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AHC Media

Trauma Updates: Fluid Resuscitation in Traumatic Hemorrhagic Shock and Blunt Cerebrovascular Injury

The American College of Surgeons in 2008 released the Advanced Trauma Life Support (ATLS) updates (eighth edition) with recommendations based on evidence-based medicine in addition to the expert consensus. A general summary and explanation of the updates were previously published in Trauma Reports.[1] *The authors provide a more detailed report of the literature since the last ATLS update on fluid resuscitation and blunt cerebrovascular injury, based on their relevance and importance to patient outcomes.*

Optimal management of hypotensive trauma patients is still under investigation, with the method of fluid delivery and the type of fluid used controversial. The concept of permissive hypotension has been around since the early 1900s, but has been revisited since military experience and landmark studies indicated that aggressive fluid resuscitation might cause more harm than originally anticipated.

Blunt cerebrovascular injury (BCVI) is another topic that has recently garnered more attention from physicians managing victims of motor vehicle collisions. For years it was thought to be a rare occurrence, but advances in imaging technology have identified BCVI as a more common entity. BCVI is catastrophic, with a high morbidity and mortality in previously young healthy people, so it is critical to identify, risk stratify, and treat these patients appropriately.

— Ann M. Dietrich, MD, Editor

Fluid Management in the Trauma Patient

Trauma is the most common cause of death among young people in the United States and around the world.[2] Hemorrhage is the most common cause of preventable death in the setting of trauma.[3-5] A long debate has ensued over the optimal fluid resuscitation regimen. More recently, damage control resuscitation has received more attention. This section will discuss permissive hypotension, also known as controlled resuscitation or delayed hypotension, and other fluid resuscitation strategies, including the choice of resuscitation fluids.

Permissive Hypotension

Permissive hypotension is defined as maintenance of lower than physiologic blood pressures while maintaining organ perfusion and preventing hemorrhage.[6] During permissive hypotension, the systolic blood pressure is maintained between 70 mm Hg and 90 mm Hg. Maintenance of blood pressure at these levels allows natural compensatory mechanisms to ensue while arrangements are made for more definitive control of blood loss, such as surgery or interventional radiologic procedures. Clot formation, vasoconstriction, and a catecholamine surge are several of the mechanisms theorized to compensate for hypovolemic shock. When fluids are administered, it is speculated that the clot may become dislodged by increased velocity of blood flow and dilatation of vasculature. Dislodgement of the clot at the site of injury can lead to rebleeding

Executive Summary

- Balanced fluid resuscitation involves correcting coagulopathies with packed red blood cells (PRBC) and fresh frozen plasma (FFP) while surgically controlling the source of bleeding and administering normal saline or lactated Ringers solutions cautiously to prevent hemodilution.
- The ideal fluid for resuscitation has not been determined since no product has been found to be superior to others.
- The evidence is clear that proper treatment of BCVI and TCAI improves mortality.
- The associated risk factors for BCVI include: any cervical spine fracture, unexplained neurological deficit, basilar cranial fracture into the carotid canal, Le Fort II and III fractures, cervical hematoma, Horner syndrome, cervical bruit, ischemic stroke, head injury with Glasgow Coma Scale (GCS) < 6, and hanging or anoxic brain injury.

and resultant decompensation. The body may also stop releasing catecholamines that are maintaining blood pressure as the body perceives a return to the normovolemic state. Permissive hypotension is part of a broader strategy called damage control resuscitation, designed to resuscitate a traumatically injured patient by preventing acidosis, hypothermia, and multi-organ dysfunction through balanced fluid resuscitation. Balanced fluid resuscitation involves correcting coagulopathies with packed red blood cells (PRBC) and fresh frozen plasma (FFP) while surgically controlling the source of bleeding and administering normal saline or lactated Ringers solutions cautiously to prevent hemodilution.[7]

The concept of permissive hypotension applies to the hypovolemic trauma patient and doesn't include those with traumatic head injury or spinal cord injury. In a head injury, brain perfusion is maintained at all costs to minimize secondary brain injury due to hypoperfusion; cerebral perfusion pressure (CPP) equals the mean arterial pressure (MAP) minus intracranial pressure (ICP). Maintenance of an adequate CPP prevents brain infarct or ischemia. Typically the lower threshold of normal CPP is 60-70 mm Hg.[8] Normal intracranial pressure is usually 7-15 mm Hg. Therefore, mean arterial pressure is typically maintained at 70-85 mm Hg to maintain CPP. The same concept applies to spinal cord injury due to a similar concern for hypoperfusion of the spinal cord. There is also concern that permissive hypotension should not be applied to the elderly due to their decreased hemodynamic reserve and compensation mechanisms compared to younger people. There hasn't been conclusive research on permissive hypotension in pregnant women, but an opinion article supports the use of damage control resuscitation strategies in hemorrhaging pregnant women requiring massive transfusion.[9]

General Fluid Management

Pathophysiology. The pathophysiology of aggressive fluid resuscitation is currently under investigation. Studies are examining the best composition of resuscitation fluid as well as the optimal rate of administration. Crystalloid can rapidly expand intravascular volume so tissue perfusion can be restored; however, in excess, it can lead to cell edema, resulting in acute respiratory distress syndrome (ARDS), acute lung injury, and compartment syndromes of the extremities or the abdomen. Colloid in excess leads to increased infection rates and multi-organ system dysfunction.

