

UPPER AND LOWER GI BLEEDING

Done by group B3

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UPPER GI BLEEDING

- Bleeding that arise above the ligament of **Treitz** including the Esophagus, Stomach and duodenum
- The ligament of **Treitz** is a thin band of tissue (peritoneum) that attaches the duodenojejunal flexure to the right crus of the diaphragm

UPPER GI BLEEDING

LIGAMENT OF TREITZ:



- **Hematochezia** bright fresh blood from the rectum due to a **massive UGIB**
- **Melena**: Black tarry stool
- **Hematemesis** or coffee ground emesis
- Signs of **shock**: hypotension, tachycardia

CLINICAL PRESENTATION

- **Esophagus:**

- Esophageal varices
- Mallory Weiss tear
- Esophagitis
- Esophageal mass

- **Stomach:**

- Gastric ulcer
- Gastric mass

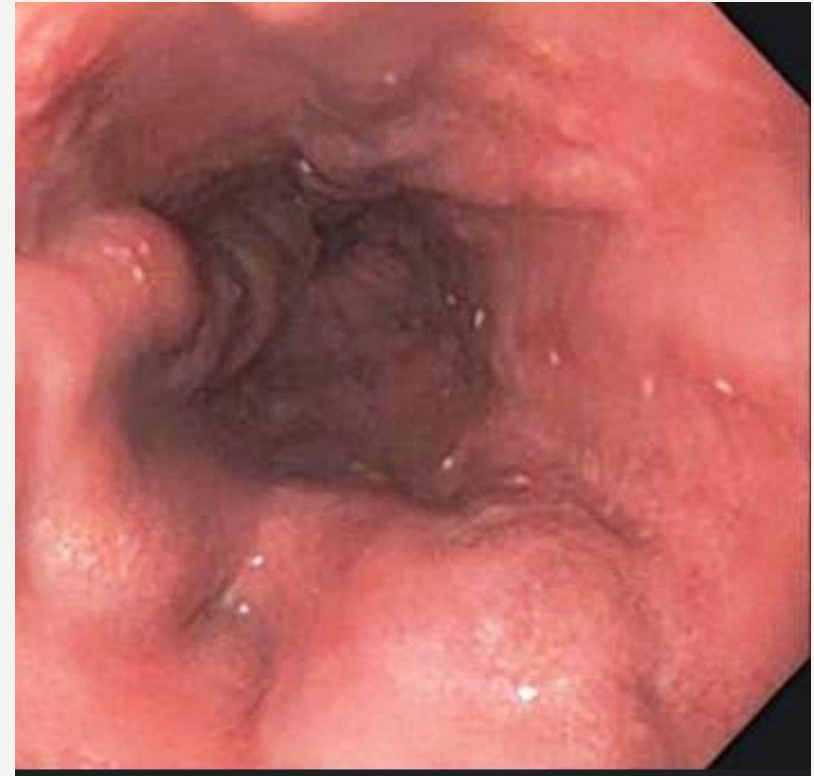
- **Duodenum:**

- Duodenal ulcer

CAUSES

ESOPHAGEAL VARICES

- Portal hypertension increases the pressure in the left gastric vein causing a backflow into the submucosal veins leading to dilation and formation of varices making them prone to rupture and life-threatening bleeding
- Assess patient for cirrhosis.



