Clin Chest Med 24 (2003) 623–632

CLINICS IN CHEST MEDICINE

Critical issues in digestive diseases

Deborah D. Proctor, MD

Section of Digestive Diseases, Department of Internal Medicine, Yale University School of Medicine, 1080 LMP, New Haven, CT 06520, USA

Gastrointestinal (GI) diseases are often encountered in the intensive care unit (ICU) setting, either as the major cause that prompted admission to the ICU or as a comorbid complication of another primary disease process. The most common digestive disease that is seen in the ICU is acute GI bleeding; upper GI bleeding occurring five times more commonly than lower GI bleeding. This article discusses acute nonvariceal and variceal upper GI bleeding and lower GI bleeding. Prophylaxis against GI hemorrhage in the ICU setting is also discussed. Acute pancreatitis is another common cause that mandates ICU admission and is reviewed in this article. Lastly, acalculous cholecystitis is an example of a disease that is usually a comorbid complication in critically ill patients and is often a difficult diagnosis to make. Diagnosis and treatment of acalculous cholecystitis is discussed.

Acute gastrointestinal bleeding

Acute GI bleeding is bleeding that has occurred in the past 24 to 48 hours and implies a temporally circumscribed event. The quantity and rapidity of blood loss and the patient's cardiovascular system determine the degree of hemodynamic instability. Evaluation and treatment is aimed initially at the immediate assessment and stabilization of the patient's hemodynamic status, followed by identifying the source of bleeding, stopping any active bleeding, treating any underlying abnormalities, and preventing any recurrent bleeding.

History, physical examination, and laboratory evaluation

A history helps the clinician with the initial assessment of the site, severity, and cause of the bleeding. Significant GI blood loss is manifested by hematemesis, melena, or hematochezia. At the same time that the history is being obtained, the patient's heart rate and blood pressure, including orthostatic measurements, should be assessed. The physical examination also should include complete heart, lung, and abdominal examinations, as well as an examination of the skin and mucus membranes. Clues to underlying liver disease, portal hypertension, or underlying vascular diseases can be identified here. A rectal examination should be done and the stool should always be visually inspected.

With significant GI bleeding, laboratory tests are less important than the history and physical examination. A complete blood cell count, serum urea nitrogen, creatinine, prothrombin time, partial thromboplastin time, and liver tests should be obtained. The patient should be typed and crossed in anticipation of blood transfusions. The hematocrit should be interpreted in light of the onset of the bleeding episode, with the understanding that it requires up to 72 hours for equilibration with the intravascular space [1]. In an acute bleeding episode, the anemia will usually be a normocytic one.

Special note should be made of patients who have renal impairment or who are on dialysis. These patients have platelet abnormalities; therefore, any lesion in the GI tract will be more likely to bleed. Consideration should be done to doing a bleeding time or administering desmopressin acetate (DDAVP) [2].

E-mail address: deborah.proctor@yale.edu

0272-5231/03/$ – see front matter
doi:10.1016/S0272-5231(03)00103-5

Initial assessment and resuscitation

Postural changes in the patient's heart rate and blood pressure are the most important evaluation tools for assessing the severity of the bleeding and should be evaluated immediately upon presentation. The degree of resuscitation should be proportional to the severity of the bleeding and the hemodynamic instability of the patient. Two 16-gauge or larger intravenous (IV) catheters should be inserted immediately in patients who are unstable. The initial infusion of colloid solution should be done as rapidly as possible with the goal to re-establish normal vital signs. Oxygen should be administered and vital signs and urine output should be closely monitored. ICU monitoring is indicated in all patients who are hemodynamically unstable, patients who have stigmata on endoscopy of a high-risk of recurrent bleeding (eg, spurting artery, visible vessel, adherent clot [see later discussion]), or those who meet any BLEED (ongoing bleeding, low systolic blood pressure, elevated prothrombin time, erratic mental status, unstable comorbid disease) criteria [3]. Blood transfusions with packed red blood cells should be done in patients who are hemodynamically unstable with the goal of reaching and maintaining a hematocrit of 30% in the elderly or patients who have heart disease, 20% to 25% in younger patients, and 25% to 28% in patients who have portal hypertension. Patients who are on anticoagulants should have their coagulopathy reversed as much as possible.

Identification of the source of bleeding and stopping any active bleeding

After a complete history and physical examination has been done, the next step is to insert a nasogastric tube and perform a lavage. This can aid in differentiating between an upper and a lower GI bleed. The color and rapidity of clearing with lavage can predict outcomes [4] and lavage can clear the field in anticipation for an esophagogastroduodenoscopy (EGD).

After initial hemodynamic stabilization of the patient, the next step in a presumptive upper gastrointestinal bleed is to perform an EGD. At the time of the EGD, different modalities to stop active bleeding and prevent recurrent bleeding can be done, depending on the source of the bleeding. These include injection therapy, bipolar or multi-polar electrocoagulation, heater probe, laser, rubber band ligation, argon plasma coagulation, and metal clips [5]. If the EGD is unsuccessful in stopping the bleeding, then angiography or surgery might be indicated, depending on the bleeding source.