A lethal triad of hypothermia, coagulopathy, and acidosis has been described as the cause of morbidity and mortality in trauma. This damage is related to the amount of fluid given during resuscitation.[10] Coagulopathy is of particular importance during fluid resuscitation because it can theoretically be prevented by adding FFP, PRBC, platelets, or other blood products to prevent hemodilution. About one-third of trauma patients will develop a coagulopathy if their hemorrhage leads to multiple organ failure.[11]

Diagnostic Evaluation

Diagnostic studies for all hypotensive trauma patients should include CBC, chemistry, PT, INR, PTT, type, and screen. A pregnancy test and toxicology studies can be included in select cases to investigate other sources of hypotension or complicating factors in care. Important laboratory studies that aid in identifying the complications of massive blood transfusion include calcium, potassium, and chloride levels.[12] Lactate and base deficit are useful in identifying underresuscitation as well as the initial severity of shock. Stored packed red blood cells are anticoagulated with citrate, which binds calcium and can lead to hypocalcemia with multiple transfusions. During transfusion, calcium levels need to be monitored and IV calcium administered as needed.[10] Intravenous calcium gluconate or chloride can be used to replace deficient levels. Potassium levels should be monitored and treated appropriately. A large amount of tissue injury in trauma can induce hyperkalemia. This should be treated with hydration, albuterol, insulin, dextrose, kayexalate, and calcium gluconate if it is severe. Transfusion with PRBCs can cause hyperkalemia because of lysis of stored cells and is treated in the same manner. Hypokalemia can also develop due to hemodilution and metabolic causes related to the stress of the trauma. Hyperchloremia

induced by normal saline administration is an iatrogenic finding that should be addressed by decreasing normal saline administration and starting a maintenance fluid, avoiding normal saline. Low fibrinogen levels and thrombocytopenia have also been used as markers of dilution coagulopathy.[13] Although this is not a comprehensive discussion of laboratory markers of fluid resuscitation in the trauma patient, key electrolytes should be monitored during fluid resuscitations, and abnormalities should be treated accordingly.

Imaging should include a chest and pelvis X-ray and focused assessment with sonography in trauma (FAST). When working at a facility where transfer to a trauma center may be needed, transfer of care may take precedence over CT scans and other diagnostic studies.

Differential Diagnosis

The differential diagnosis of hypotension is broad; however this section specifically discusses traumatic hypovolemic shock or hemorrhagic shock. Consideration should be given to the other forms of shock, including neurologic, cardiac, and distributive/septic. Other common diagnoses in the setting of traumatic hypotension include pericardial tamponade, tension pneumothorax, and cardiac contusion. Drugs, alcohol, medications, and baseline health conditions should also be considered in the differential diagnosis.

Management

Optimal management of hypotensive trauma patients is still under investigation. This article will review both the method of fluid resuscitation, rate of infusion, and the types of fluids used during the resuscitation. All of these factors impact morbidity and mortality and are important components of the initial resuscitation of traumatic shock patients.

Method of Fluid Resuscitation

Recent studies have demonstrated the efficacy of permissive hypotension as a method of fluid resuscitation.[3,14,15] Randomized, controlled trials of U.S. civilians have been difficult to complete, so most of the recent literature on this topic comes from animal studies, international studies, or military initiatives. Multiple animal studies support the use of permissive hypotension.[16-18] These studies indicate that a target MAP of 40 mm Hg allows for maintenance of splanchnic perfusion, tissue oxygenation, and decreased blood loss. Some of these animal studies demonstrate less acidemia, hemodilution, thrombocytopenia, and coagulopathy. These physiologic improvements were associated with less tissue and cellular injury. However, there is a limit to the amount of time tissues can sustain the lower MAP. In one study, after 8 hours there was an increase in the physiologic complications and tissue/cell injury. Based on the findings of these various studies, ATLS guidelines state that, "In penetrating trauma with hemorrhage, delaying aggressive fluid resuscitation until definitive control may prevent additional bleeding."[19-23] This guideline is based on a key study that demonstrated a clear survival advantage using delayed resuscitation for young patients with penetrating torso trauma.[24] The European Multidisciplinary Committee for Advanced Bleeding Care published recommendations in the 2010 edition of *Critical Care* that include minimizing the time elapsed between injury and definitive surgical management.[11]

ATLS continues to support the use of a 3-for-1 rule (3 mL of crystalloid should be used as replacement for every 1 mL of blood loss), but also encourages frequent reassessments if large amounts of crystalloid are not providing adequate resuscitation. ATLS also dictates treatment based on the class of hypovolemic shock. In class I and II, crystalloid is the preferred fluid, with an initial bolus of 2 L normal saline or lactated Ringers. For class III and IV, the patient should receive colloid in addition to crystalloid.[25]

The standard of care has been the institution of massive transfusion protocols for patients who have the anticipated need of more than 10 units PRBCs. A typical massive transfusion protocol would include 6-10 units PRBC, 4-6 units FFP, and 1-2 units of platelets.[26-28] Another method attempted for massive transfusion is a 1:1 ratio of blood to plasma, which has been found to improve survival in combat; however, other studies show higher incidences of septic complications and organ failure with this formula.[25] The standard of care is to institute a massive transfusion protocol at this time.

