







Intra-abdominal & Lower Gastrointestinal Bleeding

Objectives

• Intra-abdominal hemorrhage

The student is expected to describe and explain the etiology and clinical features of the following conditions:

- o Ruptured abdominal aortic aneurysm
- Ruptured spleen
- Ruptured ectopic pregnancy
- Ruptured ovarian cyst
- Ruptured liver adenoma
- Gastrointestinal hemorrhage

- o <u>Ruptured hepatocellular carcinoma</u>
- Ruptured visceral aneurysm (<u>splenic</u>, hepatic and mesenteric)
- Retroperitoneal hemorrhage (over anticoagulation)

The student is expected to describe and explain the etiology, clinical features and complications of the following condition:

Lower gastrointestinal bleeding

- Angiodysplasia
- Diverticulosis
- Inflammatory bowel diseases
- Anorectal conditions
- Colitis (infectious and ischemic)
- Colorectal tumors
- Colorectal polyps
- Meckel's diverticulum (covered in abdominal pain teamwork)

Colour Index

- Main Text
- Males slides
- Females slides
- Doctor's Notes (438)
- Doctor's Notes (439)
- TextbookImportantGolden notesExtra

Summary File

Editing File



Useful resources:



Ninja Nerd's Video (very helpful)



Ninja Nerd's Notes

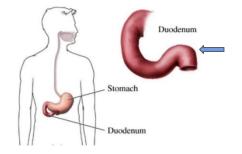


Online Meded's Summary

Terminology		
Ligament of Treitz	Formed by a peritoneal fold between the right crus of the diaphragm and the duodenojejunal flexure.	
Upper GIT bleeding	The bleeding is proximal to the ligament of Treitz. It accounts for more than 80% of acute GIT bleeding. Peptic ulcer disease and variceal bleeding are the most common causes.	
Lower GIT bleeding	The bleeding is distal to the ligament of Treitz. Colon diverticula and angiodysplasia accounting for most of the cases. The small intestine is the source of bleeding in less than 5% of patients.	
Overt GI bleeding	Macroscopically observable bleeding with accompanying clinical symptoms (e.g., anemia, tachycardia)	
Occult GIT bleeding	This is the bleeding which is not apparent to the patients or physician, and the patient present with symptoms of iron deficiency anemia with or without a positive fecal occult blood test. Bleeding in quantities too small to be macroscopically observable (requires chemical tests or microscopic examination to be detected)	
Obscure GIT bleeding	This is the bleeding that recurs or persists after a negative initial evaluation using endoscopy and radiologic imaging.	



Upper VS lower GI bleeding:



Blue arrow Anatomical landmark: 4th part of duodenum is suspended by Ligament of Treitz (anything above its level is considered upper GI and anything below is lower GI)



Another reason for this classification is that upper GI scopes can't exceed the 4th part of duodenum and lower GI scopes can't reach the terminal ileum. (We start the management with scoping) Even though nowadays we have techniques that can scope the small intestines like capsule endoscopy and balloon follow through, their use is limited to diagnosis and biopsies.

"Determination of the site of bleeding is important for directing diagnostic interventions with minimal delay. However, attempts to localize the source should never precede appropriate resuscitative measures"

GI bleedings are a TOP EMERGENCY!

Sebastian textbook of surgery 20th edition





General Approach for trauma and GI bleeding patients:

1. ABCs

Once airway and breathing are ensured, the patient's hemodynamic status becomes the main concern and forms the basis for further management.

2. Assessing magnitude of bleeding

Try to estimate it by asking the patient and through some signs the severity of the bleeding can be determined based on simple clinical parameters:

- Obtundation (reduced level of consciousness)
- Agitation
- Hypotension (systolic blood pressure <90 mm Hg in the supine position)
- Resting heart rate over 100 beats/min
- Cool clammy extremities
- Decreased pulse pressure
- Postural changes in blood pressure should be elicited (fall in BP > 10 mm Hg or increase in pulse
 20 beats/min reflects significant blood loss)

3. Resuscitation

- Use two large-bore intravenous lines.
- If patient is stable, begin resuscitation with isotonic crystalloid solution
- For patients in hemorrhagic shock, initiate a balanced blood component resuscitation.
- Place Foley catheter to facilitate monitoring of intravascular volume status
- When patient with trauma came the ER we give him 1 bolus (2L N.S I.V) STAT (in 15 min)
- Ex. When a patient come with blood pressure of 60/40, that's mean he lost half of his blood volume, you should give him 5-6 blood units on large gauge needle (14)
- The role is whenever a patient loses a fluid it should be replaced with a similar fluid, blood should be replaced with blood, burned patients lost plasma so you should replace it with plasma.



