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In Shortly about Upper GI Bleeding

Siniša Franjić

Independent Researcher

*Corresponding author
Siniša Franjić, Indepenent Researcher

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Abstract

Upper gastrointestinal bleeding is bleeding in the upper gastrointestinal tract from the esophagus, stomach or duodenum. Blood may be noticed in vomiting or in altered form as black stools. Depending on the amount of blood loss, symptoms may include shock. Upper gastrointestinal bleeding can be caused by stomach ulcers, gastric erosion, esophageal varices, and less common causes such as stomach cancer. Initial assessment includes measurement of blood pressure and heart rate, as well as blood tests to determine hemoglobin.

Keywords: GI Bleeding, Diagnosis, Endoscopy

Introduction

Nonvariceal upper GI bleeding is a common reason for emergency department visits and admissions to the hospital [1]. It has been estimated that upper GI bleeding is responsible for over 300,000 hospitalizations per year in the United States. An additional 100,000–150,000 patients per year develop upper GI bleeding during hospitalizations for other reasons.

The source of upper GI bleeding is by definition proximal to the ligament of Treitz. The natural history of nonvariceal upper GI bleeding is that approximately 80% of patients will stop bleeding spontaneously and in this group, no further urgent intervention will be needed. However, if a patient rebleeds, there is a 10-fold increased mortality rate.

Upper gastrointestinal bleeding can occur from multiple sources [2]. Broadly, upper gastrointestinal bleeding can be divided into variceal and nonvariceal etiologies. The suspected source of bleeding will drive the initial resuscitation and management. Adequate resuscitation, management of the patients prior medications, initiating medications to affect gastric pH, initiating vasoactive drugs, or administering antibiotics all occur prior to endoscopy. Endoscopy is important in the diagnosis and management of upper gastrointestinal bleeding. Pharmacologic agents are useful during the endoscopy to help achieve hemostasis. Finally, once hemostasis is achieved, decisions must be made regarding reinitiation of previous mediations like antithrombotics and which medications can be used acutely and chronically to reduce the risk of rebleeding and mortality from upper gastrointestinal bleeding.

The investigation of gastrointestinal bleeding (GI) involves answering two major questions: (1) is the patient still bleeding? and (2) where is the bleeding site? Frank bleeding from the upper gastrointestinal tract, that is a site proximal to the duodenojejunal junction or ligament of Treitz, is usually obvious, presenting as a haematemesis or melaena stools [3]. Fresh blood in the stools usually indicates rectal or colonic disease. However, it is possible for a bleeding lesion in the upper gastrointestinal tract to present with the passage of red blood per rectum; similarly a bleeding lesion in the caecum or ascending colon may cause melaena stools. The factors determining the degree of alteration of the blood in the gut include the site of the bleeding, the amount of blood lost and the motility of the bowel.

Symptoms

The clinical presentation of bleeding should be characterized [1]. Hematemesis is overt bleeding with vomiting of fresh blood or clots. Melena refers to dark black and tarry-appearing stool, with a distinctive smell. The term “coffee grounds” describes gastric aspirates or vomitus that contains dark specks of old blood. Hematochezia is the passage of fresh blood or clots per rectum. Although bright red blood per rectum is usually indicative of a lower GI source, it may be seen in patients with brisk upper GI bleeding.

Concurrent with the initial evaluation of patients with suspected upper GI bleeding attention needs to be paid to resuscitation, with the goal of achieving hemodynamic stability. The evaluation must assess vital signs, the presence or absence of shock and hypovolemia, and medical comorbidities (malignancy, chronic obstructive pulmonary disease, coronary artery disease, etc). Patients with postural hypotension have a significant blood volume loss of at least 10% and those with shock have a blood volume loss of at least 20%, a predictor of poor outcome.

Incidence

Acute gastrointestinal bleeding is a common reason for presentation to emergency departments and continues to be associated with significant morbidity and mortality [4]. The site of bleeding can usually be predicted by history with the broad categories of bleeding from the upper or lower gastrointestinal tracts. These categories are important as they influence the evaluation and management of the patient. The incidence of upper gastrointestinal bleeding is approximately 40 to 150 cases per 100,000 per year in Western populations and is associated with significant medical costs. A common subclassification for upper gastrointestinal bleeding is division into nonvariceal and variceal bleeding as the latter often causes more severe bleeding, has distinctive therapy and is associated with a higher mortality.

In almost all surveys, the most common cause of upper gastrointestinal bleeding is peptic ulceration. This diagnosis applies in 30–40% of patients. The second most common cause is equally shared between hemorrhagic gastritis and esophageal varices. Although cirrhosis is the usual cause of varices, patients with cirrhosis can have alternative causes for bleeding such as peptic ulceration, gastroduodenal erosions, portal gastropathy and Mallory-Weiss tears. An additional issue, particularly in elderly patients, is prolongation of bleeding by a variety of anti-platelet drugs, warfarin and direct oral anticoagulants.