Type of Fluid for Resuscitation

In addition to the amount of fluid resuscitation and blood pressure goal, the type of fluids used in the resuscitation is an area of ongoing debate. Systematic reviews indicate there is inadequate evidence to support the advantages of one fluid resuscitation method over another.[3,14,15] A Cochrane article determined that there is inadequate evidence to support one form of fluid resuscitation over another, so the expert consensus provided by ATLS is the current standard.[14] The following sections will review different types of fluid used for resuscitation and will discuss caveats and benefits.

Crystalloid. Various types of crystalloid have been proposed for fluid resuscitation. Hypertonic saline with or without dextran, colloid, and crystalloid have all been studied and are all non-superior in preventing mortality.[3,15,29-35] In addition, all of the studies demonstrated that fluid type did not significantly influence the transfusion requirements.[3,29-34,36-37] Multiple animal and human studies have demonstrated that extensive crystalloid resuscitation may increase the incidence of metabolic abnormalities and decrease survival. Some studies support the use of L-isomer LR because it decreases inflammation, immune dysfunction, and electrolyte abnormalities, but this is based on bench science as opposed

to actual mortality rates in human studies.[14] The Cochran review concluded there were not enough data to support use of hypertonic crystalloid for trauma resuscitation.[38]

Colloid: Packed Red Blood Cells, Fresh Frozen Plasma, and Platelets. Blood transfusion is an independent predictor of trauma mortality.[10] Mortality rates are almost 50% for patients who require massive blood transfusion.[3,39] Use of larger amounts of FFP and platelets improves mortality, but also increases risk of infection and ARDS.[13] Reducing the number of units of platelets and FFP transfused to patients, however, did not improve mortality.[40] The principal risks of massive transfusion protocols are infection, mortality, systemic inflammatory response syndrome (SIRS), and multiple organ failure.[10] Other complications of massive blood transfusions are acute hemolytic transfusion reactions, febrile nonhemolytic transfusion reaction, transfusion-related acute lung injury, transfusion-associated circulatory overload, allergic reactions, hypocalcemia, hypokalemia, hyperkalemia, acidosis, hypothermia, dilutional coagulopathy, and thrombocytopenia.[10] The delayed complications associated with massive blood transfusion are hemolytic transfusion reaction, transfusion-related immune modulation, transfusion-transmitted disease, and post-transfusion graft-versus-host-disease.[10] The military has reported the best results with whole blood for resuscitation; however, this is not feasible in the civilian population given the risk of infectious transmission.[41]

Albumin. Albumin has been identified as nonsuperior to other fluid resuscitation products.[42-46] The SAFE trial investigated albumin compared to normal saline in critically ill patients and found there was no difference in mortality overall; however, there was an association with increased mortality in the albumin group in patients with traumatic brain injury. Therefore, albumin is not recommended in patients with potential brain trauma, and since albumin is more expensive and does not have a survival benefit, it is not commonly used or recommended in critically ill patients.[46] A Cochrane review summarized that albumin may increase mortality and should not be used outside of tightly controlled clinical trials.[47] PRBC, FFP, platelets, or colloid and crystalloid products have been just as successful, if not better, in resuscitation and are less expensive and easier to obtain. The use of albumin is not recommended.

Other Fluid Resuscitation Blood Products. Other options for managing traumatic hemorrhage are emerging and deserve mention, specifically the antifibrinolytics and antihemorrhagics. The CRASH-2 study indicates that tranexamic acid (TXA), an antifibrinolytic agent, may be beneficial in controlling trauma hemorrhage.[3,13,48,49] TXA improved mortality and was not associated with an increase in vascular occlusions, while it decreased the amount of blood products necessary to resuscitate patients.[49,50] A Cochrane review concluded that "TXA safely reduces mortality in bleeding trauma patients without increasing the risk of adverse events."[50] Recombinant activated factor VII (RFVIIa), an antihemorrhagic, led to improved coagulopathy and decreased blood requirements, but did not affect or improve mortality, so it is not recommended at this time.[13,51]

Summary

In summary, the ideal fluid for resuscitation has not been determined since no product has been found to be superior to others.[13,14] The choice of fluid used for resuscitation does not appear to affect morbidity or mortality in the trauma patient. A combination of crystalloid and colloid may be of value and warrants further study. Minor injury may be managed better with crystalloids, while colloids are necessary for moderate to severe injury. TXA can be considered in severely hemorrhaging patients.

