the exsanguinating patient and should be carried out before the patient has reached physiological exhaustion.

CRITICAL DECISION

What treatment should be initiated immediately?

The underlying principle in treating hemorrhagic shock is to optimize tissue perfusion through source control and adequate resuscitation. Particularly in cases of trauma-related shock, treatment is targeted at preventing and treating the lethal triad of hypothermia, coagulopathy, and acidosis. This is accomplished by stopping the bleeding and reversing hypoperfusion.

The initial care of all critically ill patients should focus on stabilizing the airway, breathing, and circulation, and identifying any nonhemorrhagic causes of shock that require immediate intervention (eg, tension pneumothorax or cardiac tamponade). Patients should be placed on a cardiac monitor and undergo frequent blood pressure and pulse oximetry measurements. Intravenous (IV) access must consist of at least two large-bore peripheral lines; unstable patients should receive a large sheath introducer. Local hemostasis can be obtained with a blood pressure tourniquet, direct pressure, or wound repair with sutures or staples.

Any suspected unstable pelvic fractures should be bound with a commercial pelvic binder or with bedsheets. Any obvious extremity deformities should be reduced and stabilized. Pregnant patients can be turned to the left lateral decubitus position to relieve the compression of the inferior vena cava by the gravid uterus and to improve venous return.[2] Unless a urethral injury is suspected, a urinary catheter should be placed to monitor hourly urine output and assess for hematuria.[2]

Based on the mechanism of injury or the hemorrhage source, emergency physicians should involve consultants as early as possible to facilitate the definitive arrest of bleeding and repair of the injured organ. In cases of post-traumatic hemorrhagic shock, trauma surgery should be involved early, even if the source of bleeding has not yet been identified. Other consultants, including interventional radiologists and orthopedic surgeons, should be involved, as needed. In other nontrauma-related situations, the definitive repair and cessation of hemorrhage require the expertise of consultants such as obstetricians, general surgeons, vascular surgeons, orthopedic surgeons, interventional radiologists, or gastroenterologists.

Evidence has not yet been established for the safety and efficacy of using temporizing measures such as balloon tamponade for esophageal varices (eg, a Sengstaken-Blakemore tube) or resuscitative endovascular balloon occlusion of the aorta (REBOA) in the emergency department before definitive care can be performed.[25,26]

CRITICAL DECISION

What are the components of a massive transfusion protocol?

A massive transfusion, defined as more than 10 units of packed red blood cells within the first 24 hours, incorporates the transfusion of plasma and platelets.[21] The potential dangers of these protocols are well established. In addition, massive transfusions consume significant hospital resources, so patients who are most likely to benefit should be identified early and carefully. Several scoring systems were developed to recognize patients who are likely to require a massive transfusion (*Table 5*). The assessment of blood consumption (ABC) score is a practical tool for assessing these cases, as the data

TABLE 5. Massive Transfusion Scoring Systems

Scoring System	Factors
Trauma-associated severe hemorrhage (TASH)[27]	SBP HR Gender Hemoglobin FAST Base excess Pelvic/femur fractures
McLaughlin score[28]	HR >105 bpm SBP <110 mm Hg pH <7.25 Hematocrit <32%
ABC[29]	Penetrating mechanism ED SBP <90 mm Hg ED HR ≥120 bpm Positive FAST

SBP = systolic blood pressure; HR = heart rate; FAST = focused assessment with sonography in trauma; ED = emergency department

Pearls

- While recognizing hemorrhagic shock is important, the emergency physician must concurrently consider other etiologies that can cause or contribute to the patient's state of shock.
- Early overly aggressive resuscitation with crystalloids should be avoided.
- The administration of tranexamic acid within 3 hours of injury is associated with improved outcomes.
- A massive transfusion of blood products can cause hypocalcemia and hypothermia.
- A tachycardic patient who presents with trauma is in shock until proven otherwise.

points are available immediately. The trauma-associated severe hemorrhage (TASH) and McLaughlin scores require laboratory tests and/or diagnostic imaging.[27-29]

Hemostatic resuscitation relies on packed red blood cells, plasma, and platelets, but the precise ratio of these products remains a subject of investigation. In a large multicenter study, the PROPPR trial compared a 1:1:1 ratio of plasma, platelets, and red blood cells to a 1:1:2 ratio.[30] Although there were no differences in all-cause mortality (24-hour or 30-day), the 1:1:1 group experienced greater rates of hemostasis and fewer deaths due to exsanguination within 24 hours. The development of massive transfusion protocols at most trauma centers has provided an expedited process for mobilizing resources and minimizing the time required to access blood products.