Risk

Factors that predict a good or a poor prognosis in patients with nonvariceal upper GI bleeding can be used to appropriately triage and manage patients [1]. The role of nasogastric tube aspiration is controversial and currently is not recommended as part of the routine initial assessment. Although, the nasogastric tube aspirate can provide useful information depending on its contents, results can be falsely negative; false negatives may occur in patients with bleeding from duodenal ulcers due to spasm of the pylorus and can occur in other conditions, including gastric ulcers and, rarely, esophageal varices (if the tube is positioned in a nondependent area of the stomach). However, patients often find it very uncomfortable and recent data suggest that the performance of nasogastric tube lavage does not alter patient outcomes.

UGIH

Upper gastrointestinal haemorrhage (UGIH) is a frequent cause of acute hospital admission, and in the United Kingdom, it accounts for 70,000 admissions per year with the majority of cases being non-variceal [5]. Gastroduodenal (peptic) ulcers are the most common cause and account for well over 50% of admissions. This is despite the ready availability of proton pump inhibitors and the recognition of the role of Helicobacter pylori and nonsteroidal anti-inflammatory drugs (NSAIDs) in their genesis.

Although there have been significant advances in endoscopy and interventional radiology (IR), UGIH remains a significant cause of morbidity and mortality. In fact, the 30-day mortality seems unchanged at a level of around 11% as the patients tend to be older with more serious co-morbidities. Management has evolved over the last few decades with fewer cases requiring surgery, but therapy will depend on the place of treatment as smaller hospitals may not have the equipment or expertise to use the most modern techniques. In addition, in some health systems, gastroenterologists manage the patient, and surgical involvement is limited to severe cases requiring operation that have failed endoscopy and IR.

UGIH is defined as bleeding proximal to the ligament of Treitz and may present with melaena or haematemesis. Milder forms may present with anaemia and non-specific symptoms. The majority of cases are due to peptic ulceration, but the initial management and general response to non-surgical treatments are similar irrespective of the aetiology. This does not include variceal haemorrhage due to portal hypertension as this requires a different approach from the outset although patients with varices can also bleed from other lesions.

Simple luminal interventions are commonly performed for GI diseases [6]. Nasogastric tube suction decompresses the upper gut in ileus or mechanical obstruction. Nasogastric lavage of saline or water in the patient with upper GI hemorrhage determines the rate of bleeding and helps evacuate blood prior to endoscopy. Enteral feedings can be initiated through a nasogastric or nasoenteric tube. Enemas relieve fecal impaction or assist in gas evacuation in acute colonic pseudoobstruction. A rectal tube can be left in place to vent the distal colon in colonic pseudoobstruction and other colonic distention disorders.

In addition to its diagnostic role, endoscopy has therapeutic capabilities in certain settings. Cautery techniques can stop hemorrhage from ulcers, vascular malformations, and tumors. Injection with vasoconstrictor substances or sclerosants is used for bleeding ulcers, vascular malformations, varices, and hemorrhoids. Endoscopic encirclement of varices and hemorrhoids with constricting bands stops hemorrhage from these sites, while endoscopically placed clips can occlude arterial bleeding sites. Endoscopy can remove polyps or debulk lumen-narrowing malignancies. Endoscopic sphincterotomy of the ampulla of Vater relieves symptoms of choledocholithiasis. Obstructions of the gut lumen and pancreaticobiliary tree are relieved by endoscopic dilation or placement of plastic or expandable metal stents. In cases of acute colonic pseudoobstruction, colonoscopy is employed to withdraw luminal gas. Finally, endoscopy is commonly used to insert feeding tubes.

Radiologic measures also are useful in GI disease. Angiographic embolization or vasoconstriction decrease bleeding from sites not amenable to endoscopic intervention. Dilation or stenting with fluoroscopic guidance relieves luminal strictures. Contrast enemas can reduce volvulus and evacuate air in acute colonic pseudoobstruction. CT and ultrasound help drain abdominal fluid collections, in many cases obviating the need for surgery. Percutaneous transhepatic cholangiography relieves biliary obstruction when ERCP (endoscopic retrograde cholangiopancreatography) is contraindicated. Lithotripsy can fragment gallstones in patients who are not candidates for surgery. In some instances, radiologic approaches offer advantages over endoscopy for gastroenterostomy placement. Finally, central venous catheters for parenteral nutrition may be placed using radiographic techniques.

The major factors that determine the diagnostic and therapeutic approach are the amount and rate of bleeding [7]. Estimates of both should be made promptly and monitored and revised continuously until the episode has been resolved. It is important to know at the outset that bleeding stops spontaneously in 75% of cases;

the remainder includes those who will require surgery, experience complications, or die.