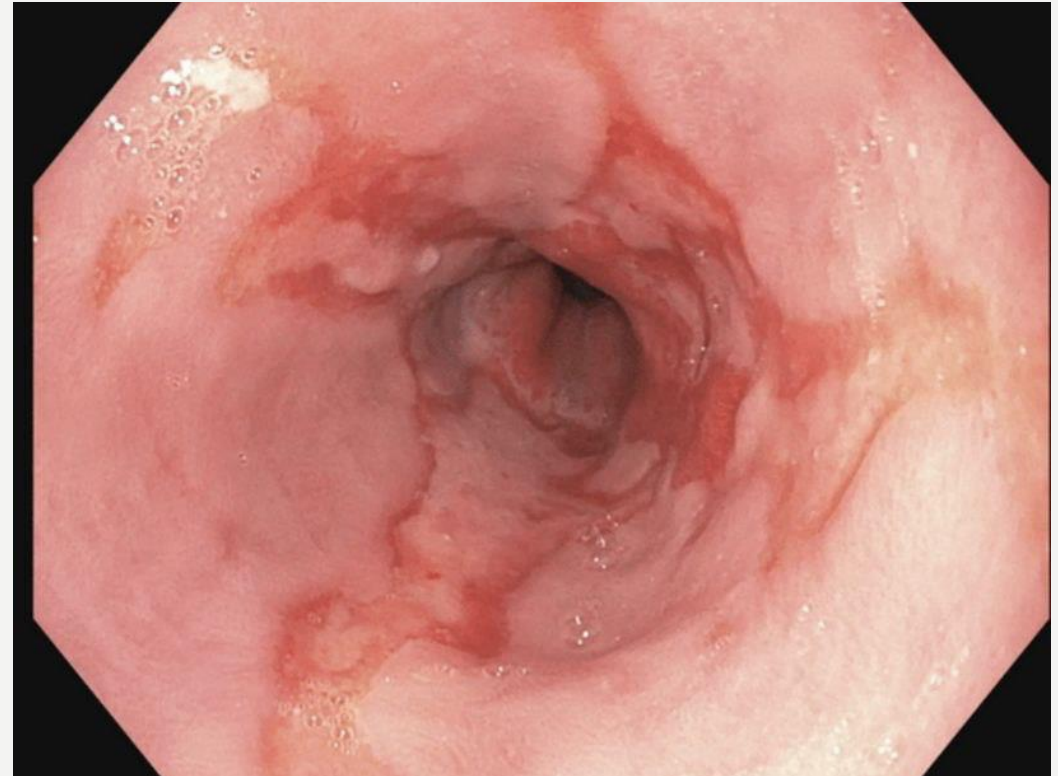
MALLORY WEISS SYNDROME

- it's a longitudinal esophageal tear caused by increased intra- esophageal pressure
- most often caused from violent coughing or vomiting
- It's commonly associated with drinking a lot of alcohol but any disorders that cause vomiting can induce the mucosal tear



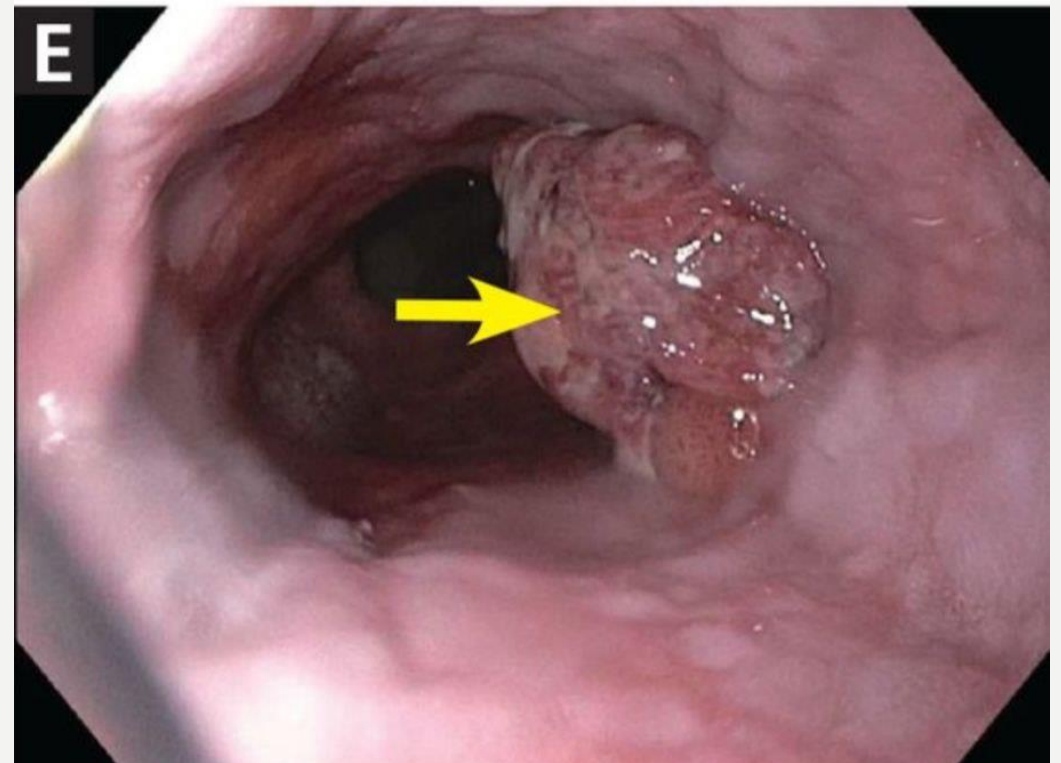
ESOPHAGITIS

- it's a mucosal injury(erosion) in the esophagus
- Assess for prior history of GERD or medications as NSAIDS ,Anti-inflammatories or chemical intake



