Approach to the Bleeding Trauma Patient
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Trauma can result in life-threatening injuries. Obvious injuries and bleeding can point you in the right direction but may also distract and lead you astray. Internal bleeding may be more difficult to detect and pose a potentially greater threat. Some patients have clinical signs related to anemia such as lethargy, weakness, pallor, or collapse while others have organ dysfunction related to the location of the bleeding such as respiratory distress, neurologic signs, and blindness. Severity can range from minor to life-threatening. Bleeding is possible in all trauma patients and a simple and logical approach should be used on every trauma patient to rule out the presence of bleeding.

Hemostasis

Hemostasis is a balance of coagulation (primary and secondary hemostasis), anticoagulation, and fibrinolysis. Coagulation involves the formation of a platelet plug and a fibrin-stabilized clot. The fibrinolytic system breaks down the clot allowing the restoration of blood flow. Primary hemostasis involves the formation of the platelet plug and requires adequate platelet numbers, platelet function, and Von Willebrand factor levels. Other factors include collagen (bind platelets), fibrinogen (bridges platelets), and endothelial cells. Secondary hemostasis involves stabilization of the plug with fibrin and relies on a series of enzymatic reactions involving coagulation factors, tissue factor, fibrinogen, and calcium. Traditionally, secondary hemostasis is split into intrinsic, extrinsic, and common pathways. While these pathways allow for an easier understanding of coagulation, they oversimplify the process. In fact, primary and secondary hemostasis occur simultaneously and is represented better by the cell-based model of coagulation, which also incorporates the endothelium's role in hemostasis. Lastly, the fibrinolytic system breaks down the clot allowing the restoration of blood flow. Renewed attention is being given to the fibrinolytic system, especially in trauma patients as an overzealous fibrinolytic system can cause/exacerbate bleeding.

Patient History

Although the history is typically straightforward for trauma cases, a detailed and accurate history is still essential as other complicating factors may be present. Other primary or secondary hemostasis defects, congenital vs acquired defects, and focal vs systemic processes may be present.

Physical Examination

Physical exam findings may better characterize the cause of bleeding. Primary hemostasis defects (e.g. platelets) result in mucosal bleeds: petechiae to ecchymosis, epistaxis, hyphema, hematuria, and melena. Secondary hemostasis defects lead to cavitary bleeds: hematomas, hemothorax, hemoabdomen, SQ tissues, muscles, and joints. Petechiae commonly occur on pressure points, mucous membranes, sclera, and ventral abdomen. Epistaxis, hematemesis, hematuria, and melena can result from either defect. Lastly, an animal's signs may be related to

the degree of blood loss (anemia, hypovolemia) or the location of the bleeding (brain, spinal cord, joint, pericardial space).

Important body systems to triage in trauma include the brain, heart, and lungs as injury to these areas can quickly become life-threatening. Common sites of hemorrhage include the abdominal cavity and fractures to the pelvis and femur. Hemothorax is also possible and usually results in cardiovascular compromise (hemorrhagic shock) before respiratory compromise (pleural space disease).

Diagnostics

Point of care ultrasound (POCUS) is instrumental in trauma patients and can help rapidly identify and monitor bleeding patients. B-lines in the lungs may indicate pulmonary contusions. Hemothorax can typically be rapidly identified. Hemorrhage in the abdomen can typically be found around the liver and urinary bladder. Those with an abdominal fluid score of 2 or less are considered “low volume bleeders” and unlikely to be anemic purely from the abdominal bleeding. Anemia in a “low volume bleeder” should prompt further investigation. Any fluid found should be characterized. Trauma can also lead to leakage of urine, bile, etc.

PCV and TP should always be evaluated and interpreted in combination. With acute hemorrhage, splenic contraction may normalize PCV values. However, total protein drops rapidly and should be low (<6 g/dL) and should alert the clinician that hemorrhage is a possibility. With time, hemorrhage will cause both PCV and TP to be abnormally low.

Platelet numbers should always be verified via a manual platelet count (blood smear). Remember to check for platelet clumping along the feathered edge and that 1 platelet seen on high powered field is approximately 15K platelets. Clinical signs typically do not result unless <30,000-50,000. Primary hemostasis can be evaluated by performing a buccal mucosal bleeding time (BMBT). While the results of the BMBT must be interpreted cautiously, prolonged bleeding times may indicate a defect in platelet number or function, von Willebrand factor, or the endothelium. Disorders of secondary hemostasis should maintain a normal BMBT unless severe. Common tests to assess secondary hemostasis include prothrombin time (PT), activated partial thromboplastin time aPTT), activated coagulation time (ACT), and fibrinogen. Specific clotting factors can be measured by specific laboratories. Fibrinolysis can be assessed by measuring clot breakdown products (FDPs, D-dimers). Other tests are available but are often unavailable, impractical, or cost-prohibitive.