A subgroup of patients are those who have presumptive or certain myocardial ischemia or infarction, either secondary to their GI bleeding or coincidental with it. These patients can safely undergo an EGD with appropriate monitoring in an ICU setting [6].

In a presumptive lower GI bleed, after initial stabilization of the patient, most endoscopists would perform an urgent colonoscopy after a colonic purge. Often, an EGD is done first to exclude an upper GI source. As with an upper GI source, if the cause of the bleeding is found, injection or cautery can be done to stop any active bleeding and to prevent recurrent bleeding. Successful therapeutic colonoscopy is done much less frequently than an upper endoscopy, however.

Acute upper gastrointestinal bleeding

Bleeding from the upper GI tract is five times more common than from the lower GI tract [5]. The incidence is estimated to be 50 to 100 per 100,000 patients per year with 100 per 100,000 hospital admissions. Thirty percent of patients who have an upper GI bleed are older than 65 years [5]. The prognosis for upper GI bleeding depends upon the cause of the bleeding (Box 1) and any underlying comorbid conditions. Eighty percent of upper GI bleeding episodes are self-limited [7]. Recurrent bleeding occurs within 48 to 72 hours and the 20% of patients who have recurrent bleeding have a poorer prognosis. Older age, comorbid conditions, large ulcers, and the onset of bleeding during hospitalization are all associated with a poorer prognosis. Variceal hemorrhage carries a 30% in-hospital mortality and 60% 1-year mortality. Upper GI bleeding can be divided into nonvariceal and variceal bleeding.

Acute nonvariceal bleeding

Upper GI bleeding is most commonly due to mucosal erosive disease (ie, gastric or duodenal ulcers). Significant GI bleeding usually occurs when the ulcer base erodes into an arteriole that is visible on EGD as active spurting, a visible vessel, or a blood clot. Other causes include esophageal ulcers, Mallory-Weiss tears, gastric or duodenal vascular ectasias, malignancies, Dieulafoy's lesions, aorto-enteric fistula, hemobilia or hemosuccus pancreaticus (bleeding from peri-pancreatic blood vessels into a pancreatic duct visualized as blood coming from the pancreatic duct on endoscopy).

After the initial assessment and resuscitation, a nasogastric lavage should be done and the stomach cleared of as much blood as possible. If the bleeding is significant or if the patient has an altered mental

Box 1. Causes of acute gastrointestinal bleeding

Upper GI bleeding

- Ulcers: duodenal, gastric, esophageal
- Varices: esophageal, gastric, duodenal
- Mallory-Weiss tear
- Dieulafoy's lesions
- Arteriovenous malformations
- Portal hypertensive gastropathy
- Gastric antral vascular ectasias (watermelon stomach)
- Erosions
- Aorto-enteric fistula
- Crohn's disease
- Malignancy
- Hemobilia
- Pancreatic source
- Foreign body ingestion or bezoar
- Caustic ingestion
- No site found

Lower GI bleeding

- Diverticula
- Arteriovenous malformations
- Neoplasia
- Postpolypectomy bleeding
- Ulcers
- Colitis: inflammatory or infectious
- Solitary ulcers
- Dieulafoy's lesions
- Intussusception
- Varices
- Anal fissures
- Hemorrhoids
- Ischemia
- Radiation proctitis/enteritis
- Medications, including nonsteroidal anti-inflammatory drugs (NSAIDs), aspirin, and vasospastic drugs
- Meckel's diverticulum
- Endometriosis
- Upper GI bleeding
- Small bowel source
- No site found

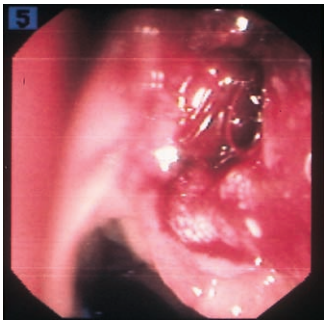

Fig. 1. Endoscopic photograph of a duodenal bulb ulcer with a spurting artery.

status, the patient should be intubated for airway protection before any EGD attempts.

The initial diagnostic procedure should be an EGD. At the time of the EGD, the cause of the upper GI bleeding can be visualized and treated. There is consensus that the most effective method to control active ulcer bleeding is with endoscopic therapy. Meta-analysis showed that endoscopic therapy prevents rebleeding (odds ratio [OR], 0.38; 95% confidence interval [CI], 0.32–0.45), reduces the need for surgery (OR, 0.36; 95% CI, 0.28–0.45), and improves mortality (OR, 0.55; 95% CI, 0.40–0.76) [8]. In addition, early EGD can aid in triage, including early discharge if stigmata indicate a low probability of rebleeding [9]. Endoscopic criteria for an increased risk of rebleeding include: spurting artery (85%) (Fig. 1), visible vessel (50%), adherent clot (30%–40%), and red or black spot (10%–15%) (Fig. 2) [10]. A clean-based ulcer has a less than 5% risk of rebleeding and does not need to be endoscopically treated [10]. Endoscopic choices depend on local expertise and availability and include injection, cautery, coagulation, and metal clips. Most studies showed that coagulation or cautery, either alone or in combination with injection therapy, will

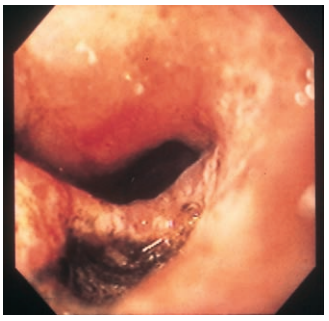

Fig. 2. Endoscopic photograph of a gastric antral ulcer with a black spot.

decrease the risk of rebleeding to less than 10% (see references [8,10,11]). The most recent study on endoscopic therapy showed that with a nonbleeding, adherent clot, manipulation to remove it and treat the underlying vessel with combination injection and coagulation decreased the risk of rebleeding from 35% to 0% [11].

