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AHC Media

Pancreatitis

Epidemiology

Pancreatitis is common disease that causes significant morbidity and mortality. In the United States, pancreatitis frequently leads to emergency department (ED) visits and subsequent hospitalization. It is estimated that more than 200,000 patients are admitted to the hospital with pancreatitis each year.[1]

Gallstones and excessive alcohol use are the two most common causes of acute pancreatitis. Patients at increased risk of developing gallstone pancreatitis include those older than the age of 60 years and those who have stones that are smaller than 5 mm.[2,3] While gallstones are the most common cause of pancreatitis, only 4% of patients with gallstones will develop pancreatitis.[4]

Excessive alcohol use is the second most common cause of pancreatitis. Casual alcohol use is not a risk factor for pancreatitis. There is considerable debate about the role that alcohol plays in the development of pancreatitis. In patients who drink more than five drinks per day, alcohol is thought to have a dose-dependent relationship with the risk of pancreatitis. Other risk factors include smoking and HIV.[5] Patients with HIV have a risk that is 35-800 times compared to otherwise healthy patients.[4]

Behind gallstones and alcohol, idiopathic pancreatitis is the third most common cause of inflammation. Patients in this category typically have no obvious explanation for their symptoms; however, recent data have suggested that these patients may have inflammation from microlithiasis, or non-visualized gallstones, within their biliary system.[3] If this theory holds, this third category of patients may be combined with the larger subset of patients suffering from gallstone pancreatitis.

Post-operative pancreatitis can be caused by a variety of surgical procedures and can present a specific diagnostic challenge. Surgeries involving the hepatobiliary system are the most common cause of post-operative pancreatitis. In patients who have had recent surgery, it can be difficult to differentiate between typical post-operative pain and pain that may be caused by an episode of acute pancreatitis. In addition to abdominal surgeries, procedures such as cardiac valve surgery and renal transplant can increase the patient's likelihood of developing acute pancreatitis. Chung et al found that 5.9% of patients who underwent cardiac valve replacement developed acute pancreatitis.[6] For reasons that are somewhat unclear, patients with post-operative pancreatitis tend to have more severe disease course and a higher rate of complications. Bragg et al reported that 53% of patients with post-operative pancreatitis developed significant complications, such as pseudocysts, abscesses, and fistulas.[7]

Certain medications increase the risk of acute pancreatitis. Corticosteroids and less widely used medications such as didanosine and pentamidine, used to treat HIV, are known to cause pancreatitis. Additionally, cases of pancreatitis have been attributed to the use of valproic acid, various chemotherapeutic agents, and assorted statin agents. Interestingly, estrogen has been linked to cases of pancreatitis and is thought to cause inflammation indirectly by increasing triglycerides.[8]

Executive Summary

- Pancreatitis results from the release of trypsin. There are many causes of pancreatitis, but gallstones are the most common.
- Post-operative pancreatitis can be seen after many different types of surgery, but is most common after surgery on the hepatobiliary tree. Such patients may be difficult to distinguish from those with post-operative pain.
- Measuring amylase in addition to lipase adds little, if anything, in establishing the diagnosis or prognosis of pancreatitis.
- Patients with chronic pancreatitis present with pain. Most have a history of alcohol abuse. Many will have normal amylase and lipase levels.

A recent study found diabetic patients who took glucagon-like peptide-1-based therapies such as exenatide and a dipeptidyl peptidase 4 inhibitor, sitagliptin, had twice the risk of hospitalization for acute pancreatitis when compared to a control group of diabetic patients who were not taking these agents. Many media outlets have reported on the results of this study, and patients taking these agents should be educated about the symptoms associated with acute pancreatitis and should be closely monitored.[9]

Genetic cases of pancreatitis are thought to be caused by defects in several genes. In these patients, trypsinogen is activated inappropriately and the pancreas undergoes auto-digestion.[10]

The remaining known causes for pancreatitis are varied and include autoimmune disorders such as systemic lupus erythematosus, hypertriglyceridemia, hypercalcemia, hyperparathyroidism, infection, and traumatic, iatrogenic, vascular, and environmental causes, including cases reported after bites from scorpions and Gila monsters.[11] Two lesser known risk factors for developing pancreatitis are pregnancy and type 2 diabetes. In pregnancy, the risk of pancreatitis is thought to be related to an increase in the level of triglycerides. While the mechanism of pancreatitis is less clear in type 2 diabetics, these patients have a 2.8-times greater risk of developing acute pancreatitis when compared to non-diabetic patients.[12]

Pathophysiology

Pancreatitis occurs as a result of an inflammatory process in the pancreas. Initially, trypsin, a proteolytic enzyme, is inappropriately activated within the pancreas. Typically this enzyme aids digestion in the duodenum, but when activated in the pancreas, it causes intrapancreatic inflammation and local cell damage.[13] Next, the pancreas activates the inflammatory cascade, releasing cytokines that can spread the inflammatory process to other organ systems, including the kidneys and lungs. Ten percent to 15% of patients with pancreatitis develop systemic inflammatory response syndrome (SIRS).[14] Patients with more severe cases of pancreatitis are at risk for developing acute respiratory distress syndrome (ARDS). This may occur when inflammatory mediators released by the pancreas cause vasodilatation and pulmonary inflammation, resulting in respiratory distress and potential respiratory failure. In addition, the patients with acute pancreatitis can develop overwhelming sepsis and multi-system organ failure.[15] The mortality rate approaches 50% in patients with multi-organ failure and pancreatitis.[16]

There is typically an inciting event that triggers the release of trypsin from the pancreas. In gallstone pancreatitis, the biliary stones lodge within the common bile duct. As bile starts to accumulate behind the impacted stone, the intraductal pressure increases, which can lead to trypsin release. In cases of alcohol-induced pancreatitis, the trigger mechanism is not as well understood.