In addition to blood products, tranexamic acid (TXA) is an antifibrinolytic agent that should be given as early as possible (within 3 hours of trauma).[31,32] TXA is administered as a loading dose of 1 g over 10 minutes, then as an infusion of 1 g over 8 hours. When administered early, the drug appears to safely reduce mortality in trauma patients with bleeding. Other potentially beneficial products include recombinant factor VIIa, which may reduce blood transfusion requirements in cases of blunt trauma, and cryoprecipitate, which can be used to treat fibrinogen deficiency.[33,34]

Throughout any resuscitation, emergency physicians should continue to monitor for hypothermia, acid-base imbalance, and electrolyte disturbances. Calcium chelation by ethylenediaminetetraacetic acid (EDTA), a preservative in stored blood, can cause hypocalcemia. A Level 1 rapid transfuser or autologous transfuser device can rapidly deliver products at normothermic levels.

CRITICAL DECISION

What is the appropriate disposition for patients in hemorrhagic shock?

Definitive repair must be performed in patients with ongoing hemorrhage. Depending on the type of injury sustained, hemostasis can be achieved in the operating room or during angiography or endoscopy. If these resources are unavailable, the patient should be stabilized and transferred to a center that offers specialty services and a higher level of care. For certain hemorrhagic shock disease states, including a ruptured aortic abdominal aneurysm, delays in transfer and surgical repair are associated with a higher mortality.[35,36]

If the hemorrhage can be stabilized and does not require immediate repair, the patient should be admitted and monitored in the ICU. Despite advances in resuscitation, the risk of death for patients in hemorrhagic shock remains high; such cases require the highest level of care.[37,21]

Summary

When diagnosing hemorrhagic shock, emergency physicians must consider the complete clinical picture, including the patient's medical history, vital signs, examination findings, and laboratory data. Immediate management efforts must aim to control the source of the bleeding and achieve hemostasis. Resuscitation can begin with crystalloid solutions but ultimately necessitates the replacement of blood volume with blood products. Hospitals that have an established massive transfusion protocol can rapidly provide red blood cells, plasma, and platelets to critically ill patients. Although the exact ratio of transfusion (plasma:platelets:red blood cells) is not yet established, most patients require as close to a 1:1:1 ratio as possible. The addition of tranexamic acid within 3 hours of life-threatening hemorrhage may also be beneficial.

The appropriate discharge disposition for patients in hemorrhagic shock depends on whether the bleeding is ongoing or stabilized. In ongoing hemorrhage, damage control should be started early to prevent metabolic exhaustion. Definitive surgical repair should be initiated only after the patient has been stabilized and any physiological derangements have been corrected. Patients with a stabilized hemorrhage should be monitored closely in an intensive care setting.

Pitfalls

- Being reassured by "normal" vital signs after the administration of several liters of crystalloid fluids, and failing to recognize subtle clinical signs that the patient is in shock.
- Waiting for the patient to be stabilized before calling a consultant who can control the hemorrhage.
- Allowing an unstable patient to proceed to CT imaging in order to obtain a diagnosis.

CASE RESOLUTIONS

■ CASE ONE

The young driver with multisystem blunt trauma was determined to be in hemorrhagic shock. She was intubated immediately upon arrival, and a massive transfusion protocol was activated (1:1:1 ratio). The patient received 1 L of normal saline, 4 units of packed red blood cells, 2 units of fresh frozen plasma, and 1 unit of platelets. She was taken to the operating room for repair of a significant hemoperitoneum, which was noted on ultrasound. The patient underwent a damage-control laparotomy, which included a partial bowel resection; a splenectomy; and abdominal packing.

■ CASE TWO

Three large-bore IVs were placed immediately in the patient with an upper-GI bleed, and he was placed on a monitor. Due to persistent hypotension and a large volume of hematemesis, he underwent an endotracheal intubation for airway protection, and a massive transfusion protocol was initiated. His laboratory results were notable for a hemoglobin level of 7.4 g/dL, a platelet level of 86,000 mm^3, and an INR of 1.7.

