Upper Gastrointestinal Bleeding

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Description of the Problem

Bleeding from the GI tract is a common, life-threatening condition, with more than 500,000 hospital discharges in the United States for gastrointestinal bleeding [1]. The mortality of UGIB is between 2.2% and 10% [2, 3]. Elderly populations are disproportionally affected: patients >65 years and older account for 65% of hospitalizations for GIB, and only 10% of hospitalized patients are younger than 45 years of age [1]. Patients admitted with UGIB utilize significant hospital resources as 20–30% of hospitalized patients require six or more units of blood, but surgical intervention is required in only 4–15% of patients. However, when patients require an operation, 69% of operations are done emergently [2, 4, 5].

Upper gastrointestinal bleeding (UGIB) (Table 7.1) has various causes and is defined as any bleeding originating proximal to the ligament of Treitz which is the most common site of bleeding (45%), with lower gastrointestinal bleeding (24%) being less common and the source being unspecified in 31% [1]. The incidence of UGIB appears to be decreasing, with an estimated annual incidence of UGIB reported as 108/100,000 hospitalizations per population in 1995 compared to 78/100,000 in 2015 [6, 7].

The care of patients with upper GI bleeding is multidisciplinary and requires a team approach. Teams involved include gastroenterologists, emergency medicine physicians, interventional radiologist, critical care physicians, and surgeons. Acute care surgeons have the unique potential to manage these patients from beginning to end and may be involved at any stage of the disease process.

Approaching the UGIB Patient

History and Physical Exam

Upon presentation, vital signs should be evaluated and simultaneous resuscitation initiated in the case of instability. A quick history should be taken with special focus on the events surrounding the current UGIB, prior episodes, comorbid conditions, medications, and past surgical history. This approach will focus the diagnostic strategy and may guide initial therapy. A history of epigastric postprandial abdominal pain occurring between half an hour and 3.5 h after a meal, or pain which wakes up the patient at night, or pain relieved by food, vomiting, or antacids is suggestive of peptic ulcer disease. A history of liver disease would suggest a likely variceal bleeding source. Elements in the past surgical

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Table 7.1 Classification of UGIB based on pathophysiology and anatomy

Variceal	Non-variceal
Bleeding varices Portal hypertensive gastropathy	
	Ulcerative Gastric ulcer Duodenal ulcer Gastroduodenal Cameron lesions Stress-induced ulcer Marginal ulcer
	Erosive (caustic, infectious, peptic, iatrogenic) Gastritis Duodenitis Gastroduodenitis
	Tumors Adenocarcinoma Squamous cell carcinoma GIST Metastasis Lymphoma Benign
	Iatrogenic/traumatic/foreign body
	Vascular Arteriovascular malformation Dieulafoy's lesions
	Miscellaneous Hemobilia Hemosuccus pancreaticus Aortoenteric fistula

history such as placement of aortic graft, recent hepatic procedures, trauma, and pancreatitis, among others, will provide valuable clues as well. Medication list should stress the use of anticoagulants, antiplatelet agents, beta-blockers, calcium channel blockers, and other vasoactive medications.

The assessment should be quick and borrowed from the Advanced Trauma Life Support "ABCDE" principles. The safety of the patient's airway should be ensured. Vomiting patients and those with altered mental status should be intubated to secure the airway and expedite upcoming endoscopic evaluation. Chest roentgenogram (CXR) should be obtained if aspiration is of concern. Oxygen should be supplemented to guarantee normal oxygen saturation and to optimize oxygen-carrying capacity in the setting of acute blood loss anemia. Evaluation for shock includes baseline vital signs, orthostatic determination of postural hypotension, pallor, and mental status changes. Reliable IV access should be obtained with at least two large-bore IVs. Initial laboratory tests include complete blood counts, coagulation studies, liver function tests, and type and crossmatch to have blood available if needed. Most importantly, infusion of warm fluids should be started and the response to volume resuscitation monitored. "Responders" will stabilize after the initial bolus of fluid. "Transient responders" will decompensate once the infusion is completed, while "non-responders" fail to respond all together.

The patient should be exposed and examined for peritonitis, stigmata of liver disease, abdominal distension, and melena. Rectal examination should be done to look for easily accessible pathology such as hemorrhoids and rectal masses. Foley catheter should be placed for monitoring. Temperature should be checked and hypothermia anticipated especially in the setting of massive transfusion.

Nasogastric lavage can help rule out an UGIB source as bilious aspirates in the absence of blood significantly decrease the likelihood of UGIB. Coffee-ground aspirates will suggest subacute bleeding, while bright red blood suggests ongoing hemorrhage, particularly when that blood fails to clear with lavage.

GI bleeding patients should be treated at or transferred to a facility with critical care capability and sufficient resources to support massive transfusion protocol, advanced interventional endoscopy, and a surgeon capable of managing UGIB. On presentation, surgical consultation should be obtained even though the vast majority of patients stop bleeding after resuscitation and medical management. This ensures that the surgical team learns about the patient, follows the response to resuscitation, and tracks the results of endoscopic therapy along with the admitting team.

Resuscitation

Once the fact of UGIB is established, high-dose proton pump inhibitors (PPI) like omeprazole

should be administered as an intravenous bolus of 80 mg followed by a continuous infusion at 8 mg/h. High-dose PPI administration is cost-effective and decreases the incidence of high stigmata of bleeding at endoscopy as well as the need for endoscopic hemostasis [8] albeit without effect on rebleeding, surgery, or mortality rates [9]. However, high-dose intravenous PPI after endoscopic therapy decreases the rate of rebleeding. Therefore, double-dose oral PPI for 11 days following 72 h of intravenous PPI is recommended for high-risk patients [10].

