Hemorrhagic Shock Due to Trauma

Josey D. Frazier

MSU-CVM Class of 2018

Clinicopathologic Conference

Presented on February 9, 2018

CPC Advisor:

Marc A. Seitz, DVM, DABVP (Canine and Feline)

MISSISSIPPI STATE UNIVERSITY™
COLLEGE OF VETERINARY MEDICINE

Introduction

Severe hemorrhage is a high cause of mortality in trauma patients (5,6,7). Patients in hemorrhagic shock from trauma tend to succumb to their injuries earlier as compared to patients with septic shock and multiple organ dysfunction syndrome (3). Although there are multiple factors to consider when developing a diagnostic and treatment plan for trauma patients, identifying and controlling occult hemorrhage is important for a successful outcome, especially if hemorrhage is ongoing (8). Of all the fluid resuscitation approaches that have been practiced and accepted over the years, limited volume resuscitation is now recognized as the ideal approach to stabilizing trauma patients in hemorrhagic shock (8).

History and Presentation

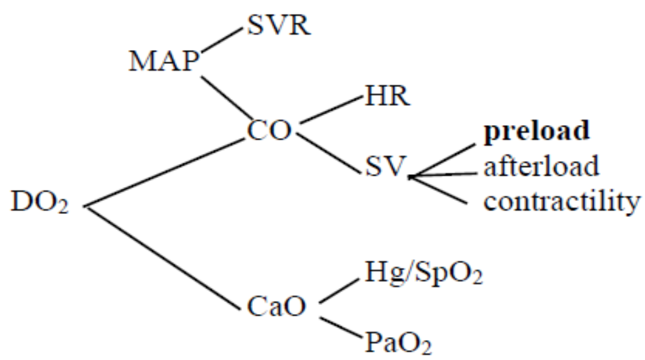
Trauma appears to be more prevalent in young to middle age dogs, with males being more affected than females (3). Trauma can cause physical injuries to any part of the body and be divided into blunt force trauma and penetrating trauma (2,6). In veterinary patients, the most common cause of blunt force trauma is motor vehicular, but other causes include high-rise falls and human mistreatment (2). Bite, stab, impalement, and gunshot wounds are examples of penetrating trauma, with bite wounds being the most common (2). Hemorrhagic shock occurs most commonly with blunt force trauma; however, it can occur if penetrating trauma injures a major vessel (2).

Clinical signs of hemorrhagic shock on presentation depend on the severity of blood loss, if the animal is actively bleeding, and the anatomic location of the blood loss (6). Animals with severe blood loss exceeding 30-40% of their total blood volume usually present in decompensated shock (3,4,6). Clinical signs include profound tachycardia, tachypnea, weak to absent pulses, pale to white mucous membrane color, prolonged capillary refill time (CRT), altered mental status, and hypothermia (6). Animals with severe ongoing hemorrhage can exsanguinate rapidly depending on the rate of blood loss (6). Patients with only mild to moderate blood loss are either stable or in compensated shock, which causes mild or even no clinical signs (6). On presentation, these animals can appear stable, but they will begin to decompensate if hemorrhage continues undetected (6). Other clinical findings correlate to the location of the blood loss, such as a distended abdomen or a fluid wave with hemoperitoneum (4).

Pathophysiology

The body constantly works to maintain homeostasis, even in times of injury (6). Oxygen delivery to the tissues via perfusion depends on two main factors: the oxygen content of the blood (CaO2) and cardiac output (CO) (6). Factors affecting CaO2 include hemoglobin concentration, hemoglobin saturation (SpO2), and the partial pressure of oxygen in arterial blood (PaO2) (6). Numerous factors affect CO, including heart rate, stroke volume, systemic vascular resistance, preload, afterload, and cardiac contractility (6,10). The interplay of these factors is illustrated well in Figure 1 (10). With trauma, oxygen delivery to tissues can become impaired due to several factors related to blood loss (6). The most important factor is hypovolemia, which decreases preload, and leads to a decrease in stroke volume and cardiac output (6,10). However, decreased hemoglobin concentration and hypoxemia (if pulmonary contusions or pneumothorax are present) contribute as well (6).

