MINISTRY OF EDUCATION AND SCIENCE OF UKRAINE
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«UZHHOROD NATIONAL UNIVERSITY»
FACULTY OF MEDICINE
DEPARTMENT OF SURGERY DISEASES

UPPER GASTROINTESTINAL BLEEDING

Methodological matherials for independent study for students

UZHHOROD

2024

UZHHOROD NATIONAL UNIVERSITY
FACULTY OF MEDICINE
DEPARTMENT OF SURGERY DISEASES

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Methodical matherials are devoted to issues of etiopathogenesis, symptoms, diagnosis and treatment methods of upper gastrointestinal bleeding (UGIB). The authors also tried to highlight different diseases and conditions which may lead to UGIB. The methodical matherials are intended for senior year students of higher medical educational institutions.

Uzhhorod

2024

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Background

Acute gastrointestinal (GI) bleeding is a potentially life-threatening abdominal emergency that remains a common cause of hospitalization. **Upper GI bleeding (UGIB)** is classified as any blood loss from a gastrointestinal source above the ligament of Treitz (esophagus, stomach, duodenum).

Upper gastrointestinal bleeding is a common problem with an annual incidence of approximately 80 to 150 per 100,000 population, with estimated mortality rates between 2% to 15%. It can manifest as hematemesis (bright red emesis or coffee-ground emesis), hematochezia, or melena. Patients can also present with symptoms secondary to blood loss, such as syncopal episodes, fatigue, and weakness. UGIB can be acute, occult, or obscure.

The severity of UGIB is defined by the patient's hemodynamic status and packed red blood cell transfusion requirements. Although patients who remain hemodynamically stable may be managed appropriately in the outpatient setting, severe UGIB requires close monitoring in the intensive care unit with early upper endoscopy.

Epidemiology

UGIB accounts for 75% of all acute gastrointestinal bleeding cases. Its annual incidence is approximately 80 to 150 per 100,000 population. In the United Kingdom, UGIB accounts for 70,000 hospital admissions each year, with the majority of cases nonvariceal in origin. In a nationwide study from Spain, UGIB was six times more common than lower GI bleeding.

UGIB is twice as common in men as in women and increases in prevalence with age (> 60 years). However, the death rate is similar in both sexes.

Patients on long-term, low-dose aspirin have a higher risk of overt UGIB compared to placebo. When aspirin is combined with P2Y12 inhibitors such as clopidogrel, there is a two-fold to three-fold increase in the number of UGIB cases.

Peptic ulcer bleeding is the most common cause of upper gastrointestinal bleeding, responsible for about 50% of all cases, followed by esophagitis and erosive disease. Variceal bleeding is the cause of bleeding in cirrhotic patients in 50-60% of cases.

Despite advances in therapy, in-hospital mortality remains high (13%). Mortality is increasing with increasing age and is significantly higher in patients who are already admitted in hospital for co-morbidity.

Rebleeding in upper gastrointestinal bleeding occurs in 7-16% of cases, despite endoscopic therapy. Rebleeding is especially high in variceal bleeding and peptic ulcer bleeding.

Risk factors for peptic ulcer bleeding are non-steroid anti-inflammatory drugs (NSAIDs) use and *H. pylori* infection. In patients at risk for gastrointestinal bleeding and using NSAIDs, a protective drug was only used in 10%. COX-2 selective inhibitors do cause less gastroduodenal ulcers compared to non-selective NSAIDs, however, more cardiovascular adverse events are reported. *H. pylori* infection is found in about 50% of peptic ulcer bleeding patients. *H. pylori* should be tested for in all ulcer patients and eradication should be given.

Anatomy

The **stomach** is an important organ and the most dilated portion of the digestive system. The **esophagus** precedes it, and the **small intestine** follows. It is a large, muscular, and hollow organ allowing for a capacity to hold food.

It is comprised of **regions:** the cardia, fundus, body, antrum and pylorus. The *cardia* is connected to the esophagus and is where the food first enters the stomach. The *fundus* follows the cardia and is a bulbous, dome-shaped, superior portion of the stomach. Followed by the fundus is the *body* or the main, largest

portion of the stomach. Following the body are the *antrum* and *pylorus*, which conically funnels food into the duodenum, or upper portion of the small intestine. The stomach is located left of the midline and centrally in the upper area of the abdomen (fig. 1).

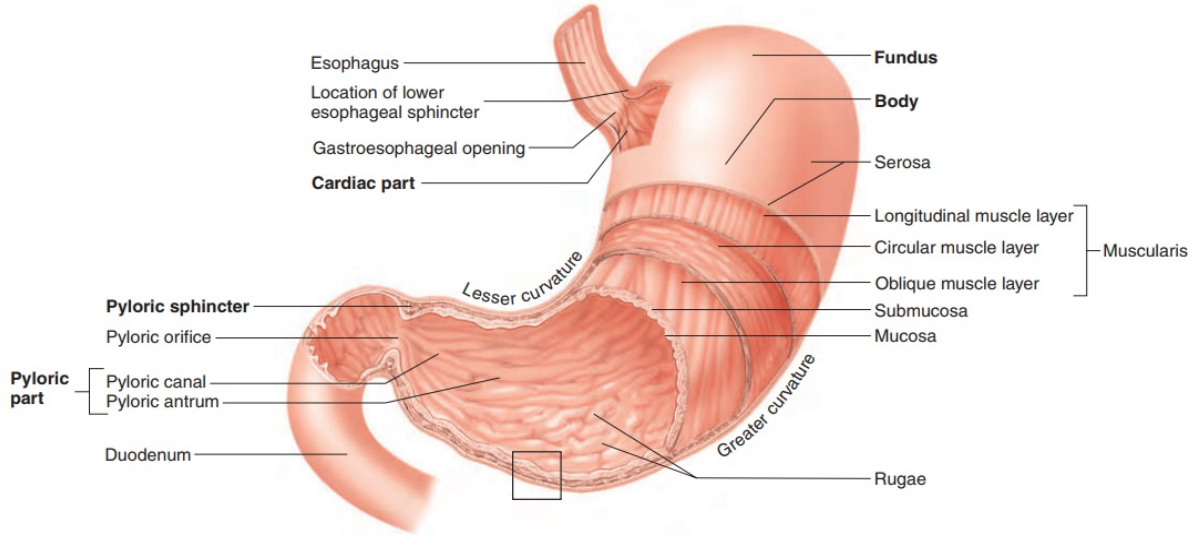

Fig. 1. Stomach anatomy.

Structures and functions. The *primary functions* of the stomach include the temporary storage of food and the partial chemical and mechanical digestion of food.

The upper portions of the stomach relax as food enters to allow for the stomach to hold increasing quantities of food. The lower portion of the stomach contracts in a rhythmic fashion (*mechanical digestion*) to aid with the breaking down of food and mixes it with stomach juices (*chemical digestion*) which also serve to break food down and prepare the mixture, termed *chyme* at this point of digestion, for further digestion. Mixing waves in the stomach are produced, and with each wave, the pyloric sphincter allows small quantities of chyme into the duodenum. Stomach juices are secreted by the fundus of the stomach and include hydrochloric acid (HCl) and the enzyme pepsin. In addition to HCl, the stomach also produces intrinsic factor (absorption of vitamin B12, which plays a role in the production of red blood cells and neurological functions) in its parietal cells.

The stomach is capable of processing food and distributing it to the duodenum on average within 2 to 4 hours. The acidic environment of the stomach may be lethal to many types of bacteria and other microorganisms that enter the body by way of ingestion, potentially protecting the body from infection and diseases.

Blood supply and lymphatics. The celiac trunk, branching directly anteriorly from the aorta provides the main *arterial blood supply*. The trunk

supplies the common hepatic artery (CHA), splenic artery, and the left gastric artery (LGA). The less curved side of the stomach is proximally supplied by a descending branch of the LGA, with its ascending branch supplying portions of the esophagus. The CHA which runs superior to the pancreas and the right branches off to the gastroduodenal artery (GDA) and continues with the branch that proceeds from the CHA being the proper hepatic artery. The right gastric artery (RGA) then branches from the proper hepatic artery. The RGA then runs from right to left across the lesser curved portion of the stomach and continues to branch into smaller vessels through the body of the stomach to join the network of smaller arteries supplying the stomach as branched off from the LGA. The posterior superior pancreaticoduodenal artery branches off of the GDA which then branches into the anterior superior pancreaticoduodenal artery and the right gastroepiploic artery, which then traverses and supplies from right to left the greater curvature of the stomach. The left gastroepiploic artery branches from the splenic artery also supplies the greater curvature body portion of the stomach. Three to five additional smaller arteries also branch from the splenic artery to supply the stomach.

The left gastric (coronary) vein and the right gastric and right gastro-omental veins all achieve *venous drainage* into different segments of the *portal vein*. The short gastric veins (the vasa brevia) and the left gastroepiploic vein achieve drainage via the splenic vein.

The *lymphatic drainage* of the stomach can be understood as 4 levels. Level 1 includes the perigastric lymph nodes and follows a path of drainage of the right pericardiac, left pericardiac, along with the less curved body portion, along with the greater curved body portion, supra-pyloric, and infra-pyloric. Level 2 is comprised of drainage along the LGA, along the CHA, along with the celiac axis, at the splenic hilum, and along the splenic artery. Level 3 is characterized by drainage in the hepatoduodenal ligament, posterior to the duodenum and pancreas head, and at the source of the small bowel mesentery. Finally, the level 4 is characterized by mesocolic and paraaortic drainage.

Nerves. The autonomic nervous system provides the stomach with the innervation via parasympathetic and sympathetic nerves. The *vagus nerve* supplies *parasympathetic innervation* via the right posterior and left vagal trunks. The left vagus nerve is anterior, while the right vagus nerve is posterior. The right vagus nerve branches to the criminal nerve of Grassi for innervation of the cardia and fundus. The trunks also follow the lesser curvature region of the stomach to form the posterior and anterior gastric nerves of Latarjet innervating the body, antrum, and pylorus. *Sympathetically,* nerves are supplied, including some fibers transmitting pain, to the celiac plexus from spinal cord segments T6 through T9.

Layers. Four main *layers* constitute the stomach wall, including the mucosa, submucosa, muscularis externa, and the serosa.

The innermost layer, the *mucosa*, is covered by epithelial tissue and is mainly comprised of gastric glands that secrete gastric juices. Particularly, the fundus releases gastric juices while the cardia secretes protective mucus which coats the inner mucosal wall of the stomach via mucus (Foveolar) cells thereby protecting the stomach muscles from being digested by the gastric juices produced by the chief cells (pepsin) and parietal cells (HCl).

The *submucosa* is comprised of dense connective tissue and contains blood and lymphatic vessels along with nerves. Together, the submucosa supports the mucosal layer and has many folds analogous to that of an accordion called rugae which allows for distension of these layers when food enters the stomach.

