Medicine
TEAM 437

MED437
KING SAUD UNIVERSITY

GI Bleeding

Objectives :

1. Recognize the clinical manifestations of upper gastrointestinal bleeding.
2. Understand the principles of managing patients with upper gastrointestinal bleeding.
3. Understand the principles of pharmacological therapy of patients with upper gastrointestinal bleeding.
4. Recognize the differences between variceal and non-variceal hemorrhage.

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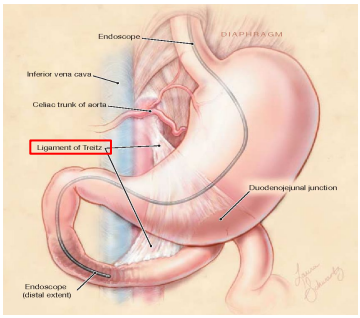
436 team + Davidsons + Kumar

Upper Vs. Lower GI Bleeding

❖ Anatomical landmarks and location of gastrointestinal bleeding:



- Upper GI bleeding : a source of bleeding above the **ligament of Treitz** (suspensory muscle of duodenum), including:
  Esophagus, stomach and duodenum.
- Lower GI bleeding : bleeding below the **ligament of Treitz,** including:
  Small & large bowel.

❖ Acute upper gastrointestinal bleeding:

(Acute UGIB is a common medical emergency that has 11% hospital mortality rate)
The cardinal features are **haematemesis** (vomiting of blood) and **melaena** (the passage of black tarry stools, the black colour being due to blood altered by passage through the gut). Melaena can occur with bleeding from any lesion proximal to the right colon. Rarely, melaena can also result from bleeding from the right colon. Following a bleed from the upper gastrointestinal tract, unaltered blood can appear per rectum, but **the bleeding must be massive** and is almost always accompanied by shock. The passage of dark blood and clots without shock is always due to lower gastrointestinal bleeding.

❖ Acute lower gastrointestinal bleeding:

Massive bleeding from the lower gastrointestinal tract is rare and is usually due to **diverticular disease** or ischaemic colitis. Common causes of small bleeds are haemorrhoids and anal fissures.

Etiology

- The most common cause of **upper GI bleeding** is **peptic ulcer disease**
- The most common cause of **lower GI bleeding** is **Diverticulosis**[1]

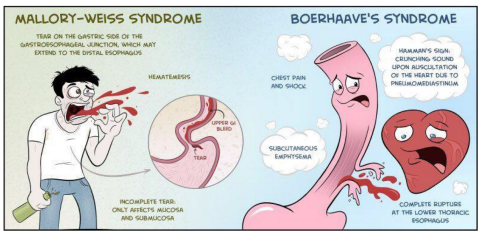
[1] Diverticulosis is the condition of having multiple pouches (diverticula) in the colon that are not inflamed. These are outpockets of the colonic mucosa and submucosa through weaknesses of muscle layers in the colon wall.They typically cause no symptoms.Diverticular disease occurs when diverticula become inflamed, known as diverticulitis, or bleed.

Etiology

Upper GI Bleeding

1. **Peptic ulcer disease** (duodenal and gastric ulcers). Most common
2. Esophagitis / Gastritis / duodenitis
3. Variceal bleeding it depends on geographic location, where alcohol consumption is seen/ schistosomiasis.
4. Mallory weiss tear and its severe form:

Boerhaave syndrome (usually lethal), mucosa get damage from the string and lead to bleeding; for example in pregnant ladies when they vomit a lot they could have muscle tearing in the region between the esophagus and



Other uncommon causes:

- Arteriovenous malformation
- Gastric antral vascular ectasia (GAVE)
- Dieulafoy's lesion (vessel that bleed and disappear)
- Malignancy

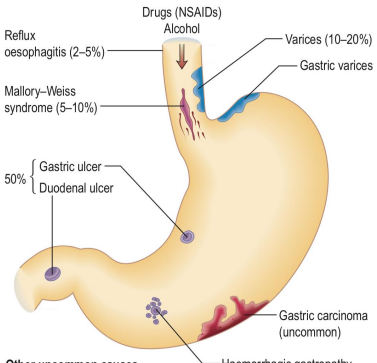
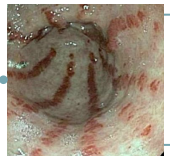


Figure 6.21 Causes of upper gastrointestinal haemorrhage. The approximate frequency is also given.



Watermelon stomach
Come with elderly, renal dialysis & chronic melena. dilated small blood vessels in the pyloric antrum, result in intestinal bleeding.

Lower GI Bleeding

1. **Diverticular disease** (40%) most common source of GI bleeding in patients over age of 60, usually painless.
2. Angiodysplasia (AVM) (40%) second most common source in patients over age of 60.
3. IBD (UC, Crohn's disease) Colorectal carcinoma Colorectal adenomatous polyps Ischemic colitis
4. Haemorrhoids, anal fissures
5. Small intestinal bleeding diagnosed by

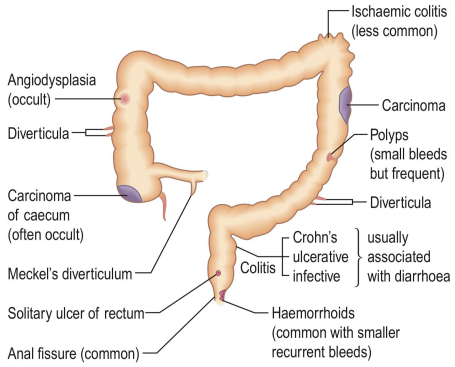


Figure 6.22 Causes of lower gastrointestinal bleeding. The sites shown are illustrative – many of the lesions can be seen in other parts of the colon.