Figure 1: Oxygen Delivery (10)



When blood loss initially occurs, the body compensates through numerous physiologic processes in an attempt to maintain oxygen delivery (6). Due to the presence of aortic and carotid baroreceptors, the central nervous system can detect and immediately respond to changes in blood volume and hence pressure (6). When stretched, baroreceptors stimulate the sympathetic nervous system to increase CO by increasing the heart rate, systemic vascular resistance, and myocardial contractility (6). As hypotension becomes more severe, the sympathetic nervous system releases catecholamines, the renin-angiotensin-aldosterone system is activated, and vasopressin is released, all causing vasoconstriction in order to maintain arterial blood pressure (6). Blood is also shunted away from areas of less importance to more vital organs such as the brain, heart, and lungs (6). Paradoxically, local factors work to maintain circulation in key tissues at the microcirculatory level by altering vessel tone and causing vasodilation (6). As hemorrhage continues, the body's mechanisms of compensation begin to fail causing tissues to become hypoxic and convert to anaerobic metabolism for energy production (6). Anaerobic metabolism leads to a buildup of lactic acid and hydrogen ions in the blood (6). Chemoreceptors in the periphery sense changes to oxygen in the blood and pH (6). These receptors allow movement of fluid from the interstitial space into the intravascular space to compensate for intravascular volume loss (6). Together, these response mechanisms work to maintain adequate perfusion throughout the body (6). Even though the body can compensate for some time, if hemorrhage and tissue damage plateau, these mechanisms can be disturbed by acidosis, electrolyte abnormalities, ischemia, hypothermia, certain drugs due to receptor alterations, response to the sympathetic nervous system, and poor perfusion making it more difficult for the body to compensate appropriately (6). Hypoxia and acidemia, as well as the release of pro-inflammatory mediators, can lead to further injury and worsen perfusion due to a disruption in microcirculation and a decrease in the affinity for oxygen by red blood cells (6). If hypoxia persists or worsens, organ failure and eventual death of the animal occurs (6).

Acute traumatic coagulopathy (ATC) following trauma characterized by hypocoagulation and hyperfibrinolysis is a potential sequela that typically occurs within 1 hour following injury in patients in hemorrhagic shock (8,9). It is thought to be a variant of disseminated intravascular coagulation (DIC) (9). However, numerous other factors also contribute including decreased tissue perfusion, hypothermia, widespread inflammation, metabolic acidosis, tissue damage, and hemodilution (8). Identification of the role of hemodilution has shifted strategies in fluid resuscitation, and it was discovered that historically used large volume fluid resuscitation worsened ATC by diluting clotting factors, potentiating inflammatory mediators and metabolic acidosis, and disrupting clot formation (9). Diagnosis can be difficult, but recognizing it early based on history, physical exam, and diagnostics can have a significant impact on patient outcome if managed appropriately (9).

Differentials Diagnoses and Sequelae

Trauma patients frequently present with co-morbidities, including diaphragmatic hernia, abdominal wall rupture, uroperitoneum, and coagulopathies (ATC and/or DIC) (2,7,9). Because trauma can cause a variety of injuries, diagnostics are important for ruling out other possible underlying injuries in these patients (2). If a history of trauma is unknown, similarly presenting critical conditions include septic shock, multi-organ dysfunction syndrome, gastric dilatation with volvulus, ruptured peritoneal neoplasia, and any other cause of severe anemia (2,7).

Diagnostic Approach/Considerations

With trauma patients, triage and initial diagnostics at initial presentation are vital for determining patient stability, baseline values, severity of injuries, and developing a treatment plan (2). Immediately upon presentation, a rapid triage exam should be performed to evaluate the respiratory, cardiovascular, and nervous systems, with brief attention given to the musculoskeletal and integumentary systems (2). Initial diagnostics that complement the triage exam include electrocardiogram (ECG), pulse oximetry (Sp02), and blood pressure (4,5). Electrocardiogram evaluates the patient for arrhythmias, of which ventricular tachyarrhythmias are the most common with trauma (2,4). In patients with suspected lung trauma, pulse oximetry is a reasonable screening tool for hypoxemia that results from contusions or pneumothorax (1). Blood pressure monitoring is crucial for patients in hemorrhagic shock and evaluates for hypotension (2). If trauma patients present with clinical signs suspicious for hemorrhagic shock, diagnostics to further evaluate the patient for evidence of either controlled or ongoing hemorrhage are warranted (2). For example, the abdominal and thoracic cavities should be evaluated for the presence of free fluid, likely to be hemorrhage (2). An important tool that is now readily available and commonly used during the triage exam is focused assessment with sonography in trauma, or FAST (2,5).

Abdominal FAST is a quick and useful tool that can be used to evaluate the presence of free fluid within the abdomen (5). This diagnostic tool has been commonly used in human trauma patients for years, and it has a higher sensitivity and specificity compared to diagnostic peritoneal lavage and radiographs (5). Abdominal FAST assesses four quadrants of the abdomen where free fluid would most likely be found if present (2,5). Patients are given an abdominal fluid score (AFS) of 0-4, corresponding to the presence of fluid in each quadrant assessed (5). Dogs with blunt force trauma with an AFS of three or four (out of four) are more likely to develop anemia and more likely to require blood transfusions as compared to lower scores (5).