The *muscularis externa* is the next layer and is comprised of the 3 sub-layers: running longitudinally, obliquely, and circularly as part of the stomach wall. The inner oblique layer is unique to the stomach and is primarily responsible for the churning, mechanical digestion of food. The middle circular layer is concentric with the stomach's longitudinal axis and thickens in the region of the pylorus to form the pyloric sphincter responsible for regulating the output from the stomach into the duodenum. The next layer is the outer longitudinal layer, but between this layer and the middle circular layer, is *Auerbach's (myenteric) plexus*, which is a region of innervation for the two adjacent muscular layers. The outer longitudinal layer facilitates the movement of food in the direction of the pylorus via muscular shortening.

The final layer, the *serosa*, is made up of multiple layers of connective tissue which also connect continuously with the peritoneum.

Etiology

Upper gastrointestinal bleeding is a common medical condition with various etiologies (fig. 2).

Ulcer-related UGIB. Bleeding peptic ulcers account for the majority of patients presenting with acute upper gastrointestinal bleeding (40-50% of the cases). As previously mentioned, peptic ulcer disease is strongly associated with *H. pylori* infection. The microorganism causes disruption of the mucous barrier and has a direct inflammatory effect on the gastric and duodenal mucosa.

In cases of ulcer-associated UGIB, as the ulcer burrows deeper into the gastroduodenal mucosa, the process causes weakening and necrosis of the arterial wall, leading to the development of a pseudoaneurysm. The weakened wall ruptures, producing hemorrhage.

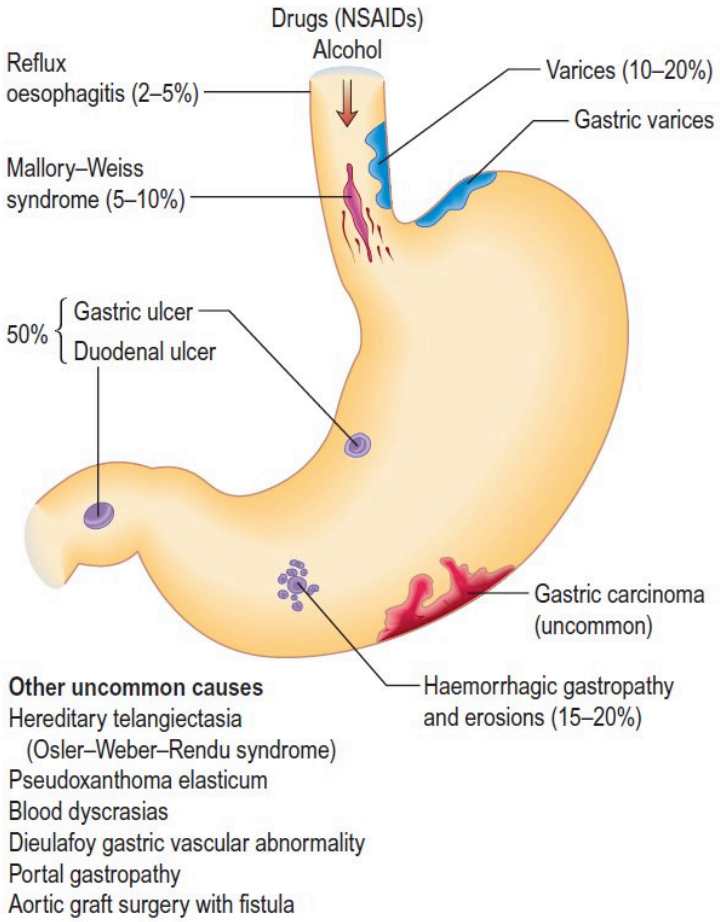

Fig. 2. Common causes of UGIB.

The flow through a vessel varies with the fourth power of the radius; thus, small increases in vessel size can mean much larger amounts of blood flow and bleeding, with more severe hypotension and more complications, especially in older patients. Visible vessels usually range from 0.3-1.8 mm.

Exsanguinating hemorrhage has been reported from larger vessels. The larger vessels are located deeper in the gastric and duodenal submucosa and serosa. Larger branches of the left gastric artery are found high on the lesser curvature, while the pancreatoduodenal artery and its major branches are located posteroinferiorly in the duodenal bulb.

Vomiting-related UGIB. During vomiting, the lower esophagus and upper stomach are forcibly inverted. Vomiting attributable to any cause can lead to a mucosal tear of the lower esophagus or upper stomach. The depth of the tear determines the severity of the bleeding. Rarely, vomiting can result in esophageal rupture (*Boerhaave syndrome,* fig. 3), leading to bleeding, mediastinal air entry, left pleural effusion (salivary amylase can be present) or left pulmonary infiltrate, and subcutaneous emphysema.

Mallory-Weiss tears in UGIB. Mallory-Weiss tears (syndrome) account for 8-15% of acute upper GI hemorrhage. Kenneth Mallory and Soma Weiss first

described the syndrome in 1929. The occasionally massive UGIB results from a tear in the mucosa of the gastric cardia. Like many upper GI tract lesions, the Mallory-Weiss tear may stop bleeding spontaneously 85-90% of the time.

This linear mucosal laceration is the result of forceful vomiting, retching, coughing, or straining. These actions create a rapid increase in the gradient between intragastric and intrathoracic pressures, leading to a gastric mucosal tear from the forceful distention of the gastroesophageal junction. In 80-90% of cases, this is a single, 1.75- to 2.5-cm mucosal tear along the lesser curve of the stomach just distal to the gastroesophageal junction (fig. 3). The presence of a hiatal hernia is a predisposing factor and is found in 35-100% of patients with Mallory-Weiss syndrome.

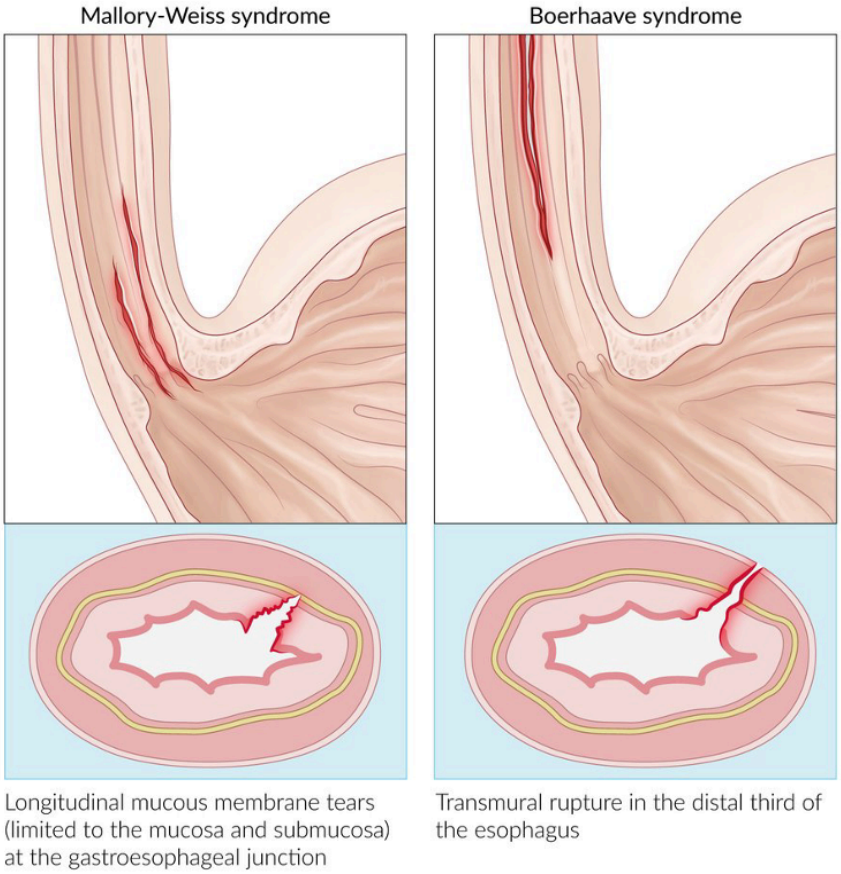

Fig. 3. Mallory-Weiss and Boerhaave syndromes.

Acute stress-related mucosal disease in UGIB. Acute stress-related mucosal disease (or stress ulcer) results from predisposing clinical conditions that have the potential to alter the local mucosal protective barriers, such as mucus, bicarbonate, blood flow, and prostaglandin synthesis. Any disease process that disrupts the balance of these factors results in diffuse gastric mucosal erosions.

This is most commonly observed in patients who have undergone episodes of shock, multiple traumas, acute respiratory distress syndrome (ARDS), systemic respiratory distress syndrome, acute renal failure, and sepsis. The principal

mechanisms involved are decreased splanchnic mucosal blood flow and altered gastric luminal acidity.

Dieulafoy lesions in UGIB. The Dieulafoy lesion, first described in 1896, is a vascular malformation of the proximal stomach, usually within 6 cm of the gastroesophageal junction along the lesser curvature of the stomach. However, it can occur anywhere along the GI tract. This lesion accounts for 2-5% of acute UGIB episodes.

Endoscopically, the lesion appears as a large submucosal vessel that has become ulcerated. Because of the large size of the vessel, bleeding can be massive and brisk. The vessel rupture usually occurs in the setting of chronic gastritis, which may induce necrosis of the vessel wall. Alcohol consumption is reportedly associated with the Dieulafoy lesion. The lesion mostly occurres in men and mostly in those in their third to tenth decade.

NSAIDs in UGIB. NSAIDs cause gastric and duodenal ulcers by inhibiting cyclooxygenase, which causes decreased mucosal prostaglandin synthesis and results in impaired mucosal defenses. Daily NSAID use causes an estimated 40-fold increase in gastric ulcer creation and an 8-fold increase in duodenal ulcer creation.

Long-term NSAID use is associated with a 20% incidence in the development of mucosal ulceration. Medical therapy includes avoiding the ulcerogenic drug and beginning a histamine-2 (H2) – receptor antagonist or a proton pump inhibitor (PPI) that provides mucosal protection.

Esophagogastric varices. Esophagogastric variceal bleeding is the second most common cause of UGIB and should always be considered in patients with a history of cirrhosis with portal hypertension (fig. 4).