The author recommends two separate EGD attempts at stopping the bleeding from an upper GI bleed before moving onto other modalities. If endoscopic therapy is unsuccessful, either on the initial EGD or subsequent EGDs, then the next modality should be angiography with embolization [12]. In addition, empiric embolization can be done based on endoscopic findings, even if no abnormality is seen on the angiogram. For example, if bleeding is seen in the duodenal bulb, embolization of the right gastroepiploic artery by way of the gastroduodenal artery can be done with coils [12]. If angiography by interventional radiologists is not locally available, surgical therapy with oversewing the bleeding area or resection is indicated, depending upon the location of the bleeding site [5].

In conjunction with direct endoscopic therapy to stop the bleeding, medication to heal the mucosal erosions or ulcers, and, therefore, to prevent recurrent disease and hemorrhage, should be instituted. Proton pump inhibitors significantly decreased recurrent bleeding and the need for surgery in patients who had bleeding ulcers, especially when combined with endoscopic therapy, and should be administered intravenously upon presentation [13]. Numerous trials of H2-receptor antagonists in patients who had bleeding ulcers were not beneficial, presumably because they do not provide optimal acid suppression [14]. If *Helicobacter pylori* is present, this bacteria should be treated with appropriate antibiotics to prevent recurrent ulcer disease [15]. Patients who are on aspirin or NSAIDs should have their medication discontinued. If it is absolutely imperative to continue one of these medications, then a proton pump inhibitor should also be used. In patients who required continuous treatment with NSAIDs, omeprazole, 20 mg/day, by mouth, healed ulcers or erosions in 80% of patients compared with 63% of patients who were given ranitidine, 150 mg orally, twice a day ($P < 0.001$) over an 8-week time period [16].

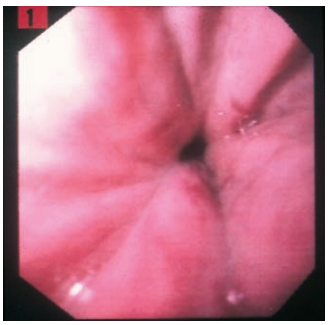

Fig. 3. Endoscopic photograph of esophageal varices with stigmata of recent bleeding.

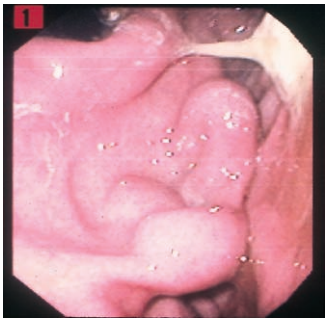

Fig. 4. Endoscopic photograph of varices in the gastric fundus.

Acute variceal bleeding

Portal hypertension can directly cause bleeding from esophageal, gastric, or duodenal varices or portal hypertensive gastropathy. Varices rarely bleed when the hepatic venous pressure gradient is less than 12 mm Hg [17]. Depending upon the population surveyed, the incidence of upper GI bleeding that is due to varices ranges from 10% (large national surveys) to 30% (inner city populations) [5].

Endoscopic therapy is the treatment of choice in the management of esophageal variceal hemorrhage (Fig. 3) and was effective in stopping active bleeding and preventing rebleeding [18]. Most endoscopists preferentially use endoscopic variceal ligation (band ligation) over endoscopic sclerotherapy because of the ease of therapy and fewer side effects [19]. Over the past few years, technology has improved band ligation with the multiband ligators and a wider field of vision than with the original banding devices. Endoscopic therapy stops bleeding in 80% to 90% of esophageal variceal hemorrhages [19]. Gastric varices are more difficult to treat endoscopically (Fig. 4). If an attempt is made, injection with a sclerosing agent will be more beneficial than band ligation, especially with large gastric varices.

Pharmacologic therapy is best used in conjunction with endoscopic therapy. Vasopressin is a potent vasoconstrictor, but side effects are seen in approximately 25% of patients; the risk of myocardial infarction is the greatest concern. Because of the high risk

of side effects and to potentially further lower portal pressures, nitroglycerin was combined with vasopressin. Three trials showed a trend toward improved control of hemorrhage, as well as fewer side effects in combination vasopressin/nitrate therapy versus vasopressin alone; therefore, vasopressin should never be used as monotherapy [20]. Octreotide, the only somatostatin analog that is available in the United States, showed varied results in clinical trials. One recent meta-analysis showed that octreotide had no benefit when used alone [20]. A second meta-analysis showed that octreotide improved the control of variceal bleeding, but did not demonstrate an improvement in mortality [21]. Somatostatin [22] and terlipressin [20] also were shown to be effective in controlling acute variceal hemorrhage, but neither is currently available in the United States. Because the risk of rebleeding is highest in the first few days, the use of vasopressin/nitrates or octreotide, in combination with endoscopic therapy, currently offers the best option for acute variceal hemorrhage. Octreotide has fewer side effects and should be used if available; if it is not available, combination vasopressin/nitrates should be used.