4. History

Onset

Today vs 2 months ago

Frequency

Massive Hemorrhage: It is defined as:

- Continued bleeding with hemodynamic instability (Hypovolemic shock, Irreversible shock "In the ER there
 is 1 golden hour between Hemorrhage and irreversible shock", Multi-organ failure, Acute respiratory
 distress syndrome, Death)
- Signs of poor perfusion
 - HB drop more than 2 gm/dl
 - Transfusion requirement of more than 2 units PRBC in 24 hrs)
 - Blood loss of the patient's circulating volume in a 24-hour period
 - 50% of total blood volume lost in less than 3 hours
 - Bleeding in excess of 150 ml/min

Types

- **Hematemesis** vomiting fresh blood usually caused by upper GIT bleeding although, rarely, bleeding from the pharynx or nose can be responsible. It may be bright red or older (coffee grounds appearance which is due to hemoglobin to methemoglobin oxidation)
- **Melena** passing black dark stool per anus sticky and foul smelling (upper GI or proximal colon bleed) due to oxidizing of iron and partly digested HB by bacteria(heme to hematin)
- The stool greenish character of patients on iron supplements should not be confused with melena. This can be distinguished by performing a guaiac test that detects the presence of fecal occult blood, which tests negative in those on iron supplementation.
- Hematochezia passage of fresh blood per anus bright red bloody stool mainly from <u>distal</u> GIT but it can happen if the patient is suffering from <u>proximal</u> GIT severe bleeding, So first thing you want to rule out massive GI bleeding (IF THE PATIENT IS HYPOTENSIVE AND NOT STABLE, MOSTLY IT'S A MASSIVE UGI BLEEDING, so in this case you do endoscopy not colonoscopy)

Chronic blood loss present with symptoms of anemia

- Fatigue
- Pallor
- Cold extremities (hands and feet)
- Shortness of breath

- Dizziness
- Palpitation
- Syncope, angina, and even myocardial infarction

Present with symptoms of haemorrhage

- Tachycardia (is the FIRST clinical feature of haemorrhage. Even if the patient clinically looks fine)
- Sweating (any patient you see sweating in the ER is in shock)
- Confusion
- Cold extremities (hands and feet)
- Decreased urine output
- Hypotension (the LAST clinical feature of haemorrhage. The range of systolic 100-140, The range of diastolic
 is 60-90, so the cutoff is 100/60. The blood pressure usually starts to decrease after 50% (2.5L) of blood loss)

Types of shock

- Hypovolemic shock
- Low blood pressure and raised blood rate: Mild hypovolemia (<20%), Moderate hypovolemia (20-40%), Severe hypovolemia (>40%)

Click here for hypovolemic shock classification

5. Physical Examination

- Epigastric tenderness = peptic ulcer disease or gastritis
- Stigmata of liver disease jaundice, spider nevi, palmar erythema, flapping tremor and caput medusae may suggest variceal bleeding
- Telangiectasias can be found in any part of GIT
- Masses
- Splenomegaly with portal hypertension
- Adenopathy indicates malignancy
- Rectal examination is a part of the abdominal examination (don't forget for your OSCE and career)

Signs of the presence of blood in the abdomen:

- 1- Abdominal Tenderness
- 2- Distension
- 3- Rebound tenderness

(When you remove your hand. The abdominal wall shakes causing severe somatic pain)

- 4- Guarding and rigidity
- 5- Decreased or absent bowel sounds
- 6- Bleeding PR

6. Localization of the site of bleeding



Nasogastric tube aspiration:

- Insertion of a nasogastric tube and examination of the aspirate is important in distinguishing upper from lower GIT bleeding. Useful to evaluate the rate of ongoing bleeding and to be used for gastric lavage and removal of the blood from the stomach to permit endoscopy.
- Gross blood or coffee grounds appearance: likely upper GI source.
- Bile, but no blood: strongly suggestive of lower GI source.
- Clear nonbilious effluent: cannot rule out a duodenal source of bleeding.
- Although the findings of the nasogastric tube aspirate could be helpful in localizing the source of bleeding, almost all patients with significant GIT bleeding should undergo upper GIT endoscopy.