ESOPHAGEAL MASS

- Esophageal mass with rich vascular blood supply ruptured into the lumen
- Assess the constitutional symptoms , dysphasia and anemia



PEPTIC ULCER

Comparison Between Duodenal Ulcer and Gastric Ulcer

Feature	Duodenal Ulcer	Gastric Ulcer
Age	Peak from 30 to 50 years of age	Increases with age (> 50 years)
Blood Group	Common in group O	Common in group A
Cause	Associated with increased serum pepsinogen caused by H. pylori infection	Less related to H. pylori than duodenal ulcers (about 80%), but NSAID use increases the risk of gastric ulcers by three to four folds.
Most Common Site	First part of the duodenum	Lesser curvature of the stomach
Complications	- Anteriorly: perforation - Posteriorly: bleeding from gastroduodenal artery	- Perforation - Bleeding from left gastric artery
Pain		
Pain Location	Above the umbilicus to the right side of the midline	Around the middle line in the epigastric region
Pain Time	Starts 2-3 hours after meal & persists until the next meal (hunger pain)	Usually starts 30m - 1 hour after meal (related to food)
Food Effect	Fasting, antacids, food	Food, vomiting, less relieved by antacids
Appetite	Good	Bad
Weight	Weight gain	Weight loss → Sitophobia (cibophobia): fear of eating because it causes pain (PYQ)
Nausea/Vomiting	Does not occur except in pyloric stenosis	Very common as it relieves pain
Additional Notes		10-20% of patients with a gastric ulcer have a concomitant duodenal ulcer

PEPTIC ULCER

- Causes:
- NSAIDs
- H-pylori(mc cause)
- Risk factor:
- smoking
- alcohol abuse
- emotional stress and dietary factors



HELICOBACTER PYLORI

The prevalence of *H. pylori* is high in developing countries (80–90% of the population) and much lower (20–50%) in developed countries. Infection is usually acquired in childhood; although the exact route is uncertain, it may be faecal–oral or oral–oral.

H.Pylori

- **Gram-negative**
- **spiral-shaped bacterium**
- **circular, double-stranded DNA genome**
- **Slow growing**

This genome contains the genes necessary for its survival in the acidic environment of the stomach:

- **flagella for movement**
- **surface adhesins**
- **urease production**
- **and virulence factors like CagA and VacA that contribute to its pathogenicity**

1- For survival in gastric environment

- Urease Production:

The bacteria form alkaline microenvironment around itself through urease enzyme .

- Mucus Penetration:

It uses its flagella to move through the acidic gastric juice and burrow into the protective mucus layer, where the pH is more neutral.

- Adherence to Gastric Epithelial Cells :

H. pylori uses adhesins to bind to gastric epithelial cells and establish a stable colonization .

This adherence helps the bacteria resist being flushed out by stomach acid.

1. CagA (Cytotoxin-Associated Gene A)

leading to:

- Increased inflammation (stimulating IL-8 production).
- Altered cell shape (elongation) and function.
- Increased risk of gastric cancer.

2. VacA (Vacuolating Cytotoxin A)

VacA is a toxin that forms pores in host cell membranes, leading to vacuole formation (cellular damage) .It also helps the bacteria evade the immune system by suppressing T-cell activity.

H. pylori strains that carry **CagA** (CagA-positive strains) are more virulent and have a stronger link to gastric ulcers and stomach cancer.

2- Production and secretion of Virulence Factors (**VacA and CagA**)

Disruption of the mucosa and causing gastritis , which lead to :

Corpus-predominant H. pylori infection → disruption of parietal cells
→ less acidity → Gastric atrophy and cancer risk.

Antral-predominant H. pylori infection → Increased gastrin production →
High acid → ulcers (duodenal mostly)

** chronic gastritis

** usually asymptomatic

- Gastric cancer:

1. Gastric adenocarcinoma .

2. Gastric B-cell lymphoma;

Over 70% of patients with gastric B-cell lymphomas(MALT) have H. pylori.