Hematemesis or melena is present except when the rate of blood loss is minimal. Hematemesis of either brightred or dark blood indicates that the source is proximal to the ligament of Treitz. It is more common from bleeding that originates in the stomach or esophagus. In general, hematemesis denotes a more rapidly bleeding lesion, and a high percentage of patients who vomit blood require surgery. Coffee-ground vomitus is due to vomiting of blood that has been in the stomach long enough for gastric acid to convert hemoglobin to methemoglobin.

Most patients with melena (passage of black or tarry stools) are bleeding from the upper gastrointestinal tract, but melena can be produced by blood entering the bowel at any point from mouth to cecum. The conversion of red blood to dark depends more on the time it resides in the intestine than on the site of origin. The black color of melenic stools is probably caused by hematin, the product of oxidation of heme by intestinal and bacterial enzymes. Melena can be produced by as little as 50–100 mL of blood in the stomach. When 1 L of blood was instilled into the upper intestine of experimental subjects, melena persisted for 3–5 days, which shows that the rate of change in character of the stool is a poor guide to the time bleeding stops after an episode of hemorrhage.

Examination

The clinical history can yield useful information suggesting the site of gastrointestinal bleeding as well as the specific lesion [8]. The medical history should include questions about prior episodes of upper gastrointestinal bleeding (ulcers or varices), liver disease, intestinal polyps or cancer, and blood transfusions. Pertinent symptoms include abdominal pain, nausea, vomiting, hematemesis, early satiety, anorexia, or weight loss. Medication history should include aspirin, nonsteroidal antiinflammatory drugs (NSAIDs), and anticoagulation therapy (warfarin or heparin). Social history should focus on intravenous drug use, alcohol abuse, and sexual partners.

Physical examination should include a rectal examination, a nasogastric tube aspiration, assessment of stigmata of chronic liver disease (jaundice, spider telangiectasias, ascites). Melena is suggestive of upper gastrointestinal bleeding, although it may be seen with small bowel bleeding or right-sided colonic lesions. Bright red blood per rectum or clot suggests a lower gastrointestinal bleeding source, although massive upper gastrointestinal bleeding may also present in this manner. A positive nasogastric aspirate yielding coffee ground material, clots, or bright red blood is highly suggestive of a recent upper gastrointestinal bleed. A negative nasogastric aspirate yielding clear fluid without bile will not exclude duodenal lesions, such as an ulcer.

Laboratory data can be useful for assessing the degree of bleeding, determining possible sources of bleeding, and guiding therapy. Laboratory studies should include a complete blood count (CBC), an automated chemistry panel that includes liver function studies [alanine aminotransferase (ALT), aspartate aminotransferase (AST), bilirubin, albumin, and total protein], and coagulation studies [prothrombin time (PT), partial thromboplastin time (PTT)]. It is important to keep in mind that the hematocrit may significantly underestimate the amount of blood loss during an acute bleed.

Serial hematocrits may be an indicator of ongoing bleeding or rebleeding and can help guide transfusions with packed red blood cells, fresh frozen plasma, and platelets. An electrocardiogram (ECG) should be obtained for all patients with significant cardiac risk factors. An upright chest and abdominal x-ray should be performed in patients with suspected intestinal perforation, obstruction, or pulmonary aspiration.

Diagnosis

Initial assessment includes a medical history, vital signs, physical examination (including digital rectal examination), and nasogastric lavage in an attempt to localize the source of melena or hematochezia to the upper GI tract [9]. Patients should be asked questions that can help determine the diagnostic possibilities for the bleeding source. For example, peptic ulcer bleeding should be suspected in patients taking daily aspirin or nonsteroidal antiinflammatory drugs (NSAIDs). For patients with known or suspected liver disease, bleeding related to portal hypertension (such as varices or portal hypertensive gastropathy) should be strongly considered. Heavy alcohol intake or vomiting should suggest a Mallory-Weiss tear. A feeding tube or a chronic nasogastric tube and a history of gastroesophageal reflux disease raise the suspicion for severe erosive esophagitis. The physician should check the vital signs with attention to signs of hypovolemia, such as hypotension, tachycardia, and orthostasis. The patient's skin should be examined for petechiae, purpura, spider angiomas, and palmar erythema, and the abdomen should be assessed for ascites, hepatomegaly, or splenomegaly, which may indicate portal hypertension. Tenderness or a mass may indicate an intra-abdominal tumor.

Routine placement of a nasogastric or orogastric tube is not recommended for patients with upper GI hemorrhage. Such a tube may, however, potentially be useful in selected patients to: localize bleeding to the upper GI tract in patients with hematochezia, hemodynamic instability, and absence of overt signs of upper GI bleeding; help clear gastric blood for better endoscopic visualization; and minimize the risk of aspiration.