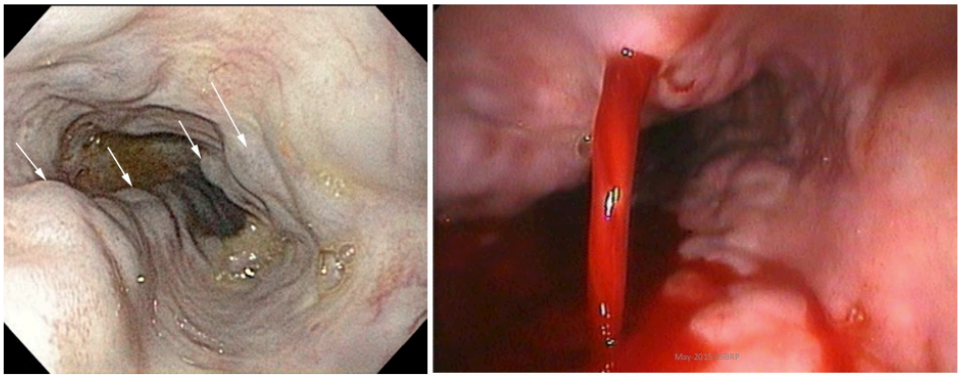

Fig. 4. Esophageal variceal bleeding. *A – esophageal varices (arrows); B – bleeding varices.*

Approximately half the patients with cirrhosis have gastroesophageal varices as a consequence of an elevated hepatic venous pressure gradient (>10-12 mm

Hg). However, it is important to consider that approximately 60% of UGIB cases in patients with cirrhosis are unrelated to portal hypertension.

The severity of the underlying cirrhosis (Child-Pugh score) is directly related to the probability that the patient will have varices. The physical examination should focus on identifying stigmata of chronic liver disease suggestive of portal hypertension (ie, ascites, caput medusae, spider angiomata).

Esophagitis accounts for approximately 10% of UGIB cases. Severe gastroesophageal reflux disease and alcohol abuse are the 2 most common risk factors for erosive esophagitis complicated by bleeding. Other causes include pill esophagitis and infectious esophagitis. Although rare, ischemia may lead to esophageal necrosis (black esophagus) and should be suspected in a patient with a history of hemodynamic instability preceding UGIB.

Other causes. *Arteriovenous malformations* are often found on routine endoscopy and are typically innocuous. Nonvariceal causes of UGIB in patients with underlying liver disease include *gastric antral vascular ectasias*, a relatively uncommon pathology that causes red streaking and bleeding extending from the pylorus to the antrum, and *portal hypertensive gastropathy*, in patients with concomitant portal hypertension. *Esophageal, gastric,* and *duodenal malignancies* can also lead to UGIB, although these are a relatively uncommon cause of an acute bleeding.

Aortoenteric fistulas can occur as a late complication of abdominal aortic surgery or vascular reconstruction, with the duodenum as the most common site of involvement. The classic presentation involves a “herald bleed” usually manifesting as an episode of hematemesis or hematochezia, followed by a grace period of several days, with massive bleeding and cardiovascular collapse.

Classification

1. By etiology:
   - ulcerative;
   - vascular;
   - traumatic;
   - iatrogenic;
   - portal hypertension.
2. By the source of bleeding:
   - esophageal;
   - gastric;
   - duodenal.

3. By the classes of blood loss:

- I class – blood loss volume up to 750 mL;
- II class – blood loss volume 750-1500 mL;
- III class – blood loss volume 1500-2000 mL;
- IV class – blood loss volume over 2000 mL.

Clinical symptoms and physical examination

The history and physical examination of the patient provide crucial information for the initial evaluation of persons presenting with a gastrointestinal tract hemorrhage. Important information to obtain includes potential comorbid conditions, medication history, and any prior history of GI bleeding, as well as the severity, timing, duration, and volume of the bleeding.

History findings include weakness, dizziness, syncope associated with **hematemesis** ("coffee ground" vomitus), and **melena** (black stools with a rotten odor).

Occasionally, a brisk UGIB manifests as **hematochezia** (red or maroon stools); the redder the stool, the more rapid the transit, which suggests a large upper tract hemorrhage.

Patients may have a history of **dyspepsia** (especially nocturnal symptoms), **ulcer disease**, early satiety, and **nonsteroidal anti-inflammatory drug**, **antiplatelet therapy**, or **aspirin** use. A history of recent aspirin ingestion suggests that the patient may have NSAID gastropathy with an enhanced bleeding diathesis from poor platelet adhesiveness.

Many patients with UGIB who are taking NSAIDs present without dyspepsia but with hematemesis or melena as their first symptom, owing to the analgesic effect of the NSAID. Low-dose aspirin (81 mg) has also been associated with UGIB, with or without the addition of NSAID therapy. Using the lowest effective dose for both short-term and long-term users is recommended.

Patients with a history of ulcers are at an especially increased risk for UGIB when taking steroids, aspirin, dual antiplatelet therapy (DAPT) (eg, addition of clopidogrel to aspirin), or NSAID therapy. These high-risk individuals should receive continuous acid suppression with a proton pump inhibitor. The patient's ulcer history is also important because recurrence of ulcer disease is common, especially there has not been successful eradication of an *H. pylori* infection.

Patients may present asymptomatically or in a more subacute phase, with a history of dyspepsia and **occult intestinal bleeding** manifesting as a positive fecal occult blood test result or as **iron deficiency anemia**.

A history of chronic **alcohol use** of more than 50 g/d or **chronic viral hepatitis (B or C)** increases the risk of variceal hemorrhage, gastric antral vascular ectasia, or portal gastropathy. Alcohol also interferes with cyclooxygenase-1 receptor enzymes which reduce the production of cytoprotective prostaglandin and alters gastric mucosal protection.

The finding of subcutaneous emphysema with a history of **vomiting** is suggestive of Boerhaave syndrome (esophageal perforation) and requires prompt consideration of surgical therapy.

The presence of **postural hypotension** indicates more rapid and severe blood loss.

Incidence of common acute UGIB symptoms are as follows:

- Hematemesis: 40-50%.
- Melena: 70-80%.
- Hematochezia: 15-20%.
- Either hematochezia or melena: 90-98%.
- Syncope: 14%.
- Presyncope: 43%.
- Dyspepsia: 18%.
- Epigastric pain: 41%.
- Heartburn: 21%.
- Diffuse abdominal pain: 10%.
- Dysphagia: 5%.
- Weight loss: 12%.
- Jaundice: 5%.

The importance of the above clinical signs/symptoms in determining the source of GI bleeding is demonstrated in the table below (tab. 1).

Table 1.

Probable source of GI bleeding

Clinical symptom	Probability of Upper GI sourse	Probability of Lower GI sourse
Hematemesis	Almost certain	Rare
Melena	Probable	Possible
Hematochezia	Possible	Probable
Blood-streaked stool	Rare	Almost certain
Occult blood in stool	Possible	Possible

Physical examination. The goal of the patient's physical examination is to evaluate for shock and blood loss. Patients present with an ulcer that has bled or is actively bleeding (although approximately 80% of ulcers stop bleeding).

Hematemesis and melena are the most common presentations of acute UGIB, and patients may present with both symptoms.

Hematemesis is the overt bleeding with vomiting of fresh blood or clots. The term "coffee-grounds" describes gastric aspirate or vomitus that contains dark specks of old blood.

Melena refers to dark and tarry-appearing stools with a distinctive smell. The more proximal the bleeding site, the more likely melena occur.

Hematochezia is the passage of fresh blood per rectum. The latter is usually a reflection of lower gastrointestinal bleeding (LGIB) but may be seen in patients with brisk UGIB.

Assessing the patient for **hemodynamic instability** and clinical signs of poor perfusion is important early in the initial evaluation to properly triage patients with massive hemorrhage to intensive care unit (ICU) settings.

Worrisome clinical signs and symptoms of hemodynamic compromise include **tachycardia** of more than 100 beats per minute (bpm), **systolic blood pressure** of less than 90 mm Hg, cool extremities, syncope, and other obvious **signs of shock**, ongoing brisk hematemesis, or the occurrence of maroon or bright-red stools, which requires rapid blood transfusion.

Pulse and **blood pressure** should be checked with the patient in supine and upright positions to note the effect of blood loss. Significant changes in vital signs with postural changes indicate an acute blood loss of approximately 20% or more of the blood volume.

Signs of **chronic liver disease** should be noted, including spider angiomata, gynecomastia, increased luneals, splenomegaly, ascites, pedal edema, and asterixis.

Signs of **tumor** are uncommon but portend a poor prognosis. Signs include a nodular liver, an abdominal mass, and enlarged and firm lymph nodes. The finding of telangiectasias may indicate the rare case of Osler-Weber-Rendu syndrome.

Rectal examination helps evaluate consistency and color of stool, exclude rectal sources of bleeding.

Diagnosis

Approach considerations. A complete blood cell count (CBC) with platelet count and differential is necessary to assess the level of blood loss in a patient with UGIB. Where possible, having the patient's previous results as a baseline is useful to gauge this loss. The CBC count should be checked frequently (every 4-6 h initially), depending on the severity of the bleeding, clinical stability, and apparent rate of blood loss.

Assessing patients' *calcium* levels is useful in identifying individuals with hyperparathyroidism, but it is especially helpful in monitoring calcium in patients receiving multiple transfusions of citrated blood. Hypercalcemia increases acid secretion.

A *gastrin* level may identify the rare patient with gastrinoma as the cause of UGIB and multiple ulcers. It is important to recognize that moderately high elevations of gastrin are seen in patients taking PPIs.

Electrocardiography (ECG) should be considered, especially in those with underlying cardiac disease or risk factors. Close measurement of vital signs, including continuous pulse and blood pressure monitoring, is important and may alert clinicians to important changes in the patient's clinical stability. Careful and ongoing monitoring of volume resuscitation is essential to avoiding end-organ injury, especially acute myocardial infarction due to hypotension.

Assessment of hemorrhagic shock. Patients who present in hemorrhagic shock have a mortality rate of up to 30%. Hemorrhage may be classified based on the amount of blood loss, as noted in the following table 2.

Table 2.

Classification of hemorrhagic shock

	Class 1	**Class 2**	**Class 3**	**Class 4**
Blood loss, mL (%)	< 750 (< 15 %)	750-1500 (15-30 %)	1500-2000 (30-40 %)	> 2000 (> 40 %)
Heart Rate, bpm	< 100	100-120	120-140	> 140
Blood Pressure	Normal	Normal	Decreased	Decreased
Pulse Pressure	Normal	Narrowed	Narrowed	Narrowed
Respiratory Rate, breaths/min	14-20	20-30	30-40	> 40
Urine Output, mL/h	> 30	20-30	5-15	Negligible
CNS/Mental Status	Slightly anxious	Mildly anxious	Anxious, confused	Confused, lethargic
Fluid Replacement	Crystalloid	Crystalloid	Crystalloid and blood	Crystalloid and blood

bpm = beats per minute; CNS = central nervous system.

This classification scheme aids in understanding the clinical manifestations of hemorrhagic shock. In early class 1 shock, the patient may have normal vital signs, even with a 15% loss of total blood volume. As the percentage of blood

volume loss increases, pertinent clinical signs, symptoms, and findings become more apparent.

Although early cardiovascular changes occur as blood loss continues, urine output, as a sign of end organ renal perfusion, is only mildly affected until class 3 hemorrhage has occurred.