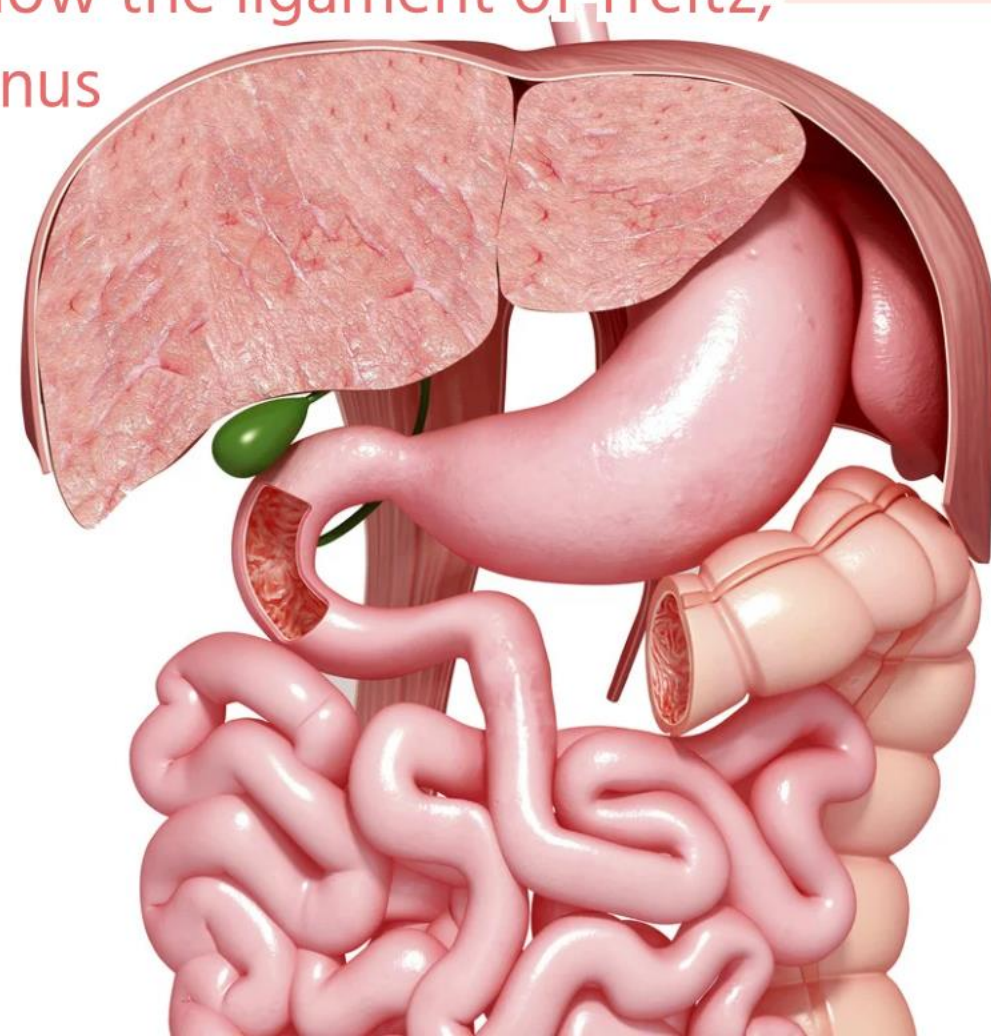
LOWER GI BLEEDING

1. INTRODUCTION

Definition: LGIB refers to bleeding occurring below the ligament of Treitz, involving the jejunum, ileum, colon, rectum, or anus

Epidemiology:

- Accounts for ~20% of all GI bleeds.
- More common in older adults.