22.20 Causes of lower gastrointestinal bleeding

Severe acute

- Diverticular disease
- Angiodysplasia
- Ischaemia
- Meckel's diverticulum
- Inflammatory bowel disease (rarely)

Moderate, chronic/subacute

- Fissure
- Haemorrhoids
- Inflammatory bowel disease
- Carcinoma
- Large polyps
- Angiodysplasia
- Radiation enteritis
- Solitary rectal ulcer

Extra

Clinical features:

1. Type of Bleeding:	
Hematemesis	• Vomiting fresh, red blood; suggests upper GI bleeding (bleeding proximal to ligament of Treitz). Indicates moderate to severe bleeding that may be ongoing.
"Coffee grounds" emesis (5-10 ml)	• Suggests upper GI bleeding as well as a lower rate of bleeding (blood has been oxidized in the stomach so it appears as "coffee grounds")
Melena black, tarry, liquid, foul-smelling stool	• Caused by degradation of hemoglobin by bacteria in the colon; presence of melena indicates that blood has remained in GI tract for several hours. • The further the bleeding site is from the rectum, the more likely melena will occur. • Note that dark stools can also result from bismuth, iron, spinach, charcoal, and licorice. • **Melena suggests upper GI bleeding 90% of the time**. Occasionally, the jejunum or ileum is the source. It is unusual for melena to be caused by a colonic lesion, but if it is, the ascending colon is the most likely site.
Hematochezia	• This usually represents a **lower** GI source (typically left colon or rectum). • Consider diverticulosis, arteriovenous malformations, hemorrhoids, and colon cancers. • **It may result from massive upper GI bleeding** that is bleeding very briskly (so that blood does not remain in colon to turn into melena). • This often indicates heavy bleeding, and patient often has some degree of hemodynamic instability. An upper GI source is present in about 5% to 10% of patients with hematochezia.
Occult blood in stool	• Source of bleeding may be anywhere along GI tract. Invisible blood in the stool detected by fecal occult blood test FOBT.

2. Signs of volume depletion (depending on rate and severity of blood loss). Increased pulse rate (>100 per minute), decreased blood pressure (systolic blood pressure < 100 mmHg), Increased respiratory rate, decreased urine output, decreased mental status.

3. Symptoms and signs of anemia (e.g., fatigue, pallor, exertional dyspnea).

Sources of GI Bleeding

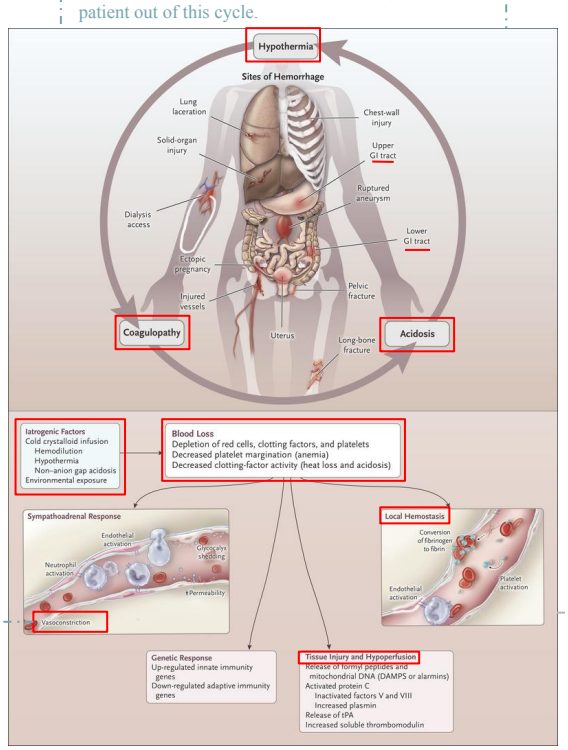
	Esophagus	Stomach	Duodenum	Small Intestine[a]	Right Colon	Left Colon
Hematemesis	X	X	X	—	—	—
Coffee-ground emesis	X	X	X	—	—	—
Melena	X	X	X	X	X	—
Guaiac-positive stool	X	X	X	X	X	X
BRBPR	(If severe)	(If severe)	(If severe)	(If severe)	X	X

Upper GI Bleeding	Lower GI Bleeding
Hematemesis Coffee ground emesis Melena	Hematochezia (BRBPR) bright red blood per rectum Melena

★ Treat upper GI bleeding as hypovolemic shock

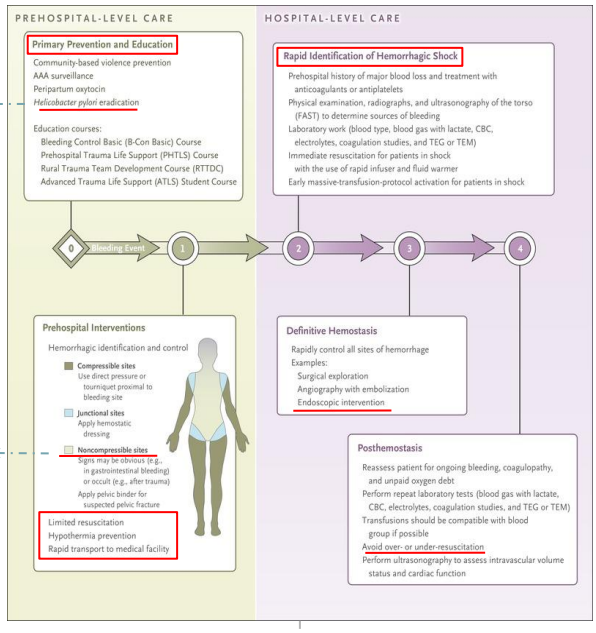
Vicious cycle

This cycle occurs when there is hypovolemia which will cause acidosis, and then you give IV fluids and cold blood products which in turn will cause hypothermia which will lead to coagulopathy.
And the cycle continues, so our aim is to get the patient out of this cycle.



For preventing second bleeding

GI compartments



These two pictures were explained by Dr. AlMadi only
Knowing the pathophysiology of shock is part of the objectives do NOT skip it.

Cause Cold periphery

➢ Hypovolemic shock signs, symptoms & fluid replacement

Blood loss (mL)	<750	750–1500	1500–2000	>2000
Blood loss (%)	<15	15–30	30–40	>40
Pulse rate	<100	>100	>120	>140
Blood pressure	Normal	Normal	Decreased	Decreased
Pulse pressure	Normal or increased	Decreased	Decreased	Decreased
Respiratory rate	14–20	20–30	30–40	>35
Urine output (mL)	>30	20–30	5–15	Negligible
Mental status	Slightly anxious	Mildly anxious	Anxious and confused	Confused and lethargic
Fluid replacement	Crystalloid	Crystalloid	Crystalloid and blood	Crystalloid and blood

Table 2. Classification of Hemorrhagic Shock.*

Shock Class	Blood Loss†	Heart Rate	Blood Pressure	Pulse Pressure	Respiratory Rate	Mental Status
	ml (%)	*beats/min*			*breaths/min*	
I	<750 (15)	<100	Normal	Normal	14–20	Slightly anxious
II	750–1500 (15–30)	100–120	Normal	Narrowed	20–30	Mildly anxious
III	1500–2000 (30–40)	120–140	Decreased	Narrowed	30–40	Anxious, confused
IV	>2000 (>40)	>140	Decreased	Narrowed	>35	Confused, lethargic

* Data are from the American College of Surgeons Committee on Trauma.[42]
† Blood-loss volume and percentage of total blood volume are for a male patient with a body weight of 70 kg.