Cases of pancreatitis tend to fall into one of two categories: edematous and necrotizing. Edematous pancreatitis, which makes up approximately 80% of the cases, is typically a benign process and has a mortality of less than 1%. Necrotizing pancreatitis, which accounts for the remaining 20% of the cases, is much more severe and has a mortality rate of 10-24 %.[17] During the initial assessment in the emergency department, it is difficult to distinguish between these two types.

Differential Diagnosis

Epigastric pain can be caused by disorders of the heart, esophagus, stomach, and aorta. Patients with any component of chest pain or epigastric discomfort may have acute coronary syndrome. Aortic dissection should be considered, particularly when there are symptoms such as a "tearing" or "ripping" pain that radiates to the back. Gastroesophageal reflux is a more benign cause of epigastric pain that can mimic pancreatitis. In some cases, pancreatitis can cause peri-umbilical or lower abdominal pain. In these patients, diseases such as appendicitis, diverticulitis, mesenteric ischemia, and other potentially dangerous intra-abdominal issues need to be considered.

Diagnosis

Clinical Features. The classic presentation of pancreatitis involves a fairly abrupt onset of upper abdominal pain. Pain will typically reach a point of maximal intensity fairly suddenly, often within an hour, and will persist for more than 24 hours in a majority of cases.[15] While the pain can present in the right or left upper quadrant, pain will often localize to the epigastric or peri-umbilical

region. The pain is often associated with nausea and vomiting. The pain will radiate to the back in approximately 50% of patients; however, patients can also experience radiations to the lower abdomen and up into the chest.[18]

In mild cases, patients may have some mild abdominal tenderness to palpation and, as the severity of the disease progresses, this may develop into significant abdominal tenderness with guarding and rebound. In more severe cases, patients may become critically ill and develop hemodynamic instability in the form of fever, tachycardia, and hypotension.

Laboratory Tests. Amylase is secreted by the acinar cells in the pancreas and rises within 6-24 hours. Amylase will stay elevated for anywhere from 3-7 days.[19] While an elevated amylase can be suggestive of pancreatitis, a significant number of patients with pancreatitis will have a normal amylase level in the ED. Up to 30% of patients who present with abdominal pain and have a normal amylase are actually suffering from pancreatitis.[20] Amylase is a less reliable indicator of pancreatic inflammation in patients who are chronic, heavy drinkers.

Lipase is also released from the acinar cells in the pancreas. Serum lipase levels rise as early as 4-8 hours after the onset of disease, peak at around 24 hours, and remain elevated for up to 14 days. This early rise in serum lipase is helpful in patients who present early in their disease course. In addition, lipase stays elevated for a significantly longer period than amylase. Lipase is more sensitive for pancreatitis in patients who have inflammation from chronic alcohol abuse.[21] Lipase is now the preferred laboratory test to use when evaluating patients for pancreatitis. Patients who have a lipase value that is three times the upper limits of normal very likely have pancreatitis, and a lipase level of greater than five times normal is almost 100% specific for acute pancreatitis.[22]

While lipase appears to be a superior test, both lipase and amylase can be used to identify pancreatitis in patients. Unfortunately, neither of these tests can be used to risk-stratify patients in terms of their risk of developing severe pancreatitis.

Despite the superior test characteristics of lipase over amylase, it is still common practice to order both tests for patients who present with abdominal pain. Studies have shown that adding an amylase value to a lipase value in the workup of a patient offers no diagnostic advantage over ordering a lipase.[23,24] This simply adds unnecessary cost. Over the course of the year, eliminating amylase reduced departmental charges by $350,000.[25]

In patients with pancreatitis, low volume status has been shown to be a significant risk factor for death. A recent study found that a BUN of greater than 20 mg/dL was associated with an odds ratio of 4.6 for mortality. Patients with an elevated hematocrit had a more severe disease course.[26] In patients with no signs of hemoconcentration and a hematocrit below 44%, the risk of developing pancreatic necrosis is lowered.[27]

In patients without a history of alcohol abuse, an alanine aminotransferase (ALT) value three times normal has a 95% positive predictive value for gallstone pancreatitis.[28] While an elevated ALT can help identify patients with gallstone pancreatitis, a patient with biliary pancreatitis can have completely normal liver function tests. Up to 20% of patients with acute gallstone pancreatitis will have normal liver function tests.

Patients with acute pancreatitis may have low serum calcium. The exact mechanism behind this hypocalcemia is unclear but is due, in part, to the chelation of calcium by fatty acids released when the pancreas becomes inflamed. Patients with low calcium levels may complain of paresthesias and have hyperreflexia and QT prolongation on their EKG. These patients should receive calcium replacement as clinically indicated.[15,29]

A CRP of greater than 130 mg/dL can be indicative of patients who are at increased risk of developing a complicated disease course. Typically this value is obtained at 72 hours, which limits its use in the emergency department. Additionally, CRP is

Table 1: Ranson's Criteria

Ranson's Criteria on admission
• Age greater than 55 years
• A white blood cell count > 16,000/μL
• Blood glucose > 11 mmol/L (> 200 mg/dL)
• Serum LDH > 350 IU/L
• Serum AST > 250 IU/L
Ranson's Criteria after 48 hours of admission
• Fall in hematocrit by more than 10%
• Fluid sequestration of > 6 L
• Hypocalcemia (serum calcium < 2.0 mmol/L) (< 8.0 mg/dL)
• Hypoxemia (PO2 < 60 mmHg)
• Increase in BUN to > 1.98 mmol/L (> 5 mg/dL) after IV fluid hydration
• Base deficit of > 4 mmol/L
The prognostic implications of Ranson's Criteria are as follows:
• Score 0 to 2: 2% mortality
• Score 3 to 4: 15% mortality
• Score 5 to 6: 40% mortality
• Score 7 to 8: 100% mortality

Adapted from: Ranson JH, Rifkind KM, Roses DF, et al. Prognostic signs and the role of operative management in acute pancreatitis. *Surg Gynecol Obstet* 1974;139:69-81.