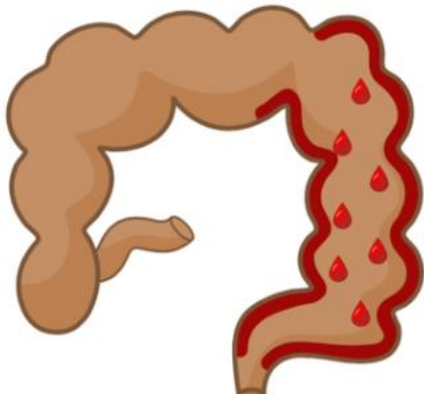


2. CAUSES OF LGIB

a) Large Intestinal Bleeding:

1. IBD

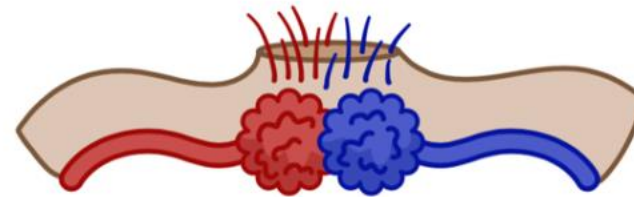
- Erosions/ulcers of **ileal mucosa (Crohn's)** or **colonic mucosa (Ulcerative Colitis)** extends to intestinal arteries
 - Assess for prior history of IBD
 - RLQ pain, watery diarrhea with fistulas, abscess, malabsorption, and strictures → **Crohn's Disease**
 - LLQ pain, Tenesmus, Colitis, or Toxic Megacolon → **Ulcerative Colitis**



*History of UC
(Hematochezia,
LLQ pain,
Tenesmus)*

2. AVM

- Nidus of artery-vein network ruptures into lumen of large intestine (Typically, the right colon)
 - Assess for prior history of **Aortic Stenosis** and **CKD**



*Associated with
aortic stenosis
and CKD*

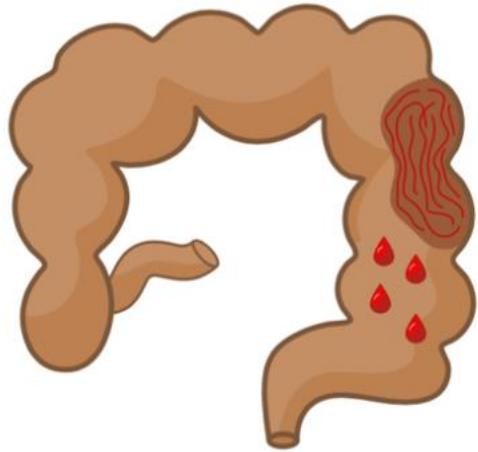


2. CAUSES OF LGIB

a) Large Intestinal Bleeding:

3. Colorectal cancer

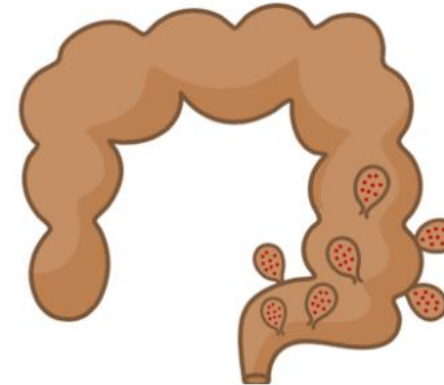
- Colorectal mass with rich vascular blood supply may rupture into lumen
 - Assess for prior history of **weight loss** and **anemia**



Weight loss
and anemia

4. Diverticulosis

- **Diverticular outpouching** shears the arteries in the diverticula wall → Bleeding into the **sigmoid colon lumen**
 - Typically, asymptomatic



Most common
cause

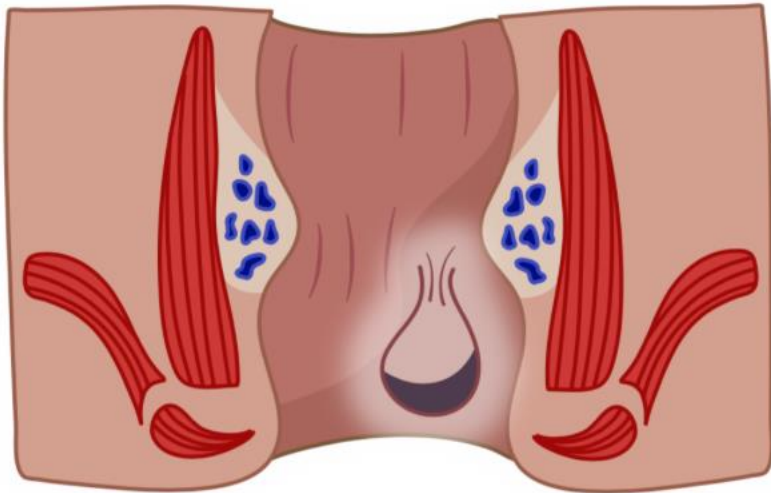


2. CAUSES OF LGIB

b) Anal Bleeding

1. Hemorrhoids

- **Varicose veins** in the anus that rupture and bleed
 - Assess for visible mass in or out of anus that may be painful (e.g. external)



2. Anal fissure



3. CLASSIC FINDINGS

Hematochezia

- Bright red/maroon blood in stool (non-oxidized).
- Suggests active lower GI bleeding.

