

Approach Upper GIT bleeding

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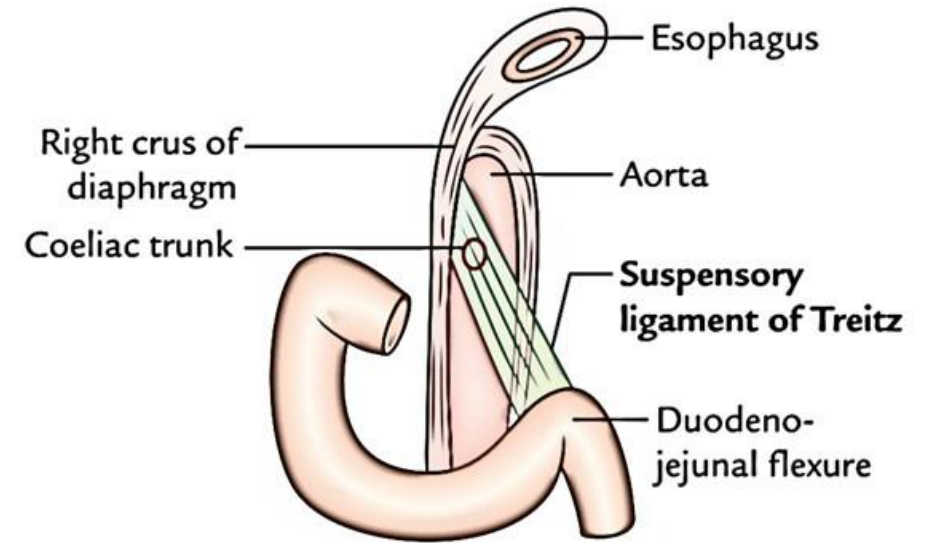


Introduction

- **Acute upper gastrointestinal bleeding is the most common GI emergency.**

Defined as: Bleeding occurring proximal to the ligament of Treitz, which anatomically separates the duodenum from the jejunum.

UGIB is 4 times as common as bleeding from lower GIT, with a higher incidence in males.



• CAUSES OF UPPER GASTROINTESTINAL HEMORRHAGE

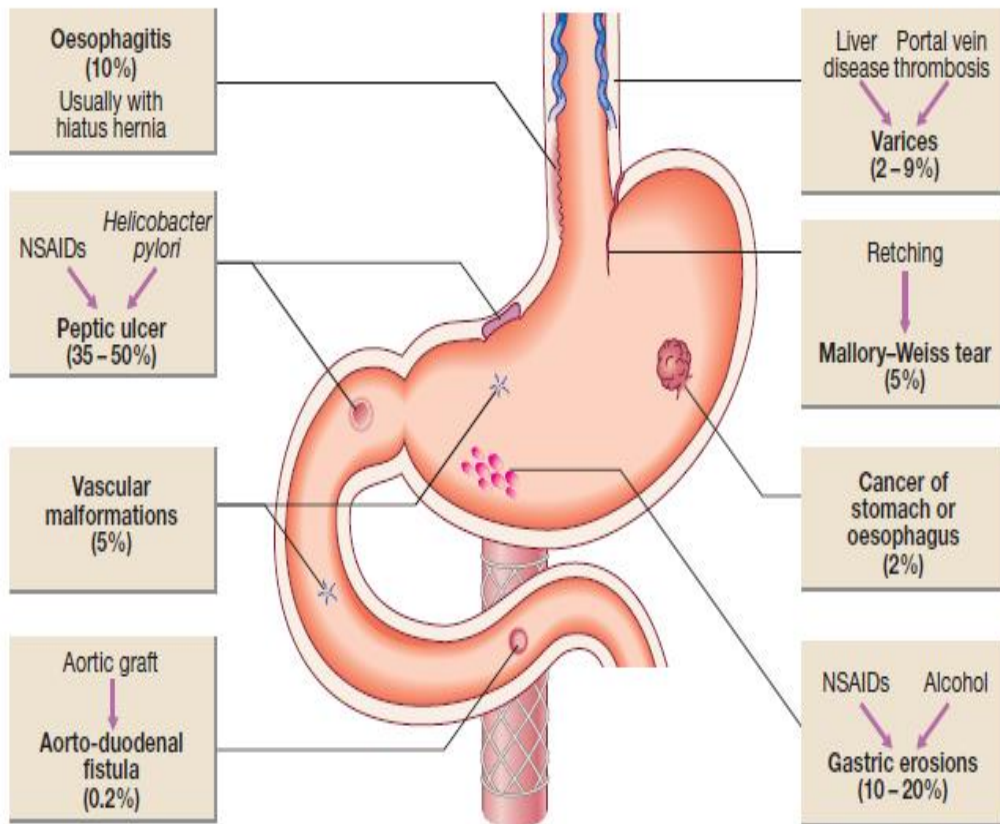


Fig. 21.19 Causes of acute upper gastrointestinal haemorrhage. Frequency is given in parentheses. (NSAIDs = non-steroidal anti-inflammatory drugs)

- The most common causes of UGIB include the following:
 - peptic ulcer disease
 - Severe or erosive gastritis/duodenitis
 - Severe or erosive esophagitis
 - Esophagogastric varices
 - Portal hypertensive gastropathy
 - Angiodysplasia (also known as vascular ectasia)
 - Mallory-Weiss syndrome
 - Mass lesions (polyps/cancers)
 - No lesion identified (10 to 15 percent of patients)

Other less common causes of UGIB include:

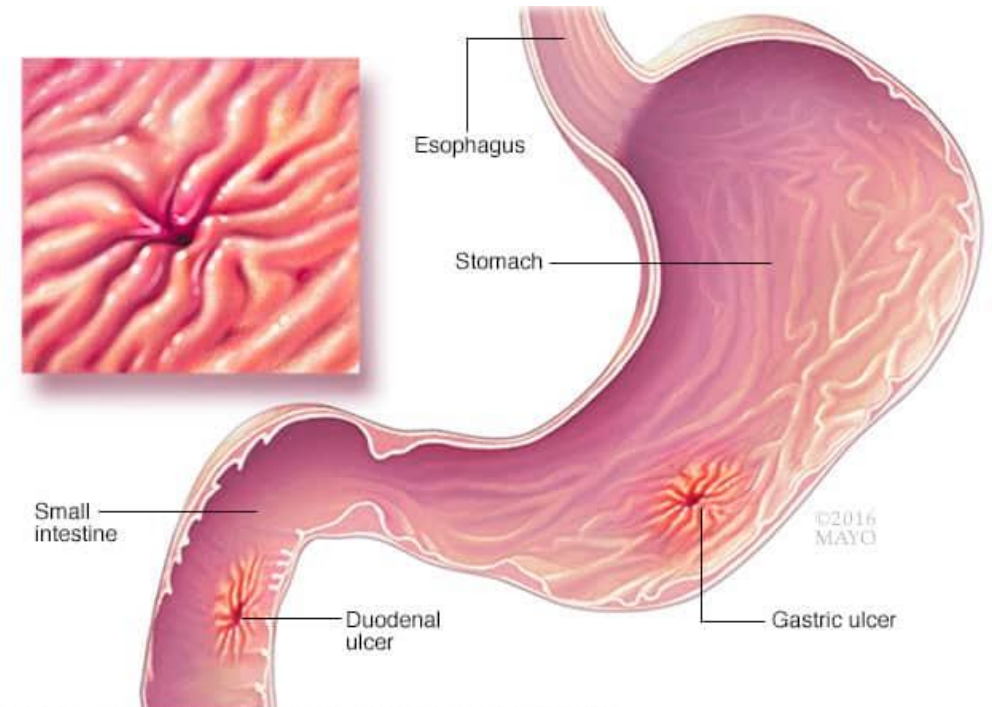
- Dieulafoy's lesion
- Gastric antral vascular ectasia
 - Hemobilia
- Hemosuccus pancreaticus
- Aortoenteric fistula
- Cameron lesions
- Ectopic varices
- Iatrogenic bleeding after endoscopic interventions

Peptic ulcer:

-The term 'peptic ulcer' refers to an ulcer in the lower esophagus, stomach or duodenum, in the jejunum after surgical anastomosis to the stomach or, rarely, in the ileum adjacent to a Meckel's diverticulum.

-The four major causes of peptic ulcer disease :

- Helicobacter pylori** infection
- Nonsteroidal anti-inflammatory drugs (NSAIDs)**
- Physiologic stress**
- Excess gastric acid**



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Clinical features:

Gastric Ulcer	Duodenal Ulcer
<ul style="list-style-type: none">• In the junction between the antrum and the acid secretory mucosa• Can happen in the fundus but rare• H. pylori infection in 70%	<ul style="list-style-type: none">• 95% in the first part of the duodenum• Most common type of ulcer• H. pylori infection in almost 100%
<ul style="list-style-type: none">• Gastritis, shallow or deep ulcer	<ul style="list-style-type: none">• Usually small <1 cm• Sharply demarcated lesion
<ul style="list-style-type: none">• Inflammatory cells	<ul style="list-style-type: none">• Eosinophils specially on the base of the ulcer
<ul style="list-style-type: none">• Can cause hematemesis or melena• Vomiting occurs	<ul style="list-style-type: none">• Can cause melena or hematoschezia
<ul style="list-style-type: none">• Pain starts and aggravated after a meal 1-2 hours (weight loss)	<ul style="list-style-type: none">• Pain starts after 3-5 hours after the meal• Pain is relieved by food and increase in intensity during the night (weight gain)
<ul style="list-style-type: none">• High risk of malignancy	

- Abdominal pain: epigastric
- ,Occasionally there is a discomfort localizes to the right or left upper quadrants of the hypochondrium
- bloating
- abdominal fullness, nausea, and early satiety that may be provoked by eating.
- Gastroesophageal reflux may coexist but may or may not be related to the peptic ulcers

Investigations:

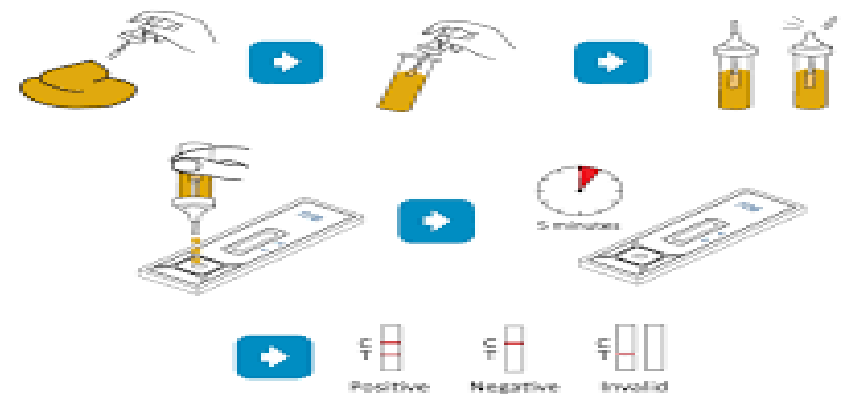
-Endoscopy is the preferred investigation: Gastric ulcers may occasionally be malignant and therefore must always be biopsied and followed up to ensure healing.