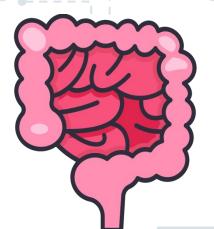


Endoscopy:

- How can we inspect the upper GIT? **EGD**, How far can you go in endoscopy? **To the duodenum**
- How do you inspect the lower GIT? **colonoscopy**, How far can you go in colonoscopy? **To the distal ileum**
- How can you examine the lumen of GIT that the endoscopy and colonoscopy can't reach? By the **capsule endoscopy** (camera takes a lot of pictures then it comes out).

Esophagogastroduodenoscopy (EGD):

- For upper GI source, it has 95% diagnostic accuracy if used within the first 24 hours.
- Should be performed within 24 hours from the arrival of the patient to the emergency, even in stable patients.
- If negative, It direct the attention to a lower GIT source and performance of colonoscopy.
- May also be used as a therapeutic modality: sclerotherapy, thermal coagulation, clipping or banding of varices.



Capsule endoscopy:

- Camera takes a lot of pictures then it comes out.
- Their use is limited to diagnosis and biopsies.
- The appropriate study for obscure bleeding, usually the small bowel where endoscopy and colonoscopy can't reach.

Anoscopy/Proctoscopy/sigmoidoscopy/colonoscopy:

- Overall diagnostic accuracy is up to 97%
- Done in stable patients non-urgently.
- Lack of adequate bowel preparation in the acute setting may render these tests inconclusive.

6. Localization of the site of bleeding cont.

The following measures are used when endoscopy is nondiagnostic



Angiography:

- May be required in cases of massive bleeding from the upper or lower GIT, precluding endoscopy.
- To visualize the bleeding by selective angiography, the bleeding must be relatively brisk (approximately 0.5 to 1.0 mL/minute) "in Davidson greater than 1 mL/min"
- Indicated as the initial test in patients with suspected LGIB and hemodynamically instability refractory to resuscitation and as workup of patients with ongoing bleeding and negative endoscopy.
- Can be used for therapeutic interventions, including vasopressin infusion and embolization of bleeding vessels.



Technetium-labelled RBC scan (tagged RBC scan):

- RBCs scan in nuclear medicine they inject the patient with tagged RBCs and then scan it and it tells you where the site of bleeding is.
- When colonoscopy is nondiagnostic bleeding at a rate 0.1 to 0.5 mL/min is required to identify the source
- Most sensitive test for ongoing bleeding.



CT angiography:

- CT Angiography (give contrast and take pictures in the arterial).
- Used when bleeding source difficult to identify.
- Detects bleeds 0.3 to 0.5 ml/min



Surgery (not easy):

- Consider if other therapeutic options have failed.
- Consider in hemodynamically unstable patients with ongoing bleeding.

Click and watch how exploratory laparotomy aggressive is





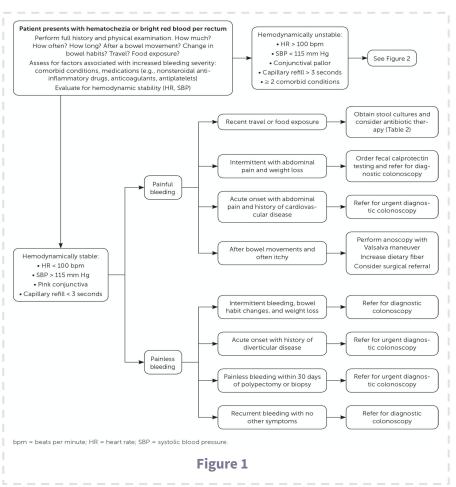
Differential diagnosis (DDx):