Nonselective β-blockers, nadolol or propranolol, with or without isosorbide mononitrate, decreased the rate of rebleeding from varices (secondary prophylaxis). They have the added advantage of reducing the risk of bleeding from portal hypertensive gastropathy. β-blockers with nitrates should be started when the patient is hemodynamically stable and preferably before the fifth hospital day [23].

If rebleeding occurs or persists after two endoscopic sessions, then transjugular intrahepatic portosystemic shunting (TIPS) or shunt surgery is indicated. TIPS is usually preferred over shunt surgery because of the preservation of the mesenteric and portal vasculature if the patient requires a liver transplant. TIPS is done by way of a transjugular approach, usually by interventional radiologists, and directly connects a branch of the portal vein with one of the hepatic veins. The success rate is greater than 90% with portal vein/hepatic vein pressure gradients being reduced to less than 12 to 15 mm Hg [24]. Acute variceal bleeding is controlled in almost all patients with a successful TIPS. If rebleeding occurs, the TIPS should be evaluated for patency; if patent, a repeat endoscopy should be done to evaluate for an alternative source of bleeding. Direct procedural complications from a TIPS are seen less than 10% of the time and include bleeding, dye-induced renal failure, hemolysis, stent migration, and puncture of the gallbladder or other organs adjacent to the liver. Because TIPS is a portosystemic shunt, complications similar to those of traditional shunt surgery are seen and include encephalopathy (20%–30%) and accelerated liver failure that is due to loss of hepatic perfusion (25%) with 5% of patients requiring urgent liver transplantation [25].

Balloon tamponade is successful in temporarily controlling variceal hemorrhage, especially gastric variceal bleeding. The patient must be intubated for airway protection before insertion of the balloon. A quadruple lumen nasogastric tube with a gastric balloon, an esophageal balloon, and two suction ports (one for esophageal secretions above the balloon and one for gastric secretions) should be used and is inserted by way of the nose and then placed into the stomach. The gastric balloon is partially inflated, a chest radiograph is obtained to verify position, and the gastric balloon is fully inflated with air with or without also inflating the esophageal balloon. The tube is secured in place. The two balloons directly compress the varices at their respective sites and control variceal bleeding. Because of the significant risk of esophageal perforation from direct pressure on the distal esophagus, the balloon should be inflated for no more than 24 hours. There is also a risk of aspiration pneumonia and asphyxiation, which is decreased by intubation, continuous monitoring, and suction of proximal esophageal secretions. Because there is a 75% rebleeding rate after balloon deflation, this is considered only a temporizing measure and the balloon should only be inserted in patients in whom more definitive therapy, such as TIPS, is indicated.

In addition to measures to control the active variceal hemorrhage, all cirrhotic patients who have variceal hemorrhage, with or without ascites, should receive short-term (7 days) antibiotic prophylaxis. The use of norfloxacin orally or ciprofloxacin intravenously decreased the risk of developing spontaneous bacterial peritonitis and other serious bacterial infections [26]. Blood products should be used judiciously and include packed red cell transfusions to maintain the hematocrit at 25% to 28% or fresh frozen plasma or platelets to correct any coagulation abnormalities. Portal pressures may increase with overly vigorous resuscitation and encourage continued variceal bleeding; therefore, the aim of resuscitation is to return to a central venous pressure and hematocrit that is adequate for tissue perfusion without overshooting the mark.

Acute lower gastrointestinal bleeding

Lower GI bleeding is less common than upper GI bleeding and accounts for 24% of all bleeding events [27]. It is bleeding that usually originates in the colon. As demonstrated in the landmark article in 1988, 74%

of severe hematochezia originated from the colon, whereas 11% was from an upper GI source, 9% from the small intestine, and 6% had no site identified [28]. The most common causes of severe hematochezia are diverticular bleeding and angiodysplasias, which together account for approximately 50% of significant lower GI bleeding (see Box 1).

Whereas endoscopic evaluation and treatment is universally preferred as an initial step for upper GI bleeding, the use of colonoscopy as the first diagnostic test in a lower GI bleed is more controversial. Many endoscopists perform an urgent colonoscopy after colonic purge. Colonoscopy in this setting is safe and will either yield or exclude diagnoses in many cases. Because of the time that is needed to cleanse the colon, an "urgent" colonoscopy includes those that are performed up to 24 hours after the acute event. Because of the time factor and the fact that a diverticular bleed will cease spontaneously in up to 80% of cases, endoscopic therapy is done much less frequently during a colonoscopy than during an EGD. Similar modalities as for EGD are available to treat colonoscopic abnormalities and include injection, cautery, and coagulation. A recent study demonstrated that in patients who had severe diverticular hemorrhage, the use of epinephrine injection or bipolar coagulation decreased the risk of rebleeding from 50% to 0%; this is similar to results that were seen in actively bleeding ulcers in the upper GI tract [29].