Other symptoms

- Tenesmus, diarrhea (if due to IBD).
- Painless bleeding (if diverticulosis or AVM).
- Weight loss (if colorectal cancer).



MCQ QUESTION

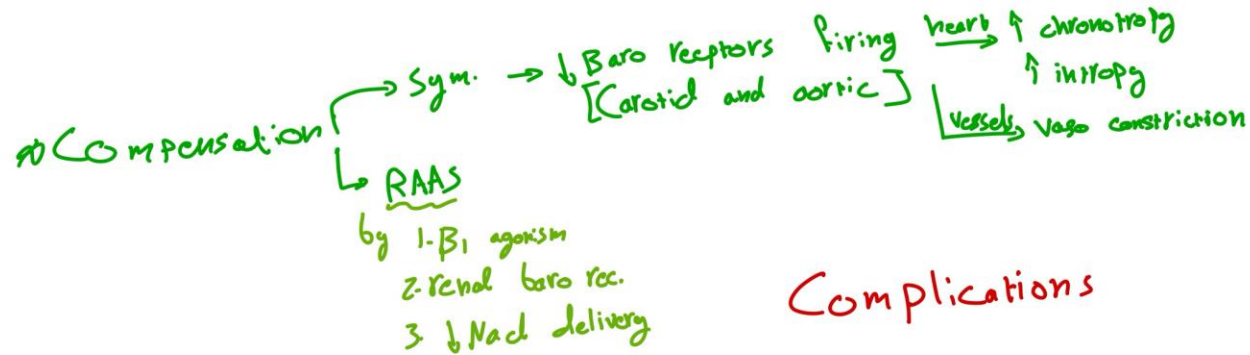
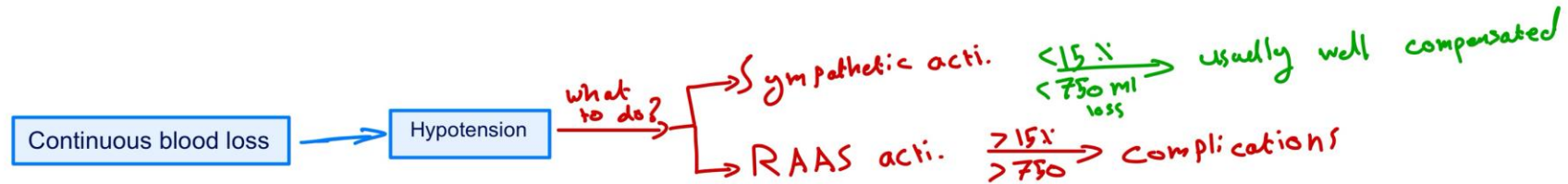
A 65-year-old man presents to the emergency department with bright red blood per rectum. He denies abdominal pain, fever, or recent changes in bowel habits. His medical history is significant for hypertension and chronic constipation. His vitals are stable, and hemoglobin is 11.2 g/dL (baseline 13.5 g/dL). Colonoscopy reveals multiple outpouchings in the sigmoid colon with no active bleeding. What is the most likely cause of his lower GI bleeding?

- A) Arteriovenous malformation (AVM)**
- B) Diverticulosis**
- C) Ulcerative colitis**
- D) Ischemic colitis**
- E) Colorectal cancer**

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COMPLICATIONS OF GI BLEEDING

Compensation or complications?



Complications

↓
Heart
↓
- ↑ HR
- ↑ demand
- ↓ O₂ supply
↓
- ischemia
- exc. of present illness

↓
Brain
↓
- watershed areas ischemia
- Vasogenic edema
- cytotoxic edema

↓
GI
↓
- watershed areas
firstly
Recto-sigmoid and Splenic flexures
↓
- Severe abd. pain and hematochezia.