Bornman et al found that rebleeding (a marker for increased mortality and need for surgery) occurres in 2% of patients without shock, in 18% with isolated tachycardia, and in 48% with shock.

Unless the patient has evidence of shock, orthostatic testing should be performed to assess and document a hypovolemic state. A positive tilt test finding is defined as a systolic blood pressure decrease of 10 mm Hg and a pulse rate increase of 20 bpm with standing compared to the supine position.

Hemoglobin value and type and crossmatch blood. The patient should be crossmatched for 2-6 units, based on the rate of active bleeding. The hemoglobin level should be monitored serially in order to follow the trend. An unstable hemoglobin level may signify ongoing hemorrhage requiring further intervention.

Patients generally require blood transfusions because of hypoperfusion and hypovolemia. Patients with significant comorbid conditions (eg, advanced cardiovascular disease) should receive blood transfusions to maintain myocardial oxygen delivery to avoid myocardial ischemia.

However, once the patient has been stabilized, controversy exists regarding strategies for transfusion of red blood cells in GI bleeding, with some studies suggesting improved outcomes with a more judicious use of blood transfusions. In the restrictive strategy, patients require transfusion when their hemoglobin level is below 7 g/dL; in the liberal strategy, patients require transfusion when their hemoglobin level is below 9 g/dL.

BMP and BUN. The basic metabolic profile (BMP) is useful in evaluating for renal comorbidity in cases of suspected upper gastrointestinal bleeding; however, blood in the upper intestine can elevate the BUN (blood urea nitrogen) level as well. Measurement of coagulation parameters is necessary to assess for continued bleeding. Abnormalities should be corrected rapidly.

The BUN-to-creatinine ratio increases with UGIB. A ratio of greater than 36 in a patient without renal insufficiency is suggestive of UGIB.

Coagulation profile. The patient's prothrombin time (PT), activated partial thromboplastin time (PTT), and international normalized ratio (INR) should be checked to document the presence of coagulopathy. The coagulopathy may be consumptive and associated with a thrombocytopenia.

A platelet count below 50 × 10^9 cells/L with active acute hemorrhage may warrant a platelet transfusion and fresh frozen plasma in an attempt to replace lost clotting factors.

The coagulopathy could be a marker of advanced liver disease. Prolongation of the PT based on an INR of more than 1.5 may indicate moderate liver impairment. A fibrinogen level of less than 100 mg/dL also indicates advanced liver disease with extremely poor synthetic function.

Endoscopy. The development of endoscopy has provided clinicians with the ability for diagnostic and therapeutic approaches to bleeding from the GI tract. Endoscopic examination of the upper GI tract provides useful information regarding the source and site of bleeding (fig. 5).

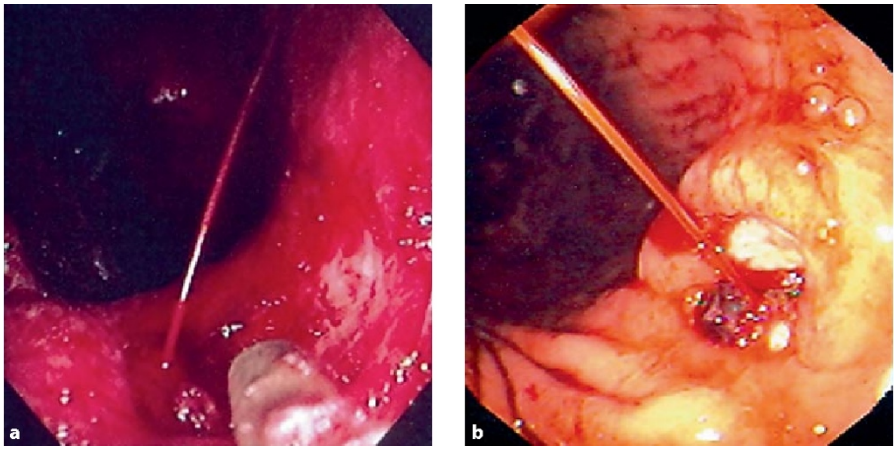

Fig. 5. Esophagogastroduodenoscopy. *Spurting ulcer. A – active bleeding from a large ulcer; B – active bleeding from a benign-appearing gastric ulcer.*

Endoscopic findings and their incidence rate in patients with UGIB include the following:

- Duodenal ulcer: 24.3%.
- Gastric erosion: 23.4%.
- Gastric ulcer: 21.3%.
- Esophageal varices: 10.3%.
- Mallory-Weiss tear: 7.2%.
- Esophagitis: 6.3%.
- Duodenitis: 5.8%.
- Neoplasm: 2.9%.
- Stomal (marginal) ulcer: 1.8%.
- Esophageal ulcer: 1.7%.
- Other/miscellaneous: 6.8%.

Endoscopy should be performed immediately after endotracheal intubation (if indicated), hemodynamic stabilization, and adequate monitoring in an intensive

care unit setting have been achieved. Endoscopy typically takes place within 24 hours. Early studies have shown that emergent endoscopy, within 12 hours or less from presentation, may reveal a higher-risk stigmata of bleeding on endoscopy and require therapeutic intervention.

When possible, it is important to take *biopsy samples* to test for *H. pylori* at the initial endoscopy procedure.

Although not a widely available or commonly employed modality in the setting of acute UGIB, *capsule endoscopy* may identify low-risk lesions in UGIB, potentially allowing a subset of patients to be safely treated as outpatients.

The **Forrest Classification** should be used to characterize all peptic ulcers, as it provides prognostic information on the need for endoscopic therapeutic intervention, the risk of rebleeding and death (tab. 3, fig. 6).

Table 3.

Forrest Classification

Stage	Characteristics	Re-bleeding risk
I – Active bleeding		
Ib	Spurting bleeding	60-100 %
Ib	Oozing bleeding	50-60 %
II – Signs of recent bleeding		
IIa	Non-bleeding visible vessel	40-50 %
IIb	Adherent clot on lesion	20-30 %
IIc	Hematin-covered flat spot (lesion)	5-10 %
III – Lesion without bleeding		
III	No signs of bleeding – clean base ulcer	< 5 %

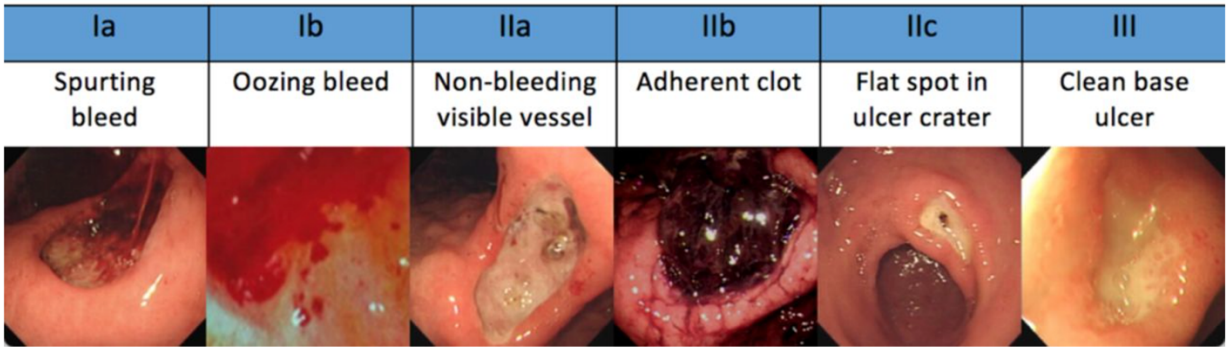

Fig. 6. Forrest classification of bleeding.

Nasogastric lavage may confirm recent upper GI bleeding (coffee ground appearance), possible active bleeding (red blood in the aspirate that does not clear), or a lack of blood in the stomach (active bleeding less likely but does not exclude an upper GI lesion).

A nasogastric tube is an important diagnostic tool, and tube placement can reduce the patient's need to vomit. Placement for diagnostic purposes is not contraindicated in patients with possible esophageal varices.

The characteristics of the nasogastric lavage fluid (eg, red, coffee grounds, clear) and the stool (eg, red, black, brown) can indicate the severity of the hemorrhage. However, a clear or bile-stained nasogastric aspirate may be seen in up to 15-18% of patients with an upper GI source.

The standard small 16-18-gauge nasogastric tube typically used for aspiration is not likely to effectively clear clots from the stomach. A large-bore orogastric tube is more likely to be successful in clearing the stomach, but the use of a large-bore orogastric tube is difficult and uncomfortable for patients and cannot be recommended routinely.

Imaging studies.

Chest radiography. If clinical suspicion is high for aspiration pneumonia, effusion, or esophageal perforation, order chest radiographs to rule out these conditions. Obtain abdominal scout and upright films to exclude a perforated viscus and ileus.

Barium contrast studies are not usually helpful in cases of suspected upper GI bleeding and can make endoscopic procedures more difficult (ie, white barium obscuring the view) and dangerous (ie, risk of aspiration).

Computed tomography (CT) scanning and **ultrasonography (US)** may be indicated for the evaluation of liver disease for cirrhosis, cholecystitis with hemorrhage, pancreatitis with pseudocyst and hemorrhage, aortoenteric fistula, and other unusual causes of UGIB. CT scanning is particularly useful for localizing obscure UGIB and for evaluating a patient with UGIB and a history of aortic reconstruction or pancreaticobiliary procedure.

CT scanning is useful in the diagnosis of aortoenteric fistula because images may reveal thickened bowel, perigraft fluid collection, extraluminal gas, or inflammatory changes in the area of the duodenum and the aortic graft. But small pseudoaneurysms, small bowel tumors, and small biliary tumors can easily be missed by CT.

CT angiography (CTA) holds promise as an initial test for acute GI bleeding because of its ubiquity, rapid performance, and potential to offer diagnostic information for management guidance (fig. 6).

CTA has the potential advantage of precisely localizing the source of GI bleeding, and to diagnose underlying pathology that may be the cause of bleeding to direct future management. Moreover, CTA can often identify causes of GI bleeding outside the GI tract (eg, hemobilia). In addition, CTA can not only define

the underlying vascular anatomy before patients undergo transcatheter angiography embolization but also identify any anatomic variants that may affect management.

Angiography is a minimally invasive method; it often allows precise localization of bleeding and enables the use of therapeutic options, which include *embolization* or *vasopressin infusion* (fig. 7).

A hemorrhage rate of 0.5-1.0 mL/min is required before it can be visualized with angiography. The detection of bleeding may be enhanced with carbon dioxide as an arterial contrast agent because of the low viscosity of the gas.