Diagnosis:

For acute bleeding especially when the bleeding is severe it is far more important to **replace fluids** and check hematocrit, platelet count and coagulation tests as the prothrombin time or INR than it is to do an endoscopy .

Laboratory tests	• **Stool guaiac for occult blood.** • **Hemoglobin/hematocrit level** (may not be decreased in acute bleeds): A hemoglobin level >7 to 8 g/dL is generally acceptable in young, healthy patients without active bleeding. However, most elderly patients (especially those with cardiac disease) should have a hemoglobin level >10 g/dL. • A low **mean corpuscular volume** is suggestive of iron deficiency anemia (chronic blood loss). Patients with acute bleeding have normocytic red blood cells. • **Coagulation profile** (platelet count, PT, PTT, INR). To rule out bleeding disorders. • **LFTs, renal function.** • The **BUN–creatinine ratio** is elevated with upper GI bleeding. This is suggestive of upper GI bleeding if patient has no renal insufficiency. The higher the ratio, the more likely the bleeding is from an upper GI source.
Upper endoscopy	• **Most accurate diagnostic test** in evaluation of upper GI bleeding. • Both **diagnostic and potentially therapeutic** (coagulate bleeding vessel). • Most patients with upper GI bleeding should have upper endoscopy within 24 hours.
Nasogastric tube	➔ This is often the **initial** procedure for determining whether GI bleeding is from an upper or lower GI source. ➔ Use the nasogastric tube to empty the stomach to prevent aspiration. ➔ **False-negative findings**: possible if upper GI bleeding is intermittent or from a lesion in the duodenum. • Evaluation of aspirate. • Bile but no blood—upper GI bleeding unlikely; source is probably distal to ligament of Treitz. • Bright red blood or "coffee grounds" appearance—upper GI bleeding. • Non-bloody aspirate (clear gastric fluid)—upper GI bleeding unlikely, but cannot be ruled out definitively (source may possibly be in the duodenum).
Colonoscopy	Identifies the site of the lower GI bleed in >70% of cases, and can also be therapeutic.
Arteriography	Definitively locates the point of bleeding. • Mostly used in patients with **lower** GI bleeding. • Should be performed during **active bleeding.** • Potentially **therapeutic** (embolization or intra-arterial vasopressin infusion).

Extra

Tests to Order in Patients With GI Bleeding: (Step up)

1. Hematemesis: Upper GI endoscopy is the initial test.
2. Hematochezia: First rule out an anorectal cause (e.g. hemorrhoids). Colonoscopy should be the initial test.
3. Melena: Upper endoscopy is usually the initial test because the most likely bleeding site is in the upper GI tract. Order a colonoscopy if no bleeding site is identified from the endoscopy.
4. Occult blood: colonoscopy is the initial test in most cases (colon cancer is the main concern). Order an upper endoscopy if no bleeding site is identified .

Management

This is EXTRA from Davidsons to arrange your information

1. Intravenous access

- The first step is to gain intravenous access using at least one large-bore cannula.

2. Initial clinical assessment

- ***Define circulatory status.*** Severe bleeding causes tachycardia, hypotension and oliguria. The patient is cold and sweating, and may be agitated.
- ***Seek evidence of liver disease.*** Jaundice, cutaneous stigmata, hepatosplenomegaly and ascites may be present in decompensated cirrhosis.
- ***Identify comorbidity.*** The presence of cardiorespiratory, cerebrovascular or renal disease is important, both because these may be worsened by acute bleeding and because they increase the hazards of endoscopy and surgical operations.

3. Basic investigations

- ***Full blood count.*** Chronic or subacute bleeding leads to anaemia, but the haemoglobin concentration may be normal after sudden, major bleeding until haemodilution occurs. Thrombocytopenia may be a clue to the presence of hypersplenism in chronic liver disease.
- ***Urea and electrolytes.*** This test may show evidence of renal failure. The blood urea rises as the absorbed products of luminal blood are metabolised by the liver; **an elevated blood urea with normal creatinine concentration implies severe bleeding.**
- ***Liver function tests***. These may show evidence of chronic liver disease.
- ***Prothrombin time.*** Check with clinical suggestion of liver disease or in anticoagulated patients.
- ***Cross-matching.*** At least 2 units of blood should be cross-matched.

4. Resuscitation

- Intravenous crystalloid fluids should be given to raise the blood pressure, and blood should be transfused when the patient is actively bleeding with low blood pressure and tachycardia. Comorbidities should be managed as appropriate. Patients with suspected chronic liver disease should receive broad-spectrum antibiotics. Central venous pressure (CVP) monitoring may be useful in severe bleeding, particularly in patients with cardiac disease, to assist in defining the volume of fluid replacement and in identifying rebleeding.

5. Oxygen

- This should be given to all patients in shock.

6. Endoscopy

- This should be carried out **after** adequate resuscitation, ideally within 24 hours, and will yield a diagnosis in 80% of cases. Patients who are found to have major endoscopic stigmata of recent haemorrhage can be treated endoscopically using a thermal or mechanical modality, such as a 'heater probe' or endoscopic clips, combined with injection of dilute adrenaline (epinephrine) into the bleeding point ('dual therapy'). This may stop active bleeding and, combined with intravenous proton pump inhibitor (PPI) therapy, prevent rebleeding, thus avoiding the need for surgery. Patients found to have bled from varices should be treated by band ligation.

7. Monitoring

- Patients should be closely observed, with hourly measurements of pulse, blood pressure and urine output.

8. Surgery

- Surgery is indicated when endoscopic haemostasis fails to stop active bleeding and if rebleeding occurs on one occasion in an elderly or frail patient, or twice in a younger, fitter patient. If available, angiographic embolisation is an effective alternative to surgery in frail patients.
- The choice of operation depends on the site and diagnosis of the bleeding lesion. Duodenal ulcers are treated by under-running, with or without pyloroplasty. Under- running for gastric ulcers can also be carried out (a biopsy must be taken to exclude carcinoma). Local excision may be performed, but when neither is possible, partial gastrectomy is required. Following surgery for ulcer bleeding, all patients should be treated with H. pylori eradication therapy if they test positive for it, and should avoid NSAIDs. Successful eradication should be confirmed by urea breath or faecal antigen testing.