Table 2: Common Causes of Acute Pancreatitis

- Gallstones
- Alcohol abuse
- Hypertriglyceridemia (greater than 1000 mg/dL)
- Autoimmune
- Drug-induced
- Post-procedural (after endoscopic retrograde cholangiopancreatography)
- Infections
- Trauma
- Idiopathic

Adapted from: Mitchell RM, Byrne MF, Baillie J. Pancreatitis. *Lancet* 2003;361: 1447-1455.

fairly non-specific and can be elevated by other disease processes such as pneumonia or cholangitis.[30] Given these limitations, CRP is not part of the ED workup.

Trypsinogen activation peptide (TAP) is typically elevated in patients with acute pancreatitis. TAP levels at the time of admission correlate with disease severity; however, this test is not available to most emergency department practitioners.[31] Urine trypsinogen 2 is measured using a point-of-care urine dipstick. While more research is needed, early data show this urine dipstick outperforms CRP in its ability to predict cases of severe pancreatitis. Additional markers such as pro-calcitonin and interleukin have been studied in the evaluation of patients with possible pancreatitis.[32]

Imaging

A computed tomography (CT) scan can help identify more severe cases of pancreatitis. Not all patients with acute pancreatitis need imaging during the ED workup; decisions to obtain these studies should be made on a case-by-case basis. Some patients may require emergent imaging to eliminate other potential causes of abdominal pain, while other patients may be appropriately treated and admitted without having any imaging performed.

Transabdominal ultrasound is readily available in most emergency departments and can evaluate patients for possible gallstone pancreatitis. Diagnosing common bile duct stones by ultrasound has a low sensitivity but a high specificity. The presence of common bile duct dilation without common bile duct stones lacks the sensitivity and specificity necessary to identify patients with biliary pancreatitis. Findings such as the presence of mobile gallstones or sludge in the gallbladder are suggestive of gallstone pancreatitis but lack the diagnostic accuracy necessary to confirm the diagnosis.[33]

In addition to evaluating the gallbladder, transabdominal ultrasound can be used to evaluate the pancreas itself. While views of the pancreas are often limited due to bowel gas, ultrasound can show pancreatic enlargement, echotextural changes, and fluid surrounding the pancreas.[34] While not available to emergency practitioners, endoscopic ultrasound is emerging as a highly accurate means of identifying stones in the common bile duct. With increasing use of endoscopic ultrasound, patients who previously have been thought to have idiopathic pancreatitis are now being correctly diagnosed with having a biliary source to their inflammation.[35]

In the emergency department, practitioners should have a high index of suspicion for patients who present with acute pancreatitis and have no history of alcohol abuse. A reasonable approach would be to obtain a transabdominal ultrasound in these patients.

Contrast-enhanced CT is the standard imaging technique used to identify acute pancreatitis.[36] CT scans in patients with acute pancreatitis serve two purposes. First, the CT findings can be used to predict disease severity. Additionally, a CT scan can help identify complications that arise from pancreatic inflammation.[33]

Balthazar et al have developed a CT Severity Index (CTSI) to categorize the severity of acute pancreatitis using CT findings. The CTSI looks at two categories. First, the severity of the pancreatic inflammation is given a grade of A through E, which corresponds to scores of 0 to 4. The second half of the CTSI looks for the presence of pancreatic necrosis. The extent of necrosis is described as none, less than one-third of the tissue, one-half, or greater than one-half. These four categories are given scores of 0, 2, 4, and 6. The extent of inflammation and the evaluation of necrosis are combined and patients are given a cumulative score ranging from 0 to 10.[37] Patients with a CTSI score of greater than 5 have an associated mortality rate that is 15 times higher than patients who have a score of less than 5. In addition to this increased mortality rate, patients who scored greater than 5 had a higher rate of complications and a longer hospital length of stay when compared to patients with a lower score.[38] In an observational study, CTSI was found to predict complications and mortality more reliably than the Ranson's criteria.[39] (*See Table 1.*)

One of the limitations of CTSI is that it is based on CT findings on scans performed 72 hours after admission. Patients who are scanned earlier in the course of their disease may not have reached the most severe point in their disease and may not have reached the full extent of pancreatic inflammation or necrosis. While these patients may have lower scores than they would receive if evaluated at 72 hours, a recent study has shown a correlation between the CTSI and the rate of mortality and complications when patients are evaluated at 48 hours.[40]

In the emergency department, the majority of patients who present with acute pancreatitis do not need a CT scan. Flezler et al evaluated the role that CT plays during the emergency

department assessment of patients with acute pancreatitis. In their study group, patients who underwent CT scanning had a longer emergency department length of stay and a prolonged stay in the hospital.[41] However, CT scan can be useful in patients with acute pancreatitis who appear severely ill. If severe inflammation, early necrosis, or other complications are seen, these patients may require admission to an intensive care unit or transfer to a facility that has readily available gastroenterology and surgery. In addition, CT can be useful in identifying other disease processes in patients when the source of their abdominal pain is somewhat unclear.

Scoring Systems

The majority of patients with pancreatitis have a very low rate of morbidity and mortality; however, approximately 20% of patients will have more severe complications. Often it is difficult to identify this at-risk subset of patients based on their presentation in the emergency department. Various authors have developed scoring systems that attempt to identify patients who are at increased risk of having a bad outcome during their episode of pancreatitis. While most of these scoring systems can be difficult to apply during the patient's initial evaluation in the emergency department, recently several scores have been proposed that are applicable for emergency practitioners.