Volume resuscitation should be initiated as soon as IV access is obtained. This can be achieved using crystalloids and colloids initially while waiting for blood products, or blood products can be started immediately if they are available. In hemorrhagic shock, multiple endpoints are pursued to assess adequate resuscitation and the patient's overall response to therapy. Hemodynamic parameters such as central venous pressure (CVP), mean arterial pressure (MAP), and cardiac output/index along with lactate, central venous oxygen saturation ($ScvO_2$), urine output, and normalization of coagulation studies should be considered. The goals of resuscitation need not be the restoration of normal blood pressure. Until definitive hemorrhage control, principles of "hypotensive resuscitation" should be followed, allowing mean arterial pressures as low as 50 mmHg as long as there is evidence of adequate end-organ perfusion. This strategy has been shown to be safe and may reduce the risk of postoperative coagulopathy and death in trauma patients with hemorrhagic shock [11].

Unstable patients, transient responders, nonresponders, symptomatic patients, or patients with massive hemorrhage should receive blood transfusion as soon as possible. For that purpose, crossmatched, type-specific, or type O packed red blood cells should be used in decreasing order of preference based on availability from the blood bank. Exsanguinating patients should receive type O PRBC initially and until crossmatched products are available. Any existing or developing coagulopathy should be aggressively treated via infusion of plasma, platelets, and factor concentrates as needed.

Stable UGIB in intermediate- to low-risk patients, in whom intravascular volume has been restored, will benefit from a restrictive transfusion strategy where it is recommended to transfuse for hgb < 7 [12]. This strategy has been validated among critical care patients across the board, and it was shown in a randomized controlled trial that in UGIB, patients on the restrictive transfusion strategy had a higher 6-week survival, lower adverse event, and lower rebleeding rates as opposed to patients in a more liberal transfusion strategy. Early aggressive resuscitation decreased organ failure and mortality. The abovementioned benefits were shown in both NVUGIB and VUGIB [13] (Fig. 7.1).

If the UGIB is related to portal hypertension, it is important not to over-resuscitate. Medical therapy should be instituted along with judicious resuscitation. Specifically, somatostatin or its analog (octreotide) should be started for portal pressure reduction through decrease of splanchnic blood flow.

In patients with VUGIB, besides the multisystem organ failure resulting from acute blood loss, encephalopathy, hepatorenal syndrome, and systemic infections contribute to mortality. Therefore, prophylactic antibiotics should be given because cirrhotic patients have high rate of infections from the GI tract due to bacterial translocation. Antibiotic prophylaxis in VUGIB improves survival and decreases infectious complications [14]. During resuscitation, patients with VUGIB often will require endotracheal intubation to protect airways in the setting of vomiting, encephalopathy or hemodynamic instability.

Laboratory Studies

Every patient should receive a complete metabolic panel, a complete blood count, coagulation studies and a type and crossmatch. Unstable patients should have their hemoglobin, platelets, PTT, PT, fibrinogen measured serially. Hemoglobin levels can be misleading in acutely bleeding patients because of insufficient time for the cardiovascular system to equilibrate with extravascular volume and reflect the true concentration of hemoglobin. All patients