-Patients should be tested for H. pylori infection

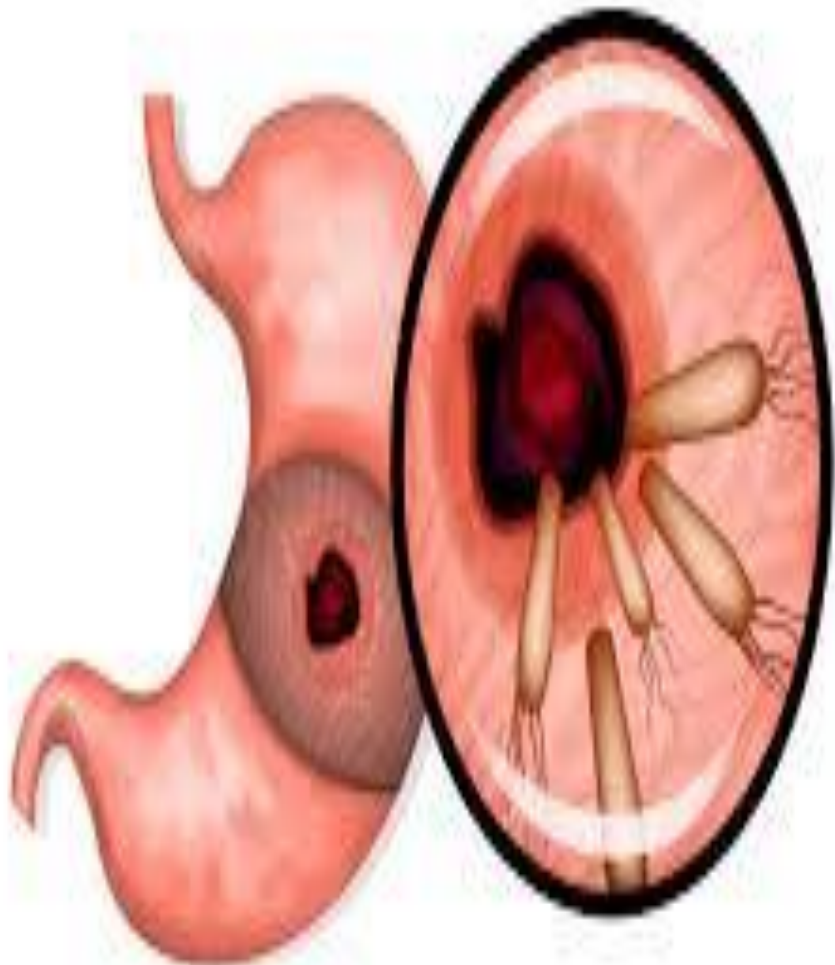
- 1. invasive :endoscopy**
- 2. noninvasive. They vary in sensitivity and specificity.**
 - Breath tests or faecal antigen tests are best because of accuracy, simplicity and non-invasiveness.**



How to use:



HELICOBACTER PYLORI



Management:

- 1- *H. pylori* eradication :** All patients with proven ulcers who are *H. pylori-positive* should be offered eradication as primary therapy.
 - Treatment is based upon a PPI taken simultaneously with antibiotics (from amoxicillin, clarithromycin and metronidazole) for 7 days. High-dose, twice-daily PPI therapy increases efficacy of treatment, as does extending treatment to 10–14 days.
 - A triple therapy includes : Amoxicillin + Clarithromycin (or levofloxacin)+ PPI.
 - A quadruple therapy regimen, consisting of omeprazole (or another PPI), bismuth subcitrate, metronidazole and tetracycline (OBMT) for 10–14 days, is recommended.
 - 14 days duration is the most recommended period.
- 2. Maintenance treatment**

For the minority who do require it, the lowest effective dose of PPI should be used.
- 3. General measures**

Stopping smoking should be strongly encouraged. Routine re-endoscopy at 6 weeks to exclude a malignant tumor of GU.

Management

- **-Patients with clinically significant UGI bleeding (ie, signs of active UGI bleeding including hematemesis, melena, or hematochezia, with or without hemodynamic instability or blood transfusion requirement) should be started on an intravenous proton pump inhibitor while undergoing their initial evaluation.**
- **Once the patient is stabilized, endoscopy is performed to diagnose high-risk lesions**
- **Ulcers that are actively bleeding and most nonbleeding ulcers that are at high risk for recurrent bleeding based upon the presence of stigmata of recent hemorrhage require endoscopic therapy.**
- **-Ulcers that lack high-risk stigmata can be managed acutely with acid suppression alone.**

-Stigmata of recent hemorrhage — Certain endoscopic findings, known as stigmata of recent hemorrhage, are associated with an increased risk of recurrent bleeding (up to 90 percent depending upon the finding) .

-The appearance of ulcers can be described using the Forrest classification:

- **Class Ia – Spurting hemorrhage**
- **Class Ib – Oozing hemorrhage**
- **Class IIa – Nonbleeding visible vessel**
- **Class IIb – Adherent clot**
- **Class IIc – Flat pigmented spot**
- **Class III – Clean ulcer base**

PHARMACOLOGIC THERAPY:

Acid suppression:The optimal approach to PPI administration prior to endoscopy is unclear. Options include giving an IV PPI every 12 hours or starting a continuous infusion.

Our approach is to give a high-dose bolus (eg, [esomeprazole](#) 80 mg)

Somatostatin and octreotide :a theoretical benefit in bleeding ulcer disease because they reduce splanchnic blood flow, inhibit gastric acid secretion, and may have gastric cytoprotective effects

Prokinetic agents: [erythromycin](#) be used before endoscopy. A reasonable dose is 250 mg intravenously over 20 to 30 minutes

endoscopic treatment:

1- injection therapy:


- Injection therapy with dilute(with saline) [epinephrine](#)
- Injection therapy should be used in conjunction with other forms of therapy, such as thermal coagulation or endoscopic clip placement

2-thermal coagulation:

- Thermal coagulation with contact probes achieves acute hemostasis and prevents recurrent bleeding by captive coagulation of the underlying artery in the ulcer base

3-hemostatic clips:

- is an alternative to thermal coagulation. Once applied, the clips achieve hemostasis in a manner similar to surgical ligation.