Permissive hypotension should be considered when resuscitating trauma patients in hypovolemic shock while waiting for prompt definitive treatment. It should not be instituted in head trauma or spinal cord injury patients. Finally, a massive transfusion protocol should be considered in severe hemorrhage.

Blunt Carotid and Vertebral Vascular Injuries

Definition of the Problem. Traumatic blunt cerebrovascular injury (BCVI), if undetected, can be a devastating diagnosis that holds great morbidity and mortality. Since trauma affects a younger population, BCVI frequently causes strokes in young previously healthy patients. BCVI is potentially treatable with anticoagulant and antiplatelet regimens or surgical intervention, making early detection paramount. Originally thought to be a rare diagnosis, BCVI is now diagnosed more often due to the advent of CTA and other screening tools. The ATLS 8th edition of 2008 added a new section on BCVI and included screening recommendations, including use of CTA for detection of BCVI.[52] This section will discuss the scope, etiology, diagnosis, and treatment of BCVI, which includes two distinct subsets: traumatic carotid artery injury (TCAI) and traumatic vertebral artery injury (TVAI).

Epidemiology

BCVI occurs in about 0.18-3% of all blunt trauma patients.[12,53-58] Prior to increased screening, the incidence of BCVI in blunt trauma was thought to be about 0.08%. The more recent prospective screening studies document a true incidence of 0.32%, and in some studies as high as 1.05%.[12,53-61] Many of the patients with this injury were not identified because of the severity of their other injuries and, therefore, the lack of a good neurological exam.

Studies vary in sample size and methodology, which results in varying improvements in mortality, but the evidence is clear that proper treatment of BCVI and TCAI improves mortality.[62-72] A study from 2011 indicates overall in-hospital

mortality for BCVI is 11%, and after following patients for 22 months, an overall mortality of 16%.[73] One study from 2002 suggests that overall mortality for TCAI was 25%, with 21% attributable to other injury or sequelae. Of those with TVAI, there was a 9% mortality.[54] Some of the earlier studies demonstrated higher mortality.[71] This higher mortality rate may be due to the decreased detection rate in patients in the earlier years, among other factors. Patients have a greatly increased mortality from TCAI if not treated, 64% versus 6.8% with treatment. Mortality for TVAI is 54% untreated, versus 2.6% with treatment. TCAI has a 13% stroke-related mortality, and TVAI has a 4% stroke-related mortality.[69] BCVI-related mortality was reduced from 59% to 21% with implementation of a more rigorous screening protocol.[74] It is important to keep in mind that these studies had different inclusion criteria, different numbers of patients included in the study, and many were retrospective trauma database searches. There are a multitude of factors influencing these results. One of the most recent retrospective reviews demonstrated 24% stroke-related deaths in untreated patients for BCVI that could have been prevented with treatment.[75]

Etiology

Risk factors for BCVI can help guide decision-making about imaging and further diagnostics. The Denver criteria for screening for BCVI include both TVAI and TCAI diagnoses and are summarized in Table 1.[76] The associated risk factors for BCVI include: any cervical spine fracture, unexplained neurological deficit, basilar cranial fracture into the carotid canal, Le Fort II and III fractures, cervical hematoma, Horner syndrome, cervical bruit, ischemic stroke, head injury with Glasgow Coma Scale (GCS) < 6, and hanging or anoxic brain injury.[76]

BCVI is most commonly associated with motor vehicle collisions, but is also associated with assault, hanging, clothesline injuries, and sports injuries such as those from swimming.[76-80] Less common associations include chiropractic manipulation or physical therapy involving neck manipulation.[81-83] Another predisposing factor is an existing connective tissue disease, since this can lead to traumatic disruptions with minor events.[84] Sometimes BCVI can occur apparently spontaneously or with very minor trauma.[81,85,86] Younger children are at higher risk of injury because of their less developed neck musculature and large head-to-body ratio.[77] There may be a predisposition for BCVI in females, with 41% of BCVI occurring in women, although only 30% of the blunt trauma population are women.[87]

Table 1. Denver Criteria for Screening[66]

Signs and Symptoms of BCVI	Risk Factors for BCVI
Arterial hemorrhage	High-energy mechanism
Cervical bruit	Le Fort II or III
Expanding cervical hematoma	Basilar skull fracture with carotid canal involvement
Focal neurological deficit	Diffuse axonal injury with GCS < 6
Neurological exam inconsistent with head CT	Cervical spine fracture: subluxation, transverse foramen, or C1-C3 vertebrae
Ischemic stroke on repeat head CT	Near-hanging with anoxic brain injury

Table 2. Grading System of BCVI[109]