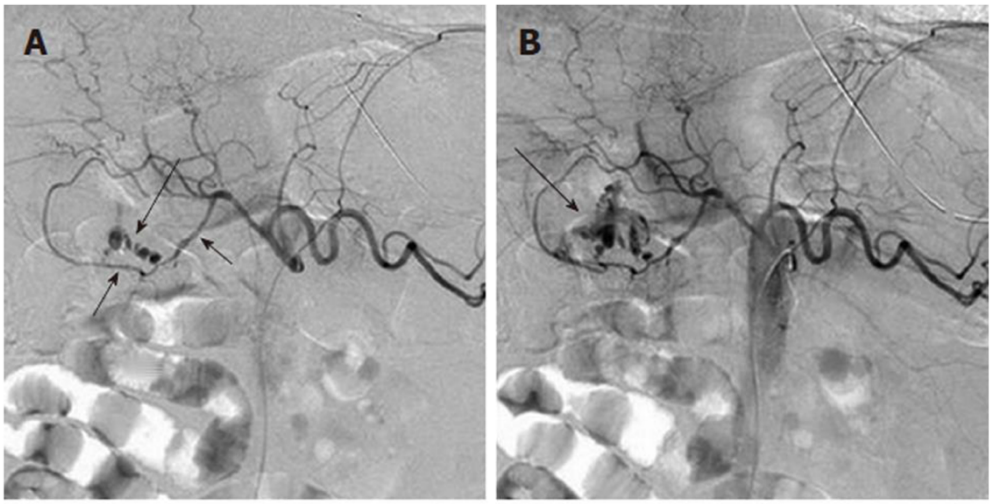

Fig. 7. Angiography of duodenal hemorrhage. *A – celiac digital subtraction angiography arteriogram obtained in a patient with copious bleeding seen endoscopically in the duodenum shows focal contrast extravasation (black arrow) arising from the gastroduodenal artery; B – an image slightly later in the arterial phase shows increasing extravasation (black arrow).*

Differential diagnosis

Diagnostic considerations. UGIB can be categorized on the basis of the following anatomic and pathophysiologic factors: ulcerative, vascular, traumatic, iatrogenic, tumors, and portal hypertension. Certain *risk factors* or *comorbidities*, such as the following, may help refine the differential diagnosis:

- a previous history of acid-peptic disease or UGIB;
- underlying cardiovascular or cerebrovascular disease (eg, valvular heart disease) or taking antithrombotic agents;
- known advanced liver disease, with possible underlying cirrhosis;
- severe systemic illness, with possible disseminated intravascular coagulation;
- having known bleeding dyscrasias, chronic kidney disease, or von Willebrand disease, which may predispose to vascular ectasias.

In individuals known to have an abdominal aortic aneurysm or an aortic graft, clinicians should strongly consider acute GI bleeding secondary to an aortoenteric fistula.

Individuals who have had previous upper GI surgery, such as Roux-en-Y gastric bypass or a Whipple procedure, are at risk for bleeding due to anastomotic or marginal ulceration; these patients should receive special consideration due to anatomic challenges to diagnosis and management.

Ulcer disease related to the Zollinger-Ellison syndrome, in which there is over-secretion of gastrin due to a gastrinoma, very rarely causes of UGIB, with an annual incidence of 0.5 to 2 per million population.

In addition to the disorders listed in the differentials section below, diagnostic consideration should be given to the following conditions in patients with symptoms of UGIB:

- Esophageal or gastric varices.
- Mallory-Weiss tear.
- Malignant neoplasm.
- Stress-related mucosal disease/stress ulcer.
- Dieulafoy lesion.
- Hemobilia.
- Pancreatic pseudoaneurysm.
- Aortoenteric fistula.
- Benign gastric tumors.
- Portal hypertensive gastropathy.
- Disseminated intravascular coagulation.
- Syncope.
- von Willebrand disease.
- Zollinger-Ellison syndrome.

Differential diagnoses:

- Abdominal aortic aneurysm.
- Acute gastritis.
- Barret's esophagus.
- Esophageal cancer.
- Esophagitis.
- Gastric cancer.
- Gastric outlet obstruction.
- Gastric ulcer.
- Gastrinoma.

Treatment

The goal of **medical therapy** in UGIB is to correct shock and coagulation abnormalities and to stabilize the patient so that further evaluation and treatment can proceed. In addition to *intravenous (IV) fluids*, patients may need transfusion of *packed red blood cells.*

High doses of *proton pump inhibitors (PPIs)* may reduce the need for endoscopic therapy. The relative efficacy of PPIs may be due to their superior ability to maintain a gastric pH at a level above 6.0, thereby protecting an ulcer clot from fibrinolysis.

Blood transfusions should be given to target a hematocrit above 20%, with a hematocrit above 30% targeted in high-risk patients, such as the elderly and patients with coronary artery disease. There is no evidence that higher targets for hematocrit goals should be sought as that higher targets can even be deleterious.

Platelet transfusion should be administered to patients with active bleeding and a platelet count less than 50×10^9 cells/l. The decision to stop or reverse anticoagulation should weigh the risks of thromboembolism against ongoing bleeding. Antiplatelet agents would likely be continued in patients with recent (< 3 months) coronary ischemia or drug-eluting coronary stent placement.

Octreotide, a somatostatin analog, is a medication used when variceal bleeding is suspected. It is given as an intravenous bolus of 20 mcg to 50 mcg, followed by a continuous infusion at a rate of 25 mcg to 50 mcg per hour.

Administration of *antibiotic drugs* (ie, ceftriaxone) to patients with cirrhosis presenting with acute UGIB regardless of the underlying etiology is associated with improved survival and decreased rebleeding.

The use of *NSAIDs* should be discontinued.

Resuscitation of a *hemodynamically unstable patient* begins with assessing and addressing the "ABCs" (ie, airway, breathing, circulation) of initial management.

Patients presenting with severe blood loss and hemorrhagic shock present with mental status changes and confusion. In these cases, patients are at an increased risk for aspiration, which is a potentially avoidable complication that can significantly affect morbidity and mortality, patients should be electively intubated in a controlled setting.

Intravenous access must be obtained. Bilateral, 16-gauge (minimum), upper extremity, peripheral intravenous catheters are adequate for volume resuscitative efforts. Foley catheter placement is helpful to allow a continuous evaluation of the urinary output as a guide to renal perfusion.

Patients with severe coexisting medical illnesses, such as cardiovascular and pulmonary diseases, may require pulmonary artery catheter insertion to closely monitor hemodynamic cardiac performance profiles during the early resuscitative phase.

Once the maneuvers to resuscitate are underway, it is often very helpful to insert a *nasogastric tube* and perform an aspirate and lavage procedure. This should be the first procedure performed to determine whether the GI bleeding is emanating from above or below the ligament of Treitz. If the stomach contains bile but no blood, UGIB is less likely. If the aspirate reveals clear gastric fluid, a duodenal site of bleeding may still be possible.

Primary surgical intervention should be considered in patients with a perforated viscus (eg, from perforated duodenal ulcer, perforated gastric ulcer, or Boerhaave syndrome). Emergency surgery in UBIG typically entails oversewing the bleeding vessel in the stomach or duodenum (usually preoperatively identified by endoscopy), vagotomy with pyloroplasty, or partial gastrectomy. Angiographic obliteration of the bleeding vessel is often considered a favorable modality in patients who are poor surgical candidates.

Contraindications to upper endoscopy include an uncooperative or obtunded patient, severe cardiac decompensation, acute myocardial infarction (unless active, life-threatening hemorrhage is present), and perforated viscus (eg, esophagus, stomach, intestine).

Hypotension may be exacerbated by sedation; therefore, patients who are clinically unstable should be carefully sedated. Continuous monitoring in the ICU is warranted and monitored anesthesia care by an anesthesia provider may improve the safety of endoscopy.

Patients with massive bleeding should be considered for intubation to reduce the increased risk of aspiration. Such patients should be treated in an intensive care setting. Ideally, the patient should be stabilized prior to endoscopy and abnormalities in coagulation should be corrected.

The UGIB *treatment algorithm* is described on fig. 8.

Therapeutic endoscopy. For several decades, endoscopy has been the primary method of evaluating and managing UGIB. Early endoscopic hemostatic therapy significantly reduces the rates of recurrent bleeding, the need for emergent surgery, and mortality in patients with acute nonvariceal UGIB.

Aside from ulcer hemorrhaging, other causes of GI bleeding, including mucosal tears in the esophagus or upper stomach due to vomiting (Mallory-Weiss tears), venous blebs, and vascular ectasias, can also be treated with endoscopic coagulation.

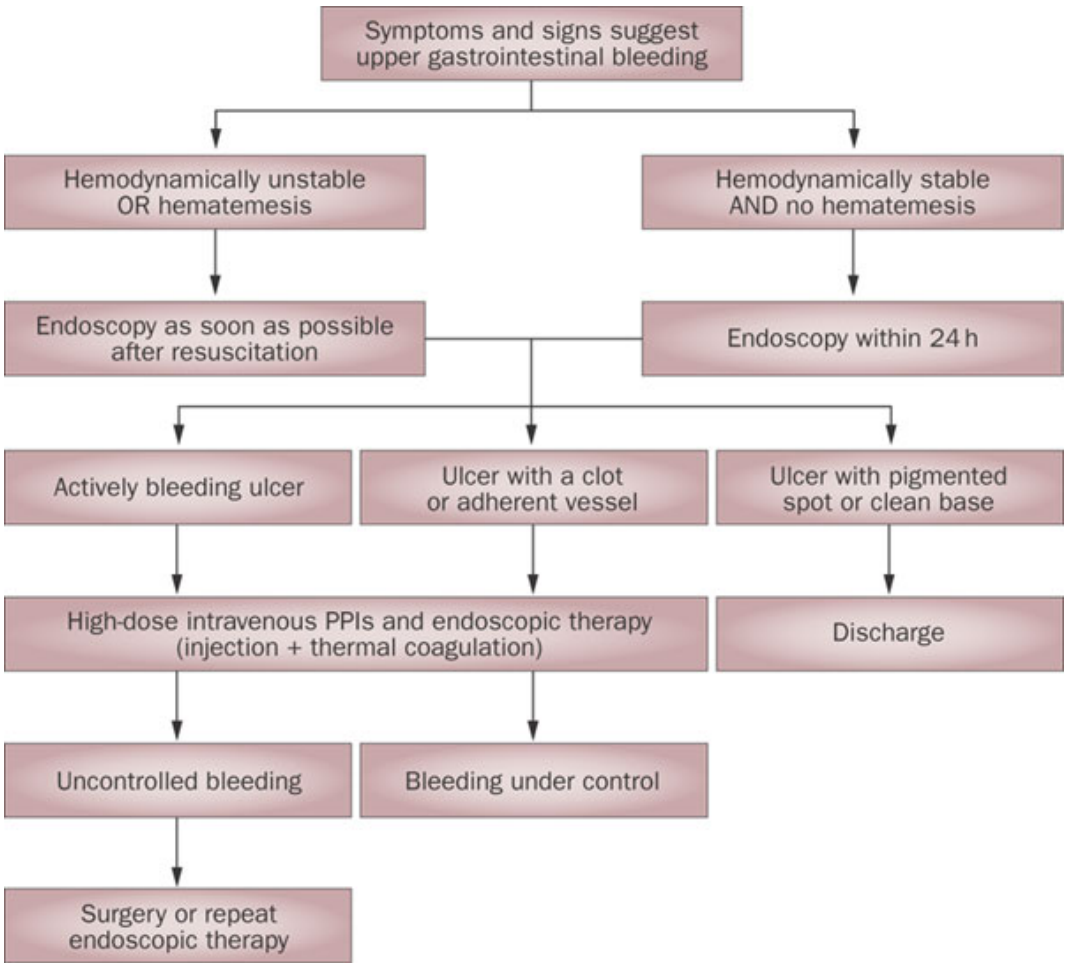

Fig. 8. Clinical algorithm for the management of upper GI bleeding.