If colonoscopy is unsuccessful in identifying the source of the lower GI bleed, then radionuclide scanning or mesenteric angiography may detect the bleeding site. Radionuclide scanning with 99mTc-labeled red blood cells can detect bleeding rates as low as 0.1 to 0.5 mL/min (Fig. 5). In addition, if the scan is initially negative, the patient can be rescanned up to 24 hours later. If the stool at the time of the radionuclide scan indicates rapid bleeding, the scan is likely to be positive. In one study, stool color was a better predictor than hemodynamic instability or number of blood transfusions required [30]. The most useful aspect of radionuclide scanning may be as a screening test for visceral angiography. In one study, a positive tagged red blood cell scan increased the yield of angiography from 22% to 53% [31]. In a large GI bleed, angiography can detect the bleeding site with rates as low as 0.5 mL/min (Fig. 6). In addition to identifying a bleeding site, selective angiography can be therapeutic by embolizing particulate matter directly into the bleeding artery, or if not possible, with vasopressin infusion.

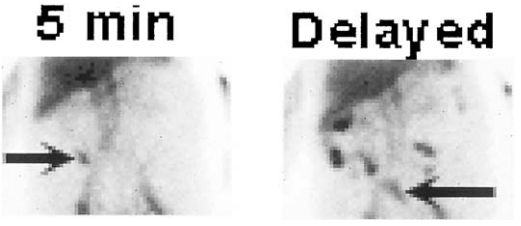

Fig. 5. Technetium-labeled red blood cell scan demonstrating active bleeding in the right lower quadrant.

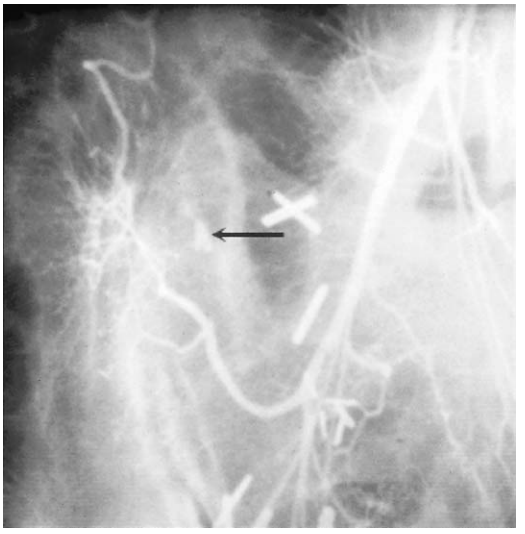

Fig. 6. Mesenteric angiogram demonstrating active bleeding in the right lower quadrant from the ileocecal artery (same patient as in Fig. 5).

Surgical therapy is necessary in 10% to 20% of patients who have significant lower GI bleeding and is indicated for continuous or recurrent bleeding, usually in patients who require more than six units of blood over 24 hours or a total of 10 units. Options include limited resection of a known bleeding source or left or right hemicolectomy. Blind subtotal colectomy for severe bleeding without a clear source was associated with a high morbidity and mortality and is used only as a last resort.

Prophylaxis against gastrointestinal hemorrhage

GI hemorrhage in critically ill patients in the ICU is caused by decreased mucosal blood flow which predisposes the gastroduodenal mucosa to develop erosions or ulcerations. More than 75% of patients in the ICU will have gastroduodenal lesions by endoscopy. The frequency of clinically important GI bleeding has declined over the past 20 years independent of ulcer prophylaxis and is probably the result of improved

medical management with earlier restitution of mucosal blood flow. The patients who are at highest risk for developing clinically significant GI bleeding are intubated patients; those who have multi-organ failure, coagulopathy, sepsis, or extensive burns; or those who have experienced head trauma or neurosurgery.

A meta-analysis of stress ulcer prophylaxis demonstrated a decreased incidence of clinically important GI bleeding with H2-receptor antagonists with a trend favoring these agents over antacids. H2-receptor antagonists and sucralfate did not obviously differ in preventing important GI bleeding. In addition, sucralfate had a lower incidence of nosocomial pneumonia and a decreased mortality rate [32]. Sucralfate does not increase the gastric pH as do H2-receptor antagonists and antacids, and, therefore, presumably, does not predispose the patient to developing a nosocomial pneumonia. Limited studies in using proton pump inhibitors for stress ulcer prophylaxis suggest a beneficial effect for these medications [33], but they predispose the patient to developing nosocomial pneumonias.

Current recommendations for prophylaxis include the following: if IV administration is necessary, H2-receptor blockers should be used in patients in the ICU who are at the highest risk for developing clinically significant GI bleeding and the gastric pH should be titrated into the 3-4 range [34]; if enteral administration is possible, sucralfate should be used, although it does have significant interactions with other medications [32,34].