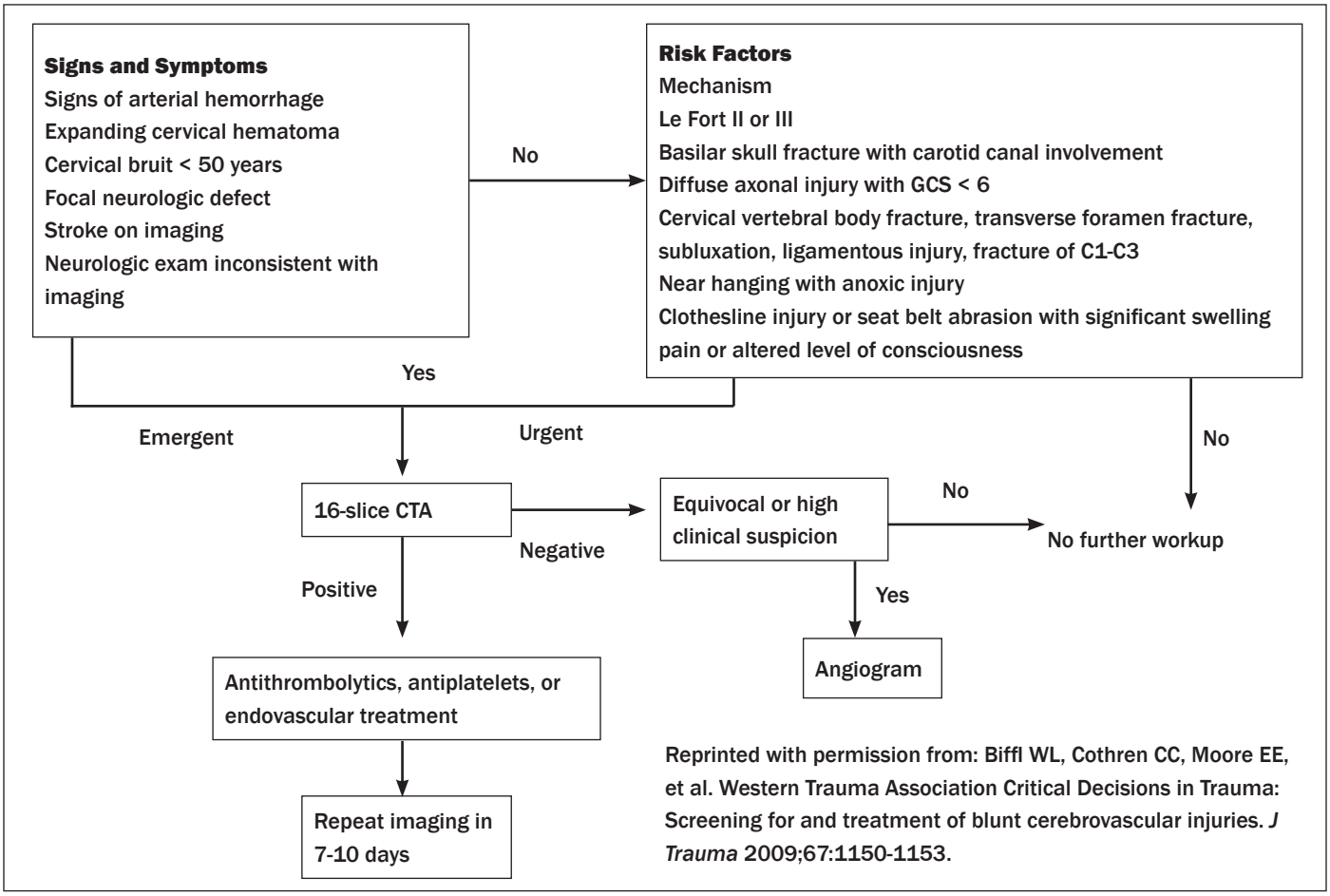
I: Irregularity of vessel wall, dissection or intramural hematoma with < 25% stenosis
II: Intramural thrombus, raised intimal flap, dissection or intramural hematoma with > 25% stenosis
III: Pseudoaneurysm
IV: Vessel occlusions
V: Vessel transection

Pathophysiology

The three forces associated with vascular injuries are rotational (lateral flexion), hyperextension, and hyperflexion.[76-78,88] In rotational forces, the carotid artery can collide against the vertebral body, especially in the upper cervical spine, or impact the styloid process, causing damage to the vessel. Hyperextension causes vessel injury by stretching the carotid artery while colliding against the cervical vertebrae. In hyperflexion, the carotid artery is forced between the cervical spine and mandible. Sometimes severe mandibular fractures lead to injury of the carotid artery. Subluxation, dislocation, or ligamentous injury allows movement of the vessels, particularly the vertebral arteries, which can then impact against bone or sustain shear forces, leading to vascular injury.[76] Both vessels are at high risk for injury when cervical spine fractures occur, most commonly C1-C3.[76] Fracture fragments from the cervical spine or skull base can directly injure the vessels. Another means of injury is direct injury to the neck, from a seat belt, sports, or clothesline injury, as well as intraoral trauma from an object.[77]

Vessel injury can lead to immediate

Figure 1. Management of BCVI[108]



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stroke, either embolic or ischemic. More often, there is a latent period during which the injury gradually progresses, due to occlusion by a clot, aneurysm, or released emboli, resulting in a stroke.