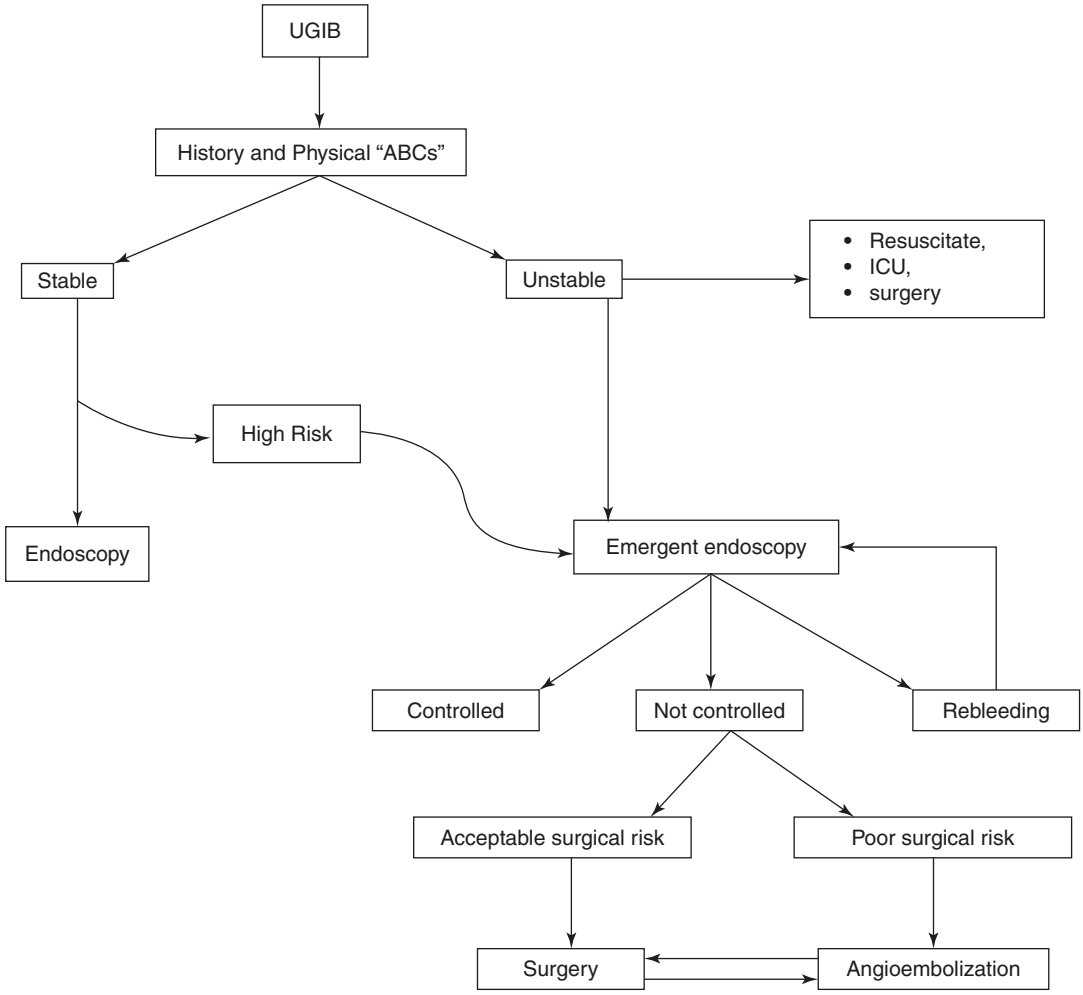

Fig. 7.1 Proposed algorithm for the management of NVUGIB

receiving large amount of transfusions could benefit from thromboelastography (TEG) if available. TEG is increasingly used as a point of care test as it simultaneously studies the integrated effects of different blood components involved in the coagulation cascade including thrombolysis [15]. Laboratory data can assist in risk stratification, bleeding localization, and guide therapy. The blood urea nitrogen (BUN) is elevated in GI bleeding [16] in general, and this is attributed to the digestion of blood in the GI tract [17] and its subsequent absorption. Furthermore, BUN to creatinine (Cr) ratio (BUN/Cr) >30 is 90% specific for UGIB with a positive likelihood ratio of 7.5 [18]. This test, nonetheless, has a low sensitivity of 39% [19]. EKG and cardiac enzymes should be sent to evaluate for myocardial ischemia.

Restoration of Coagulation

Patients with UGIB are often coagulopathic due to anticoagulant administration, consumption of coagulation factors during hemorrhage, underlying liver disease or as an effect of transfusion itself. Aggressive correction of coagulopathy decreases mortality [20]; therefore, it should be aggressively pursued. The following values should be targeted: international normalized ratio (INR) <1.5 and platelets >50 × 10^9 per liter [21].

Anticoagulation should be discontinued for patients on Coumadin, and INR should be reversed with vitamin K and FFP. Alternatively, prothrombin complex concentrate (PCC) should be used in conjunction with vitamin K for cases where rapid reversal is necessary or circulatory volume overload is a risk [22, 23] and for all direct oral anticoagulant (DOAC) reversal [14, 24]. For patients on Pradaxa, the specific reversal agent idarucizumab (Praxbind) is now available. If this agent is not available, then emergent hemodialysis is indicated to reverse the effects of Pradaxa. Low-dose aspirin for secondary cardiovascular prophylaxis in select patients may be continued [25].

Endoscopy

Endoscopy is essential for patients with UGIB to establish definitive diagnosis and guide therapy as early endoscopy improves outcomes in acute UGIB [26]. An important decision to be made is whether endoscopy needs to be done emergently or can wait for 12–24 h. For patients with severe UGIB, early upper endoscopy is recommended after hemodynamic resuscitation [25]. It is important that the endoscopist has the capability of performing the full range of therapeutic options, based on the endoscopic findings. Based on the timing of endoscopy from the time of presentation, there is *early endoscopy* which comprises (1) *very early* or *emergent* endoscopy (<8–12 h), (2) *urgent endoscopy* (12–24 h), and (3) *delayed endoscopy* (> 24 h) [25, 27]. This approach was shown to decrease mortality [28] and length of stay [29]. Very early endoscopy is indicated for "non-responders" and "transient responders" or in patients with evidence of ongoing bleeding (hematemesis, non-clearing bright red aspirates) or for patients for whom reversal of anticoagulation is not possible [25]. The advantage of second-look endoscopy is controversial and not routinely recommended. However, it may decrease the rebleeding rate of peptic ulcer bleeding in patients with unsatisfactory first endoscopic hemostasis, NSAID use, or massive transfusion [30].

Presentation and Management of Specific UGIB Etiologies

Non-variceal UGIB: Peptic Ulcers

Gastroduodenal peptic ulcers are the most frequent cause of UGIB and constitute more than 1/3 of patients with UGIB (Table 7.2). The underlying etiologies include *H. pylori* infection, NSAID use, gastrinoma, and stress. UGIB due to peptic ulcers stops spontaneously in 80% of the cases [35]. Peptic ulcers can cause eruptive bleeding when the ulcer base erodes into a blood vessel, usually the gastroduodenal artery [36]. Important risk factors include high levels of acid secretion and NSAID use, but interestingly, patients with bleeding ulcers have a lower prevalence of *Helicobacter pylori* than non-bleeding ulcers [36].

Bleeding peptic ulcers present with melena (20%), hematemesis (30%), or both (50%) [37]. Bright red blood per rectum can be from an upper gastrointestinal source when there is at least 1000 ml of blood entering the GI tract from an upper source. Bright red blood hematochezia occurring concomitantly with fresh blood

Table 7.2 Most frequent causes of UGIB

Diagnosis	Frequency of occurrence (%)
Peptic ulcer disease Duodenal Gastric	32–60 20–36 12–24
Mucosal erosive disease[a] Esophagitis	13–38 4–10
Gastroesophageal varices	4–33
Mallory-Weiss tear	3–7
Neoplasm	1–5
Angiodysplasia	1–3
Dieulafoy's lesions	1
Aortoenteric fistula	<1
Cameron lesion	<1
Hemobilia	<1
Not localized or unknown	5–25

References [7, 31–34]

[a]Mucosal erosive disease includes esophagitis, gastritis, duodenitis, and gastroduodenitis

hematemesis implies brisk UGIB and has a mortality rate of 30% [2].