After being transfused with 4 units of packed red blood cells, 3 units of platelets, and 3 units of fresh frozen plasma, the patient's blood pressure improved to 92/61, and his heart rate increased to 102. He was transferred to the medical ICU, where an esophagogastroduodenoscopy revealed esophageal varices.

■ CASE THREE

The elderly man was suspected of having a ruptured abdominal aortic aneurysm. He received two large-bore IVs and 1 L of normal saline. The blood bank was notified immediately to type and crossmatch for 2 units of type O-negative blood. A bedside ultrasound confirmed an abdominal aortic aneurysm measuring 8.1 cm and a small amount of free fluid in the right upper quadrant.

After consulting vascular surgery, the emergency physician decided to transfer the patient to a nearby quaternary care center. Upon arrival, the patient's systolic blood pressure was 82. He was taken to the operating room for repair of his aortic aneurysm rupture. His abdomen was left open, and he was transferred to a surgical ICU for further monitoring. Without evidence of further bleeding, he underwent closure of his abdomen. He was discharged to a rehabilitation hospital 20 days later.

REFERENCES

1. Gough JE. Therapeutic approach to the hypotensive patient. In: Cline DM, Ma OJ, Cydulka RK, Meckler GD, Handel DA, Thomas SH. *Tintinalli's Emergency Medicine Manual*. 7th ed. New York, NY: McGraw-Hill; 2012:47-51.
2. American College of Surgeons. *Advanced Trauma Life Support: ATLS Student Course Manual*. 9th ed. Chicago, IL: American College of Surgeons; 2012.
3. Cocchi MN, Kimlin E, Walsh M, Donnino MW. Identification and resuscitation of the trauma patient in shock. *Emerg Med Clin North Am*. 2007 Aug;25(3):623-642,vii.
4. Guly HR, Bouamra O, Little R, et al. Testing the validity of the ATLS classification of hypovolaemic shock. *Resuscitation*. 2010 Sep;81(9):1142-1147.
5. Mutschler M, Neinaber 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 Trauma Register DGU. *Crit Care*. 2013 Aug 12;17(4):R172.
6. Vandromme MJ, Griffin RL, Kerby JD, McGwin G Jr, Rue LW 3rd, Weinberg JA. Identifying risk for massive transfusion in the relatively normotensive patient: utility of the prehospital shock index. *J Trauma*. 2011 Feb;70(2):384-388.
7. Gonzalez E, Moore EE, Moore HB. Management of trauma induced coagulopathy with thromboelastography. *Crit Care Clin*. 2017 Jan;33(1): 119-134.
8. Ghane MR, Gharib MH, Ebrahimi A, et al. Accuracy of rapid ultrasound in shock (RUSH) exam for diagnosis of shock in critically ill patients. *Trauma Mon*. 2015 Feb;20(1):e20095.
9. Reed MJ, Cheung LT. Emergency department led emergency ultrasound may improve the time to diagnosis in patients presenting with a ruptured abdominal aortic aneurysm. *Eur J Emerg Med*. 2014 Aug;21(4):272-275.
10. Gutierrez G, Reines HD, Wulf-Gutierrez ME. Clinical review: hemorrhagic shock. *Crit Care*. 2004 Oct;8(5):373-381.
11. Heier HE, Bugge W, Hjelmeland K, Søreide E, Sørlie D, Håheim LL. Transfusion vs. alternative treatment modalities in acute bleeding: a systematic review. *Acta Anaesthesiol Scand*. 2006 Sep;50(8):920-931.
12. Dick F, Erdoes G, Opfermann P, Eberle B, Schmidli J, von Allmen RS. Delayed volume resuscitation during initial management of ruptured abdominal aortic aneurysm. *J Vasc Surg*. 2013 Apr;57(4):943-950.
13. Holcomb JB, Jenkins D, Rhee P, et al. Damage control resuscitation: directly addressing the early coagulopathy of trauma. *J Trauma*. 2007 Feb;62(2):307-310.
14. Jacob M, Kumar P. The challenge in management of hemorrhagic shock in trauma. *Med J Armed Forces India*. 2014 Apr;70(2):163-169.
15. Dutton RP, Mackenzie CF, Scalea TM. Hypotensive resuscitation during active hemorrhage: impact on in-hospital mortality. *J Trauma*. 2002 Jun;52(6):1141-1146.