Ranson's criteria (*see Table 1*) risk-stratify patients by several factors, both at the time of admission and then again during the initial 48 hours of the patient's evaluation.[42] The need to obtain data after admission severely limits the applicability of these criteria in the emergency department. The Acute Physiology and Chronic Health Evaluation (APACHE II) scale was derived to evaluate patients who are critically ill, and includes arterial blood gas values and the patient's past medical history as components of the score. Emergently, it can be difficult to obtain a comprehensive past medical history necessary to properly derive an APACHE II score. In addition to the practical limitations of calculating Ranson's and APACHE II scores, neither of these clinical scoring systems has been shown to accurately predict mortality or length of hospitalization in patients with acute pancreatitis.[38]

The Atlanta Classification of Severe Acute Pancreatitis was developed in an effort to take elements from various scoring systems and radiographic studies and reach a definition of what determines the severity of a course of pancreatitis. The initial criteria, developed in 1992, used the Ranson's criteria, APACHE II scale, and the presence of various CT findings.[43] The Atlanta criteria were revised in 2008 and the process of acute pancreatitis was divided into an acute phase and a subsequent phase. The acute phase refers to the first week of disease. During this time frame, the criteria focus on the presence or absence of multi-system organ dysfunction. After the first week, during the subsequent phase, more emphasis is placed on the morphologic CT findings. Patients were categorized as having "mild" or "severe" pancreatitis. These revised criteria proved to be somewhat problematic.[44]

In 2012, a determinant-based classification system was developed. This system used the presence of pancreatic necrosis and the degree of organ dysfunction as the two major data points that were necessary to categorize the severity of a patient's pancreatitis. CT scan findings were used in addition to laboratory data, physical exam findings, and clinical gestalt to help place patients in one of four categories. Patients were defined as having mild, moderate, severe, or critical acute pancreatitis.[45] While these consensus criteria have a very limited role in the emergency department, they do emphasize the risk associated with multi-organ system dysfunction and pancreatic necrosis in patients who present with acute pancreatitis. Emergency practitioners should identify patients with these two findings as being at significantly increased risk of having an adverse outcome.

There are two scoring systems that are applicable in the emergency department. The Bedside Index for Severity of Acute Pancreatitis (BISAP) (*see Table 3*) and the Harmless Acute Pancreatitis Score

Table 3: Bedside Index for Severity of Acute Pancreatitis (BISAP)

- BUN > 25
- Impaired mental status
- SIRS (> 2 criteria)
- Age > 60 yrs
- Pleural effusion on CT scan
- 1 point for the presence of each finding.

BUN, blood urea nitrogen; SIRS, systemic inflammatory response syndrome

BISAP Score	Observed Mortlity
0	0.1%
1	0.4%
2	1.6%
3	3.6%
4	7.4%
5	9.5%

Adapted from: Wu BU, Johannes RS, Sun X, et al. The early prediction of mortality in acute pancreatitis: A large population-based study. *Gut* 2008;57:1698-1703.

Table 4: Harmless Acute Pancreatitis Score (HAPS)

Assess for the following features:
- No signs of peritonitis
- Normal serum creatinine
- Normal hematocrit

If all three features are present, it is 98% accurate at identifying patients with a non-severe disease course.
"Non-severe"= no death, need for dialysis, or artificial ventilation.

Adapted from: Lankisch PG, Weber-Dany B, Hebel K, et al. The harmless acute pancreatitis score: A clinical algorithm for rapid initial stratification of nonsevere disease. *Clin Gastroenterol Hepatol* 2009;7:702-705.

(HAPS) (*see Table 4*) were designed in an effort to identify high-risk patients with pancreatitis during their initial evaluation. The BISAP has five components. Patients are given one point per variable: blood urea nitrogen greater than 25 mg/dL; the presence of the systemic inflammatory response syndrome (SIRS); age greater than 60 years; pleural effusion seen on imaging; and altered mental status, defined as a Glasgow Coma Score of less than 15.[46] Patients with scores of 3,4, and 5 have corresponding mortality of 5.3%, 12.7%, and 22.5%. In addition to predicting mortality, the BISAP has been validated to predict the risk of persistent organ failure in patients with acute pancreatitis.[47] As with all of the scoring systems for acute pancreatitis, the BISAP is not intended to fully risk-stratify patients, as patients with acute pancreatitis and a low BISAP score are not deemed low risk enough to be discharged home. Patients with an elevated BISAP score are at increased risk of a bad outcome and should receive close monitoring and management.

The HAPS uses even simpler clinical criteria. Patients are evaluated for the following three features: the absence of rebound tenderness and/or guarding, normal hematocrit, and normal serum creatinine. Patients who had all three elements of the score were thought to have a "non-severe" disease course, with 98% positive predictive value.[48] As with the BISAP, HAPS is not intended to identify patients that can be appropriately discharged home.

While the HAPS and BISAP can be used to risk-stratify patients in the emergency department with pancreatitis, it is not clear that these scoring systems guide initial therapy or provide any information that would not be apparent to the physician at the bedside. For most emergency practitioners, patients who have high scores in any of the scoring systems would likely be identified as "sick." As with any other "sick patients," common treatments such as fluid resuscitation, airway management, pain management, and admission to an appropriate level of care are reasonable for patients with acute pancreatitis.

Management

Despite the benign course that is seen with most patients, no good data exist that suggest that any subset of patients can be treated as outpatients. The main goals of hospitalization are to reduce the morbidity and mortality associated with acute pancreatitis. Patients with severe disease need to be identified and treated, while less severe cases require pain management, adequate nutrition, and proper consultation by specialists. (*See Table 5.*) In patients who develop severe disease, the role for treatments such as ERCP, MRCP, and possible surgical intervention is somewhat unclear. In addition, treatments such as enteral feeding, broad spectrum antibiotics, and pain management have all been studied in an attempt to decrease the morbidity and mortality associated with acute pancreatitis.