- 
- **4-fibrin sealant (or glue): injected fibrin sealant to achieve initial hemostasis and decrease the rate of recurrent bleeding.**

**5-Hemostatic sprays:
can be used to control active GI bleeding
in a variety of contexts, particularly when
traditional endoscopic techniques fail to
control massive GI bleeding**

6- argon plasma coagulation

Portal hypertension

Cause

1. esophageal varices
 2. PHG(portal hypertension gastropathy)
 3. gastric varices
 4. and ectopic varices.
- While most patients with portal hypertension have cirrhosis, portal hypertension can also occur in the absence of cirrhosis, a condition referred to as "noncirrhotic portal hypertension." include portal vein thrombosis, schistosomiasis, and idiopathic noncirrhotic portal hypertension.

Varices Due to portal hypertension .

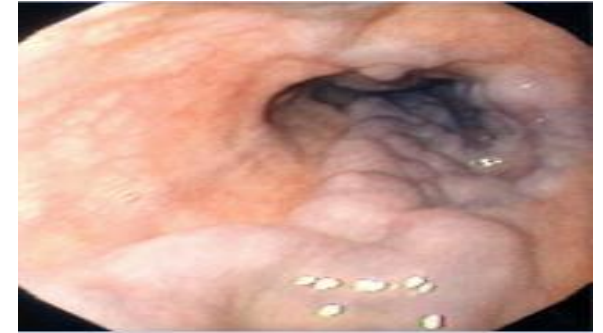
identified in the

1-esophagus

2- the stomach.

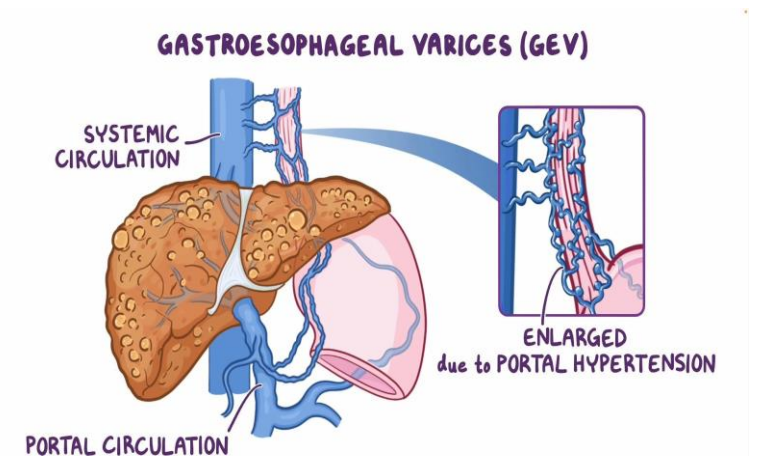
3- the small bowel (ectopic varices)

Esophageal varices



- Variceal bleeding is the most common cause of upper gastrointestinal bleeding in patients with cirrhosis and/or portal hypertension.
- Increased portal vascular resistance leads to a gradual reduction in the flow of portal blood to the liver and simultaneously to the development of collateral vessels, allowing portal blood to bypass the liver and enter the systemic circulation directly.
- Portosystemic shunting occurs, particularly in the gastrointestinal tract and especially the distal esophagus, stomach.....

endoscopy is the diagnostic



Management

- * Strategies for bleeding prevention include:
 - 1-Primary prevention of variceal bleeding
- If non-bleeding varices are identified at endoscopy, β -blocker therapy is effective in reducing portal venous pressure. Like (propranolol, nadolol).
 - 2-Preventing recurrent variceal bleeding.
- Beta-blockers are used as a secondary measure to prevent recurrent variceal bleeding. Following successful endoscopic therapy,



3-Management of acute variceal bleeding

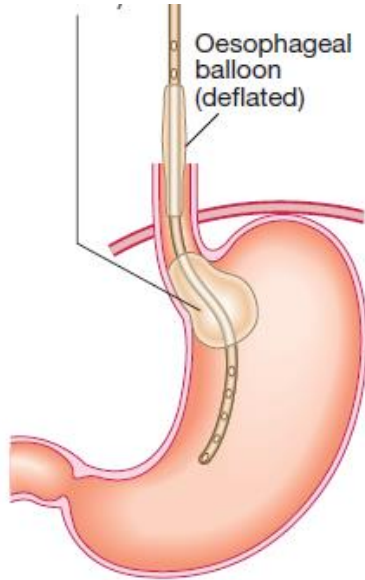
Goals of therapy — Treatment goals during an episode of acute bleeding are to:

- Restore and maintain hemodynamic stability
- Restore and maintain adequate oxygenation
- Control bleeding
- Prevent complications

1-Initial measures include intravenous access, intravenous fluids, and supplemental oxygen, and airway protection.

2-Antibiotic prophylaxis (preferably before endoscopy). We typically use a broad-spectrum antibiotic such as [ceftriaxone](#). It reduces the risk of mortality, infections (spontaneous bacterial peritonitis, urinary tract infections), and rebleeding.

3-Pharmacologic therapy for bleeding (Terlipressin, octreotide)

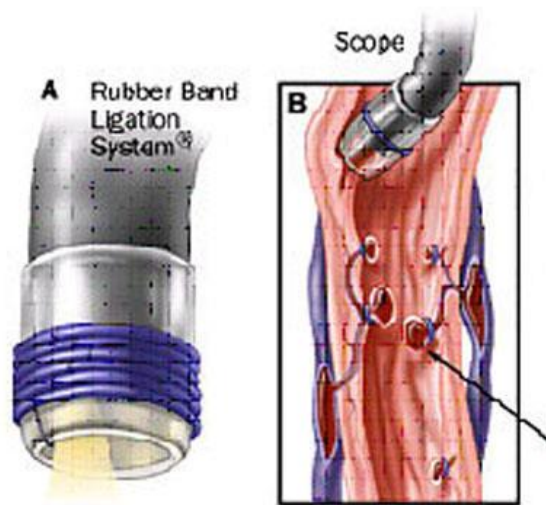


4- Variceal ligation ('banding') and sclerotherapy.