22.17 Emergency management of acute non-variceal upper gastrointestinal haemorrhage

- Gain IV access with large-bore cannula × 2
- Check full blood count, routine biochemistry and coagulation screen; cross-match blood
- Perform hourly measurements of blood pressure, pulse and urine output; consider central venous pressure monitoring in the high-dependency unit for severe bleeding
- Give IV crystalloids in patients with hypotension and tachycardia
- Transfuse with blood if blood pressure remains low and patient is actively bleeding
- Organise endoscopy for diagnosis and treatment once patient is resuscitated
- Give 72-hr proton pump inhibitor IV infusion for bleeding peptic ulcer
- Consider surgery or interventional radiological intervention (e.g. arterial embolisation) if bleeding recurs

Clinical Approach the the patient

Management:

1: Initial assessment:

- **Risk assessment tools (Increased risk of further bleeding and death)**
  - Glasgow - blatchford score most accurate and common.
  - Rockall score (clinical + endoscopic)
  - AIMS65 score

Table 1 | Glasgow–Blatchford score assessment criteria

	Risk factors at presentation	Threshold	Score
Urea	Blood urea nitrogen (mmol/l)	6.5–7.9	2
		8.0–9.9	3
		10.0–24.9	4
		≥25.0	6
CBC	Hemoglobin for men (g/l)	120–130	1
		100–119	3
		<100	6
	Hemoglobin for women (g/l)	100–120	1
		<100	6
Physical	Systolic blood pressure (mmHg)	100–109	1
		90–99	2
		<90	3
	Heart rate (bpm)	>100	1
History	Melena	Present	1
	Syncope	Present	2
	Hepatic disease	Present	2
	Cardiac failure	Present	2

Total score (0–23). Patients with scores >0 are considered to be at high risk. Permission obtained from Elsevier Ltd © Blatchford, O. *et al*. *Lancet* 356, 1318–1321 (2000).

BUN is high in upper GI bleeding b/c blood will be digested and metabolized into urea. Urea will be high disproportionate to creatinine

Score Above 0 is considered high risk and has to be admitted.

B Rockall Score

		Variable	Points
Complete Rockall Score	Clinical Rockall Score	Age	
		<60 yr	0
		60–79 yr	1
		≥80 yr	2
		Shock	
		Heart rate >100 beats/min	1
		Systolic blood pressure <100 mm Hg	2
		Coexisting illness	
		Ischemic heart disease, congestive heart failure, other major illness	2
		Renal failure, hepatic failure, metastatic cancer	3
		Endoscopic diagnosis	
		No lesion observed, Mallory–Weiss tear	0
		Peptic ulcer, erosive disease, esophagitis	1
		Cancer of upper GI tract	2
		Endoscopic stigmata of recent hemorrhage	
		Clean base ulcer, flat pigmented spot	0
		Blood in upper GI tract, active bleeding, visible vessel, clot	2

Not used

AIMS65 Score

Risk Factor	Points
Albumin <3.0 g/dL	1
INR >1.5	1
Altered mental status	1
SBP ≤90 mm Hg	1
Age >65 y	1

The following factors affect the risk of rebleeding and death:

- Age
- Evidence of comorbidity, e.g. cardiac failure, ischaemic heart disease, chronic kidney disease and malignant disease
- Presence of the classical clinical features of shock (pallor, cold peripheries, tachycardia and low blood pressure)
- Endoscopic diagnosis, e.g. Mallory–Weiss tear, peptic ulceration.
- Endoscopic stigmata of recent bleeding, e.g. adherent blood clot, spurting vessel
- Clinical signs of chronic liver disease.

Clinical Approach the the patient

2: Resuscitation If patient is unstable resuscitation is always top priority.

You have to resuscitate the pt immediately or he will go into shock.
Blood transfusion is important but you can't wait too long.

a. **Supplemental oxygen**

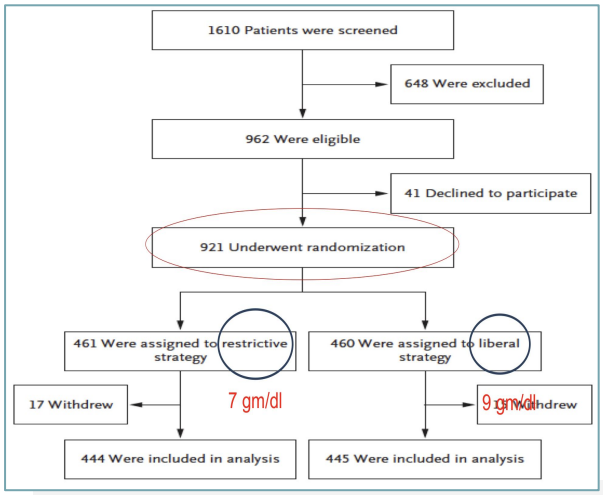
b. **Hemodynamic status**

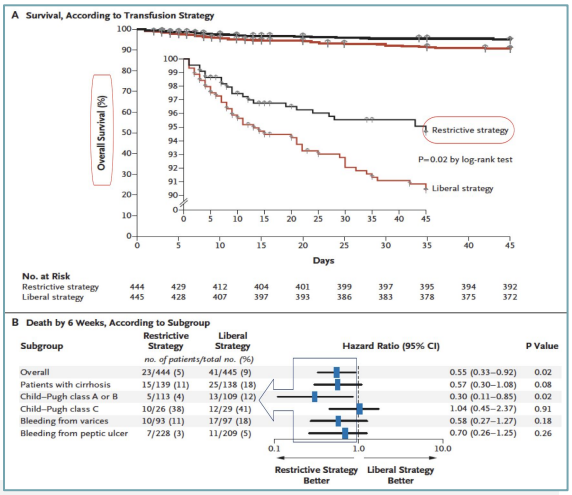
Adequate venous access, 2 large-bore peripheral venous lines (16 or 18 gauge). Isotonic intravenous fluids **(Normal saline solution)** for patients with evidence of hemodynamic instability. A bolus of 500 mL of IV isotonic fluid should be given and repeated as necessary to achieve hemodynamic stability. At the same time draw blood for hemoglobin and hematocrit, PT, PTT and platelet count.

- Blood Transfusions: The role of transfusion in clinically stable patients with mild GI bleeding remains controversial, with uncertainty at which hemoglobin level transfusion should be initiated. Literature suggesting poor outcomes in patients managed with a liberal transfusion. The restrictive RBC transfusion had significantly improved survival and reduced rebleeding.

➔ Packed red blood cells: If the hemoglobin level < 7 g/dL or If hemoglobin < 10 g/dL in patients with preexisting cardiovascular disease or patients with symptoms.

➔ Fresh frozen plasma: if PT or INR is elevated





As you can see in the graph patients who underwent restrictive transfusion (meaning only transfusion when their HB<7) had better outcomes and decreased mortality . while liberal(transfusing blood even if the hb is >7) transfusion didn't really help.
more than 7 in a previously healthy patient and more than 10 in an old patient with comorbidities.