The bleeding from gastric cancers and ulcers in leiomyomas does not usually respond to endoscopic therapy; surgical or radiologic intervention is needed.

The patient should undergo upper endoscopy prior to any operative intervention in order to diagnose and localize the bleeding site. Most patients (85-90%) respond to endoscopic therapy.

Endoscopic techniques. There are several currently widely accepted hemostatic treatment options. They inclide the following:

- Injection of epinephrine or sclerosants.
- Bipolar electrocoagulation.
- Band ligation.
- Heater probe coagulation.
- Constant probe pressure tamponade.
- Bipolar/soft coagulation hemostatic forceps.
- Argon plasma coagulation (APC).
- Laser photocoagulation.
- Rubber band ligation.

- Application of hemostatic materials, including biologic glue and tissue adhesives.
- Application of hemoclips/endoclips or over-the-scope clips.
- Application of hemostatic powder/spray.
- Doppler ultrasonographic assessment, pre- and postendotherapy.

Treatment using a combination of endoscopic therapies has become more common. For example, injection therapy can be performed prior to endoscopic placement of hemoclips.

Bipolar electrocoagulation. The bipolar probe consists of alternating bands of electrodes producing an electrical field that heats the mucosa and the vessel. The electrodes are coated with gold to reduce adhesiveness. The probes are stiff in order to allow adequate pressure to be applied to the vessel to appose the walls and thus produce coaptive coagulation when the electrical-field energy is transmitted (fig. 9). Careful technique is required to heat-seal the perforated vessel.

Injection therapy. Injection therapy involves the use of several different solutions injected into and around the bleeding lesion (fig. 9). Solutions available for injection include epinephrine, sclerosants, and various clot-producing materials, such as fibrin and cyanoacrylate glues.

The epinephrine used for injection is diluted (1:10,000). The hemostatic effect of epinephrine is due to induced vessel vasoconstriction and subsequent platelet aggregation and to the tamponade effect produced by injecting the volume of drug into the tissue surrounding the bleeding lesion. Although the epinephrine administered in injection therapy is absorbed into the systemic circulation, this does not appear to have any adverse effects on the hemodynamic status. Injecting a volume of sterile isotonic sodium chloride solution and providing a tamponade effect also leads to hemostasis, although not as effectively as does epinephrine.

The sclerosant solutions such as ethanol, polidocanol, and sodium tetradecyl sulfate are not frequently administered. The sclerosants create hemostasis by inducing thrombosis, tissue necrosis, and inflammation at the site of injection. When large volumes are injected, the area of tissue necrosis can produce an increased risk of local complications, such as perforation.

Laser therapy. Laser phototherapy is a noncontact thermal method that uses an Nd:YAG (neodymium-doped yttrium aluminium garnet) laser to create hemostasis by generating heat and direct vessel coagulation (fig. 9). It is not as effective as coaptive coagulation, because it lacks the use of compression to create a tamponade effect. An additional deterrent to its use is expense.

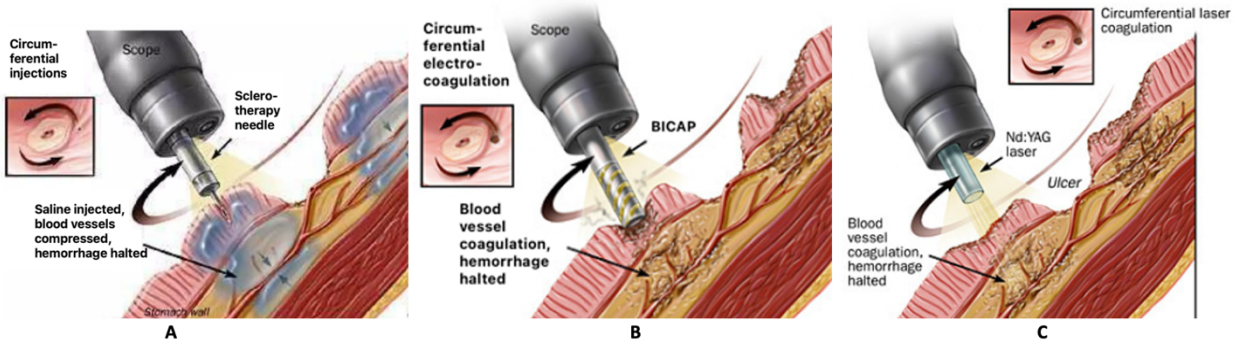

Fig. 9. Methods of endoscopic hemostasis. *A – injection therapy; B – electrocoagulation; C – laser coagulation.*

Hemostatic clips and endoclips. With careful placement of the clip, closing the defect in the vessel is possible (fig. 10). Often, depending on the lesion and progress to affect hemostasis, multiple clips are applied. Typically, they become detached and pass from the GI tract within 2 weeks. Hemostatic clips are considered MRI-conditional because they are metallic, and they can serve as radiopaque markers to direct the interventional radiologist during angiography to the relevant area if endoscopy fails to achieve adequate hemostasis.

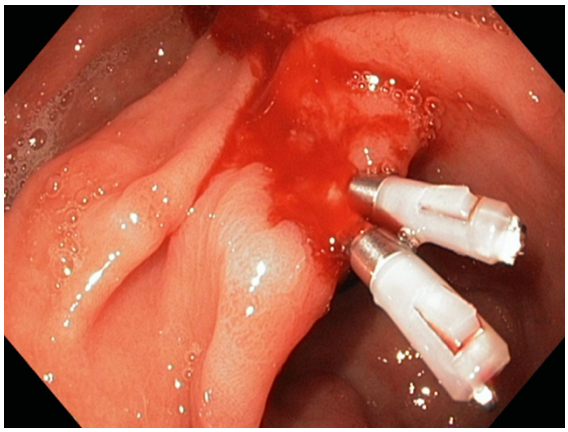

Fig. 10. Hemostatic clips.

There are some clinical settings in which endoclips may be preferred over other hemostatic methods. These include the treatment of ulcers in patients who are coagulopathic or who require ongoing anticoagulation; in such patients, electrocoagulation will increase the size, depth, and healing time of treated lesions. Endoclips may also be preferable in the retreatment of lesions that rebleed after initial thermal hemostasis.

Ulcers on the lesser curvature, the posterior duodenum, or the cardia increase the difficulty of clip deployment and clip failure rates.

Larger endoclips, such as the over-the-scope clips, have advantages over smaller hemoclips for the hemostasis of chronic ulcers, fibrotic lesions, and the

closure of larger lesions. However, the use of the over-the-scope clips can be cumbersome in upper GI bleeding and, therefore, these clips have been more commonly used in refractory bleeding or as a salvage maneuver, but there are data showing efficacy of these clips as a primary modality.

Argon plasma coagulation. APC is a technique in which a stream of electrons flows along a stream of argon gas. The coagulation is similar to monopolar cautery, with the current flow going from a point of high current density (the point of contact of the gas with the mucosa) to an area of low current density (the conductive pad on the patient's body). The current flows through the body in an erratic path to the pad.

This technique is not effective for visible vessels larger than 1 mm. Small, superficial vessels such as arteriovenous malformations, telangiectasias, and particularly gastric antral vascular ectasia respond well to treatment by APC.

Hemostatic powder/spray. These agents have primarily been used as a second-line option when other endoscopic hemostasis techniques have failed. The hemosprays have the advantage of excellent initial hemostasis, but they can also obscure the endoscopic views of the underlying lesion.

Although some trials indicate a high technical success of the hemospray for treating diffuse or refractory UGIB, there is a high rebleeding risk and further investigation might be needed.

Doppler ultrasonographic probes. There is increasing use of Doppler ultrasonographic probe-guided lesion assessment to detect significant arterial flow in the vessel at the ulcer base, thanks to the development of relatively low cost, easy-to-use Doppler units and disposable endoscopic probes.

Endoscopic treatment decisions. The choice of treatment modality is influenced by the size of the vessel. Heater probe and bipolar probe are effective for vessels as large as 2 mm in diameter. Other techniques (eg, clips, band ligation) or a combination of techniques are needed for larger vessels or vessels that are not approachable by the heater probe or bipolar probe. Surgical intervention should be considered when dealing with vessels larger than 2 mm in diameter, discounting an enlargement due to the development of a pseudoaneurysm.

Second-look endoscopy. A second attempt at endoscopic control is warranted if the initial endoscopy fails to control the bleeding. Owing to the relatively high rebleeding rate associated with ulcers, some clinicians advocate scheduled second-look endoscopy, with the intent of identifying and proactively managing persistent or recurrent bleeding. Good visualization is important. The uncleared fundal pool may obscure an ulcer, mucosal tear, gastric varices, portal gastropathy, or tumor (eg, leiomyoma, adenocarcinoma, lymphoma).

Bleeding peptic ulcer treatment. Upper GI endoscopy is the most effective diagnostic tool for peptic ulcer disease and has become the method of choice for controlling active ulcer hemorrhage. Failure of endoscopy to maintain hemostasis is one of the indications to initiate surgical intervention, especially in high-risk patients. Regardless of the endoscopic therapy, however, 10-12% of patients with acute ulcerous hemorrhage require an operation as the definitive procedure to control the bleeding ulcer. In most circumstances, the operation is performed emergently, and the associated mortality rate is as high as 15-25%.

Medical therapy used in conjunction with endoscopy involves PPI administration. PPIs decrease the rebleeding rates in patients with bleeding ulcers associated with an overlying clot or visible, nonbleeding vessel in the base of the ulcer. Consider transcatheter angiographic embolization in patients who are poor surgical candidates.

Surgical treatment. If two attempts at endoscopic control of the bleeding vessel are unsuccessful, avoid further attempts and pursue surgical intervention. The *indications for surgery* in patients with bleeding peptic ulcers are as follows:

- Severe, life-threatening hemorrhage not responsive to resuscitative efforts.
- Failure of medical therapy and endoscopic hemostasis with persistent recurrent bleeding.
- A coexisting reason for surgery, (perforation, obstruction or malignancy).
- Prolonged bleeding, with loss of 50% or more of the patient's blood volume.
- A second hospitalization for peptic ulcer hemorrhage.