More recently, global assessments of hemostasis are becoming more available. For example, viscoelastic testing can assess multiple aspects of hemostasis concurrently including fibrinolysis. Thromboelastography (TEG) and thromboelastometry (ROTEM) are the most established, but newer tests such as Entegrion’s VCM Vet have made rapid cage-side (point-of-care) viscoelastic testing possible. While the complete test may take an hour to complete, only a few minutes are typically needed to get an initial impression of coagulation status. Minimal training is required, and the cartridge-based test requires only a small volume (~0.3 mL) of native whole blood (no citrate). Abnormalities of different components of viscoelastic tracing may correlate with

transfusion requirements. Further studies are warranted to determine the indications and clinical utility of such modalities.

Acute Traumatic Coagulopathy

Severely traumatized patients may develop a syndrome referred to as acute traumatic coagulopathy, which is characterized by hypocoagulability and hyperfibrinolysis. The mechanism is likely multifactorial and is due to hypoperfusion, severe tissue injury, systemic inflammation, metabolic acidosis, and hypothermia. Human patients with ATC have a much higher mortality rate. Overzealous fluid administration may exacerbate ATC and fluids must be used cautiously. Diagnosis is based on clinical findings as well as laboratory findings. Viscoelastic testing may provide a more sensitive test, but further research is needed.

Other Causes of Bleeding

Although trauma and injury is the likely cause of bleeding, other causes may need to be considered. Thrombocytopenia may result from increased destruction (ITP, DIC), decreased production (drugs, bone marrow issues, tick-borne diseases), consumption (bleeding), or sequestration (splenic torsion). Thrombocytopathia may be congenital (Glanzmann's, Scott syndrome, Caldag-GEF-1, vWD), drug-related (NSAIDs, clopidogrel, synthetic colloids), or disease-related (uremia, liver failure). Secondary hemostasis and clotting factors issues may be due to hereditary (hemophilia A, hemophilia B), anticoagulant rodenticide, liver disease, and DIC. Coagulation may also be impaired by drugs such as heparin, fibrin degradation products (from DIC), and envenomation. Excessive fibrinolysis (hyperfibrinolysis) is becoming increasingly recognized. In addition to trauma, sepsis and DIC also contribute.

Treatment

Prevention of further blood loss and stabilization is the first step. Apply pressure or pack wounds if possible. Some deeper wounds may stop bleeding by applying deep digital pressure. Foley catheters may be utilized in these cases to provide pressure by inserting them into the wound and inflating the Foley. Consider medications such as lysine analogs (aminocaproic acid, tranexamic acid) or Yunan Baiyao for non-compressible bleeds. The lysine analogs function by discouraging clot breakdown (clot stabilizers) and gained prominence after the landmark 2013 CRASH-2 trial. These drugs can also be applied topically.

Judicious fluid therapy is warranted, but anemia is not a contraindication for fluid administration. Currently, the improvement in perfusion is thought to outweigh the dilution of red cells. The thought is that the same absolute number of red cells and hemoglobin is still there and by giving fluid, we improve the delivery of that hemoglobin/oxygen to the tissues. Hypertonic saline (7.2%, 4 mL/kg) can be used in a *low-volume resuscitation* strategy. Hypertonic saline is especially attractive in patients with head trauma as it may decrease intracranial pressure as well. *Hypotensive resuscitation* or *permissive hypotension* is another strategy closely linked to low-volume resuscitation. With hypotensive resuscitation, a lower blood pressure target (SBP =90) is chosen to avoid pushing too much fluids and causing the patient to bleed more (e.g. popping the clot), but also not so low that perfusion is completely compromised. This strategy is not the goal,

but a compromise until hemostasis is achieved (e.g. surgery for a ruptured spleen). Think of hypotensive resuscitation as a bridge to another more definitive treatment. If there is not a more definitive treatment planned, hypotensive resuscitation is not appropriate. Hypotensive resuscitation is also completely contraindicated in patients with head trauma as hypotension is associated with increased mortality. Synthetic colloids may be considered but may contribute to further bleeding and increase the risk of AKI. There is insufficient data to completely ban the use of synthetic colloids, but caution should be exercised.

If fluid therapy is not sufficient, consider transfusing blood products early. While overly simplistic, if they're losing blood, give them blood. This can be achieved with fresh whole blood if a donor is available, and the timing is practical, or component therapy with packed red blood cells and fresh frozen plasma. Some patients may require large amounts of blood products. A massive transfusion has been defined as an entire blood volume within 24 hours, half a blood volume within 3 hours, or 1.5 mL/kg/min of red cells in 20 minutes. Early transfusion of FFP may decrease the total amount of red cells needed. These patients need to be monitored closely. Electrolyte abnormalities such as hypocalcemia (from citrate) may require supplementation. Hypomagnesemia can also occur due to citrate. Hyperkalemia is also possible. Autotransfusion can improve volume and oxygen-carrying capacity but will typically not provide clotting factors or platelets. The safety of autotransfusion must also be considered as the blood may be contaminated with infectious agents or mixed with neoplastic cells.

While there are many approaches to calculating the volume for transfusions, I prefer to keep it simple. If patients need more red blood cells to carry oxygen, I will start with 10 mL/kg of pRBCs or 20 mL/kg of fresh whole blood. If patients are bleeding and need clotting factors give at least 10 mL/kg of fresh frozen plasma. For more severe cases, aim for 20-30 mL/kg of plasma.

References available from the author.