Pain Management

Patients with pancreatitis often have pain that requires narcotic analgesia. Traditionally, practitioners have been taught to limit the use of morphine out of concern that morphine and its analogs may cause a spasm of the sphincter of Oddi, which could lead to a worsening of the patient's symptoms. In an effort to avoid this potential worsening, meperidine became a popular agent used to treat patients' pain. Multiple studies have shown meperidine to have questionable efficacy and a problematic safety profile. Additionally, more recent studies have shown that morphine has a minimal impact on the sphincter of Oddi and is a suitable agent to use for pain control in the setting of pancreatitis.[49] Other opioids such as fentanyl and hydromorphone, as well as nonsteroidal anti-inflammatories, can be used. Once the patient is admitted, techniques such as thoracic epidural analgesia can be used to provide further pain control and to limit the potential for systemic effects that are common with traditional pain medications.[50]

Fluid Resuscitation

Patients with acute pancreatitis often require aggressive fluid resuscitation. Cytokines and other inflammatory mediators released from the pancreas cause vasodilatation and decrease intravascular volume, which can lead to end-organ hypoperfusion. Studies have found that patients who receive inadequate fluid resuscitation have an increased morbidity and mortality. In the emergency department, most patients with acute pancreatitis should receive a bolus of 1-2 liters. After this initial bolus, patients should be placed on an infusion with a rate of 250-300 mL/hr. Fluid therapy can be adjusted to maintain a urine output of 0.5 mL/kg/hr. There is no consensus on the most appropriate type of IV fluid to use in patients during the initial resuscitation. While most patients need aggressive fluid resuscitation, therapy should be adjusted for each individual patient. Patients who show

signs of volume overload such as hypoxia, jugular venous pulsations, or crackles on lung exam may require a less aggressive approach to fluid resuscitation.[19]

Nutrition

Traditionally it was thought that feeding these patients would cause additional stimulation to the pancreas and would worsen the course of the disease. In the emergency department, patients should be made NPO during their initial workup and treatment, and further decisions regarding nutrition should be made in consultation with the admitting service. Even in the most well-appearing patients, there are few data to suggest that patients with acute pancreatitis can tolerate early oral intake, however. Approximately 20% of patients will have a relapse of pancreatitis when they resume eating.[51]

Antibiotics

Patients with acute pancreatitis who experience pancreatic necrosis have a significant increase in mortality. Numerous studies have evaluated the role that antibiotics play in the prevention of pancreatic necrosis. In 2001, a meta-analysis found that in patients with acute necrotic pancreatitis, using prophylactic antibiotics reduced the rates of sepsis, caused a decrease in the need for surgical intervention, and led to an overall mortality benefit.[52] A more recent double-blinded, randomized, controlled trial compared patients treated with a placebo to patients given ciprofloxacin and metronidazole and showed no significant difference in the rate of complications, infection, or overall mortality. While the benefit from antibiotics in the treatment of pancreatitis is unclear, there are well-documented complications from antibiotic therapy, including adverse drug reactions and emerging rates of drug-resistant pathogens. Emergency practitioners should not use antibiotics for the treatment of acute pancreatitis without a clear consideration of potential risks and benefits.[53]

Table 5: Summary Recommendations for the Management of Acute Pancreatitis in the First 24 Hours

Intervention	Recommendation
Prognostication of severity	Use BISAP or HAPS score, monitor BUN or hematocrit
Fluid resuscitation	Use 250-300 cc/hr of IV fluids Titrate to urine output or changes in BUN or hematocrit
Prophylactic antibiotics	No indication
Early ERCP in biliary pancreatitis	ERCP only if cholangitis or worsening cholestasis with declining clinical course

Adapted from: Fisher JM, Gardner TB. The "golden hours" of management in acute pancreatitis. *Am J Gastroenterol* 2012;107:1146-1150.

Interventions

Endoscopic retrograde cholangiopancreatography (ERCP) has been used to treat acute pancreatitis thought to be secondary to gallstones. The initial theory behind this procedure was that removing obstructions within the biliary tree would relieve the upstream pressure and reduce the degree of inflammation in the pancreas. Removing these gallstones is also thought to lower the risk of associated cholangitis. Typically, ERCP is performed within the first 48-72 hours of the hospital course.

The data on the benefits of performing ERCP are somewhat varied. A 2004 Cochrane review evaluated outcomes in patients with pancreatitis who underwent ERCP. Patients with severe disease who underwent ERCP were found to have a lower rate of complications when compared to patients who were managed with more conservative non-invasive therapy. While complication rates were lower in the ERCP group, these patients had no reduction in mortality. In patients with mild pancreatitis, there was no difference in the rate of complications or the rate of mortality between the two groups.[54] While the Cochrane review failed to show a mortality benefit, later studies have shown a survival benefit in a select group of patients with severe gallstone pancreatitis.[55]

Approximately 10% of patients who undergo ERCP will have a complication from this procedure. The most common complication is the development of post-ERCP pancreatitis. While less common, other complications such as bleeding, infection, and biliary perforation are well-documented risks associated with the procedure.[56]

For emergency practitioners, it is important to consider the need for ERCP in all patients who present with acute pancreatitis. Only a small subset of these patients will actually need to have this procedure performed, but there is a subset of patients who will benefit. Patients with presumed biliary pancreatitis who show signs of biliary sepsis, cholangitis, worsening jaundice, or elevated bilirubin may benefit significantly from an ERCP.