- Patients receiving anticoagulants correction of coagulopathy is recommended , just temporarily.
- **80% of GI bleeding will stop spontaneously if the fluid resuscitation is adequate and only need supportive therapy**

Bleeding could be intermittent or duodenal, that's why nasogastric lavage will not detect it.

Initial resuscitation
- Maintain airway, breathing, and circulation
- Ensure large-bore intravenous access and consider monitored setting
- Resuscitate initially with crystalloid solution
- Send blood work including CBC, coagulation studies, and type and cross-matching

⬇

Transfusion requirements
- Transfuse red blood cells only if hemoglobin <70g/L, unless symptoms of anemia or significant cardiac disease
- Transfuse platelets only if platelet count <50 x 10^9/L or <100 x 10^9/L with suspected platelet dysfunction

⬇

Pre-endoscopic therapy
- Provide erythromycin intravenously 30 minutes prior to endoscopy
- High-dose intravenous proton pump inhibitors should be initiated
- The routine use of nasogastric lavage and/or tranexamic acid is not recommended

3: Endoscopy

- The time of endoscopy is not significant in decreasing the mortality. Most IMPORTANT thing is to stabilize the patient.
- Definition of early endoscopy: ranges from 6 to 24 hours AFTER INITIAL PRESENTATION
- Endoscopy should not be delayed for a high INR unless the INR is supratherapeutic.
- Endoscopy may need to be delayed or deferred :
    1. Active acute coronary syndrome
    2. Suspected perforation (X-ray to exclude perforation)
- Within 24 hours after appropriate resuscitation and transfusion as needed, to a hemoglobin level greater than 7 g/dL

- In high-risk endoscopic findings > give IV PPI bolus (at a dose of 80 mg) followed by a continuous infusion (8 mg per hour) for 72 hours. This will reduce the risk of further bleeding and the need for surgery.
- If bleeding recurs after first scope repeat endoscopy if failed again Transarterial therapy (Injections, Clipping, Thermal therapy or powder spray) or surgery. Nobody knows about the mechanism of the powder. Also, it's not FDA approved drug.

- High-risks lesions are those that spurt blood (Forrest grade IA, Panel A), ooze blood (grade IB, Panel B), contain a non bleeding visible vessel (grade IIA,Panel C), or have an adherent clot (grade IIB, Panel D).
- Low-risk lesions are those that have a flat, pigmented spot (grade IIC, Panel E) or a clean base (grade III, Panel F).

Hospitalization

- It takes 72 hours for most high-risk lesions to become low-risk lesions AFTER endoscopic therapy.
- 60% -76% of patients who had rebleeding within 30 days AFTER endoscopic hemostasis PLUS high-dose PPI therapy did so within the first 72 hours.

➢ Admission to a monitored setting

For at least the first 24 hours on the basis of risk or clinical condition

1. Hemodynamic instability
2. Increasing age
3. Severe comorbidity
4. Active bleeding at endoscopy
5. Large ulcer size (>2 cm)

Admit to ICU

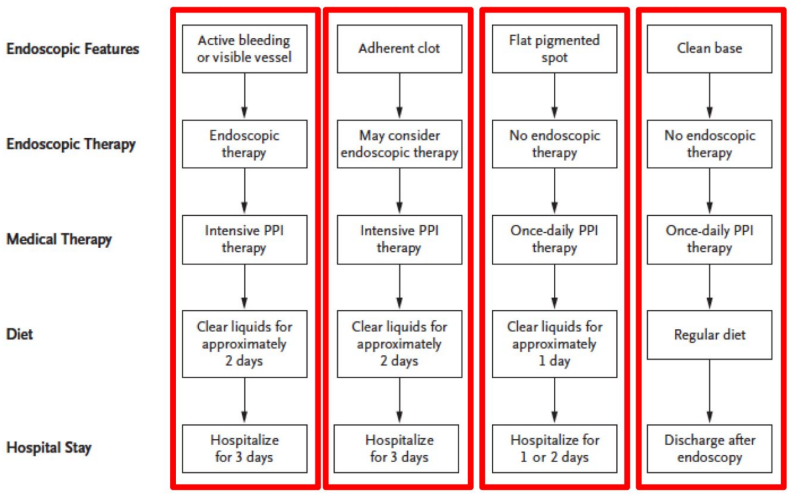


Figure 1. Initial Treatment of Patients with Ulcer Bleeding, According to the Endoscopic Features of the Ulcer.
Intensive proton-pump inhibitor (PPI) therapy is an intravenous bolus (80 mg) followed by an infusion (8 mg per hour) for 72 hours or an oral or intravenous bolus (e.g., 80 mg) followed by intermittent high-dose PPI therapy (e.g. 40 to 80 mg twice daily) for 3 days.[11] The diets shown are diets after endoscopy in patients who do not have nausea or vomiting. The duration of hospital stay after endoscopy is shown in patients who are in stable condition and do not have further bleeding or concurrent medical conditions requiring hospitalization. NEJM 2016

Pharmacological therapy

➢ Pre hospital management

***H pylori*-associated ulcer:**

- No need for continuing PPI therapy after eradication of *H pylori*

NSAID-induced ulcer:

- No need for continuing PPI therapy after discontinuation of NSAID
- If NSAID required consider COX-2 inhibitor with PPI therapy
- Use PPI with low-dose ASA if needed for secondary prevention

Idiopathic ulcers:

- PPI therapy should be prescribed indefinitely

➢ Patients with Moderate / Large Varices that have NOT Bled

Therapy	Dose	Therapy goals	Maintenance/follow-up evaluation
Propranolol[a]	20 mg orally twice a day Adjust every 2–3 days until treatment goal is achieved[a] Maximal daily dose should not exceed 320 mg	Maximum tolerated dose Aim for resting heart rate of 50–55 beats per minute	At every outpatient visit make sure that patient is appropriately β-blocked Continue indefinitely No need for follow-up EGD
Nadolol[a]	40 mg orally once a day Adjust every 2–3 days until treatment goal is achieved[a] Maximal daily dose should not exceed 160 mg	As for propranolol	As for propranolol
Carvedilol	Start with 6.25 mg once a day After 3 days increase to 12.5 mg Maximal dose should not exceed 12.5 mg/day (except in patients with arterial hypertension)	Systolic arterial blood pressure should not decrease <90 mm Hg	
EVL[b]	Every 2–4 weeks until the obliteration of varices	Obliteration varices Eradication of new varices after initial obliteration	First EGD performed 1–3 months after obliteration and every 6–12 months thereafter

NOTE. Only 1 of the 4 therapies shown in the table are recommended.
[a]Dose titration is feasible in 1–2 weeks in settings where a medical assistant is available to check the patient's heart rate. In the case of carvedilol, the dose is fixed at a maximum of 12.5 mg/day so no titration is necessary.
[b]EVL is unlikely to prevent other complications of portal hypertension.