The operative treatment options for a bleeding *duodenal ulcer* include vagotomy, gastric resection, and drainage procedures. The three most common *operations* performed for a *bleeding duodenal ulcer* are as follows:

- Truncal vagotomy and pyloroplasty with suture ligation (fig. 11) of the bleeding ulcer.
- Truncal vagotomy and antrectomy with resection or suture ligation of the bleeding ulcer.
- Proximal (highly selective) gastric vagotomy with duodenostomy and suture ligation of the bleeding ulcer.

The purpose of the *vagotomy* is to divide the nerves to the acid-producing body and fundus of the stomach. This inhibits acid production that occurs during the cephalic phase of gastric secretion, thereby decreasing the risk for recurrent ulceration. A truncal vagotomy also has effects on distal gastric motor function. It

weakens distal gastric peristalsis, thus requiring the creation of a pyloroplasty to decrease the resistance to outflow from the stomach. Proximal vagotomy abolishes gastric receptive relaxation and impairs storage in the proximal stomach. As a result, a more rapid gastric emptying of liquids occurs.

Truncal vagotomy and suture ligation of a bleeding ulcer is a frequently used operation for treating upper GI bleeding in elderly patients with life-threatening hemorrhage and shock.

The diagnosis of *H. pylori* infection is important in the management of patients with a complicated bleeding peptic ulcer. Many studies support the decision to manage the bleeding ulcer in conjunction with eradication of *H. pylori*.

Bleeding gastric ulcer treatment. The goals of surgery are to correct the underlying emergent problem, prevent recurrent bleeding or ulceration, and exclude malignancy. A bleeding gastric ulcer is most commonly managed by a distal gastrectomy that includes the ulcer, with a gastroduodenostomy or a gastrojejunostomy reconstruction.

The *common operations* for the management of a *bleeding gastric ulcer* include:

- Truncal vagotomy and pyloroplasty with a wedge resection of the ulcer.
- Antrectomy with wedge excision of the proximal ulcer.
- Distal gastrectomy to include the ulcer, with or without truncal vagotomy.
- Wedge resection of the ulcer only.

Types of gastric ulcers. The choice of operation for a bleeding gastric ulcer depends on the location of the ulcer and the hemodynamic stability of the patient to withstand an operation.

Type 1 gastric ulcers are located on the lesser curvature of the stomach, at or near the incisura angularis. These ulcers are not associated with a hypersecretory acid state. Type 2 ulcers represent a combination of 2 ulcers that are associated with a hypersecretory acid state. The ulcer locations occur in the body of the stomach in the region of the incisura. The second ulcer occurs in the duodenum. Type 3 ulcers are prepyloric ulcers. They are associated with high acid output and are usually within 3 cm of the pylorus. Type 4 ulcers are located high on the lesser curvature of the stomach and are not associated with high acid output. Type 5 ulcers are related to the ingestion of NSAIDs or aspirin. These ulcers can occur anywhere in the stomach.

Surgical management according to ulcer type. A vagotomy is added to manage type 2 or type 3 gastric ulcers. Patients who are hemodynamically stable

but have intermittent bleeding requiring blood transfusions should undergo a truncal vagotomy and distal gastric resection to include the ulcer for types 1, 2, and 3 ulcers.

In patients who present with life-threatening hemorrhage and a type 1, 2, or 3 ulcer, biopsy and oversew or excision of the ulcer in combination with a truncal vagotomy and a drainage procedure should be considered (fig. 11).

Patients with type 4 ulcers usually present with hemorrhage. The left gastric artery should be ligated, and a biopsy should be performed on the ulcer. Then, the ulcer should be oversewn through a high gastrotomy.

Rebleeding rates for the procedures that keep the ulcer in situ range from 20% to 40%. Gastric bleeding in the immediate postoperative period from recurrent peptic ulcer disease is initially best managed by endoscopic or angiographic means. If reoperation is required, gastric resection is usually indicated, because a repeat vagotomy is not reliable and a more definitive solution is warranted.

Overwesing of a bleeding peptic ulcer (fig. 11).

Ligation of the gastroduodenal artery. Oversewing the ulcer begins with exposure of the gastroduodenal artery. It is situated near the pylorus, in a projection of the prominent prepyloric vein of Mayo, and is always deeper than expected. It is often mistaken for the right gastric artery, which courses more superficially. The gastroduodenal artery is confirmed by identifying its origin from the common hepatic artery, which is palpable as a pulsating cord over the upper border of the pancreas. After exposure of the gastroduodenal artery, an Overholt clamp is passed beneath it before ligation (fig. 11-1).

Ligation of the pancreaticoduodenal artery. The superior pancreaticoduodenal artery and the right gastroepiploic artery are exposed at the level of the caudal circumference of the pylorus. Both vessels are ligated under direct vision.

Gastroduodenotomy. A longitudinal incision is made between stay sutures in the anterior wall of the stomach or duodenum, depending on the location of the ulcer. For its most common location in the postpyloric duodenum, the incision is placed predominantly in the region of the proximal duodenum, with the pylorus being included in the cranial part of the incision (fig. 11-2).

Identification of the bleeding point. The bleeding site usually lies in the posterior wall, over the course of the gastroduodenal artery. If bleeding has ceased by the time of surgery, it should be reactivated by dabbing and manipulation to Identify the bleeding point (fig. 11-3).

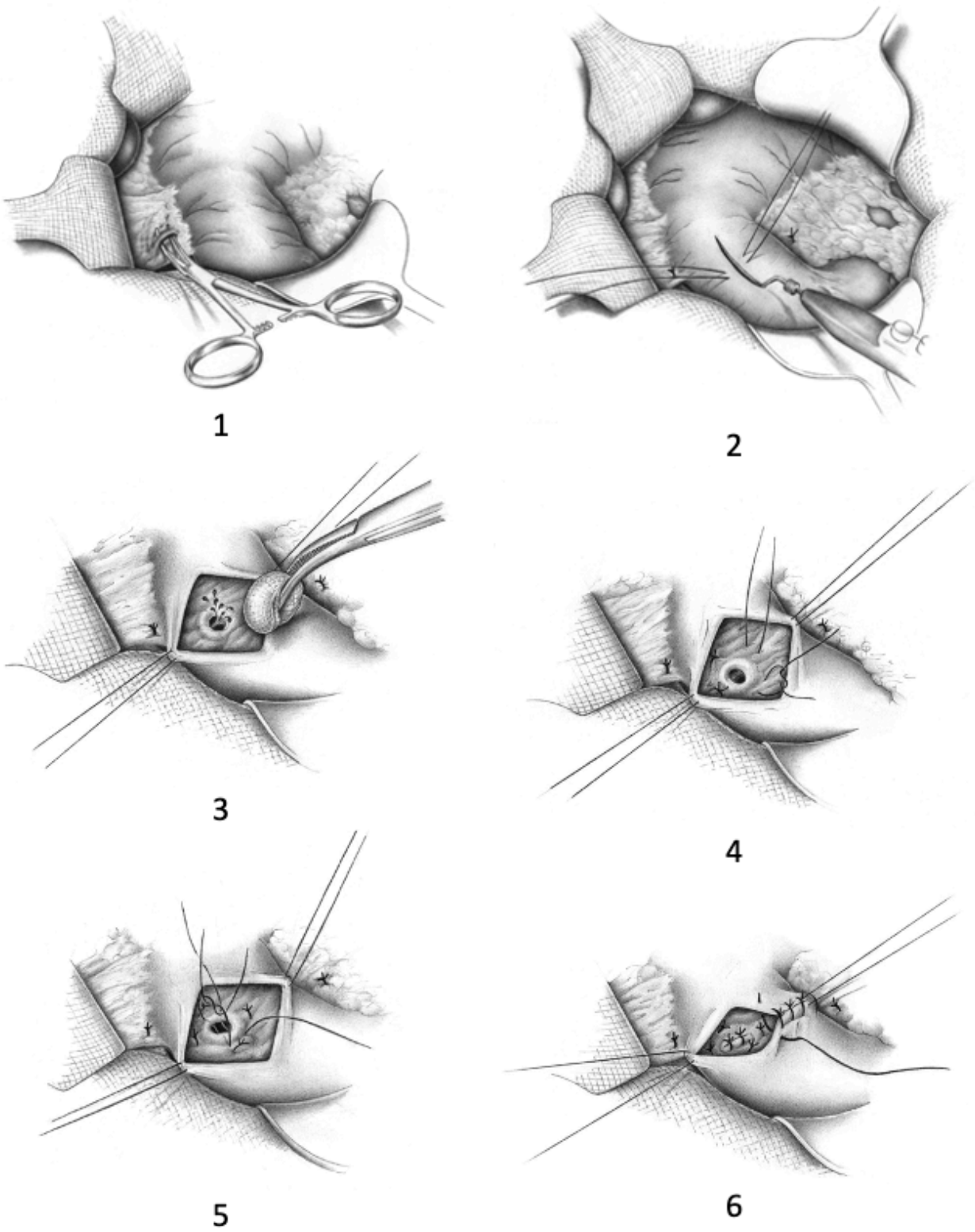

Fig. 11. Overwesing of a bleeding peptic ulcer.

Four-quadrant suture ligation. The bleeding point is transfixed with interrupted sutures, incorporate the gastroduodenal artery cranial and caudal to the ulcer. Additional branches from the stomach and duodenum are also suture ligated with interrupted stitches (3-0 PGA) in the two other quadrants (fig. 11-4).

Ulcer closure. Closure of the ulcer base is accomplished with two or three interrupted sutures to reduce the add-peptic exposure of the ulcer base (fig. 11-5).

Pyloroplasty. If the gastroduodenotomy has included the pylorus, then a Heineke-Mikulicz pyloroplasty should subsequently be performed. The longitudinal incision is closed transversely between the stay sutures. Longitudinal suture closure of a longitudinal incision is justifiable following gastrotomy. Individual cases will require a Kocher mobilization of the duodenum to achieve a tension-free suture line (fig. 11-6).

Stress ulcer treatment. Knowledge of the predisposing conditions for stress ulceration allows to identify patients at risk for developing stress ulceration and GI bleeding (respiratory failure with mechanical ventilation and coagulopathy being very prominent risk factors). Aggressive support of hemodynamic parameters ensures adequate mucosal blood flow.

Stress-related bleeding usually occurs 7-10 days after the initial insult. Initially, endoscopy is the most important diagnostic tool. The acute superficial erosions are multiple, begin in the fundus, and progress toward the antrum. 90% of patients stop bleeding with conservative medical therapy that includes gastric acid–controlling medications. PPIs are the drugs of choice for acid suppression in stress ulcer prophylaxis. Endoscopic hemostasis is attempted using traditional techniques, including electrocoagulation, APC, or injection therapy.