Thoracic FAST can also be used to quickly evaluate trauma patients for pleural and pericardial effusion, pneumothorax, and pulmonary contusions (2). For patients that are not hemodynamically stable on initial presentation, AFAST and TFAST can be useful diagnostic tools for patients who are too unstable to undergo further diagnostic imaging (5).

Serum lactate can be used to assess trauma patients with suspected hemorrhage and blood loss (4,7). Elevated lactate often indicates poor tissue perfusion and decreased oxygen delivery, which in trauma patients usually corresponds to hemorrhagic shock (4,7). Measuring lactate in veterinary patients is now quick and easy thanks to handheld point-of-care machines (7). Furthermore, lactate can be used to guide fluid resuscitation and improve outcomes in trauma patients (8).

Abdominocentesis is another useful diagnostic tool and should be performed if free fluid is suspected or confirmed with diagnostics (2). Fluid analysis can provide better understanding of what the fluid is (i.e. blood, urine, or bile) and where it may be coming from so that appropriate treatment can be initiated (2). If erythyphagocytosis is seen when analyzing the abdominal fluid sample, this is confirmatory for hemoperitoneum (2). However, in acute cases, red blood cells only may be seen in the free fluid sample, which only provides suspicion of hemoperitoneum (2). In these cases, the fluid’s packed cell volume (PCV) can be compared to the peripheral PCV to confirm hemoperitoneum (2,4). Fluid analysis can also be useful in differentiating hemorrhagic shock from septic shock (2).

Clinical laboratory tests are necessary to evaluate all patients with a history of trauma (2). A complete blood count and coagulation profile are important to assess, and common findings in hemorrhaging trauma patients include anemia, thrombocytopenia due to platelet consumption, and neutrophilia due to stress, inflammation, or less likely infection (2). Coagulation tests (e.g. PT and PTT) can be normal, but are often prolonged in patients with ACT and can change in the first 24-48 hours after admission (9). A biochemical profile is also important for assessing liver enzymes, renal values, electrolytes, and acid-base status (2). Azotemia can occur from dehydration, shock, renal injury, or urinary tract rupture (2). Urine specific gravity can help differentiate causes of azotemia and should ideally be checked prior to fluid therapy (2). Elevated liver enzymes (ALT, AST, ALP, and GGT) can indicate damage to the liver or gallbladder, both of which can occur after trauma (2).

One final clinicopathologic test useful for diagnosing blood loss and hemoperitoneum is a PCV and total solids (TS) (4). With peracute blood loss, TS will decrease while the PCV will stay the same or increase (4). As hemorrhage continues, PCV and TS will drop together, and serial PCV and TS can be used to monitor the patient for evidence of uncontrolled hemorrhage (4). Comparing free fluid PCV and PCV from a peripheral vessel can also be used determine if fluid in the abdomen is from hemorrhage (2,4). If the abdominal free fluid sample PCV is at least 25% to the peripheral vessel sample PCV, hemorrhage is likely (2,4).

Diagnostic imaging is essential for evaluating trauma patients (2). Thoracic and abdominal radiographs are important for evaluating abnormalities such as diaphragmatic or body wall hernias, pneumothorax, pleural effusion, pulmonary contusions, rib fractures, organ perforation, subcutaneous emphysema, and fluid within the pericardium, peritoneum, or retroperitoneal space (2). Free fluid in the abdomen is typically due to blood from a ruptured organ or vessel, or urine from a ruptured bladder or ureter (2). Uncommonly, bile from biliary tree rupture or an exudate from septic peritonitis secondary to gastrointestinal tract rupture can occur (2).

Treatment and Management Options

A detailed review of the treatment of all sequelae associated with trauma is beyond the scope of this paper. However, universal supportive therapies must be recognized. Immediately upon presentation, flow-by oxygen supplementation and appropriate analgesia should be provided (1). After assessing the patient's airway and ability to breathe, severe external hemorrhage can be addressed if present, and pressure, either direct and/or indirect, should be applied (1). In severe cases, visualization and ligation may be necessary to provide better control of the hemorrhage (1). If thoracic hemorrhage is present and causing hypoventilation, thoracocentesis may be warranted to allow adequate lung expansion (1). If open wounds or severe penetrating wounds are present, early administration of antibiotics is recommended (2). After stabilization of the respiratory system, immediate stabilization of the circulatory system should ensue to restore tissue perfusion (1).