16. Bickell WH, Wall MJ Jr, Pepe PE, et al. Immediate versus delayed fluid resuscitation for hypotensive patients with penetrating torso injuries. *N Engl J Med*. 1994 Oct 27;331(17):1105-1109.
17. Hébert PC, Wells G, Blajchman MA, et al. A multicenter, randomized, controlled clinical trial of transfusion requirements in critical care. *N Engl J Med*. 1999 Feb 11;340(6):409-417.
18. Kohli S, Yadav N, Singh GP, Prabhakar H. Permissive hypotension in traumatic brain injury with blunt aortic injury: how low can we go? *J Anaesthesiol Clin Pharmacol*. 2014 Jul;30(3):406-408.
19. Schierhout G, Roberts I. Fluid resuscitation with colloid or crystalloid solutions in critically ill patients: a systematic review of randomised trials. *BMJ*. 1998 Mar 28;316(7136):961-964.
20. Finfer S, Bellomo R, Boyce N, et al. A comparison of albumin and saline for fluid resuscitation in the intensive care unit. *N Engl J Med*. 2004 May 27;350(22):2247-2256.
21. Pohlman TH, Walsh M, Aversa J, Hutchison EM, Olsen KP, Lawrence Reed R. Damage control resuscitation. *Blood Rev*. 2015 Jul;29(4):251-262.
22. Rotondo MF, Schwab CW, McGonigal MD, et al. 'Damage control': an approach for improved survival in exsanguinating penetrating abdominal injury. *J Trauma*. 1993 Sep;35(3):375-382.
23. Johnson JW, Gracias VH, Schwab CW, et al. Evolution in damage control for exsanguinating penetrating abdominal injury. *J Trauma*. 2001 Aug;51(2):261-271.
24. Lamb CM, MacGoey P, Navarro AP, Brooks AJ. Damage control surgery in the era of damage control resuscitation. *Br J Anaesth*. 2014 Aug;113(2):242-249.
25. D'Amico G, Pagliaro L, Bosch J. The treatment of portal hypertension: a meta-analytic review. *Hepatology*. 1995 Jul;22(1):332-354.
26. Morrison JJ, Galgon RE, Jansen JO, Cannon JW, Rasmussen TE, Eliason JL. A systematic review of the use of resuscitative endovascular balloon occlusion of the aorta in the management of hemorrhagic shock. *J Trauma Acute Care Surg*. 2016 Feb;80(2):324-334.
27. Yücel N, Lefering R, Maegele M, et al. Trauma Associated Severe Hemorrhage (TASH)-Score: probability of mass transfusion as surrogate for life threatening hemorrhage after multiple trauma. *J Trauma*. 2006 Jun;60(6):1228-1236.
28. McLaughlin DF, Niles SE, Salinas J, et al. A predictive model for massive transfusion in combat casualty patients. *J Trauma*. 2008 Feb;64(2Suppl):S57-S63.
29. 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):346-352.
30. 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 Feb 3; 313(5):471-482.
31. Ker K, Roberts I, Shakur H, Coats TJ. Antifibrinolytic drugs for acute traumatic injury. *Cochrane Database Syst Rev*. 2015 May 9;(5):CD004896.
32. CRASH-2 trial collaborators, Shakur H, Roberts I, et al. 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 Jul 3;376(9734): 23-32.
33. Boffard KD, Riou B, Warren B, et al. Recombinant factor VIIa as adjunctive therapy for bleeding control in severely injured trauma patients: two parallel randomized, placebo-controlled, double-blind clinical trials. *J Trauma*. 2005 Jul;59(1):8-15.
34. Kirkman E, Watts S, Hodgetts T, Mahoney P, Rawlinson S, Midwinter M. A proactive approach to the coagulopathy of trauma: the rationale and guidelines for treatment. *J R Army Med Corps*. 2007 Dec;153(4):302-306.
35. Mell MW, Wang NE, Morrison DE, Hernandez-Boussard T. Interfacility transfer and mortality for patients with ruptured abdominal aortic aneurysm. *J Vasc Surg*. 2014 Sep;60(3):553-557.
36. Groves EM, Khoshchehreh M, Le C, Malik S. Effects of weekend admission on the outcomes and management of ruptured aortic aneurysms. *J Vasc Surg*. 2014 Aug; 60(2):318-324.
37. Kobayashi L, Constantini TW, Coimbra R. Hypovolemic shock resuscitation. *Surg Clin North Am*. 2012 Dec; 92(6):1403-1423.