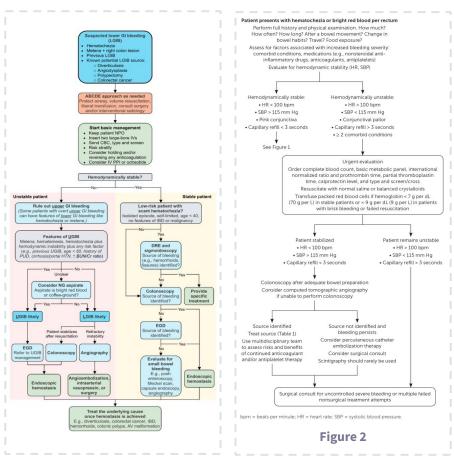
COLONIC BLEEDING	95%	SMALL BOWEL BLEEDING	5%
Diverticular disease	30%-40%	Angiodysplasias	
Anorectal disease	5%-15%	Erosions or ulcers (potassium, NSAIDs)	
Ischemia	5%-10%	Crohn's disease	
Neoplasia	5%-10%	Radiation	
Infectious colitis	3%-8%	Meckel's diverticulum	
Post-polypectomy	3%-7%	Neoplasia	
Inflammatory bowel disease	3%-4%	Aortoenteric fistula	
Angiodysplasia	3%		
Radiation colitis or proctitis	1%-3%		
Other	1%-5%		
Unknown	10%-25%		

Lower GI bleeding

- Digital rectal examination, anoscopy, and/or sigmoidoscopy is the first step in the workup to rule out an anorectal source of bleeding.
- Nasogastric tube insertion which can rule out upper GIT bleeding in most patients if the aspirate contains bile and no blood.
- Subsequent evaluation depends on the magnitude of the bleeding:
 - **Colonoscopy:** It is required when the bleeding is intermediate, where resuscitation and hemodynamic stability permit a more directed evaluation and therapeutic intervention.
 - **Angiography:** It is used as an adjunct to colonoscopy when the patient is hemodynamically stable. Angioembolization and control of bleeding can be performed during the procedure.
 - **Tagged RBC scan:** It can be used to localize the source of bleeding when the bleeding is minimal and intermittent. It is used as an adjunct to colonoscopy.
 - **Surgical intervention:** is required in major and/or persistent bleeding, an unstable patient who continues to bleed and requires ongoing aggressive resuscitation.

Approach to hematochezia or rectal bleeding:





Diverticular Disease

- Chronic constipation which increases pressure inside the lumen causing outpouching in the intestinal wall and when these outpouching raptures it results in bleeding.
- True diverticulum: all the layers are pouching e.g. Mickle's
- Pseudo-diverticulum (False diverticulum): inner layers will pouch on the outer layer.
- ★ Most common cause of significant lower GI bleeding.
- Only 3% to 15% experience any bleeding, 75-80% resolves spontaneously and 10% rebleed within a
 year.
- The right-sided diverticular disease is responsible for more than 50% of the bleeding.
- Most common risk factor is chronic constipation
 - o It is very common.
 - o Mostly stops spontaneously, low rebleeding incidence.
 - Colonoscopy is diagnostic gold standard.
 - o Once a diverticula occur, it never regress "anatomically".



Diagnosis	Best Initial, diagnostic test and treatment is Colonoscopy.
Treatment	 Epinephrine injection, electrocautery, and most recently endoscopic clips. If bleeding is massive or cannot be controlled by endoscopy treatment: Angioembolization (risk for perforation & ischemia) Surgery when other modalities have failed

	Angiodysplasia
	★ Acquired arteriovenous malformations.
	• Secondary to progressive dilation of normal blood vessels within the submucosa of the intestine. 5-10 years of chronic constipation, the blood vessel will stretch and dilate.
Etiology	Rare disease, and if present rarely causes major bleeding.
63	How to differentiate it from Hemangioma?
	 Hema is congenital (A vascular tumor that is typically benign)
	Angio is acquired
	Both are AV malformation
	 Almost uniformly found in patients > 50, 65 years.
	 Associated with aortic stenosis and renal failure. Classic presentation: elderly patient with <u>renal failure</u> or aortic stenosis.
Clinical features	 Hemorrhage tends to arise from the right side of the colon, most common location is the cecum.
icacures	Bleeding stops spontaneously in most cases.
	 Increased pressure will cause the walls of the veins and arteries to open on each other and form a Star-like sign.
Diagnosis	Colonoscopy or angiography can diagnose these lesions and treatment through.
	Colonoscopy: sclerotherapy injection or electrocoagulation.
Trootmont	Angiography: intra-arterial vasopressin, selective gel-foam embolization.
Treatment	• Surgery: If previous therapies fail, or bleeding recurs and the lesion has been localized, segmental colonic resection (most commonly right colectomy) is an effective surgical procedure.

