Acute Hemorrhage and Blood Transfusions in Horses

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KEYWORDS

• Equine • Blood • Transfusion • Hemorrhage • Hemostasis

KEY POINTS

- Acute hemorrhage can be life-threatening and may require blood transfusion, especially if greater than 25% blood loss has occurred.
- A variety of topical hemostatic agents and techniques can be used to control external hemorrhage.
- Initial transfusions may be performed without crossmatch in an emergency, and blood recipients should be monitored closely for any transfusion reactions.
- Ideally transfuse with fresh whole blood to replace 25% to 50% of the blood lost.
- Cell salvage devices can also be used to collect autologous blood in cases of intracavitary and intraoperative blood loss.

INTRODUCTION

Acute hemorrhage in horses can result in severe shock, and even when treated can be fatal. The principles of treating hemorrhagic shock have been described in the human and veterinary literature.[1,2] Adjunctive methods of controlling hemorrhage, such as newer hemostatic devices and products on the market, have potential for use in the horse. In addition, recently published information about blood storage and transfusion in human, canine, and equine medicine may help guide blood transfusion procedures. Acute hemorrhage in the horse does present some unique challenges because of the volume of hemorrhage and difficult access to sources of the bleeding.

ACUTE HEMORRHAGE

Recognizing Acute Hemorrhage in the Horse

Acute hemorrhage can be external, such as arterial lacerations or epistaxis, or internal, such as splenic lacerations or uterine artery bleeding. Blood loss requiring transfusion

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has been reported with gastrointestinal, urogenital, and sinonasal surgeries.[3–5] Internal hemorrhage may be more difficult to diagnose because of the absence of visible blood loss. Abdominal ultrasound and abdominocentesis can indentify hemoperitoneum, although the underlying cause is not always evident.

For internal and external hemorrhage, it is critical to recognize the signs of hemorrhagic shock, which include tachycardia, tachypnea, cold extremities, anxiety or depression, pale mucous membranes, and prolonged capillary refill time. Horses with hemoperitoneum may also demonstrate colic and abdominal distension.[6] Clinicopathologic findings often include hyperlactatemia, hypoproteinemia, and anemia. However, the packed cell volume (PCV) and total protein (TP) may be normal in horses with acute whole blood loss. Splenic contraction can maintain PCV in the acute stages of hemorrhage, and PCV and TP remain normal until fluid redistributes from the interstitial spaces (over the first 12 hours if intravenous fluids are not administered). Serial monitoring of PCV and TP is recommended, and emergency resuscitation and transfusion may be indicated based on clinical signs rather than a specific PCV value.

Physiologic Hemostasis and Medical Management

After initial trauma, vasoconstriction occurs initially, followed by platelet activation, adhesion, and aggregation. Disruption of the endothelium exposes tissue factor–bearing cells, leading to activation of clotting factors and production of thrombin. The activated platelet serves as a congregation site for coagulation factors, and ultimately a stable fibrin clot is formed. Coagulopathy is rarely the cause of acute hemorrhage in horses, and further diagnostics and specific treatments are needed if coagulopathy is suspected (see article on coagulopathies elsewhere in this issue).

Stabilization with intravenous fluids has the potential to worsen bleeding, although specific medications can be used to enhance clot stability. Intravenous fluids are needed to restore intravascular volume in cases of hemorrhagic shock. Unfortunately, these fluids can also dilute platelets and clotting factors and negatively affect clot formation. In vitro and in vivo studies have demonstrated the hypocoagulatory effect of hydroxyethyl starch in humans and horses.[7–9] Large volumes of fluid may also raise blood pressure and disrupt the clot; therefore, conservative fluid resuscitation is recommended over shock doses of intravenous fluids, at least until definitive surgical hemostasis can be achieved. Human trauma and animal model studies demonstrate the benefits of delayed or hypotensive resuscitation.[10,11] Prolonged hypotension has deleterious effects, and therefore a balance must be achieved between treating hypotension and stabilizing the clot.

In addition to blood products (discussed later), medications can be used to promote hemostasis. Synthetic lysine analogues, epsilon aminocaproic acid, and tranexamic acid act through reversibly blocking the lysine binding sites of plasminogen, thus inhibiting fibrinolysis. Aminocaproic acid was administered in 92% of mares with periparturient hemorrhage in a recent study.[12] Some mares in this retrospective study received 10% formalin, conjugated estrogens, and yunnan baiyao as adjunctive hemostatic treatments. Intravenous formaldehyde is thought to potentiate hemostasis through activating platelets or endothelium. However, infusion of formaldehyde in normal horses did not result in any change in hemostatic variables.[13] Naloxone has also been advocated for use during uncontrolled hemorrhage. Naloxone may lessen hypotension through counteracting the endogenous opiate vasodilation and bradycardia that can occur with severe acute hemorrhage.[14,15]