Surgical intervention becomes necessary if nonoperative therapy fails and blood loss continues. The goals of operative treatment are to control bleeding and to reduce recurrent bleeding and mortality. Simply oversewing an actively bleeding erosion is sometimes effective enough to control the bleeding. In the setting of life-threatening hemorrhage not amenable to endoscopic control, gastric resection with or without vagotomy with reconstruction may be necessary. The options are antrectomy and subtotal, near total, or total gastrectomy.

Managing the underlying insult causing the gastric stress ulcerations is also important. This involves supportive measures to maintain acceptable hemodynamic parameters, to provide adequate nutritional support in the critically ill patient, and to treat sepsis (if present).

Mallory-Weiss syndrome treatment. Distinguishing Mallory-Weiss syndrome from Boerhaave syndrome is critical. A Gastrografin swallow helps to confirm the presence of the perforation (Boerhaave syndrome) in most cases, and prompt surgical intervention is necessary to prevent mediastinitis and sepsis.

However, surgical intervention in Mallory-Weiss syndrome is required to achieve hemostasis in only 10% of cases. For patients in whom bleeding is visualized at endoscopy, the endoscopic treatment options are electrocoagulation, heater-probe application, hemoclips, epinephrine injection, or sclerotherapy.

Other available options are angiographic intra-arterial infusion of vasopressin and transcatheter embolization of branches of the left gastric artery using Gelfoam. Avoid the balloon tamponade technique using the Sengstaken-Blakemore tube in this particular circumstance, because this apparatus may extend the mucosal laceration into a transmural laceration with perforation.

Surgical intervention is indicated in patients with continued bleeding after failed attempts at nonoperative therapies – anterior gastrotomy and once the tear is localized, the bleeding is controlled by oversewing the lesion.

Dieulafoy lesion treatment. The initial endoscopic management of a Dieulafoy lesion can be highly successful (up to 96% of cases).

Contact thermal ablation with a heater probe is a very effective technique, with or without the combined use of epinephrine to slow or stop the bleeding prior to applying the heater probe. Argon plasma coagulation and endoclips have also been used successfully for hemostasis. Although surgical intervention may be required after failed endoscopic therapy, endoscopy is still an important adjunct for management, because a nonbleeding Dieulafoy lesion may be undetectable through a gastrotomy.

Angiodysplasia treatment. Because the lesions are small and superficial, endoscopic therapy is highly successful. Endoscopic treatments and devices used for hemostasis include argon plasma coagulation, contact heat probes, electrocoagulation, and injection therapy.

APC would be the treatment of choice when treating gastric antral vascular ectasia, as it allows the endoscopist to apply prompt and effective “painting” of the angiodysplastic lesions in the distal stomach.

When endoscopic techniques fail, surgical resection becomes necessary. When pangastric involvement is the source of bleeding, a total gastrectomy may be required; however, this is extremely rare. Available nonsurgical options include angiography with catheter-directed vasopressin.

Variceal bleeding (fig. 4) constitutes 70% of all upper GI bleeding episodes in patients with portal hypertension, and they result from esophageal varices, gastric varices or ectopic varices.

The *key points* of acute variceal bleeding management are: volume infusion, pharmacological and endoscopic control of hemorrhage and infection prophylaxis.

During active bleeding, the volume resuscitation should be undertaken promptly with the goal of restore blood pressure and perfusion, but it might be done carefully to avoid overload volume which could increase portal vein pressure and subsequently the risk of rebleeding.

Treat coagulopathy as necessary. Fresh frozen plasma may increase blood volume and increase rebleeding risk.

Monitor mental status. Avoid sedation, nephrotoxic drugs, and beta-blockers acutely.

Intravenous splanchnic vasoconstrictors. Three IV vasoconstrictors are recommended: terlipressin, somatostatin, or octreotide. IV octreotide to lower portal venous pressure as adjuvant to endoscopic management. IV bolus of 50 micrograms followed by a drip of 50 micrograms/hr. Terlipressin (alternative): 2 mg q4h IV for 24 to 48 hours, then 1 mg q4h.

Erythromycin 250 mg IV 30 to 120 minutes before endoscopy, than urgent *upper GI endoscopy* for diagnosis and treatment – variceal endoscopic ligation, sclerotherapy.

Variceal endoscopic band ligation (EBL) has become the treatment of choice for variceal bleeding (fig. 12). Endoscopic variceal ligation is employed as a treatment for patients with acute bleeding, as a primary preventive measure in high-risk patients, and as a secondary preventive measure for eradicating residual varices in those patients who have experienced their first bleeding episode.

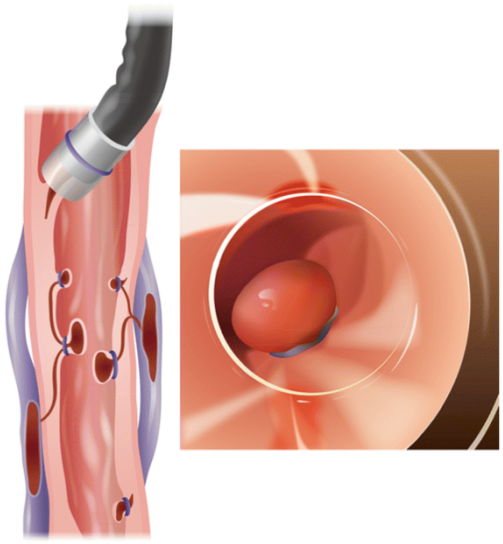

Fig. 12. Variceal endoscopic band ligation.

The equipment for EBL includes rubber rings loaded on a plastic cap attached to the tip of an endoscope. After the rubber bands are released over the esophageal varices, the ligated tissue sloughs off within days, forming shallow esophageal ulcers, which eventually decreases the size of the varices

Variceal band ligation is preferred to sclerotherapy for bleeding varices and for nonbleeding medium-to-large varices to decrease bleeding risk. Ligation has lower rates of rebleeding, fewer complications, more rapid cessation of bleeding and a higher rate of variceal eradication. Sclerotherapy may be considered in rare cases in which EBL is not technically feasible.

If endoscopic treatment fails, consider self-expanding esophageal metal stents or peroral placement of *Sengstaken-Blakemore-type* tube up to 24 hours to stabilize the patient for Transjugular intrahepatic portosystemic shunt (TIPS) – to decrease portal hypertension, subsequently decreasing pressure in esophageal varices.

Antibiotic prophylaxis has an important role in the treatment of digestive bleeding in cirrhotic patients and it should be initiated as soon as bleeding episode appears. Infection is identified in 25-50% of patients in their admission or during the length of stay for esophageal variceal bleeding, being commonly associated to spontaneous bacterial peritonitis, urinary tract infection and pneumonia.

Combined therapy with non-selective beta-blockers (NSBBs) (propranolol or nadolol) plus esophageal variceal band ligation is the first-line therapy in the prevention of re-bleeding.

UGIB treatment complications. Complications of endoscopic therapy include aspiration pneumonia and perforation (1% and 3% respectively). Bleeding can be caused by drilling into the vessel with contact thermal probes, by perforating the vessel with an injection, or by removing the clot with failure to coagulate the vessel.

Mortality in UGIB is most often from the patient's underlying comorbidities. Therefore, strategies to manage the bleeding episode and in preventing rebleeding need to include the management of the comorbidities.

Complications from emergency abdominal surgery include ileus, sepsis, poor wound healing, and myocardial infarction.

Prevention of UGIB. *H. pylori* eradication therapy should be given if *H. pylori* is present in the setting of any history of ulcer disease. Eradication of *H. pylori* has been demonstrated to reduce the risk of recurrent ulcers and, therefore, recurrent ulcer hemorrhages.

Avoid NSAIDs. If this is not possible, use the lowest dose and duration.

Proton-pump inhibitors should be used along with NSAIDs.

Multiple choice questions

1. Anatomical parts of the stomach are:
   A. Fundus, body, neck.
   B. Head, neck, body, tail.
   C. Left lobe, right lobe.
   D. Cardia, fundus, body, antrum, pylorus.
   E. Capsule, parenchyma, hilum.

2. Choose the most common cause of upper gastrointestinal bleeding:
   A. Gastric carcinoma.
   B. Mallory-Weiss tear.
   C. Esophagitis.
   D. Gastric / duodenal ulcer.
   E. Meckel's diverticulum.

3. According to Forrest Classification, Ia stage is:
   A. Spurting bleeding.
   B. Oozing bleed.
   C. Non-bleeding visible vessel.
   D. Adherent clot.
   E. Flat spot in ulcer crater.

4. Choose the therapeutic endoscopic methods of hemostasis for upper gastrointestinal bleeding:
   A. Bipolar coagulation.
   B. Argon plasma coagulation.
   C. Injection therapy.
   D. Hemostatic clips.
   E. All answers are correct.

5. Surgical treatment method of Mallory-Weiss syndrome:
   A. Distal gastric resection.
   B. Gastrotomy + oversewing the tear.
   C. Truncal vagotomy.
   D. Pyloroplasty.
   E. Gastro-enterostomy.

Clinical cases

1. A 64-year-old male patient complains of weakness, dizziness, vomiting of fresh blood. History of chronic alcohol abuse. On physical examination: pale skin, tachycardia 108 bpm, blood pressure 90/50 mm Hg, ascites, jaundiced skin, varicose veins of the abdominal wall. Blood test: hemoglobin – 88 g/l, RBC – 2.8×10^{12}/l, WBC 5.2×10^{9}/l, PLT 250×10^{9}/l, total bilirubin level – 244 mcol/l. Which instrumental diagnostic method should be carried out first of all?

A. Upper gastrointestinal endoscopy.
B. X-ray of abdomen.
C. CT of abdomen.
D. Ultrasonography.
E. Colonoscopy.

2. Patient, 48 years old, was admitted to the surgical department complaining of "coffee ground" vomiting, black stool and a significant general weakness. Physical examination: pale skin, blood pressure 80/50 mm Hg, pulse 112 bpm. What is the most likely diagnosis?

A. Pulmonary bleeding.
B. Myocardial infarction.
C. Upper gastrointestinal bleeding.
D. Acute peritonitis.
E. Pulmonary embolism.

3. Female patient, 56 y.o. is presented with clinical signs of severe upper gastrointestinal bleeding. History: liver cirrhosis due to chronic alcoholic hepatitis, no history of peptic ulcer disease. Physical examination: jaundice, ascites. What is the most likely source of bleeding?

A. Gastric ulcer.
B. Esophageal varices.
C. Duodenal ulcer.
D. Mucosal tear.
E. Gastric tumor.

MCQ answers						**Clinical cases answers**			
Question	1	2	3	4	5	Case	1	2	3
Answer	D	D	A	E	B	Answer	A	C	B

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