5-Balloon tamponade.

6-TIPSS

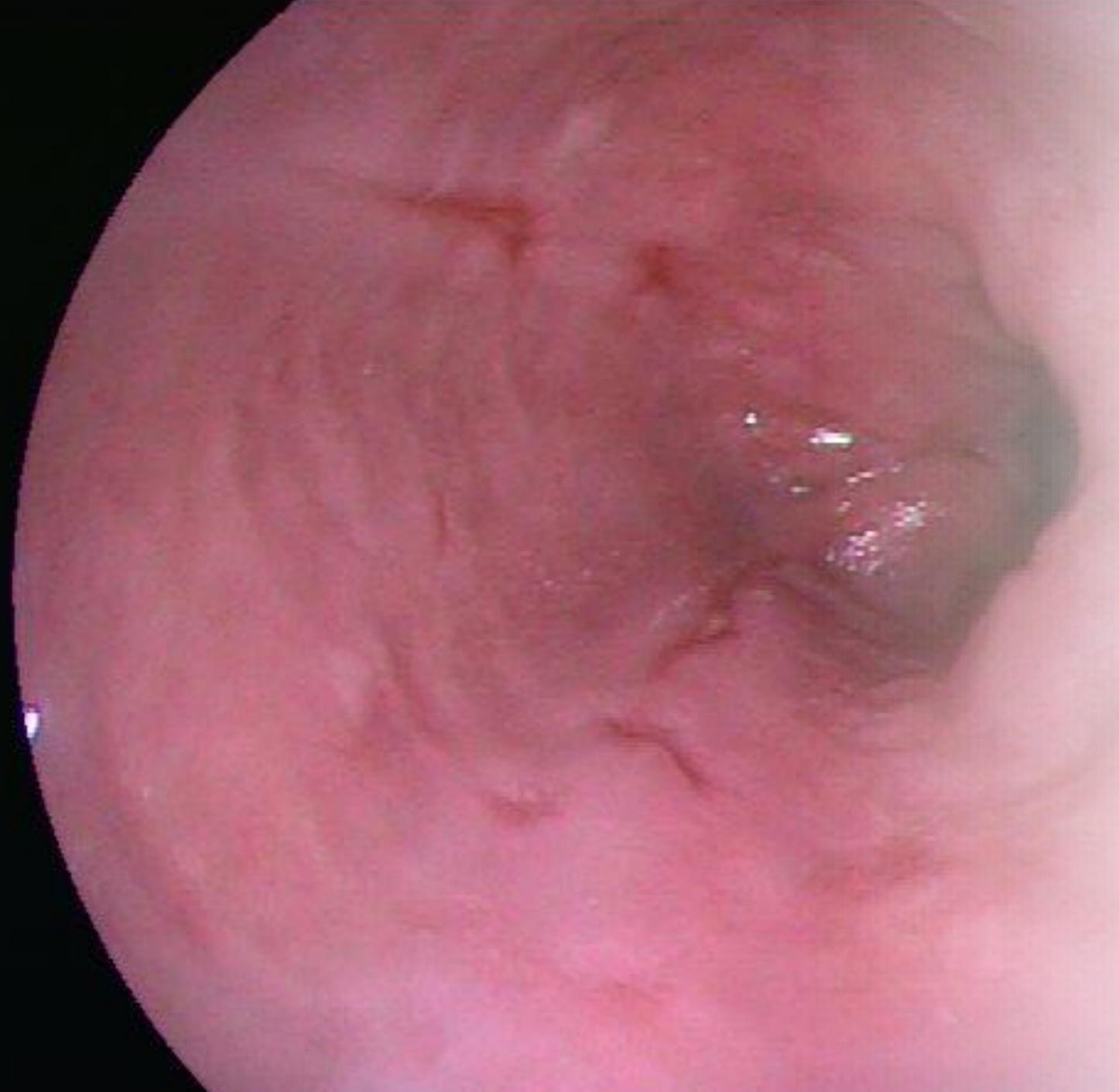
7-Portosystemic shunt surgery



FILM

Esophagitis

- Risk factor
 - history of gastroesophageal reflux disease (GERD)
 - medication use (eg, NSAIDs, oral bisphosphonates, [tetracycline](#))
 - infections (eg, *Candida*, herpes simplex virus).
- Compared with patients with UGIB due to other causes, esophagitis often have a more benign course, with shorter hospital stays, lower rebleeding rates, and lower mortality rates



Gastritis/gastro pathy and duodenitis/duo denopathy

- inflammation-associated mucosal injury.
- rarely lead to significant UGIB.
- risk factors
 - excessive alcohol consumption,
 - radiation injury
 - obesity surgery
 - and chronic bile reflux.
- Gastritis may also be found in certain autoimmune diseases.

- **Mallory-Weiss syndrome**

- longitudinal mucosal lacerations (intramural dissections)

1-distal esophagus 2- proximal stomach.

- bleeding from submucosal arteries.

- The amount of blood loss is usually small and self-limited. However, massive hemorrhage requiring transfusions and even leading to death can occur.

- tears are usually secondary to a sudden increase in intra-abdominal pressure

- Precipitating factors

1. Vomiting
2. straining at stool or lifting
3. Coughing
4. seizures
5. blunt abdominal injury
6. and gastroscopy.

- Endoscopy is the diagnostic modality of choice .

- Most tears heal spontaneously

- **treatment**

1. Endoscopic therapy is the first-line

2. for actively bleeding lacerations Several hemostatic methods have been used to control bleeding, including injection of epinephrine, thermal coagulation, endoscopic clip placement, and endoscopic band ligation.