Box 2. Causes of acute pancreatitis

- Gallstones, including biliary sludge and microlithiasis
- Alcohol
- Hypertriglyceridemia
- Hypercalcemia
- Medications
- Trauma
- Post-endoscopic retrograde cholangiopancreatography (ERCP)
- Postoperative
- Infectious
- Malignancy
- Cystic fibrosis
- Hereditary pancreatitis
- Pancreas divisum
- Vascular disease
- Autoimmune diseases
- Trinidad scorpion bite and other toxins
- Idiopathic

Acute pancreatitis

Acute pancreatitis occurs in 4 to 24 patients per 100,000 in the United States [35]. Although gallstones and alcohol are the two leading causes of acute pancreatitis, many conditions predispose to its development (Box 2). The clinical manifestations are severe, steady, upper abdominal or epigastric pain accompanied by an increase in the serum amylase or lipase.

Serum amylase and lipase are the two most commonly used blood tests to confirm the diagnosis of acute pancreatitis. Serum amylase increases in 6 to 12 hours and remains elevated for 3 to 5 days in uncomplicated acute pancreatitis. Serum lipase is elevated on the initial day and remains elevated slightly longer than amylase. If the cut-off of three times the upper limit of normal is used, the specificity of both enzymes is high [36].

CT scanning is the most important and helpful diagnostic test for acute pancreatitis and its potential complications. Spiral CT scanning should be done with oral contrast followed by IV contrast during the scan to identify any unenhancing areas that would indicate pancreatic necrosis [37]. An ultrasound can be useful to evaluate for gallstones or biliary ductal dilation. MRI with gadolinium provides the same information about the pancreas as CT scanning, but provides better information on the biliary tree. In the future, with wider availability, MRI with gadolinium may be the imaging test of choice.

A variety of methods has been use in an attempt to predict attack severity, including clinical evaluation, scoring systems, laboratory tests, and CT scanning. Clinical evaluation of a severe attack includes respiratory decompensation, peritonitis, and shock. Scoring systems that are common used include Ranson's criteria and Acute Physiology and Chronic Health Evaluation (APACHE)-II scores. Ranson's criteria is best used to exclude severe disease. APACHE-II is the most commonly used scoring system to predict severity and can be continuously used. Several serum (eg, interleukin-6, C-reactive protein, polymorphonuclear leukocyte elastase) and urinary (eg, trypsinogen activation peptide), markers have recently been studied and seem to be useful predictors of severity [38]. Before wide-spread clinical use occurs, they need to be more available or generally accepted. Lastly, unenhanced or IV contrast–enhanced CT scanning can be used to assess edematous or necrotizing pancreatitis, and, thus, the severity of the attack [37]. In summary, if

there is clinical evidence or the APACHE-II score indicates severe pancreatitis, then the patient should be admitted to the ICU as well as undergo a CT scan to determine the presence or absence of necrotizing pancreatitis [38].

Treatment of severe acute pancreatitis includes ICU monitoring and support of all major organ systems. In addition to intensive monitoring, because bacterial infections develop in 30% of patients, particularly those who have pancreatic necrosis, and significantly contribute to multi-organ failure and death, two modalities have been demonstrated to decrease this complication and should be used. The use of systemic, prophylactic antibiotics that have good pancreatic tissue penetration was shown, including a meta-analysis of eight controlled trials [39], to improve morbidity and mortality in patients who had severe, acute pancreatitis, mainly by decreasing bacterial infections [40]. Because the use of prophylactic antibiotics may increase the risk of fungal infections, the use of prophylactic antifungal therapy also has been advised, especially if antibiotics are used for longer than 1 week [41]. There is increasing evidence that the use of enteral nutrition by way of a nasojejunal tube early in the course may reduce complications by maintaining the integrity of the intestinal barrier [42]. The author advocates the use of enteral feeds, starting at 10 mL/hour and advancing to the nutritional goal, into the jejunum as soon as the ileus has resolved.

Early ERCP in the course of severe acute biliary pancreatitis was of benefit by preventing biliary sepsis [43,44]. Early ERCP should be considered in all patients who have acute pancreatitis secondary to gallstone disease and should be performed in patients who have biliary sepsis or a serum bilirubin level that is greater than 5 mg/dL. Cholecystectomy should be performed before discharge in patients who have gallstone pancreatitis.

Local complications of acute pancreatitis for which patients should be observed include pseudocysts, necrosis, ascites, hemorrhage, and abscess formation. The overall mortality for hospitalized patients is 5% to 10% and increases with severity, multi-organ failure, and local complications [38].

Acalculous cholecystitis

Acalculous cholecystitis should be suspected in any patient who is critically ill and has evidence of sepsis or evidence of hemodynamic instability with or without abnormal liver tests and with or without right upper quadrant pain. This is a difficult diagnosis to make because the patient is often intubated, ventilated, and sedated and is unable to communicate appropriate symptoms. Therefore, a high index of suspicion is needed for all patients in the ICU, especially in the setting of unexplained fever or leukocytosis [45]. The cause of acalculous cholecystitis is decreased blood flow with impaired microcirculation as a result of systemic hypotension from sepsis or other hemodynamic instability [46]. Because the patient is unable to communicate, early imaging with ultrasound is indicated and will yield the diagnosis in 67% to 92% of patients [47]. The bedside availability of ultrasound is its major advantage. Ultrasound shows gallbladder wall thickening, fluid around the gallbladder or frank perforation. Incidental gallstones may be present, but unlike calculous cholecystitis, where the cause is gallstone migration into the cystic duct and ischemia on an obstruction basis, the ischemia that causes acalculous cholecystitis is from global hypotension and poor microcirculation perfusion. CT scanning is superior to ultrasound imaging and excludes other intra-abdominal pathology, but it requires moving the patient to the scanner. CT imaging will show a thickened gallbladder wall, pericholecystic fluid, subserosal edema, intramural gas, or sloughed mucosa [47]. Treatment is with systemic antibiotics that cover enterococcus and gram negative aerobic and anaerobic enteric bacteria. In addition, percutaneous drainage is required and can be done under ultrasound guidance by interventional radiology. After the patient recovers from the underlying systemic illness, cholecystectomy can be semi-electively performed.