Patients who recently diagnosed with cirrhosis should start primary prevention by giving them propranolol to decrease portal pressure, thus, decreasing the risk of varices progression and bleeding.

➢ Most Commonly Used Vasoactive Agents in the Management of Acute Hemorrhage

Drug	Standard dosing	Duration	Mechanism of action
Somatostatin	Initial IV bolus 250 mcg (can be repeated in the first hour if ongoing bleeding) Continuous IV infusion of 250–500 mcg/h	Up to 5 days	Inhibits vasodilator hormones similar to glucagon, causing splanchnic vasoconstriction and reduces portal blood flow Facilitates adrenergic vasoconstriction
Octreotide (somatostatin analogue)	Initial IV bolus of 50 mcg (can be repeated in first hour if ongoing bleeding) Continuous IV infusion of 50 mcg/h	Up to 5 days	Same as somatostatin, longer duration of action
Terlipressin (vasopressin analogue)	Initial 48 hours: 2 mg IV every 4 hours until control of bleeding Maintenance: 1 mg IV every 4 hours to prevent re-bleeding	Up to 5 days	Splanchnic vasoconstriction The active metabolite lysine-vasopressin is released gradually over several hours in tissue, thus decreasing typical systemic vasopressin side effects

Octreotide causes vasoconstriction and decrease risk of bleeding.

➢ Drugs used in Management of Acute Esophageal Variceal Hemorrhage

Regimen	Dose	Duration	Follow-up
Vasoconstrictor			
Octreotide	Intravenous 50-μg bolus, followed by infusion of 50 μg/h	2–5 d	Bolus can be repeated in first hour if variceal hemorrhage uncontrolled; if rebleeding occurs during therapy, consider TIPS
Terlipressin	2 mg given intravenously every 4 h for first 48 h, followed by 1 mg given intravenously every 4 h	2–5 d	If rebleeding occurs during therapy, consider TIPS
Somatostatin	Intravenous 250-μg bolus, followed by infusion of 250–500 μg/h	2–5 d	Bolus can be repeated in first hour if variceal hemorrhage uncontrolled; if rebleeding occurs during therapy, consider TIPS
Antibiotic			
Ceftriaxone	Intravenous ceftriaxone at a dose of 1 g once a day	5–7 d or until discharge	No long-term antibiotics unless spontaneous bacterial peritonitis develops
Norfloxacin	400 mg given orally twice a day	5–7 d or until discharge	No long-term antibiotics unless spontaneous bacterial peritonitis develops

Antibiotics are given as prophylaxis to prevent SBP with ascites "spontaneous bacterial peritonitis"

➢ H. Pylori

Commonest cause of GIB & major cause of carcinoma. prevalence is varying with different country. Depending on the hygiene.

- Patients with bleeding peptic ulcers should be tested for H. pylori
  - Ieceive eradication therapy if present
  - Confirmation of eradication
- Negative H. pylori diagnostic tests obtained in the acute setting should be repeated.
- Eradication of H.pylori infection and confirm eradication after therapy with breath test or stool test. Stop PPI for at least 2 weeks. Stop bismuth or antibiotics for at least 4 weeks. H2-receptor antagonists are permissible .
- Discontinue NSAIDs permanently if possible. If must be resumed a combination of COX-2 selective NSAID and PPI

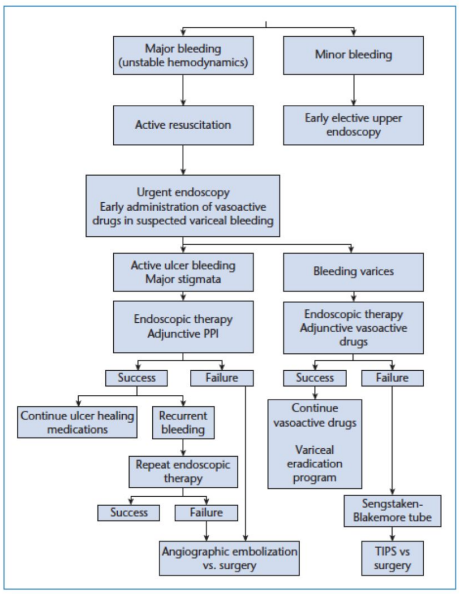
Summary of GI Bleeding Approach: **SUM UP**

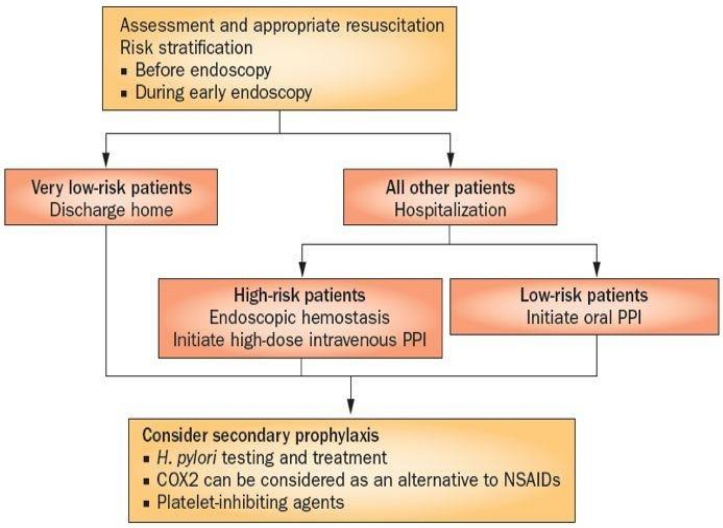
1. Initial assessment detailed history
2. Hemodynamic status and resuscitation physical examination 1/tachycardic 2/hypotensive
3. Blood transfusions
4. Risk assessment and stratification Anemia Hb<7 transfer blood + Uremia .
5. Pre-endoscopic medical therapy PPI (will stop the bleeding ulcer) + Octreotide (vasoconstriction the bleeding vessels)

   (both IV), so we can see clearly while scoping.
6. Timing of endoscopy
7. Endoscopic therapy clipping, banding, cauterizing.
8. Post-endoscopy