- **Portal hypertensive gastropathy** — PHG (congestive gastropathy),
 - while extremely common in patients with portal hypertension, is an uncommon cause of significant bleeding in these patients.
 - The severity of gastropathy is related to the level of portal pressure, the level of hepatic vascular resistance, and the degree of reduction in hepatic blood flow. PHG, most often causes occult bleeding.
 - PHG may be confused with GAVE, which may also be seen in patients with cirrhosis.

Vascular lesions — Vascular lesions in the GI tract

1. Angiodysplasia

- most common vascular anomalies in the GI tract.
- Present occult bleeding or overt bleeding .
- diagnosed by endoscopy, but in some cases, radiographic imaging or surgery may be required for detection.

1. Dieulafoy's lesion

- is a dilated aberrant submucosal vessel that erodes the overlying epithelium in the absence of a primary ulcer .
- Lesions are usually located in the proximal stomach along the lesser curvature, near the esophagogastric junction.
- The etiology is unknown.
- Bleeding episodes are often self-limited, although bleeding can be recurrent.
- can be managed by different approach such as Endoscopic hemostasis, endoscopic band ligation, argon plasma coagulation, and cyanoacrylate injection.



- **3. Gastric antral vascular ectasia**

- is an uncommon cause of UGIB that is often confused with PHG
- both of which can occur in patients with cirrhosis
- isolated problem but has been associated with cirrhosis and systemic sclerosis. The most common clinical profile of a patient older (>70 years old) woman.
- Diagnosis
- classic endoscopic appearance. confirmed with a biopsy,
- bleeding.

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Approach

The initial evaluation of a patient with a suspected clinically significant acute upper GI bleed includes a history, physical examination, and laboratory tests.

Bleeding manifestations —

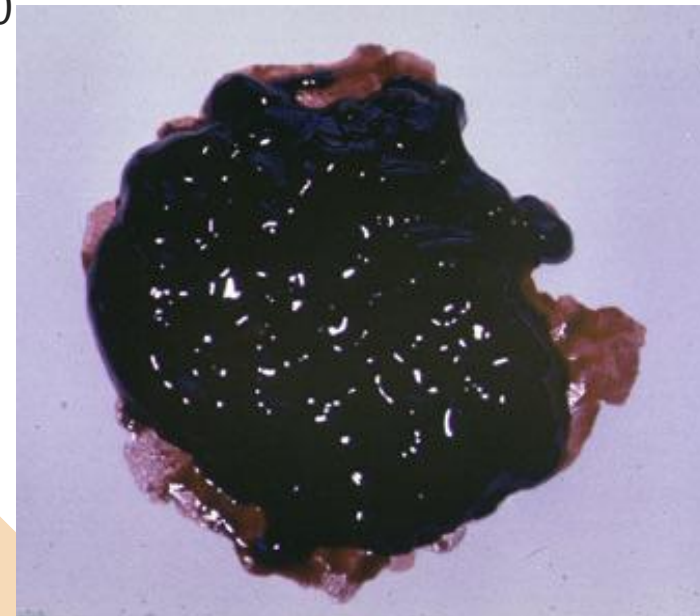
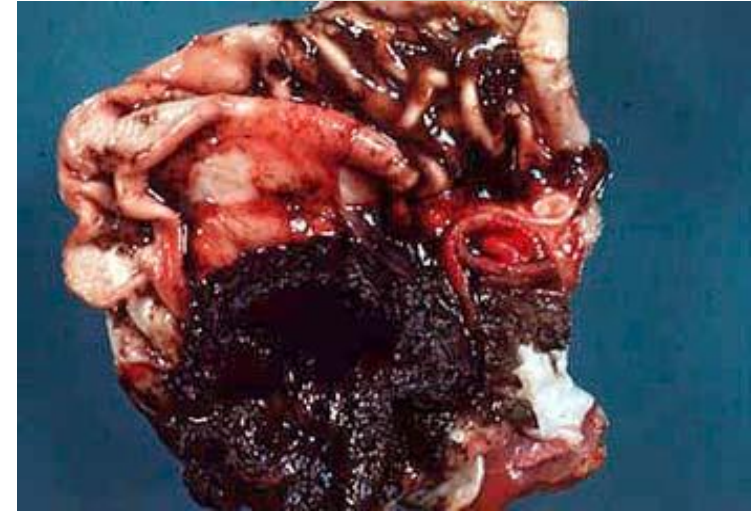
- **Hematemesis** (either red blood or coffee-ground emesis)

1. frankly bloody emesis suggests moderate to severe bleeding that may be ongoing.
2. coffee-ground emesis suggests more limited bleeding.

- **Melena** (black, tarry stool) majority originates proximal to the ligament of Treitz (90 percent) .

Melena may be seen with variable degrees of blood loss, being seen with as little as 50 mL of blood .

- **Hematochezia** (red or maroon blood in the stool) is usually due to lower GI bleeding. However, it can occur with massive upper GI bleeding.