Summary

This article discussed the diagnosis and management of acute GI bleeding, prophylaxis against GI bleeding, acute pancreatitis, and acalculous cholecystitis. These diseases are commonly encountered in the ICU setting. Acute GI bleeding is usually obvious and with GI and with available interventional radiologic techniques, patients rarely need surgery. Conversely, acalculous cholecystitis is difficult to diagnosis; therefore, a high degree of suspicion needs to be exercised with all critically ill patients.

References

[1] Ebert RA, Stead EA, Gibson JG. Response of normal subjects to acute blood loss. Arch Intern Med 1940;68: 578–80.

[2] Mannucci PM, Remuzzi G, Pusineri F, et al. Deamino-8-D-arginine vasopressin shortens the bleeding time in uremia. N Engl J Med 1983;308:8–12.

[3] Kollef MH, O'Brien JD, Zuckerman GR, et al. BLEED: A classification tool to predict outcomes in patients with acute upper and lower gastrointestinal hemorrhage. Crit Care Med 1997;25:1125–32.
[4] Peterson WL. Clinical risk factors. Gastrointest Endosc 1990;36(5):S14–5.
[5] Rockey DC. Gastrointestinal bleeding. In: Feldman M, Friedman LS, Sleisenger MH, editors. Sleisenger & Fordtran's gastrointestinal and liver disease. 7th edition. Philadelphia: WB Saunders; 2002. p. 211–40.
[6] Cappell MS. The safety and clinical utility of esophagogastroduodenoscopy for acute gastrointestinal bleeding after myocardial infarction: a six-year study of 42 endoscopies in 34 consecutive patients at two university teaching hospitals. Am J Gastroenterol 1993;88(3): 344–9.
[7] Fleischer D. Etiology and prevalence of severe persistent upper gastrointestinal bleeding. Gastroenterology 1983;84:538–43.
[8] Cook DJ, Guyatt GH, Salena BJ, et al. Endoscopic therapy for acute nonvariceal upper gastrointestinal hemorrhage: a meta-analysis. Gastroenterology 1992; 102(1):139–48.
[9] Rockall TA, Logan RFA, Devlin HB, et al. Selection of patients for early discharge or outpatient care after acute upper gastrointestinal haemorrhage. Lancet 1996;347: 1138–40.
[10] Johnston JH. Endoscopic risk factors for bleeding peptic ulcer. Gastrointest Endosc 1990;36(5):S16–20.
[11] Jensen DM, Kovacs TOG, Jutabha F, et al. Randomized trial of medical or endoscopic therapy to prevent recurrent ulcer hemorrhage in patients with adherent clots. Gastroenterology 2002;123:407–13.
[12] Lefkovitz A, Cappell MS, Kaplan M, et al. Radiology in the diagnosis and therapy of gastrointestinal bleeding. Gastroenterol Clin N Am 2000;29:489–510.
[13] Khuroo MS, Yattoo GN, Javid G, et al. A comparison of omeprazole and placebo for bleeding peptic ulcer. N Engl J Med 1997;336(15):1054–8.
[14] Collins R, Langman M. Treatment with histamine H2-antagonists in acute upper gastrointestinal hemorrhage. Implications of randomized trials. N Engl J Med 1985; 313:660–6.
[15] Graham DY, Hepps KS, Ramirez FC, et al. Treatment of *Helicobacter pylori* reduces the rate of rebleeding in peptic ulcer disease. Scand J Gastroenterol 1993;28: 939–42.
[16] Yeomans ND, Tulassay Z, Juhasz L, et al. A comparison of omeprazole with ranitidine for ulcers associated with nonsteroidal anti-inflammatory drugs. N Engl J Med 1998;338:719–26.
[17] Nevens F, Bustami R, Scheys I, et al. Variceal pressure is a factor predicting the risk of a first variceal bleeding: a prospective cohort study in cirrhotic patients. Hepatology 1998;27:15–9.
[18] De Franchis R, Primignani M. Endoscopic treatments for portal hypertension. Semin Liver Dis 1999;19:439–55.
[19] Laine L, Cook D. Endoscopic ligation compared with sclerotherapy for treatment of esophageal variceal bleeding. A meta-anaylsis. Ann Intern Med 1995;123: 280–6.
[20] D'Amico G, Pagliaro L, Bosch J. Pharmacological treatment of portal hypertension: an evidence-based approach. Semin Liver Dis 1999;19(4):475–505.
[21] Corley DA, Cello JP, Adkisson W, et al. Octreotide for acute esophageal variceal bleeding: a meta-analysis. Gastroenterology 2001;120(4):946–54.
[22] Imperiale TF, Teran JC, McCullough AJ. A meta-analysis of somatostatin versus vasopressin in the management of acute esophageal variceal hemorrhage. Gastroenterology 1995;109:1289–94.
[23] Villanueva C, Balanzo J, Novella MT, et al. Nadolol plus isosorbide mononitrate compared with sclerotherapy for the prevention of variceal rebleeding. N Engl J Med 1996;334:1624–9.
[24] Rossle M, Haag K, Ochs A, et al. The transjugular intrahepatic portosystemic stent-shunt procedure for variceal bleeding. N Engl J Med 1994;330:165–71.
[25] Rouillard SS, Bass NM, Roberts JP, et al. Severe hyperbilirubinemia after creation of transjugular intrahepatic portosystemic shunts: Natural history and predictors of outcome. Ann Intern Med 1998;128:374–7.
[26] Rimola A, Garcia-Tsao G, Navasa M, et al. Diagnosis, treatment and prophylaxis of spontaneous bacterial peritonitis: a consensus document. J Hepatol 2000;32(1): 142–53.
[27] Peura DA, Lanza FL, Gostout CJ, et al. The American College of Gastroenterology Bleeding Registry: preliminary findings. Am J Gastroenterol 1997;92:924–8.
[28] Jensen DM, Machicado GA. Diagnosis and treatment of severe hematochezia. Gastroenterology 1988;95: 1569–74.
[29] Jensen DM, Machicado GA, Jutabha R, et al. Urgent colonoscopy for the diagnosis and treatment of severe diverticular hemorrhage. N Engl J Med 2000;342(2): 78–82.
[30] Zuckerman GR, Prakash C. Acute lower intestinal bleeding. Part II: etiology, therapy, and outcomes. Gastrointest Endosc 1999;49:228–38.
[31] Gunderman R, Leef JA, Lipton MJ, et al. Diagnostic imaging and the outcome of acute lower gastrointestinal bleeding. Acad Radiol 1998;5:S303–5.
[32] Cook DJ, Reeve BK, Guyatt GH, et al. Stress ulcer prophylaxis in critically ill patients: resolving discordant meta-analyses. JAMA 1996;275(4):308–14.
[33] Levy MJ, Seelig CB, Robinson NJ, et al. Comparison of omeprazole and ranitidine for stress ulcer prophylaxis. Dig Dis Sci 1997;42:1255–9.
[34] Tryba M, Cook D. Current guidelines on stress ulcer prophylaxis. Drugs 1997;54:581–96.
[35] Go VLW, Everhart JE. Pancreatitis. In: Everhart JE, editor. Digestive diseases in the United States: epidemiology and impact. Washington DC: NIH publication no. 94–1447; 1994. p. 693.
[36] Gumaste VV, Roditis N, Mehta D, et al. Serum lipase levels in nonpancreatic abdominal pain versus acute pancreatitis. Am J Gastroenterol 1993;88:2051–5.
[37] Balthazar EJ, Robinson DL, Megibow AJ, et al. Acute