Zollinger-Ellison syndrome (ZES) causes less than 1% of peptic ulcer disease, and it is the constellation of excessive gastric acid production causing severe peptic ulcer disease and diarrhea. Gastrinoma, the neuroendocrine tumor responsible for the hypersecretion of gastrin, most commonly arises sporadically or less commonly is associated with multiple endocrine neoplasia syndrome type 1 (MEN-1). The excessive amount of gastrin secreted by gastrinoma leads to hyperplasia of the parietal cells and increased basal gastric acid output, which breach the gastric and duodenal mucosal defenses leading to ulceration. Clinically, ZES is characterized by the presence of abdominal pain and diarrhea which both improve after administration of proton pump inhibitors [38].

Endoscopic Therapy for Non-variceal UGIB

Following endoscopy therapy, about 10–30% of patients have clinical evidence of rebleeding [5]. Among patients with stigmata of recent hemorrhage who rebleed after therapeutic endoscopy, 19% go on to require surgery or interventional radiology, and 27% of those patients die [26].

The timing of endoscopy depends on the risk of mortality and rebleeding. Therefore, it becomes important to identify high-risk patients. High-risk UGIB patients require higher level of care, aggressive resuscitation, earlier consultant's involvement, and more prompt procedures (endoscopy). Prior to endoscopic evaluation, patients are risk-stratified based on clinical and laboratory data. The Forrest Classification [39] (Fig. 7.2) standardizes the description of peptic ulcer and is used to identify the patients at risk of persistent ulcer bleeding, rebleeding, and mortality [25]. Other endoscopic features that predict adverse outcome and treatment failure include (1) large ulcer (> 2 cm), (2) visible vessel, (3) blood in the gastric lumen, and (4) ulcer in the posterior duodenal wall [40]. Three-quarters of the UGIB patients have *H. pylori* infection; therefore, vigorous attempts should be made to detect the presence of *H. pylori* acutely and retest the patient later to increase the diagnostic yield [25, 41]. When *H. pylori* is found, eradication with antibiotics should be pursued, and successful eradication should be documented [36].

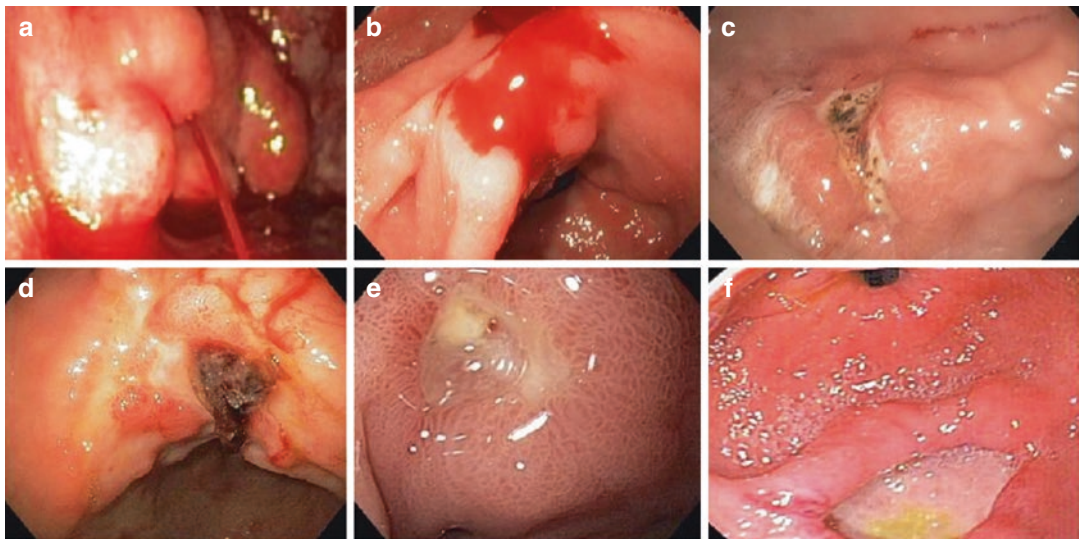

Fig. 7.2 Appearance of ulcers at endoscopy according to Forrest. Forrest Classification of ulcers: (**a**) *Forrest Ia*: ulcer spurting blood. (**b**) *Forrest Ib*: ulcer oozing blood. (**c**) *Forrest IIa*: ulcer with visible vessel. (**d**) *Forrest IIb*: ulcer with adherent clot. (**e**) *Forrest IIc*: ulcer with flat pigmented spot. (**f**) *Forrest III*: ulcer with clean base. (Pictures courtesy of Sven Hida, MD)

Once the bleeding is located, endoscopic therapeutic measures are taken for high-risk ulcers. Endoscopic therapies include:

(a) Injection therapy, with saline or vasoconstricting agents like epinephrine, sclerosing agents like ethanolamine.
(b) Thermal therapy is achieved by contact using a heater probe, a bipolar electrocautery, or argon plasma coagulator.
(c) Mechanical therapy involves using band ligation, clipping.
(d) Newer technologies include endoscopic spraying of topical hemostatic agents [42].

Surgical Management for NVUGIB

Indications for Surgical Intervention

Indications for surgery for UGIB are (1) hemorrhage not amenable to endoscopic control, (2) hemorrhage with post-endoscopy transfusion requirements *>4 units* [43, 44], (3) lack of endoscopic capacity, (4) recurrent bleeding after two attempts at endoscopic control, (5) lack of transfusion capabilities or limited supply, (6) absence of consent to transfuse as in the case of Jehovah's Witnesses, (7) repeated hospitalization for UGIB, and (8) concurrent indication of laparotomy such as perforation or obstruction [45, 46].