Magnetic retrograde cholangiopancreatography (MRCP) is a noninvasive test that can be used to evaluate patients for possible biliary obstruction. Known as the "pancreatogram," MRCP provides views similar to the ERCP. Unlike the ERCP, which can be both diagnostic and therapeutic, MRCP does not allow for any interventions. MRCP is also limited in its ability to detect small biliary stones and ductal strictures, which are seen more easily on ERCP. While it is not needed in every patient who presents with pancreatitis, when available, MRCP may provide an alternative diagnostic test in patients with possible biliary pancreatitis who are unable to undergo an ERCP.[57]

Endoscopic ultrasound is an emerging technology that may

benefit select patients. Endoscopic ultrasound is helpful in identifying biliary stones and tumors and has been used to identify patients who might benefit from an ERCP.[58]

Surgical Intervention

Patients who present with gallstone pancreatitis need urgent evaluation by a surgery team. Up to 50% of patients with gallstone-induced pancreatitis will have a recurrence of pancreatitis within two months unless they undergo a cholecystectomy. While cholecystectomy will eliminate the source of future gallstones, patients with retained gallstones may have recurrent pancreatitis after having their gallbladder removed. Patients with retained stones may benefit from ERCP. Cholangiography allows the provider to evaluate the biliary system for retained stones, and is similar to ERCP and endoscopic ultrasound in terms of its accuracy in identifying biliary obstructions.[59] A recent randomized, controlled trial evaluating patients with gallstone pancreatitis found that early cholecystectomy reduced hospital length of stay by approximately 1.5 days.[60]

Apart from performing cholecystectomy and cholangiography, there is a limited role for surgical intervention in the treatment of acute pancreatitis. Patients with significant pancreatic necrosis may benefit from surgical debridement. Patients who have infected pancreatic necrosis and show signs of clinical deterioration may need urgent surgical intervention. For patients who are thought to have sterile necrosis, there is little role for urgent instrumentation. Malangoni et al found that delaying or foregoing surgical debridement for patients with sterile necrosis resulted in a decreased mortality rate. Likewise, patients who underwent surgical intervention had a significantly higher rate of nosocomial infection. In addition, patients who undergo hepatobiliary surgery are at risk of developing worsening pancreatitis. Procedures such as pancreatic biopsy and interventions on the common bile duct run a higher risk of causing post-operative pancreatitis, but most other intra-abdominal procedures do carry a risk of causing worsening pancreatitis.[35] While not all patients with suspected gallstone pancreatitis will need emergent intervention, it is prudent to consult both gastroenterology and surgery services during the intitial workup for co-management and consideration for procedural intervention.

Cutting Edge/ Controversy

Chronic Pancreatitis. Patients with chronic pancreatitis pose an interesting, and at times frustrating, challenge for emergency practitioners. The etiology of chronic pancreatitis is unclear, and to date there are no readily available diagnostic tests used to identify this subset of patients. In addition, patients with chronic pancreatitis are heavy users of health care resources. These patients often require significant pain medications.

Chronic pancreatitis is thought to be caused by progressive and often increasing levels of inflammation in the pancreas. Typically, patients have several years of recurrent episodes of acute pancreatitis before developing chronic pancreatitis. In a study of chronic pancreatitis in Europe, the average patient was 50-70 years of age when they were diagnosed with chronic disease. Despite this predominance, there are case reports of patients developing chronic pancreatitis between the ages of 20 and 30 years.[61] Ongoing alcohol abuse is thought to be the causative factor of ongoing inflammation in up to 70% of patients with chronic pancreatitis. As with acute pancreatitis, alcohol is responsible for most of the cases of chronic pancreatitis; however, conditions such as autoimmune disease, hypertriglyceridemia, and ongoing pancreatic duct obstruction can cause a patient to develop chronic pancreatitis. Notably, up to 30% of patients who develop chronic pancreatitis are diagnosed as having idiopathic pancreatitis and have no clear etiology identified.[62]

For patients with chronic pancreatitis, pain is often the predominant initial symptom.[63] Amman et al identified these two subsets of patients and found that patients with short painful episodes were at low risk of developing complications and had essentially no need for surgical intervention. Patients with constant pain were at increased risk of developing structural changes, including pseudocyst formation, biliary strictures, and pancreatic stones, all of which might improve with surgical intervention.[64] It is thought that as the chronic changes progress, patients will have a reduction in the severity and frequency of their pain; however, research in this area is limited.[65]

It is difficult to identify patients with chronic pancreatitis. To date, no readily available laboratory tests show enzymatic changes suggestive of chronic pancreatic dysfunction. Thirty percent to 50% of patients with chronic pancreatitis will develop diabetes from endocrine dysfunction. In addition, patients with chronic pancreatitis may develop malabsorption due to a deficiency of enzyme release due to chronic changes. It is estimated that patients must lose 90% of their pancreatic function before they develop any demonstrable loss of enzyme function.[66]

As with acute pancreatitis, there is poor correlation between laboratory values and the severity of disease in patients with chronic pancreatitis. Patients may report having a "burned out" pancreas that, while capable of producing pain, is not capable of elevating the levels of amylase and lipase.

It can be difficult to treat patients with chronic pancreatitis. One of the obstacles to treating patients with chronic pancreatitis is the overlap between substance abuse and the development of a chronically painful condition. By definition, patients who develop chronic pancreatitis due to ongoing alcohol abuse have a predilection toward abusing addictive substances. To treat chronic pancreatitis, patients typically receive narcotic analgesia, which has a well-documented and significant risk

of causing desensitization and a need for increasing doses to obtain adequate levels of pain control. In addition, patients who are prone to addiction may be at increased risk of developing addiction to other substances.[67] Up to 70% of patients with chronic pancreatitis have problems with alcohol abuse. As a result, a significant portion of patients with chronic pancreatitis are at increased risk of becoming addicted to other substances, including the medications used to treat their chronic disease state. This risk of co-dependency is poorly studied; however, providers should be cognizant of this risk when treating patients who present with chronic pancreatitis and a history of substance abuse.

The mainstay of treatment for chronic pancreatitis is to limit exposure to the offending agent. Patients are advised to avoid dietary fat and alcohol in an effort to limit ongoing inflammation and pancreatic damage. Pancreatic enzyme supplementation has been studied in patients with chronic pancreatitis, but to date, the data to support their use are limited.[68]

Despite these attempts to reduce pain, patients frequently require narcotic analgesia to treat their symptoms. Given the risk of addiction associated with narcotics, patients with chronic pain may be best managed in a pain clinic. In addition to narcotic analgesia, patients with chronic pain may be treated with non-narcotic modulators of chronic pain such as gabapentin and selective serotonin inhibitors like paroxetine. While these agents have well-established efficacy in the treatment of chronic pain, their use is beyond the scope of practice of most emergency practitioners.