Algorithm for the management of acute GI bleeding

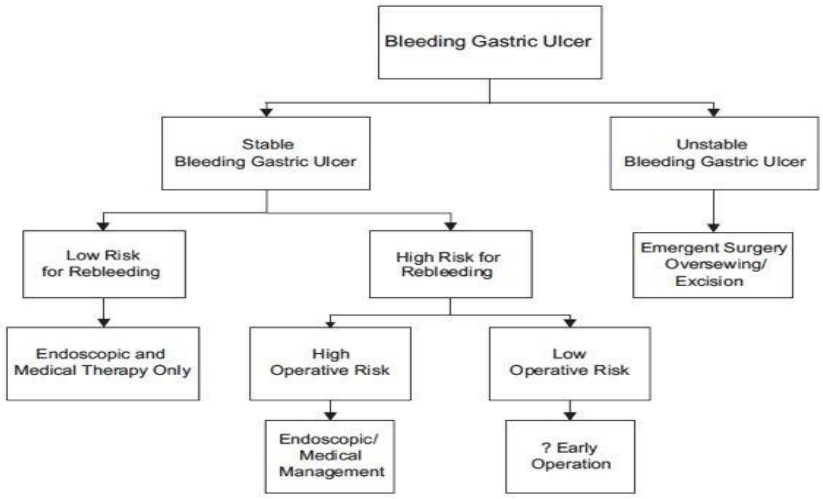
DON'T SKIP, READ IT !

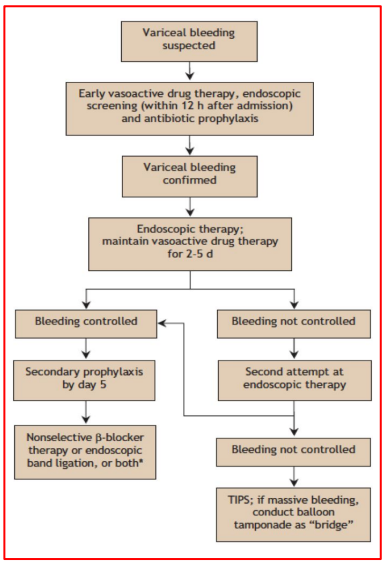


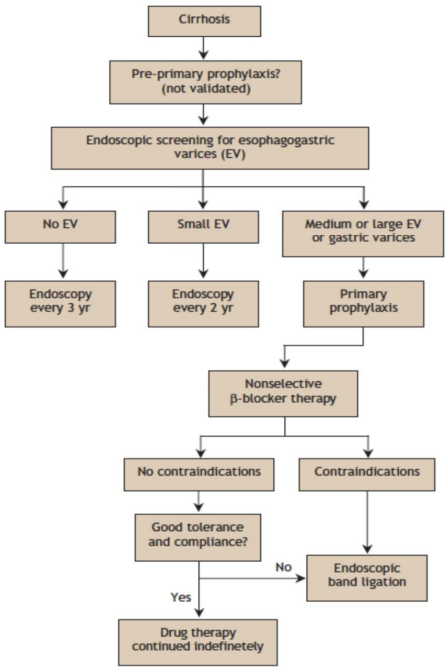


A young man who is known to have heartburn, with no comorbidities came with minor bleeding, book him and early elective endoscopy, you don't have to admit him.

When to go to surgery?







Conclusions

- Resuscitation should be initiated prior to any diagnostic procedure
- Gastrointestinal endoscopy allows visualization of the stigmata, accurate assessment of the level of risk and treatment of the underlying lesion
- Intravenous PPI therapy after endoscopy is crucial to decrease the risk of cardiovascular complications and to prevent recurrence of bleeding
- Helicobacter pylori testing should be performed in the acute setting

Case 1

A **65** y/o (old) male referred for evaluation of 4 months (chronic) HX of weight loss, (alarming symptom) fatigue and weakness (anemia) . He also gave history of passing dark stool intermittently for the last 3 months. He is known DM on insulin, hyperlipidemia on statin and occasionally aspirin.

ESSENTIALS OF DIAGNOSIS

- Symptoms: Coffee ground vomiting, hematemesis, melena, hematochezia, anemic symptoms
- Past medical history: Liver cirrhosis, use of non-steroidal anti-inflammatory drugs
- Signs: Hypotension, tachycardia, pallor, altered mental status, melena or blood per rectum, decreased urine output
- Bloods: Anemia, raised urea, high urea to creatinine ratio
- Endoscopy: Ulcers, varices, Mallory-Weiss tear, erosive disease, neoplasms, vascular ectasia, and vascular malformations

What else do you want to ask him?

1. Trauma (abdominal aortic aneurysm) but not suitable with Hx of 3 months
2. Other symptoms like odynophagia or dysphagia (with solids or fluids) for esophageal pathology\ abdominal symptoms \ past medical "reflux" \ other GIB symptoms (in the 'essentials of diagnosis' table)
3. **Anemic symptoms:** fatigue, SOB, dizziness, palpitation.
4. **Hypotension:** in severe presentation not like this case (3 months)
5. **Raised urea:** b\c of Hgb degraded in GIT then reabsorbed as urea. So high urea in relation to creatinine telling me that it's not AKI.

- **What is the likely diagnosis?** Gastric cancer
- **What will be the next step?** Endoscopy

A 69-year-old woman comes to the ER with multiple red/black stools over the last day. Her past medical history is significant for aortic stenosis. Her pulse is 115 per minute and her BP is 94/62 mm Hg. The physical examination is otherwise normal. What is the most appropriate next step in the management of this patient?

A. Colonoscopy
B. NGT
C. Upper endoscopy
D. Bolus of normal saline
E. CBC

Answer: D. The precise etiology of severe GI bleeding is not as important as a fluid resuscitation. There is no point in checking for orthostasis with the person's systolic BP under 100 mm Hg or when there is a tachycardia at rest. Endoscopy should be performed, but it is not as important to do first as fluid resuscitation. When BP is low, **normal saline or Ringer lactate** are better fluids to give than 5% dextrose in water (D5W). D5W does not stay in the vascular space to raise BP as well as NS.

Case 2

A **42** years old male complaining of chronic recurrent epigastric pain which worsen recently especially when he is fasting (may indicate duodenal ulcer) For the last 2 days he started to have frequent vomiting associated with blood. He is not known to have any chronic medical problems and not on any medications.

- **What is the best next step in the approach of such patient?**
  - It's an **acute** presentation so start with ABC
  - Detailed HX, Full Physical examination (vital sign, lock for clubbing, spider nevi, fluid thrill, splenomegaly, lymph nodes…)
- **How would you assess the bleeding severity? By Risk Stratification**
  - Glasgow- Blatchford Score (GBS) the classical one and most commonly used and most accurate.
  - Rockall Score
  - Modified-GBS
  - AIMS65 (A= albumin. I= INR, M= mental status, S=sBP, 65=age) , easiest to remember
- **What is the diagnosis and the associated risk factors?** Peptic ulcer "duodenal", All these are consider risk factors.