Clinical Features

Patients may present with stroke symptoms on arrival to the emergency department, but a majority of patients have what has been described as a latent period. This is a period of time after the initial injury during which patients are asymptomatic. The duration of the latent period is variable, with 23-50% of patients developing BCVI stroke symptoms more than 12 hours after the event.[56,66,86,88-92] This provides a window of opportunity to treat the injury if the diagnosis can be made promptly.

One study that followed patients after hospital discharge found that of the 40 BCVI patients with a CVA, 17 presented with symptoms on arrival, 8 developed symptoms before treatment, 4 had symptoms after failure of medical or endovascular treatment, and 6 developed symptoms after discharge (3%).[73] Another study indicated that of patients without neurologic symptoms within the first two hours of injury, there was a mean delay to stroke of 75 hours.[93] In this study, there were no reported strokes after discharge; however, the patients were not followed. Yet another study demonstrated that neurological injury could occur 4-75 days after the insult.[94] Due to the possibility of delayed stroke, patients diagnosed with a vessel injury are typically anticoagulated for 7-10 days until re-imaging is performed and, if there is evidence that the injury has resolved, anticoagulation may be stopped. Anticoagulation is maintained for 3-6 months if the injury does not resolve. For patients undergoing endovascular stenting, 6-12 months of anticoagulation is the typical treatment regimen.

TVAI symptoms typically include headache, neck pain, gait and sensory abnormalities, dizziness, nausea, vomiting, altered level of consciousness, speech abnormalities, and visual disturbances. One of the keys to identify a syndrome consistent with TVAI is looking for symptoms of posterior circulation ischemia.[76] Signs of posterior circulation ischemia include dysarthria, balance problems, ataxia, visual field deficits, diplopia, nystagmus, Horner's syndrome, hiccups, lateral or medial medullary syndrome, cranial nerve palsies, pupil

abnormalities, and altered level of consciousness.

TCAI symptoms can include face/arm/leg weakness, difficulty with speech, sensory abnormalities, headache, and neck pain. Key findings with TCAI for anterior circulation or anterior cerebral artery ischemia include contralateral leg weakness. The middle cerebral artery typically causes contralateral face and arm weakness or language deficits. Signs of TCAI include abnormal pulses, Horner's syndrome, and other neurological disturbances, and they occur most commonly in the anterior circulation. Sometimes a bruit or a hematoma may be observed.

If patients do not present with obvious neurological deficits, other clinical features should make the clinician suspicious of BCVI. Thoracic trauma of any kind is often present when BCVI is identified.[12] A higher injury severity score (ISS) is associated with BCVI. Patients with BCVI typically have an ISS in the range of 28-32 compared to a statistically significant lower score in non-BCVI patients.[59,73,94]

The Denver criteria, discussed earlier, are useful for BCVI screening.[56] (*See Table 1.*) Approximately one-third of patients fitting one or more of these criteria will have an associated vascular injury on angiography.[52,56,60,61,91,95-97] Based on the Denver criteria, there was a 33-48% risk of BCVI with one risk factor, 56-74% with two risk factors, 88% with three risk factors, and 93% with greater than four risk factors.[56]

The Memphis criteria are a second screening tool for BCVI. These criteria are similar to the Denver criteria and include cervical spine fracture, neurologic findings not explained by brain imaging, Horner's syndrome, Le Fort II or III, skull base fractures involving the foramen lacerum, and neck soft-tissue injury such as a seat belt or hanging injury.[54] Patients with one of the Memphis criteria had a 29% risk of BCVI.[54,77]

Screening criteria are insensitive and can miss up to 20% of injuries. Despite not being included in the published screening criteria, one study identified isolated cervical spine fractures at the level of C4-C7 with a 9% risk of BCVI. Another study demonstrated cervical spine fractures, basilar skull fractures, mandible fractures, higher ISS, and lower GCS as high-risk criteria for BCVI.[74] In a meta-analysis of the current literature, only cervical spine fracture and thoracic injury were associated with BCVI.[12] It has also been suggested that petrous bone fractures and any basilar skull fractures, even if they don't include the carotid canal, should be added to the screening criteria, but there is insufficient evidence for conclusive recommendations at this time.[98,99] The evidence suggests that any cervical spine fractures should be considered as independent criteria for screening for BCVI, since they would have been otherwise missed by the currently available screening criteria.[59,74,81,100] Both Eastern and Western trauma associations agree that any cervical spine fracture should be included in the high-risk group and should be evaluated for BCVI.

A cervical seat belt sign has been described as a mark or contusion on the neck induced by a seat belt. The seat belt sign has been documented in several studies to be present in 0.76-3% of patients with BCVI.[7,101,102] An expanding neck hematoma is a hard sign that requires further imaging to exclude BCVI by angiography or CTA. It is typically more difficult to determine the need for imaging with an abrasion or contusion on the neck. Clinical experience and correlation with neurologic symptoms and other injuries will assist the clinician in determining the need for further evaluation. Some studies report that the seat belt sign is very poorly correlated with BCVI when used as an isolated risk factor, so it should be used in combination with other risk factors.[101,102] In conclusion, conservative management would include the seat belt sign as a criterion for further evaluation of BCVI, especially when associated with other injuries.