↓
Kidney
↓
↓ RBF
↓
↓ GFR
↓
↑ Na⁺/H₂O reabs. and Urea.
- Urine osm. > 500
Na⁺ < 20
- Serum Bun/Cr > 20

3/11/2025
* 60 sec. before loss of contractility

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DIAGNOSTIC APPROACH

HISTORY AND PHYSICAL

We need to know :

- What is the patient having
- What is the most probable underlying cause
- What complications the patient is having

HISTORY AND PHYSICAL

In History we focus on

- The presenting complain of the patient (Hematemesis, Hematochezia, Melena...)
- The duration and the onset in terms of what brought it up (vomiting, medications...)
- The color and amount of blood (fresh, bright red, dark...)
- Number of episodes
- Bleeding elsewhere, bruising
- Any symptoms of shock
- Chang in bowel habits, tenesmus, change in stool caliber
- Constitutional symptoms

HISTORY AND PHYSICAL

In History we focus on

- Previous episodes
- PMH (PUD, liver problems, coagulopathy...)
- Medications (NSAIDs, Warfarin...)
- Social history (smoking, alcohol)
- FHx (cancer, liver problems, coagulopathy)

The physical exam could reveal the severity of the bleeding and denote any abnormality that could be related to the causes of GI bleeding

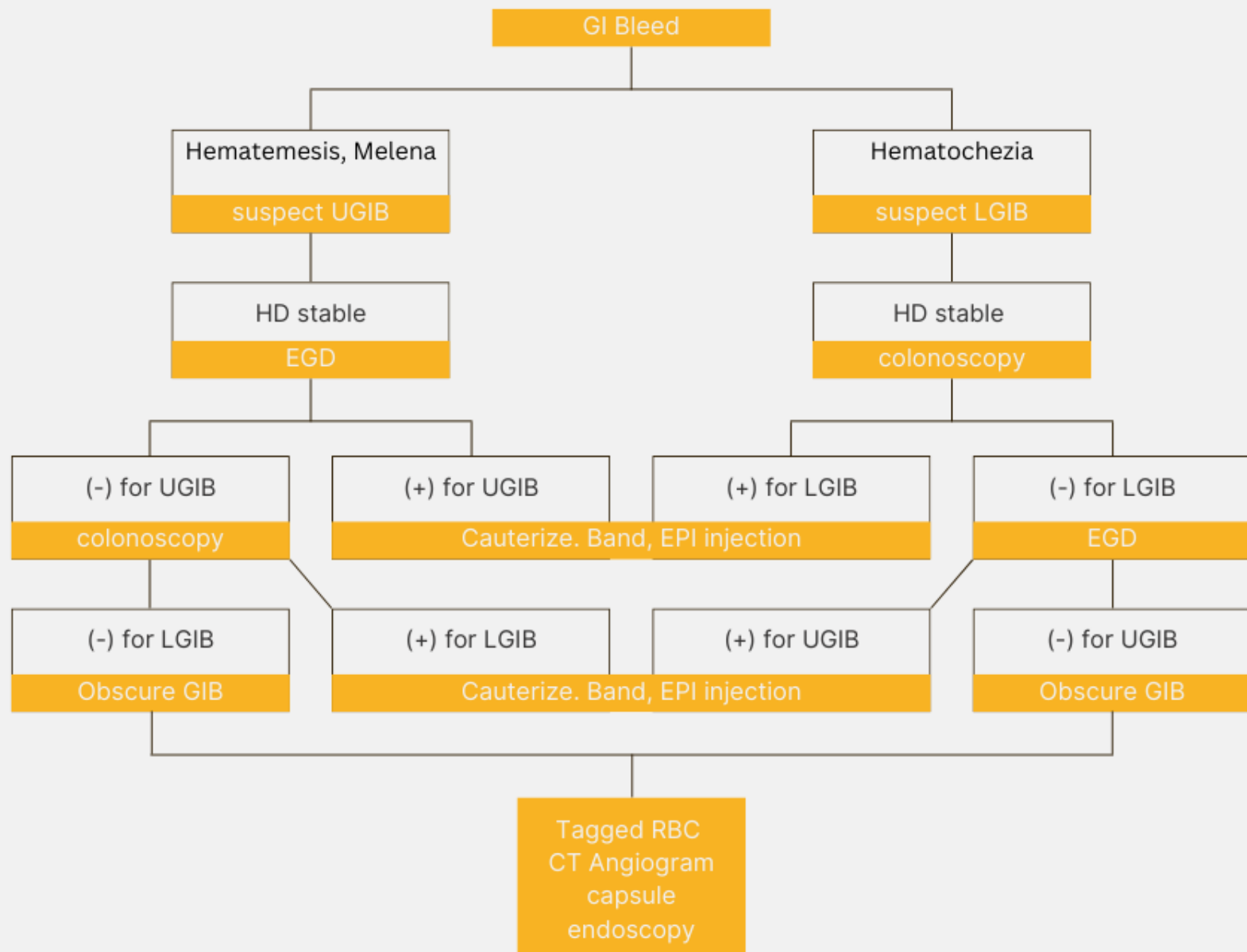
INVESTIGATIONS

A stepwise Approach in the investigation of GI bleeding is directed to assess

- The severity of the bleeding and hemodynamic stability
- Determine the source and the cause of the bleeding

So we start with

- Vital signs
- CBC
- Coagulation profile
- Liver function test





MANAGEMENT

ESOPHAGEAL VARICES TREATMENT

- Initial resuscitation with intravenous fluids and blood transfusions.
- Octreotide infusion initiated to reduce portal pressure.
- Urgent endoscopic band ligation performed to control active bleeding.

MALLORY WEISS SYNDROME TREATMENT

- Surgery, but this is rarely necessary
- Acid suppression is used to promote healing as H₂-receptor blockers ,Proton pump inhibitors

PU TREATMENT

- Discontinue aspirin/NSAIDs.
- Restrict alcohol use
- Stop smoking, decrease emotional stress.
- Avoid eating before bedtime

Acid suppression therapy

- PPIs-omeprazole , lansoprazol.
- H2 receptor blockers-cimetidine and ranitidine . Less effective than PPIs.