Surgical Management of Bleeding Gastric Ulcer

Options for surgical management of bleeding gastric ulcer include (1) oversewing of the bleeding ulcer through a surgical gastrostomy. Biopsy of the ulcer should be performed at the time of the surgery. Other options include (2) gastric resection for giant ulcers located on the lesser curvature (Pauchet procedure) and (3) partial gastrectomy for ulcer at the antrum. Other maneuvers to control the bleeding gastric ulcer are (4) simple ulcer excision [46] and (5) total gastrectomy for massively bleeding erosive gastritis. In the situation of diffusely, massively bleeding gastric erosions in an unstable patient, damage control principles can be utilized. It could require gastrostomy with packing the stomach with or without hemostatic agents and temporarily closing the gastrostomy. After resuscitation and rewarming, the patient is taken back for a second-look procedure where the packs are removed [47, 48]. Another option is to perform catheter-directed intra-arterial delivery of vasopressin [49].

Surgical Management of Bleeding Duodenal Ulcers

First of all, the surgeon needs to have a confirmation of the location of the ulcer from the endoscopist report or be present for the esophagogastroduodenoscopy (EGD). This will avoid the mistake of performing an unnecessary duodenostomy and extending it into a gastroduodenostomy. Surgical options for bleeding duodenal ulcers include (1) simple suture ligation, (2) suture ligation with drainage procedure and truncal vagotomy, (3) suture ligation and antrectomy, and (4) suture ligation and highly selective vagotomy. The ulcer is usually located at the first portion of the duodenum and sometimes at the proximal second portion of the duodenum. Kocher maneuver is necessary to mobilize the duodenum. A 3 cm pyloromyotomy should be performed, and if the ulcer is not in the duodenum, that incision should be extended to get more exposure in either direction. Intraoperative gastroscopy should be considered to look for a gastric source if not identified after duodenotomy.

Bleeding is initially controlled by applying direct pressure. Using a heavy braided suture on a non-cutting needle, three U-sutures should be placed around the gastroduodenal artery (GDA) proximally and distally at the 12 and 6 o'clock positions and around the transverse pancreatic branch at the 3 o'clock position to control the bleeding from the transverse pancreaticoduodenal artery (Fig. 7.3). If the ulcer is found and there is no active bleeding, suture ligation should still be performed. Care should be taken to avoid the common bile duct which runs deeper.

The longitudinally oriented incision should be closed transversely with a standard Heineke-Mikulicz pyloroplasty. Historically, a vagotomy has been used to reduce acid secretion; however, with the availability of proton pump

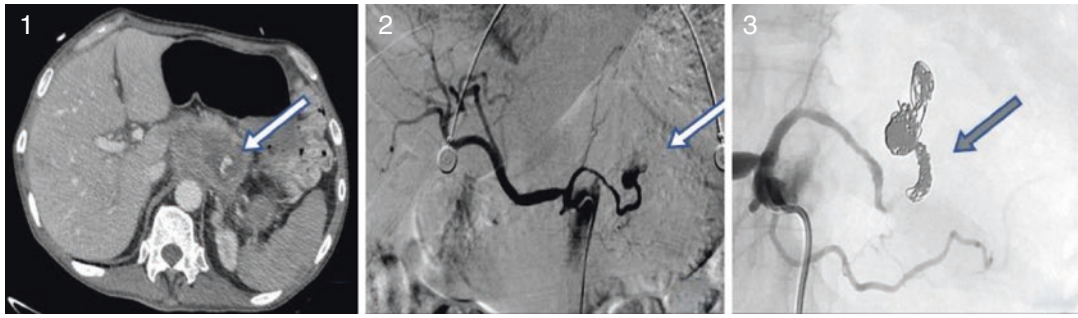

Fig. 7.3 Transcatheter angioembolization of bleeding gastric ulcer. 1. Computed tomography scan showing bleeding originating from the left gastric artery. 2. Angiogram showing pseudoaneurysm arising from the left gastric artery. 3. Coils in the artery

inhibitors and *H. pylori* treatment, vagotomy is not indicated unless the patient is noncompliant, will likely require NSAID treatment or has recurrent bleeding. There is evidence that a more extensive procedure, such as ligation with antrectomy, may have a lower incidence of rebleeding, but the higher morbidity associated with resection hence the advent of effective medical treatment make this approach rarely necessary [50].

Other Causes of NVUGIB and Their Managements

Mucosal Erosive Disease

Mucosal erosive disease of the upper gastrointestinal tract is the second most common cause of UGIB [33]. Esophagitis, gastritis, and duodenitis arise from alterations resulting in a break in the mucosa that does not extend to the muscularis mucosae and that may be infiltrated by inflammatory cells on histology. On endoscopy, mucosal erosive disease has the appearance of diffuse erythema, without significant depth erosions and mucosal hemorrhages.

Esophagitis accounts for approximately 10% of UGIB, but typically it is self-limited and carries a low morbidity and mortality [7, 31–34, 51]. Elderly and critically ill patients are at higher risk [52]. Reflux esophagitis is the most common cause, but another important subtype is infectious esophagitis, which includes viral (herpes simplex virus or CMV) or fungal or bacterial infections, all affecting immunocompromised hosts.

Gastritis and duodenitis most commonly cause bleeding in the setting of coagulopathy and are diagnosed by endoscopy which has the benefit of excluding other causes of bleeding. Causes of gastritis and duodenitis [53] include NSAID use, alcohol intake, portal gastropathy, and stress. Nearly all patients (>80%) with critical illness develop gastroduodenal erosions [54, 55]. Among patients admitted to the intensive care unit (ICU), 16% will still develop UGIB, despite receiving stress ulcer prophylaxis. Fortunately significant bleeding will develop in only 6% of these patients. Stress gastritis occurs in critically ill patients after stress events such as trauma, shock, sepsis, severe head trauma (Cushing's ulcers), and burns (Curling's ulcers). The pathogenesis is multifactorial and includes mucosal ischemia and reperfusion caused by fluctuation of splanchnic blood flow and perhaps an overactive parasympathetic system (vagus) causing hypersecretion of acid and pepsin [56, 57]. About 50–77% of ICU patients with UGIB may die of other causes, such as multiple system organ failure or underlying disease [58–60]. Risk factors for bleeding due to stress ulcers include respiratory failure, coagulopathy, older age, repair of abdominal aortic aneurysm, severe burns, multiple organ failure, neurological trauma, sepsis or septic shock, and high-dose corticosteroid. Respiratory failure requiring mechanical ventilation for more than 48 h or coagulopathy is a very strong risk factor for clinically relevant UGIB [61].