pancreatitis: value of CT in establishing prognosis. Radiology 1990;174:331–6.
[38] DiMagno EP, Chari S. Acute pancreatitis. In: Feldman, M, Friedman LS, Sleisenger MH, editors. Sleisenger & Fordtran's gastrointestinal and liver disease. 7th edition. Philadelphia: WB Saunders; 2002. p. 913–41.
[39] Golub R, Siddiqi F, Pohl D. Role of antibiotics in acute pancreatitis: a meta-analysis. J Gastrointest Surg 1998; 2:496–503.
[40] Luiten EJ, Hop WC, Lange JF, et al. Controlled clinical trial of selective decontamination for the treatment of severe acute pancreatitis. Ann Surg 1995;222:57–65.
[41] Grewe M, Tsiotos GG, Luque de-Leon E, et al. Fungal infection in acute necrotizing pancreatitis. J Am Coll Surg 1999;188:408–14.
[42] Kalfarentzos F, Kehagias J, Mead N, et al. Enteral nutrition is superior to parenteral nutrition in severe acute pancreatitis: results of a randomized prospective trial. Br J Surg 1997;84:1665–9.
[43] Fan S-T, Lai ECS, Mok FPT, et al. Early treatment of acute biliary pancreatitis by endoscopic papillotomy. N Engl J Med 1993;328:228–32.
[44] National Institutes of Health State-of-the-Science Conference Statement. Endoscopic retrograde cholangiopancreatography (ERCP) for diagnosis and therapy. Available at: http://consensus.nih.gov/ta/020/020sos_statement.htm. Accessed June 10, 2002.
[45] Johnson LB. The importance of early diagnosis of acute acalculous cholecystitis. Surg Gynecol Obstet 1987; 164:197–203.
[46] Hakala T, Nuutinen PJ, Ruokonen ET, et al. Microangiopathy in acute acalculous cholecystitis. Br J Surg 1997; 84:1249–52.
[47] Mirvis SE, Vainright JR, Nelson AW, et al. The diagnosis of acute acalculous cholecystitis: a comparison of sonography, scintigraphy, and CT. Am J Roentgenol 1986; 147:1171–5.