Disposition Decision. Well-appearing patients who present with frequent episodes of alcohol-induced pancreatitis can be particularly difficult to manage in the emergency department. Often these patients will have a history of alcohol use, abdominal pain, and a slightly elevated lipase, but will otherwise have a benign appearance. The difficulty with this approach is that, to date, there is almost no literature to support outpatient management of any patient who presents with pancreatitis. Despite significant efforts to risk-stratify patients with acute pancreatitis, there are no historical elements, physical exam findings, laboratory values, or imaging studies that can reliably identify a subset of patients that is safe to discharge from the emergency department.

Sphincter of Oddi Dysfunction. Patients with sphincter of Oddi dysfunction represent a different subset of patients who present with chronic upper abdominal pain and may complain of symptoms consistent with chronic pancreatitis. These patients have typically undergone a cholecystectomy and have chronic episodes of upper abdominal pain with no other signs of gastrointestinal distress. This poorly defined patient population is thought to have a functional biliary disorder. Patients will typically have undergone extensive hepatobiliary testing, including ERCP and HIDA scan, and will have an essentially normal workup. In the emergency department it is important to recognize the existence of these chronic pain patients, treat their pain adequately, while at the same time ensuring that no other emergent disease process is causing their symptoms. Beyond their course in the emergency department, these patients can be difficult to identify and manage, and their treatment often involves a multidisciplinary approach.[69]

Pancreatitis is a common cause of abdominal pain in patients who present to the emergency department. The majority of patients with acute pancreatitis will have a benign clinical course; however, it is difficult to identify the small subset of patients who are at increased risk of having a complicated course and an adverse outcome. In the emergency department, the primary goals of treatment include providing adequate analgesia, limiting oral intake, and admitting the patient to the appropriate level of care. Tests such as ultrasound, CT scan, and emergent ERCP may be helpful in some patients, but are not a necessary part of the workup for the large majority of patients.

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Physician CME Questions

1. What is the most common cause of acute pancreatitis?
   A. alcohol use
   B. gallstones
   C. drug-induced
   D. autoimmune

2. Which of the following drugs can increase the likelihood of a patient developing pancreatitis?
   A. alprazolam
   B. didanosine
   C. verapamil
   D. methadone

3. Which of the following tests should be ordered to evaluate a patient with potential pancreatitis?
   A. lipase
   B. blood urea nitrogen
   C. hematocrit
   D. all of the above

4. Which of the following scoring systems can be used to accurately identify patients with acute pancreatitis who can be managed as an outpatient?
   A. Ranson's
   B. APACHE II
   C. HAPS
   D. none of the above

5. Which of the following medications should *not* be used routinely in patients with acute pancreatitis?
   A. morphine
   B. hydromorphone
   C. fentanyl
   D. meperidine

6. In the emergency department, what is the appropriate goal urine output for patients with acute pancreatitis?
   A. 1 mL/kg/hr
   B. 0.5 mL/kg/hr
   C. 0.25 mL/kg/hr
   D. 2 mL/kg/hr

7. What nutrition status is most appropriate for patients in the emergency department with acute pancreatitis?
   A. clear liquids only
   B. NG tube feeds
   C. NPO
   D. normal diet

8. What is the rate of recurrence of pancreatitis for patients with gallstone pancreatitis who do not undergo a cholecystectomy?
   A. 20%
   B. 30%
   C. 40%
   D. 50%

9. Which of the following laboratory tests accurately predicts disease severity?
   A. amylase
   B. lipase
   C. sedimentation rate
   D. none of the above

10. What is the rate of alcohol abuse in patients with chronic pancreatitis?
    A. 40%
    B. 50%
    C. 70%
    D. 90%

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Emergency Medicine Reports
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Pancreatitis

Ranson's Criteria

Ranson's Criteria on admission

- Age greater than 55 years
- A white blood cell count > 16,000/µL
- Blood glucose > 11 mmol/L (> 200 mg/dL)
- Serum LDH > 350 IU/L
- Serum AST > 250 IU/L

Ranson's Criteria after 48 hours of admission

- Fall in hematocrit by more than 10%
- Fluid sequestration of > 6 L
- Hypocalcemia (serum calcium < 2.0 mmol/L) (< 8.0 mg/dL)
- Hypoxemia (PO2 < 60 mmHg)
- Increase in BUN to > 1.98 mmol/L (> 5 mg/dL) after IV fluid hydration
- Base deficit of > 4 mmol/L

The prognostic implications of Ranson's Criteria are as follows:

- Score 0 to 2: 2% mortality
- Score 3 to 4: 15% mortality
- Score 5 to 6: 40% mortality
- Score 7 to 8: 100% mortality

Adapted from: Ranson JH, Rifkind KM, Roses DF, et al. Prognostic signs and the role of operative management in acute pancreatitis. *Surg Gynecol Obstet* 1974;139:69-81.

Common Causes of Acute Pancreatitis

- Gallstones
- Alcohol abuse
- Hypertriglyceridemia (greater than 1000 mg/dL)
- Autoimmune
- Drug-induced
- Post-procedural (after endoscopic retrograde cholangiopancreatography)
- Infections
- Trauma
- Idiopathic

Adapted from: Mitchell RM, Byrne MF, Baillie J. Pancreatitis. *Lancet* 2003;361: 1447-1455.

Bedside Index for Severity of Acute Pancreatitis (BISAP)

- BUN > 25
- Impaired mental status
- SIRS (> 2 criteria)
- Age > 60 yrs
- Pleural effusion on CT scan
- 1 point for the presence of each finding.

BUN, blood urea nitrogen; SIRS, systemic inflammatory response syndrome

BISAP Score	Observed Mortlity
0	0.1%
1	0.4%
2	1.6%
3	3.6%
4	7.4%
5	9.5%

Adapted from: Wu BU, Johannes RS, Sun X, et al. The early prediction of mortality in acute pancreatitis: A large population-based study. *Gut* 2008;57:1698-1703.