Peripheral blood should be sent for standard hematology, chemistry, liver, and coagulation studies as well as for typing and crossmatching for packed red blood cells. Hemoglobin concentration and hematocrit may not accurately reflect blood loss because equilibration with extravascular fluid requires 24 to 72 hours. A low platelet count suggests chronic liver disease, dilution, drug reaction, or a hematologic disorder. In upper GI bleeding, the blood urea nitrogen level typically increases to a greater extent than the creatinine level owing to increased intestinal absorption of urea after the breakdown of blood proteins. However, this phenomenon can be misleading in the setting of renal insufficiency or rapid transit of blood. An elevated international normalized ratio can be observed in chronic liver disease and in patients who are taking warfarin.

Endoscopy

The goal of endoscopic therapy is to eliminate persistent bleeding and prevent rebleeding [1]. Rebleeding is the greatest contributor to both morbidity and mortality. The ability to achieve sustained hemostasis is dependent on controlled access to the bleeding ves-

sel, a relatively small vessel size, and the absence of significant coagulation defects.

Upper endoscopy provides diagnosis, prognosis, and therapy in patients with acute upper GI bleeding and is reasonably safe to perform in patients with upper GI bleeding (50% of complications are cardiopulmonary). Upper endoscopy is 90–95% sensitive at locating the bleeding site. Sensitivity increases when the procedure is done closer to the onset of the bleeding and decreases with increasing duration from time of onset. Most patients with upper GI bleeding should undergo an upper endoscopy within 24 hours of presentation as is currently recommended by guidelines. Studies in patients with nonvariceal upper GI bleeding have not found an overall advantage of early endoscopy (within 12 hours) in terms of rebleeding rate, need for surgery, or mortality, however patients with active upper GI bleeding may benefit from an early intervention. Endoscopy can offer therapeutic options including injection, cautery, placement of hemoclips, or a combination of therapies. Endoscopy can also predict the likelihood of persistent or recurrent bleeding based on recognition of the endoscopic stigmata of recent hemorrhage.

GBS

GBS (Glasgow-Blatchford score) was developed to identify patients presenting with upper gastrointestinal tract bleeding that would require intervention [10]. It incorporates simple clinical and laboratory parameters to identify low-risk patients. In a large study of over 3,000 participants, patients scoring ≤ 1 on the GBS had a 0.4% mortality rate and 1.4% required endoscopic intervention. The score comprises urea, blood pressure, haemoglobin, pulse, syncope, melaena, history of liver disease, and cardiac failure.

The GBS is a validated pre-endoscopic risk score for predicting the need for intervention in upper gastrointestinal bleeding. Very low-risk patients are unlikely to require intervention and thus are suitable for consideration of early discharge. Very low risk was originally defined in guidance from the United Kingdom's National Institute for Health and Care Excellence as a score of 0. However, more recent European Society of Gastrointestinal Endoscopy guidance recommends ≤ 1.

Upper and Lower GI Bleeding

Hematemesis indicates an upper GI source of bleeding (above the ligament of Treitz) [11]. Melena indicates that blood has been present in the GI tract for at least 14 h. Thus, the more proximal the bleeding site, the more likely melena will occur. Hematochezia usually represents a lower GI source of bleeding, although an upper GI lesion may bleed so briskly that blood does not remain in the bowel long enough for melena to develop. When hematochezia is the presenting symptom of UGIB (Upper Gastrointestinal Bleeding), it is associated with hemodynamic instability and dropping hemoglobin. Bleeding lesions of the small bowel may present as melena or hematochezia. Other clues to UGIB include hyperactive bowel sounds and an elevated blood urea nitrogen level (due to volume depletion and blood proteins absorbed in the small intestine).

A nonbloody nasogastric aspirate may be seen in up to 16% of patients with UGIB— usually from a duodenal source. Even a bile-stained appearance does not exclude a bleeding postpyloric lesion since reports of bile in the aspirate are incorrect in about 50% of cases. Testing of aspirates that are not grossly bloody for occult blood is not useful.

Conclusion

All patients with suspected upper GI bleeding should be subjected to nasogastric aspiration and lavage. A bloody nasogastric aspirate indicates active upper GI tract bleeding, but about 10% of patients with upper GI tract bleeding have no blood in the nasogastric aspirate. Vomiting of contents such as black coffee grounds indicates bleeding that has slowed or stopped. In case of bleeding from the upper GI tract, it is necessary to perform an examination of the esophagus, stomach and duodenum. Since gastroscopy can be diagnostic and therapeutic, it should be done quickly in case of heavy bleeding, but it can also be postponed for 24 hours if the bleeding stops or is minimal.

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