The treatment for mucosal erosive disease is supportive along with acid suppressive therapy

using proton pump inhibitors (PPI). Provocating agents such anticoagulation and nasogastric tube should be eliminated. For infectious esophagitis, antibiotics should be added.

Mallory-Weiss Lesions

Mallory-Weiss lesions are longitudinal lacerations in the gastric and/or esophageal mucosa near the gastroesophageal junction caused by mechanical forces of increasing intra-abdominal pressure like in forceful vomiting or retching. Other causes of these lacerations have been described and include coughing, hiccups, CPR, and colonoscopic preparation. Diagnosis is made with endoscopy. The bleeding is self-limiting in 90% of the cases [62]. Endoscopic therapies mostly used are epinephrine injection, heater probe, and band ligation. Surgery may be required for oversewing the laceration [62].

Dieulafoy's Lesions

Dieulafoy's lesions are large submucosal arteries close to the surface usually found in the proximal stomach along the lesser curvature but can be found anywhere else in the GI tract, with the duodenum being the next most common location [63]. Hemorrhage usually occurs after the vessel perforates. It is thought to be a pressure ulceration of the epithelium overlying a dilated artery [64]. Patients present with melena, hematemesis, followed by recurrent intermittent bleeding without a prior history or classic risk factors for GIB. The diagnosis is made by endoscopy, but unfortunately multiple endoscopies may be required to locate the bleed. Endoscopic therapy, usually with sclerotherapy, is curative in 95% of the cases [65]. Surgery is indicated if endoscopic treatment fails, but the lesion should be marked, and the location should be known, and operative therapy will consist of underrunning the blood vessel. In the case where the lesion cannot be found intraoperatively, endoscopic ultrasound can be used.

Hemobilia

Hemobilia is a gastrointestinal bleeding emanating from the biliary tree that comes through the ampulla of Vater [66]. Common causes include biliary tract procedures, trauma, biliary obstruction, cholangitis, cholecystitis, and pancreatitis. Classically, hemobilia presents with right upper quadrant abdominal pain, GI bleeding, and jaundice, with or without melena and/or hematemesis. CT scan and MRI are the diagnostic tools of choice, and blood from the papilla can be seen with endoscopy using a side-viewing scope. Treatment is by angiography with percutaneous trans-arterial catheter embolization. Surgery may be necessary (rarely) for failed angiography, and depending on the situation, options will include cholecystectomy with ligation of the relevant hepatic artery branch or resection by hepatectomy.

Hemosuccus Pancreaticus

Hemosuccus pancreaticus is another rare form of GI bleeding where there is transpapillary pouring of blood into the GI tract. In this situation, the gastrointestinal hemorrhage results from the erosion of the blood vessel into a pancreatic pseudocyst that communicates with the pancreatic duct. Like in hemobilia, the diagnosis can be made by CT scan and MRI with bleeding from the pancreatic duct which can be visible from the ampulla of Vater at endoscopy with a side-viewing scope. The preferred treatment is angiographic embolization.

Aortoenteric Fistula

Aortoenteric fistula constitutes the majority of the fistula between an artery and the GI system. Other communications have been described with the esophagus, the stomach and the small bowel, and the artery including the aorta. But the most common is aortoenteric fistula between the duodenum and the aorta. It can form from pressure necrosis of the bowel caused by the aortic aneurysm for primary aortoenteric fistula or the aortic graft for secondary aortoenteric fistula (most often due to fistula formation secondary to aortic infection). Patients present with back pain, fever, and hematemesis with or without hematochezia. These are "herald bleeds" before the ultimate massive GI bleed. A pulsatile mass may be present on physical examination. In the presence of a previous aortic graft, and an UGIB, aortoenteric

fistula should be suspected. Endoscopy is primarily performed to rule out other causes of GI bleeding and may visualize the fistula, adherent clot, or the aortic graft. The diagnostic test of choice is CT scan which will demonstrate signs of inflammation between the aorta or the graft and the duodenum. The treatment consists of antibiotics, emergent graft explantation with extra-anatomical bypass, and closure of the enterotomy.

Cameron Lesions

Cameron lesions are erosions or ulcerations of the gastric mucosa found within a hiatal hernia. Cameron lesions exist in up to 5% of hiatal hernias and are responsible for about 0.2% and 3.8% of overt and occult UGIB, respectively [67]. The incidence of these lesions is proportional to the size of the hernia [68].

Variceal Upper Gastrointestinal Bleeding

In patients with liver cirrhosis (90%) or hepatic vein obstruction (non-cirrhotics), portal hypertension worsens over time, leading to the formation of esophageal and gastric varices. Further increase in portal pressure causes the rupture of varices and subsequent bleeding [69]. Risk factors for variceal bleeding include variceal size, presence of red marks on varices, and high Child classification [70]. Patients with variceal UGIB have a mortality three times higher than that of non-variceal VUGIB [2, 3], and it could be as high as 15–30% [71]. For variceal UGIB, the Model for End-Stage Liver Disease (MELD) score is accurate in predicting risk of mortality [72]. Management of VUGIB along with ressuscitation includes vasoactive drug therapy (nitrates, beta-blockers, somatostatin/octreotide) antibiotic prophylaxis endoscopy.