Harmless Acute Pancreatitis Score (HAPS)

Assess for the following features:

- No signs of peritonitis
- Normal serum creatinine
- Normal hematocrit

If all three features are present, it is 98% accurate at identifying patients with a non-severe disease course.
"Non-severe"= no death, need for dialysis, or artificial ventilation.

Adapted from: Lankisch PG, Weber-Dany B, Hebel K, et al. The harmless acute pancreatitis score: A clinical algorithm for rapid initial stratification of nonsevere disease. *Clin Gastroenterol Hepatol* 2009;7:702-705.

Summary Recommendations for the Management of Acute Pancreatitis in the First 24 Hours

Intervention	Recommendation
Prognostication of severity	Use BISAP or HAPS score, monitor BUN or hematocrit
Fluid resuscitation	Use 250-300 cc/hr of IV fluids Titrate to urine output or changes in BUN or hematocrit
Prophylactic antibiotics	No indication
Early ERCP in biliary pancreatitis	ERCP only if cholangitis or worsening cholestasis with declining clinical course

Adapted from: Fisher JM, Gardner TB. The "golden hours" of management in acute pancreatitis. *Am J Gastroenterol* 2012;107:1146-1150.

Supplement to *Emergency Medicine Reports*, March 10, 2013: "Pancreatitis." *Authors:* **Matthew DeLaney MD**, Assistant Professor, Department of Emergency Medicine, University of Alabama at Birmingham; and **Carl Germann, MD, FACEP**, Assistant Professor, Tufts University School of Medicine, Attending Physician, Maine Medical Center, Portland, ME.
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Trauma Reports

EVIDENCE-BASED MEDICINE FOR THE ED

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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]

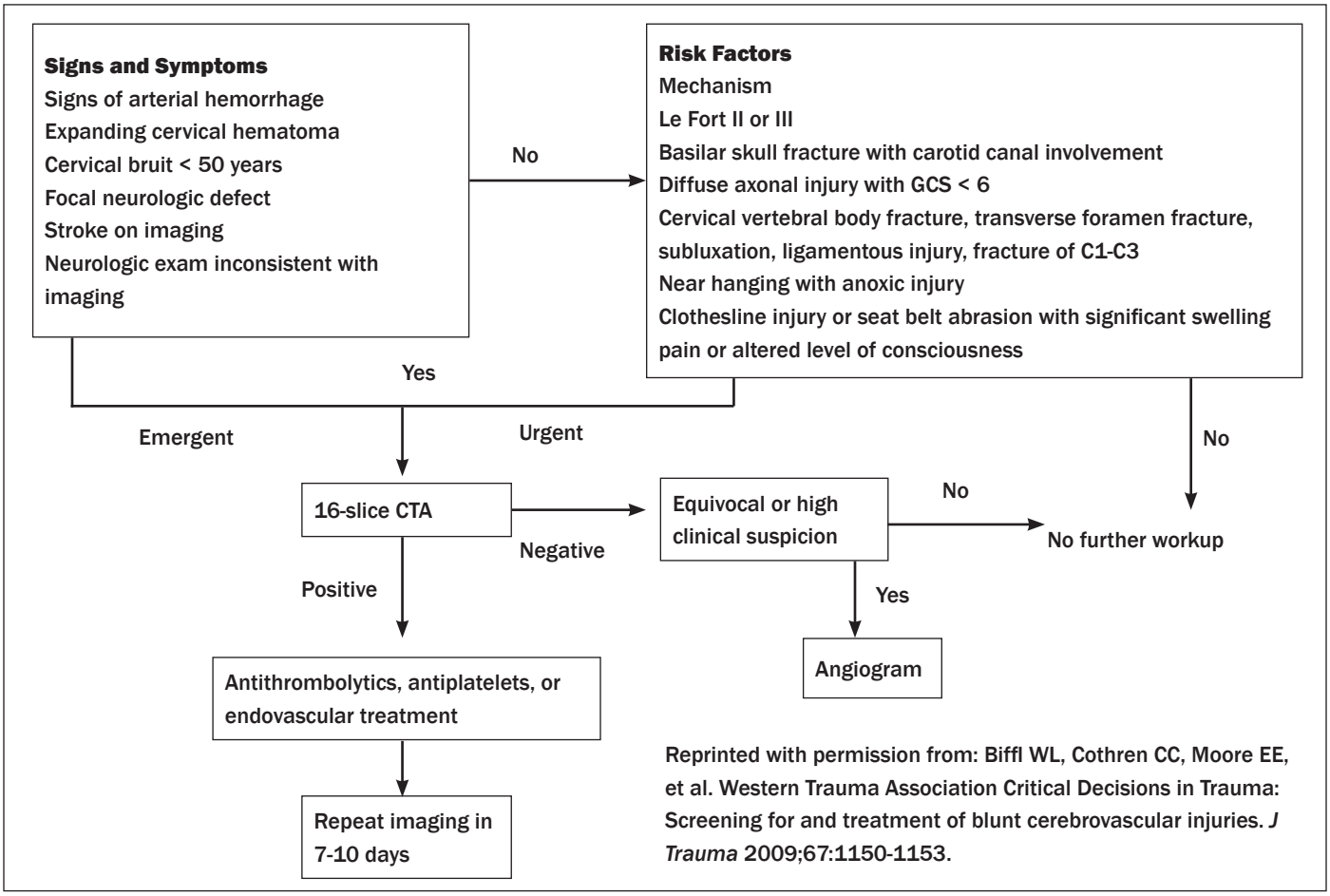
I: Irregularity of vessel wall, dissection or intramural hematoma with < 25% stenosis
II: Intramural thrombus, raised intimal flap, dissection or intramural hematoma with > 25% stenosis
III: Pseudoaneurysm
IV: Vessel occlusions
V: Vessel transection

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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