Fluid resuscitation is essential for treating patients in hemorrhagic shock and should be started immediately to correct hypoperfusion and shock in these patients (1,2). The type and severity of injury as well as the type and severity of hemorrhage should be considered when determining how to best treat these patients (8). Limited volume intravenous fluid resuscitation is now considered the initial mainstay of treatment in patients with hemorrhagic shock (8). Limited volume resuscitation uses a synthetic colloid or hypertonic saline bolus combined with a balanced crystalloid to lower the total volume of fluid needed to achieve hemodynamic stability (8). Doses are listed in table 1 for reference (10). Current recommendations are to fluid resuscitate to a blood pressure on the low end of normal (i.e. systolic of 90mmHg and a mean of 60mmHg) (8). This practice is in contrast to previous theories that used excessive crystalloid volumes or hypotensive targets, both of which led to worse outcomes (8).

Table 1: Doses of fluids used during hemorrhagic shock resuscitation (10). All doses are given intravenous over 10-20 minutes as a bolus (10).

Fluid Type	Dog	Cat
Balanced crystalloid	20-30ml/kg	10-20ml/kg
Synthetic colloid	5-10ml/kg	3-5ml/kg
Hypertonic saline	4-7ml/kg	3-4ml/kg
Whole blood	20ml/kg	20ml/kg
Packed RBC's and Plasma	10ml/kg	10ml/kg

If judicious fluid use cannot achieve hemodynamic stability, or in patients with severe blood loss or coagulopathies, blood products are necessary (8). Currently, component therapy is considered standard of a care in transfusion medicine, and recommends replacing the blood component that has been lost (8). For the bleeding traumatic patient, fresh whole blood is logically the most effective blood product for stabilization (8). If availability is limited, stored blood products such as packed red blood cells and plasma may be combined, but are less ideal than fresh whole blood (8). The ideal ratio of red cells to plasma is considered 1:1 (8). For patients with a coagulopathy and stable PCV, plasma may be given. When administering blood products, these patients should be closely monitored for adverse reactions (10).

Surprisingly, many patients with internal abdominal hemorrhage can be stabilized and medically managed with fluid therapy and blood products alone (5). Although not an ideal first choice, autotransfusion can also be performed as a more conservative management option when blood products are unavailable (2,4). For patients that fail to respond to fluids and blood products, emergency exploratory surgery of the affected cavity (i.e. thorax or abdomen) may be required (2).

Expected Outcome and Prognosis

The outcome for patients in hemorrhagic shock depends on a variety of factors including severity of injuries, co-morbidities, degree of shock, amount of blood loss at presentation, and how quickly treatment can be initiated (2,8). Negative prognostic indicators in patients with hemorrhage include failure to normalize hyperlactatemia, acidosis, hypothermia, coagulopathies, and hypocalcemia, and mortality rates tend to be higher in these patients (2,8). Patients sustaining injuries from blunt trauma can have good survival rates if they can get to a veterinarian for evaluation and treatment soon following the event (2).

Conclusion

Patients in hemorrhagic shock from trauma can present in a variety of ways and at multiple ends of the spectrum. Rapid evaluation via a triage exam and diagnostics helps direct therapy. Limited volume resuscitation is considered standard of care and uses synthetic colloids and/or hypertonic saline with balanced crystalloids to achieve hemodynamic stability at the lowest effective volume (8).

References

1. Crowe DT. Assessment and management of the severely polytraumatized small animal patient. J Vet Emerg Crit Care 2006; 16:264-275.

2. Culp WTN, Silverstein, DC. In: Silverstein, DC, Hopper K. eds. Small Animal Critical Care Medicine. 2nd ed. St. Louis, Mo: Saunders/Elsevier, 2015; 728-733.

3. Hall KE, Sharp CR, Adams CR, et al. A novel trauma model: naturally occurring canine trauma. Shock 2014; 41:25-32.

4. Herold LV, Devey JJ, Kirby R, et al. Clinical evaluation and management of hemoperitoneum in dogs. J Vet Emerg Crit Care 2008; 18:40-53.

5. Lisciandro GR, Lagutchik MS, Mann, KA, et al. Evaluation of an abdominal fluid scoring system determined using abdominal focused assessment with sonography for trauma in 101 dogs with motor vehicle trauma. J Vet Emerg Crit Care 2008; 19:426-437.

6. Muir W. Trauma: physiology, pathophysiology, and clinical implications. J Vet Emerg Crit Care 2006; 16:253-263.

7. Nye CJ, Musulin SE, Hanel, RM et al. Evaluation of the Lactate Plus monitor for plasma lactate concentration measurement in dogs. J Vet Emerg Crit Care 2017; 27:66-70.

8. Palmer L. Fluid management in patients with trauma, restrictive versus liberal approach. Vet Clin Small Anim 2017; 47:397-410.

9. Palmer L, Martin L. Traumatic coagulopathy-part 1: pathophysiology and diagnosis. J Vet Emerg Crit Care 2014; 24:63-74.

10. Proulx A, Silverstein D. Resuscitative fluid therapy for circulatory shock. Today's Veterinary Practice 2011; 1:54-60.