Age >65
Previous peptic ulcer
Previous ulcer-related upper GI complication
High-dose NSAIDs
Multiple NSAID use
Selection of NSAID (e.g., COX-1 vs. COX-2 inhibition)
NSAID-related dyspepsia
Aspirin (including cardioprotective dosages)
Concomitant use of
NSAID plus low-dose aspirin
Oral bisphosphonates (e.g., alendronate)
Corticosteroids
Anticoagulant or coagulopathy
Antiplatelet drugs (e.g., clopidogrel)
Selective serotonin reuptake inhibitor
Chronic debilitating disorders (e.g., cardiovascular disease, rheumatoid arthritis)
Helicobacter pylori infection
Cigarette smoking
Alcohol consumption

[a]Combinations of risk factors are additive.

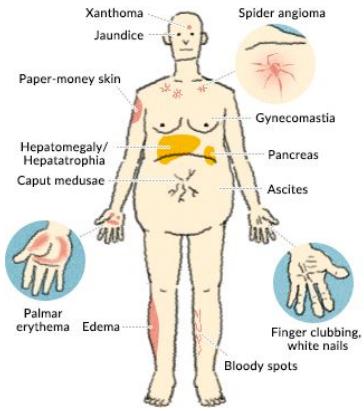
Data from references 1, 12–15, 20, and 29.

Case 3

A 52 years old lady presented to ER with one day history of vomiting of fresh blood (hematemesis). She also notices passing black tarry stool (melena). She is feeling **dizzy and unwell** (severity). Past HX of jaundice no other medical problems and not on any medications. Clinically jaundiced and pale. Vital signs BP 100/70 pulse **110/min** Abdomen examination showed liver span of 7 cm and spleen felt 3 fingers below costal margin (enlarged, normally not felt) with few spider nevi seen over chest. Clearly she has liver disease, which increases the risk of GI varices and eventually bleeding

Case 3

- **What is the likely diagnosis of this case and list 4 common aetiology ?**
  - Drug induced hepatitis (alcohol, acetaminophen)
  - Viral hepatitis B, C
  - Autoimmune hepatitis
  - NASH
  - hemolysis disease (Sickle cell..)
- **What is the priority in the management of this patient?** IV Fluid Resuscitation
- **What is the target Hb and INR prior to the endoscopy for this case?** Target Hb is 7 g/dL.



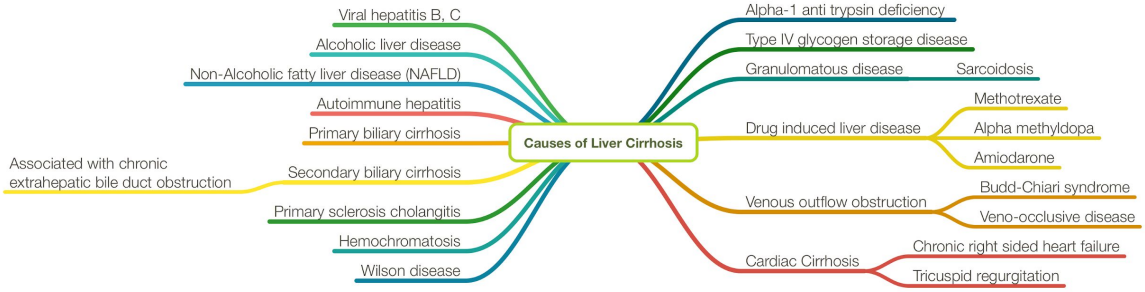
Symptoms of liver cirrhosis

- General malaise, fatigue
- Anorexia / weight loss
- Feeling of enlarged abdomen
- Swollen abdomen / legs
- Nose bleed / bleeding from lower limbs
- Jaundice / itch
- Hand tremors

Physical findings

- Skin pigmentation
- Xanthoma
- Spider angioma
- Palmar erythema
- Finger clubbing (hepatopulmonary syndrome)
- Caput medusae
- Gynecomastia
- Fever
- Hepatoceleoma
- Hepatic halitosis (dimethyls -ulphide, ketons in the expired breath)
- Jaundice
- Ascites, lower thigh edema
- Hepatic encephalopathy
- Bleeding plaque / purpura

Akuko Wakuta etc., Hepatobiliary and pancreas, 73(6), 979-984, 2016 (Partially modified)



Case 4

A 47 years old male known to have alcoholic liver disease presented with hematemesis of large amount and dizziness after resuscitation an upper GI endoscopy done which showed multiple large esophageal varix which was banded, however 12 hrs post endoscopy he continued to have melena with drop of Hb and hypotension.

- **What is the next step in the patient management?** since it's persistent you can do surgery .

Summary

GI Bleeding		
	Upper GI Bleed	**Lower GI Bleed**
Etiology	**1. Peptic ulcer disease** 2. Esophagitis, gastritis, duodenitis 3. Variceal bleeding 4. Mallory weiss tear 5. Dieulafoy's lesion (vessel that bleed and disappear) 6. Malignancy	**1. Diverticular disease** 2. Angiodysplasia 3. IBD 4. Colorectal carcinoma 5. Colorectal adenomatous polyps
Clinical features	● Type of bleeding: ○ Hematemesis → vomiting fresh red blood ○ "Coffee grounds" emesis → upper GI bleed with low rate of bleeding ○ Melena → black, tarry, foul smelling / suggest **upper GI bleed 90%** of time. ○ Hematochezia → usually a lower GI source, may result from **massive upper GI bleed (5 - 10%).** ● Sign of **volume depletion** (tachycardia, hypotension, low urine output, etc..) ● Symptoms and signs of **anemia** (fatigue, pallor, exertional dyspnea, etc..)	
Diagnosis	● Laboratory tests: ○ CBC → hemoglobin, hematocrit level. ○ Coagulation profile ○ LFTs and renal function ○ BUN-creatinine ratio ● **Upper endoscopy** ● Nasogastric tube ● Colonoscopy ● Arteriography	
Management Approach	1. Risk assessment (**AIM65 score**). 2. Resuscitation: Hemodynamic status (give Packed RBC if **Hb < 7 g/dL**). 3. Endoscopy (early endoscopy from 6 to 24 hrs after initial presentation) → may be delayed if pt has active ACS or suspected perforation (do x-ray to exclude). 4. Prevent recurrence.	