The Eastern Association for Trauma Surgery also encourages the use of the same criteria for selection of pediatric patients for further imaging because there are no pediatric-specific screening criteria.[103] Some of the most commonly identified associated pediatric injuries were cervical spine fracture, thoracic and abdominal injury, and traumatic brain injury.[104] BCVI was identified in 72% of asymptomatic children using the adult screening criteria, but more than 75% of patients with neurologic symptoms were not otherwise identified by the adult criteria.[104] This study found a strong association in these patients with thoracic trauma, non-basilar skull fractures, and traumatic brain injury.[104] All children involved in a high-risk mechanism trauma or direct injury to the neck need to be considered for imaging to exclude BCVI.[104]

Diagnostic Studies

Digital subtraction angiography is consider the gold standard diagnostic study for BCVI, but is limited by a 0.5% risk of stroke, risk of bleeding into the thigh or retroperitoneum, expense, and accessibility.[59,76]

Acceptable alternatives include CTA and MRA.[59,76] MRA has not been systematically analyzed for detection of BCVI, and small studies indicate it is inferior to CTA.[54] CTA is typically the first-line screening tool for suspected BCVI. Angiography should be pursued in all suspect cases with a high level of suspicion with negative or inconclusive CT imaging.[87,103] The sensitivity and specificity of CTA vary widely in the literature.[59-61,87,97,105,106] Two studies indicate that specificity and sensitivity can reach 100% with properly trained radiologists,[97,105] but there are also two studies indicating that sensitivity is just 51% and 68%.[59,60,87] A 16-slice CT scanner or greater is the screening modality of choice recommended by both the Western and Eastern trauma associations and is supported by a study comparing 4-slice to 16-slice CT scanners.[60] A 32-slice scanner does not appear to capture more BCVI diagnoses.[87] Ultrasonography of the vessels prevents radiation exposure, but is

limited by the bone obscuration and neck immobility. In one ultrasound study, the sensitivity was 38.5% with a specificity of 100%.[107] At this time, CTA is considered by most experts to be the screening tool of choice for noninvasive diagnostics.

Differential Diagnosis

When evaluating a patient with suspected BCVI, other diagnoses should be considered, including CVA, subdural hemorrhage, intraparenchymal hemorrhage or epidural hemorrhage, drugs, alcohol, overdose, spinal cord contusions or syndromes, seizure, and Todd's paralysis. This differential is not all-inclusive, but provides a basis for a complete evaluation of a suspected BCVI patient.

Management

All patients with symptoms or signs of BCVI should receive emergent imaging by CTA. A management algorithm has been developed by the Western Trauma Association.[108] (*See Figure 1.*) Once identified, treatment of BCVI is based on the Denver grading system.[76,109] The grading (*see Table 2*) correlates with prognosis and clinical severity of the injury only in traumatic carotid artery injury. Grade I includes irregularities of the vessel wall with less than 25% occlusion of the lumen. Grade II includes a comparable injury to grade I, but also has greater than 25% occlusion of the blood vessel lumen. Grade III encompasses pseudoaneurysms, and grade IV describes a complete occlusion, while grade V is a complete transection.

Intravenous heparin therapy is generally regarded as the standard of treatment with a goal PTT of greater than 40 seconds (40-60 seconds) or 1.5 to 2 times normal.[73] Patients are typically observed as inpatients while receiving anticoagulation with heparin and then are transitioned to warfarin for a total of 3-6 months of therapy.[73,87] It has been suggested that they should be followed up with imaging in 7-10 days to look for improvement or worsening by possible fistula or aneurysm formation. If the imaging shows significant improvement, the anticoagulation may be stopped; however, if there is development of a pseudoaneurysm (lesion progresses to a grade III), the patient should be treated with heparin and a stent. If the lesion remains stable, the patient should be treated with warfarin for 3-6 months with re-imaging. If surgery is required, the heparin drip can be held 2 hours preoperatively and 4 hours postoperatively.[73]

The next most common regimen is antiplatelet drugs such as aspirin 325 mg.[55] Antiplatelet therapy is most often used for small vessel irregularities or grade I lesions.[87] In studies comparing antiplatelet therapy and heparin, antiplatelet therapy was noted to be noninferior and seemingly equivalent in terms of mortality and stroke prevention.[12,89,103] Clopidogrel 300 mg can be used in place of aspirin if needed.

The third-line choice of agents is both an anticoagulant and an antiplatelet agent together.[59] If an endovascular approach is taken with placement of a stent, the patient can be started on aspirin 325 mg prior to stent placement. After the procedure, the same dosage is continued with the addition of heparin.