Endoscopic Therapy for Variceal Bleeding

In general, emergent EGD is required for VUGIB, both for diagnosis and therapy. Endoscopic therapy for VUGIB consists primarily of endoscopic sclerotherapy (EST) or endoscopic band ligation (EBL). The therapies work by interrupting the flow through the esophageal or gastric system of venous collaterals. EBL is the treatment of choice due to lower complication profile, rebleeding rates, and number of treatments required to eradicate varices as compared to EST [73]. These therapies are less successful with gastric varices due to the profound depth of varices. Complications include ulceration, perforation, stricture formation, dysphagia, chest pain, worsening of the portal hypertensive gastropathy, and systemic embolization of sclerosing agent. EST and EBL have shown the ability to control active bleeding at the first treatment in 77% and 86% of the time [73] with a 21% and 12% rebleeding rate, respectively [74]. Overall, a 10–20% failure of medical and endoscopic treatment is expected. EBL should be repeated if the patient is stable and the bleeding is mild. For refractory bleeding varices in an unstable patient's balloon, tamponade may be achieved with the Sengstaken-Blakemore tube [75] or self-expanding metal stent (SEMS) [76]. In the past, the use of Sengstaken-Blakemore tube was 60–90% effective at controlling variceal bleeding [77] but should be used for less than 24 h. It should be used as a bridge to definitive treatment, because bleeding will recur after the release of tamponade in half of the patients. Major complications of balloon tamponade occur in 10–20% of cases and include aspiration, esophageal rupture, and airway obstruction [78, 79].

Surgical Therapy for Variceal Bleeding

Following endoscopic therapy or temporizing measure with balloon tamponade, definitive control should be achieved by decompressing the varices. This is achieved by diverting the flow of blood away from the portal toward the systemic circulation using a shunt. Operative portosystemic shunts are now of historic interest, and the shunt of choice today is the transjugular intrahepatic portosystemic shunt (TIPS). TIPS is less invasive and consists of placing fluoroscopically a large-bore stent

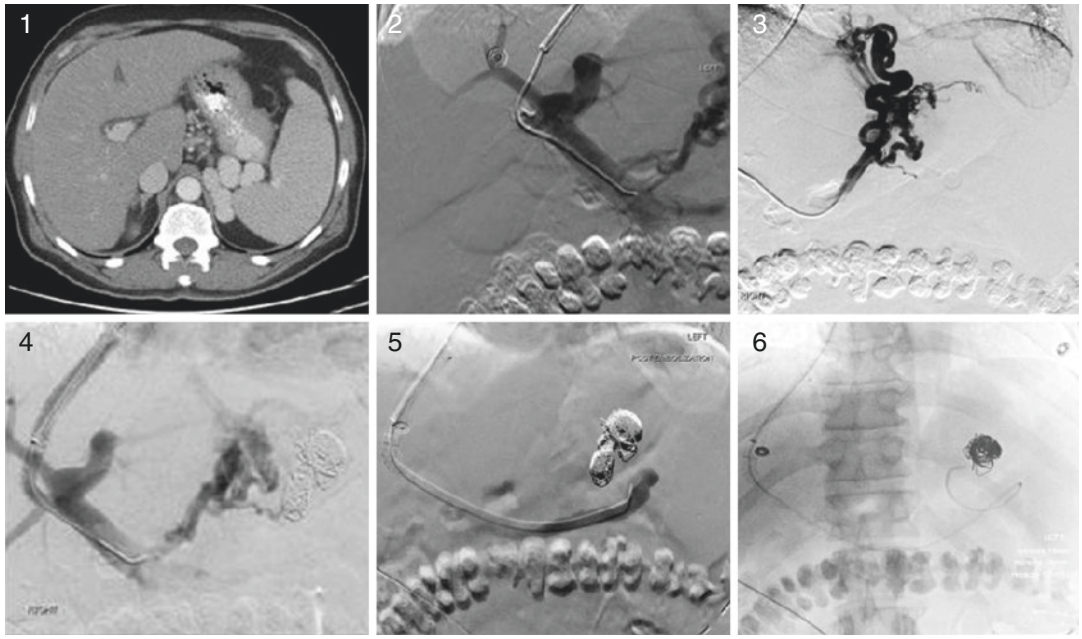

Fig. 7.4 Diagnostic and therapeutic angiography for variceal bleeding. 1. Multiple gastroesophageal varices secondary to portal HTN. 2. 3. Access gained into the portal venous system through the hepatic vein, liver parenchyma. 4. 5. Varices catheterized and embolized. 6. Transjugular intrahepatic portosystemic shunt (TIPS) placed. (Images courtesy of Gary Siskin, MD)

between the hepatic veins and the portal veins within the liver (Fig. 7.4). In VUGIB, TIPS is indicated for (1) s*alvage TIPS*, refractory active variceal hemorrhage despite medical and endoscopic therapy, (2) recurrent variceal hemorrhage despite medical and endoscopic therapy, and (3) *early TIPS,* now proposed after the initial variceal bleeding episode for Child B cirrhotics and selected Child C patients. Significant reductions in treatment failure (97% vs 50%) and mortality were shown when compared to medical therapy plus endoscopy [80]. Unfortunately, TIPS can worsen encephalopathy due to impaired hepatic protein metabolism and ensuing hyperammonemia. Operative portocaval shunting (end-to-side or splenorenal shunt) is rarely needed. In esophageal devascularization and transection, "Sugiura procedure" is a last-ditch treatment for refractory bleeding when shunting is not possible. The mortality for the Sugiura procedure is extremely high [78].

Patients with refractory VUGIB with encephalopathy along with refractory ascites or hepatorenal syndrome should be referred to a transplant center for consideration for liver transplant.

In non-cirrhotic patients, sinistral portal hypertension (SPH) should be suspected. SPH manifests as bleeding gastric varices in the setting of patent portal vein, normal hepatic function, and splenic vein thrombosis caused by pancreatic pathology. Causes include trauma, pancreatitis, or cancer. Splanchnic arteriography is necessary for accurate diagnosis. Splenectomy is curative [81].