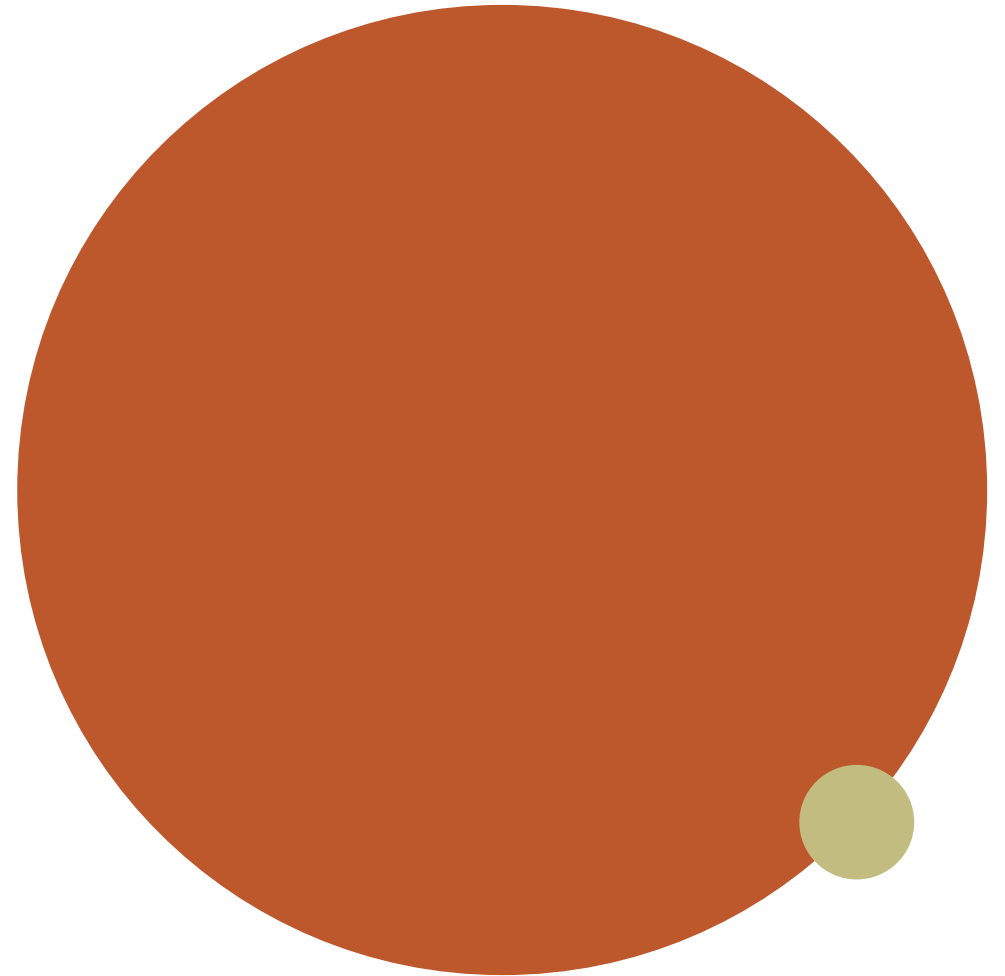


Symptom assessment —

bleeding is severe including orthostatic dizziness, confusion, angina, severe palpitations, and cold/clammy extremities.

Specific causes of upper GI bleeding may be suggested by the patient's symptoms :

- Peptic ulcer – Upper abdominal pain
- Esophageal ulcer – Odynophagia, gastroesophageal reflux, dysphagia
- Mallory-Weiss tear – Emesis, retching, or coughing prior to hematemesis
- Variceal hemorrhage or portal hypertensive gastropathy: Jaundice, abdominal distention (ascites)
- Malignancy – Dysphagia, early satiety, involuntary weight loss, cachexia





- **Past medical history**

- Varices or portal hypertensive gastropathy -----liver disease or excess alcohol use
- Aorto-enteric fistula -----abdominal aortic aneurysm or an aortic graft
- Angiodysplasia ----- renal disease, aortic stenosis, or hereditary hemorrhagic telangiectasia
- Peptic ulcer disease -----*Helicobacter pylori* (*H. pylori*) infection, nonsteroidal anti-inflammatory drug (NSAIDs) use, antithrombotic use, or smoking
- Malignancy -----smoking, excess alcohol use, or *H. pylori* infection
- Marginal ulcers (ulcers at an anastomotic site) -----gastroenteric anastomosis

- Drug history :

Use of: NSAIDs, aspirin, anticoagulants, antiplatelet agents



- **Physical examination** — assessment of hemodynamic stability.
- Signs of hypovolemia include :
 - ●Mild to moderate hypovolemia (less than 15 percent of blood volume lost) – Resting tachycardia.
 - ●Blood volume loss of at least 15 percent – Orthostatic hypotension (a decrease in the systolic blood pressure of more than 20 mmHg and/or an increase in heart rate of 20 beats per minute when moving from recumbency to standing).
 - ●Blood volume loss of at least 40 percent – Supine hypotension.
- The presence of abdominal pain, especially if severe and associated with rebound tenderness or involuntary guarding, raises concern for perforation. If any signs of an acute abdomen are present, further evaluation to exclude a perforation is required prior to endoscopy.



- **Laboratory data —**

- complete blood count.
- Hb level
- should initially be monitored every two to eight hours, depending upon the severity of the bleed.
- The initial Hb level ----- at the patient's baseline (because the patient is losing whole blood)
- . With time Hb ----- level will decline (as the blood is diluted by the influx of extravascular fluid into the vascular space and by fluid administered during resuscitation)
- MCV
- Normal MCV -----Acute bleeding
- If the MCV is low -----(iron deficiency due to chronic bleeding).
- serum chemistries.
- liver tests. Kidney test
- coagulation studies (PT, PTT, INR)
- serial ECG and cardiac enzymes may be indicated in patients who are at risk for a myocardial
- Cross match
- #acute upper GI bleeding typically have an elevated blood urea nitrogen (BUN)-to-creatinine or urea-to-creatinine ratio. The higher the ratio, the more likely the bleeding is from an upper GI source





- **GENERAL MANAGEMENT**

- Closely monitor airway, clinical status, vital signs, cardiac rhythm, urine output
- Do **NOT** give the patient anything by mouth
- Intravenous access : Establish two large bore IV lines (16 gauge or larger) larger intravenous catheters.
- Provide supplemental oxygen (goal oxygen saturation $\geq 94\%$ for patients without COPD)
- **Fluid resuscitation** —prior to endoscopy
- Treat hypotension initially with rapid, bolus infusions of isotonic crystalloid (normal [saline](#) or [lactated Ringer's](#) solution over 30 minutes 500 to 1000 mL per bolus; use smaller boluses and lower total volumes for patients with compromised cardiac function)
- **Nasogastric lavage** — The use of nasogastric tube (NGT) placement in patients with suspected acute upper GI bleeding is not recommended,

• 7- Blood Transfusion

- transfusion should be guided by hemodynamic parameters (eg, pulse and blood pressure), rather than by serial hemoglobin measurements.
- If the initial hemoglobin level is low (<7 g/dL) transfusions should be initiated .
- In an acutely hemorrhaging patient, however, transfusion support should not be delayed while awaiting laboratory test results.