Each grade of injury is treated slightly differently. Grade I injuries are typically managed medically with aspirin, but could also be treated with heparin. Grade II lesions can also be managed medically, more likely with heparin than aspirin, or with an endovascular intervention. Typically any patient with a grade III injury will receive heparin, but can also be managed with endovascular interventions.[87,109] Medical management is often used because the vessel may be too friable for successful endovascular intervention. Typically, grade IV lesions are treated with coil embolization or stent placement.[89] Grade V lesions typically require surgery. Ultimately, the treatment regimens will be determined on a case-by-case evaluation at the discretion of the interventional, vascular, or trauma physician. The final decision on the best method depends on the patient's inherent vasculature anatomy and location of injury.

Absolute contraindications to anticoagulation include bleeding, anticipated surgery, or bleeding diatheses. Relative contraindications include external ventricular drain, intracerebral contusion, and subdural hematoma.[76] Bleeding is the most common complication from antiplatelet or anticoagulant therapies.[66,77,93] This includes rebleeding from an intracranial injury or other visceral or orthopedic injuries. Typically, patients with BCVI and concomitant severe trauma cannot be anticoagulated, so consideration should be given to surgical intervention by stenting, coil embolization, or arterial occlusion.

Treatment of BCVI is effective and decreases morbidity and mortality. Patients who are not effectively managed have up to a 30% risk of death within 6 hours of admission or development of a massive stroke.[89] The rate of stroke with BCVI was 12.2% if treated by some method, whether stenting, embolization, antiplatelet, or anticoagulants, compared to 26% if left untreated, with 50% mortality among patients who had a stroke.[89] Another study demonstrated a 21% incidence of stroke if BCVI was left untreated, with a mortality rate of 30%.[93] One study has demonstrated that it is cost effective to screen for BCVI given the high risk of morbidity and mortality.[67] Thus, it is imperative to quickly identify an injury and institute treatment.

Disposition

All patients with risk factors for TCAI or TVAI should be admitted for further observation if urgent imaging for BCVI cannot be completed in the ED. If a CTA is negative, the overall clinical condition of the patient should be assessed to determine further investigation for BCVI with angiography. If angiography is negative for disruption, the patient can be discharged with instructions describing neurological warning signs. Otherwise, the

patient will be admitted with BCVI and managed as described above.

Summary

Defined screening criteria for BCVI have been published, but clinicians should have a high index of suspicion in all trauma patients, especially those with chest, head, and neck injuries. If suspected, the patient should receive a CTA. An angiogram, which is the gold standard, may be ordered in patients with a suspicion for BCVI even with a negative CTA. Typical treatment includes anticoagulants, antiplatelets, and endovascular stenting or embolization, depending on the grade of the injury. Extensive morbidity and mortality can be prevented with early identification and treatment.

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CNE/CME Questions

1. What is an indication for CTA radiological evaluation for carotid artery injury in trauma?
   A. transverse foramen injury
   B. C1-C3 fractures
   C. C-spine subluxation
   D. all of the above
2. What is the most commonly used SBP range for permissive hypotension?
   A. 80-90 mm Hg
   B. 80-100 mm Hg
   C. 70-90 mm Hg
   D. 60-70 mm Hg
3. What is the ideal fluid for resuscitation of trauma patients?
   A. lactated Ringers
   B. normal saline
   C. PRBC
   D. an ideal fluid has not been definitively determined
4. When is permissive hypotension contraindicated?
   A. penetrating trauma
   B. long bone fractures
   C. head trauma
   D. splenic or liver injury
5. What is the most common treatment for blunt cerebrovascular injury?
   A. heparin
   B. aspirin
   C. endovascular stenting
   D. clopidogrel
6. Which of the following is a contraindication to the treatment of blunt cerebrovascular injury with anticoagulants?
   A. visceral injury requiring surgery
   B. hypotension
   C. intracranial hemorrhage
   D. all of the above
7. What is the usual mechanism of BCVI injury?
   A. hyperflexion, hyperextension
   B. lateral rotation
   C. axial compression
   D. A and B
8. When should BCVI patients be reimaged to determine if the lesion is healing?
   A. 3 days
   B. 1 week
   C. 4 weeks
   D. 6 weeks
9. What is an adverse effect associated with colloid treatment of traumatic injury?
   A. infectious disease transmission
   B. hypocalcemia
   C. ARDS
   D. all of the above
10. What are some of the adverse effects of crystalloid treatment in excess?
   A. cell edema
   B. acute respiratory distress syndrome
   C. compartment syndromes of the extremities
   D. all of the above

CNE/CME Objectives

Upon completing this program, the participants will be able to:

- discuss conditions that should increase suspicion for traumatic injuries;
- describe the various modalities used to identify different traumatic conditions;
- cite methods of quickly stabilizing and managing patients; and
- identify possible complications that may occur with traumatic injuries.

CNE/CME Instructions

HERE ARE THE STEPS YOU NEED TO TAKE TO EARN CREDIT FOR THIS ACTIVITY:

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