Diagnostic and Interventional Radiology for UGIB

Endoscopy is nondiagnostic in 10–15% and non-therapeutic in 20% of cases, respectively [4]. Where traditional surgery was the logical next step, angioembolization has been used

particularly when patients are too sick to undergo a surgical intervention. The use of radiology for the localization of bleeding and achieving hemostasis in UGIB has increased.

Although rarely used, nuclear medicine studies may have a role in detecting intermittent bleeding and can detect bleeding with as little as 0.1 ml/min. Technetium-99m-labeled erythrocyte scan is preferred over the technetium-99m-labeled colloid because it remains in the intravascular space for 24 h allowing for repeated scanning [82].

Hemodynamically stable patients in the appropriate clinical setting (pancreatitis, following percutaneous hepatobiliary procedures, tumor) can have their UGIB localized by contrast-enhanced computed tomography angiography (CTA) scan. CTA scan detects bleeding as slow as 0.3 ml/h [83] (Fig. 7.3), and it has the advantage of localizing the source and defining the etiology at the same time. Angiographic examination for suspected UGIB source requires celiac trunk angiography and selective angiography of the gastroduodenal artery and left gastric artery. The key is to get the patient to the angiography suite as soon as possible when ongoing bleeding is suspected even if the patient is coagulopathic as a bleeding rate of at least 0.5 ml/h is required for the bleeding to be detected.

Portography not only permits TIPS creation to decrease portal venous pressures but will allow the visualization of gastric varices and potential embolization of bleeding varices [84] (Fig. 7.4). Angiographic therapy is indicated for severe, persistent bleeding after failure of endoscopic therapy in patients for whom surgery is not an option either because of the high risk of surgery or its unavailability [85]. The use of angiography and radiography-guided angioembolization is required in l% of admissions or less [3, 86] (Figs. 7.5 and 7.6). There are case series of positive experience with transcatheter angioembolization (TAE) used to treat refractory massive UGIB with a technical success ranging from 52% to 98% [85]. One of those groups reports complications and 1-month mortality rates of 10% and 26.7%, respectively, with a rebleeding rate of 28% and an 11.6% rate of surgery. Although the rebleeding rates are high, these patients could avoid the higher mortality of surgery [5]. Complications of TAE include access site hematoma, arterial dissection, contrast nephrotoxicity, and bowel ischemia [88].

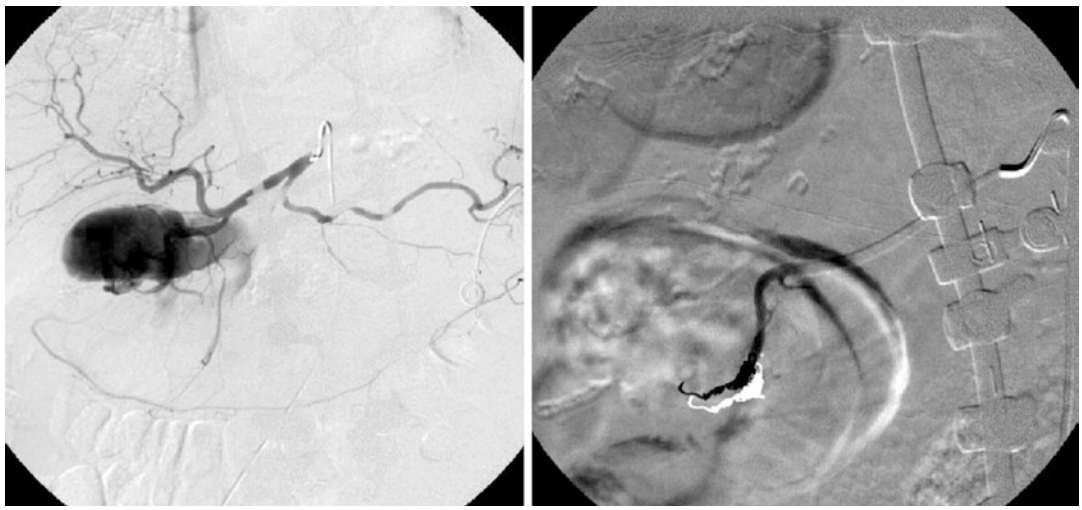

Fig. 7.5 Transcatheter angioembolization of bleeding duodenal ulcer. 1. Angiogram showing bleeding duodenal ulcer through gastroduodenal artery. 2. Coils placed in the gastroduodenal artery

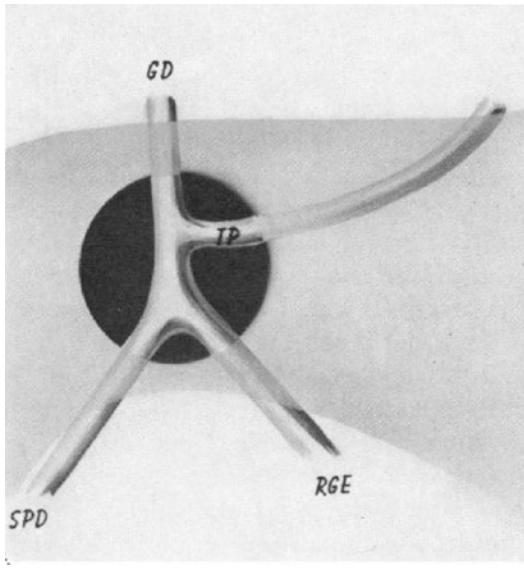

Fig. 7.6 Gastroduodenal artery complex. The two major types of "T" intersections are illustrated. Arteries: GD gastroduodenal, TP transverse pancreatic, SPD superior pancreaticoduodenal, RGE right gastroepiploic. (Reproduced from Berne and Tosoff [87])

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