- Antacids-somewhat outdated for primary therapy and more appropriately used

for adjunctive therapy/symptomatic relief. Example bismuth subsalicylate (Pepto-Bismol).

- Eradicate *H. pylori* with triple or quadruple therapy
- For initial therapy, **triple therapy** (PPI, amoxicillin, and clarithromycin) for 14 days.
- **Quadruple therapy** (PPI, bismuth, metronidazole, and tetracycline) indicated for patients with risk factors for macrolide resistance.
- Also begin with acid suppression therapy

- H. pylori-negative ulcers that are **NOT** caused by NSAIDs can be treated with antisecretory drugs (either H2 blockers or PPI).

GASTRITIS TREATMENT

- If H. pylori infection is confirmed, antibiotic therapy is indicated
- If stress induced, avoid it and give H2 antagonist

GASTRIC CA TREATMENT

1. Surgical resection with wide (>5 cm) margins (total or subtotal gastrectomy) with extended lymph node dissection.
2. Chemotherapy may be appropriate in some cases.

IBD – CHRON'S TREATMENT

- **Medical**
 - a. Systemic corticosteroids (prednisone)-used as initial therapy for low-risk patients with diffuse disease or left-sided colon disease.
 - b. Budesonide-used in low-risk patients with mild disease in ileum or right colon.
 - c. Biologic agent (TNF inhibitor) can be used as monotherapy or in combination with thiopurine (azathioprine) or methotrexate in patients with moderate to severe disease.
- **Surgical** (eventually required in most patients) Involves segmental resection of involved bowel

IBD –ULCERATIVE COLITIS TRET..

- 5-Aminosalicylic Acid (5-ASA) Compounds (First-Line Therapy)

Sulfasalazine (Topical & Oral)

- Mainstay of treatment for mild to moderate UC.
- Preferred over topical steroids (better for maintenance therapy).

- Corticosteroids (For Moderate to Severe Flares)

- Used when 5-ASA therapy fails.
- Oral (prednisone) for moderate UC.
- IV (methylprednisolone, hydrocortisone) for severe UC

- Immunomodulators & Biologics (For Severe/Refractory UC)

TNF Inhibitors (*Infliximab, Adalimumab, Golimumab*)

- Used in steroid-dependent or refractory UC.
- Infliximab is preferred for severe disease.

Thiopurines (*Azathioprine, 6-Mercaptopurine*)



THANK YOU