Indication :

For severe, ongoing bleeding, immediately transfuse blood products in 1:1:1 ration of RBCs, plasma, and platelets, as for trauma patients

For hemodynamic instability despite crystalloid resuscitation, transfuse 1 to 2 units RBCs

For hemoglobin <8 g/dL (80 g/L) in high-risk patients (eg, older adult, coronary artery disease), transfuse 1 unit RBCs and reassess the patient's clinical condition

For hemoglobin <7 g/dL (70 g/L) in low-risk patients, transfuse 1 units RBCs and reassess the patient's clinical condition

Avoid over-transfusion with possible variceal bleeding

Give plasma

- coagulopathy (if high PT ,PTT, or (INR) greater than 1.5 times normal)
- transfusing four units of RBCs

give platelets

- thrombocytopenia (platelets <50,000) (we attempt to raise the platelet count to >50,000/microL prior to endoscopy.)
- platelet dysfunction (eg, chronic aspirin therapy)
- after transfusing four units of RBCs

Obtain immediate consultation with gastroenterologist; obtain surgical and interventional radiology consultation for any large-scale bleeding

8-Pharmacotherapy

- Give a proton pump inhibitor:
 1. Evidence of active bleeding (eg, hematemesis, hemodynamic instability), give esomeprazole or pantoprazole, 80 mg IV
 2. No evidence of active bleeding, give esomeprazole or pantoprazole, 40 mg IV
 3. Endoscopy delayed beyond 12 hours, give second dose of esomeprazole or pantoprazole, 40 mg IV

Pharmacotherapy for known or suspected esophagogastric variceal bleeding and/or cirrhosis:

1. Give somatostatin or an analogue (eg, octreotide 50 mcg IV bolus followed by 50 mcg/hour continuous IV infusion)
2. Give an IV antibiotic (eg, ceftriaxone or fluoroquinolone)
3. Balloon tamponade may be performed as a temporizing measure for patients with uncontrollable hemorrhage likely due to varices using any of several devices (eg, Sengstaken-Blakemore tube, Minnesota tube); tracheal intubation is necessary if such a device is to be placed; ensure proper device placement prior to inflation to avoid esophageal rupture

9- DIAGNOSTIC STUDIES

Upper endoscopy :diagnostic modality of choice ,therapeutic endoscopy Early endoscopy (within 24 hours) is recommended for most patients .For patients with suspected variceal bleeding, we perform within 12 hours of presentation.

prokinetic agent such as [erythromycin](#) or to irrigate the stomach prior to endoscopy to help remove residual blood and other gastric contents.

A colonoscopy is generally required for patients with hematochezia and a negative upper endoscopy unless an alternative source for the bleeding has been identified. In addition, patients with melena and a negative upper endoscopy frequently undergo colonoscopy to rule out a colonic source for the bleeding, as right-sided lesions may present with melena.

Other diagnostic tests for acute upper GI bleeding include CT angiography and angiography, which can detect active bleeding ,deep small bowel enteroscopy, and rarely, intraoperative enteroscopy Small bowel capsule endoscopy has also been employed to help localize bleeding in patients with acute gastrointestinal bleeding without hematemesis.

. Upper GI [barium](#) studies are **contraindicated**

10- Treatment of the underlying lesion

Triage

- All patients with hemodynamic instability or active bleeding should be admitted to an intensive care unit for resuscitation
close observation
 1. automated blood pressure monitoring
 2. electrocardiographic monitoring,
 3. pulse oximetry.
- Other patients can be admitted to a regular medical ward, though we suggest that all admitted patients with the exception of low-risk patients receive electrocardiographic monitoring.
- Outpatient management may be appropriate for some low-risk patients.
- Determining the appropriate site of care for a patient can be facilitated using risk stratification scores, such as the Glasgow-Blatchford score.
- Patients with a Glasgow Blatchford Score (GBS) of 0-1 may be considered for out-patient management

Low risk if Score is “0”

High risk is any score > 0 needing medical intervention, transfusion, endoscopy or surgery

ICU admission is indicated if GBS ≥ 8

Score is "0" only if all the following are present :

1. Hemoglobin level > 12.9 g/dL (men) or > 11.9 g/dL (women)
2. Systolic blood pressure > 109 mm Hg
3. Pulse < 100 /minute
4. Blood urea nitrogen level < 39 mg/dL
5. No melena or syncope
6. No past or present liver disease or heart failure.

The Rockall score

- Scoring systems have been developed to assess the risk of rebleeding or death, which based on clinical and endoscopy findings .

Rockall Scoring System				
Variable	Score=0	Score =1	Score =2	Score =3
Age (years)	<60	60-79	>80	
Comorbidity			Congestive heart failure, ischemic heart disease	Renal failure, liver disease, metastatic disease
Shock	No shock	Pulse > 100 bpm	Systolic BP <100 mmHg	
Source of bleeding	Mallory-Weiss Tear	All other diagnoses: e.g., esophagitis, gastritis, peptic ulcer disease, varices	Malignancy	
Stigmata of recent bleeding	None		Adherent clot or spurting vessel	

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Rockall score < 3 good prognosis

Rockall score > 8 High risk of death

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Thanks