Neoplasia	7
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Lower GI malignancies are common to bleed				
Colorectal carcinoma		Polyps	Gastrointestinal Stromal Tumor (GIST)	Juvenile polyps
 Uncommon cause of significant lower GIT bleeding but is the most important diagnosis to rule out. Painless Intermittent Slow → iron deficiency anemia Rarely right colon cancer is palpable Age >50 (Do colonoscopy even if you see by your eyes bleeding hemorrhoids!) some will also have colorectal cancer. 		 Can bleed usually occurs after endoscopic polypectomy. Most commonly after polypectomy. 	 Anywhere in the GIT Can be associated with massive bleeding. 	 2nd most common cause of spontaneous bleeding in patients 20 years. 1st most common is Meckel's diverticulum.
 Colonoscopy is the best diagnostic tool. If the polyp is the source of bleeding, it can be treated with endoscopic therapy by burning or clipping it. Surgical intervention may be required for bleeding polyp or tumor as an emergency procedure or elective after endoscopic control or spontaneous cessation of bleeding. 				
		ergency		

Internal hemrrhoid (vascular structures in the anal canal) Painless o Often accompanied by prolapsing tissue that reduces spontaneously or has to be manually reduced by the patient. Major causes **Anal fissures:** o A tear in the anal canal extending from below the dentate line to the anal verge. o Painful bleeding after a bowel movement. Pathophysiology: Overdistension or disease of the anal mucosa → laceration of the anoderm Colorectal neoplasia Low-volume bleeding. Bright red blood per rectum seen in the toilet bowl and on the toilet paper. Clinical features Tenesmus: feeling of incomplete evacuation. Chronic hemorrhoids or fissures can cause anemia. It doesn't cause shock. Constipation Obesity **Risk factors** Pregnancy Straining during defecation Liver cirrhosis

		Anorectal diseases
Approach	Auscopy &Exclude ot	nsive Hx & physical examination. rectoscopy (if needed). her diagnoses. (e.g. colorectal cancer, Crohn disease) dietary changes, toileting behavior education, fiber supplements.
	mucous m does not c • External h of the lowe	remorrhoids develop above the dentate line covered by nembrane of the upper half (dilated veins) (not innervated by cutaneous nerves; distension ause pain) Best next step when finding an internal hemorrhoids is colonoscopy. The covered with the skin er half. (innervated by cutaneous nerves; distention of this lakin due to thrombosis results in severe pain)
Internal hemorrhoids grading &	Grade I	 Hemorrhoids don't prolapse (only project into the anal canal): above the dentate (pectinate) line: reversible, often bleed. Management: Conservative therapy with supplements, dietary & lifestyle changes. (By decreasing the risk factors such as as losing weight, stopping smoking and treat straining and constipation by fibers or sitting in warm water with salt to relax the sphincter and release the pressure "Sitz bath")
management	Grade II	 Prolapse when straining, but spontaneously reduce at risk . Management: Office-based treatments (e.g. rubber band ligation)
	Grade III	 ★ Prolapse when straining, only reducible manually. ★ Next best step when finding a 3rd degree hemorrhoid is colonoscopy • Management: Rubber band ligation, consider surgical management.
	Grade IV	 Irreducible prolapse: may be strangulated and thrombosed with possible ulceration. Surgical management (hemorrhoidectomy and stapled hemorrhoidopexy)
Management	• The anal softeners	ation is a feature, bulk laxatives or stool softeners may be indicated. fissure can be treated with stool-bulking agents such as psyllium (Metamucil), stool, increased water intake, and topical nitroglycerin ointment or diltiazem to relieve anal spasm and promote healing

The rule of anal fissure **D**'s:

Distally to the Dentate line; bleeDing During Defecation; Dull puDenDal pain; Diet low in fiber



Ninja Nerd's Notes on IBD





Online Meded summary on IBD



For more info about IBD from the textbook (make sure to



check it out)





Radiation **Proctitis**

Causes of Colitis:







Bowel Disease		Proctitis	
	Ulcerative colitis	Crohn's disease	
Location	 Starts distally in the rectum (proctitis). Progresses proximally to occasionally involve the entire colon. 	Can affect the entire GI tract. Most common site for crohn disease is the terminal ileum.	
Pattern	 Superficial diffuse continuous ulcerative lesions. Mucosal disease and sometimes submucosal (Not full thickness inflammation) 	 Skip lesions Transmural thickening Granuloma formation 	
Bleeding	 More likely Can present with up to 20 bloody bowel movements / day. 	 Very rare Positive fecal-occult blood Not with bright red blood. 	
Associated Symptoms	 Crampy abdominal pain in LLQ Tenesmus. All extraintestinal manifestations are improved by control of colitis or colectomy except hepatobiliary manifestations (primary sclerosing cholangitis) 	 Central or rt. Iliac fossa pain Distinguishing Sx: occurrence of repeated episodes of diarrhea in the weeks before the attack. Runs a chronic course: long Hx of colicky central/lower and pain coming on every 15-20 min associated with diarrhea. Mucus-filled bowel movements 	
Diagnosis	 Careful history Flexible endoscopy with biopsy. Colposcopy finding: superficial continuous ulcer 	 Endoscopy Biopsy Contrast studies. MR or CT enterography 	
	Colectomy can be curative	Colectomy can't be curative	
	 Symptomatic disease unresponsive to, or poorly controlled by, medical manage Chronic relapsing disease on discontinuation of medical management and steroid dependency. 	 Chronic subacute obstruction due to fibrotic strictures, adhesions or refractory disease. Symptomatic disease unresponsive to, or poorly controlled by, medical management. Chronic relapsing disease on discontinuation of medical 	

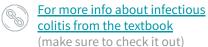
	sclerosing cl	nolangitis)	Mucus-filled bowel movements
Diagnosis	 Careful history Flexible endoscopy with biopsy. Colposcopy finding: superficial continuous ulcer 		 Endoscopy Biopsy Contrast studies. MR or CT enterography
		• Colectomy can be curative	Colectomy can't be curative
Treatment (Surgical)	Elective	 Symptomatic disease unresponsive to, or poorly controlled by, medical manage Chronic relapsing disease on discontinuation of medical management and steroid dependency. Complications of medical management Concerns about long term immunosuppression, risk of malignancy and viral/atypical infections Severe dysplasia on surveillance biopsies of colorectal epithelium Onset of colorectal adenocarcinoma Rarely, control of debilitating extracolonic manifestations such as iritis and sacroiliitis 	 Chronic subacute obstruction due to fibrotic strictures, adhesions or refractory disease. Symptomatic disease unresponsive to, or poorly controlled by, medical management. Chronic relapsing disease on discontinuation of medical management and steroid dependency. Complications of medical management (e.g., osteoporosis) Concerns about long-term immunosuppression, risk of malignancy and viral/atypical infections. Perianal sepsis and fistula Enterocutaneous fistula Onset of malignancy, including colorectal adenocarcinoma and small bowel lymphoma. Rarely, control of debilitating extracolonic manifestations such as iritis and sacroillitis.
	Emergency	 Fulminant colitis unresponsive to maximal medical management Toxic megacolon Free perforation 	 Fulminant colitis or acute small bowel relapse unresponsive to medical management Acute bowel obstruction Life-threatening haemorrhage

Life-threatening hemorrhage

Acute complications of medical management

Abscess or free perforation

Perianal abscess.





Causes of Colitis:









	C. difficile ³ Opportunistic	Cytomegalovirus (CMV)
	Gram-positive bacillus which can proliferate after antibiotics therapy and can cause antibiotics associated diarrhea and pseudomembranous colitis.	
Transmission	 Community acquired: fecal oral route. Hospital acquired: via contaminated surfaces and medical equipment. 	 Blood transfusion Body fluids Sexual transmission Transplacental Perinatal Transplant transmitted infection
Risk factors	 Prior antibiotic use IV Abx Hospitalization 	Immunocompromised patient
Symptoms	 Abdominal pain Fever Diarrhea: Explosive (≥ 3 stools per day) Foul-smelling May develop into toxic megacolon 	Bloody Diarrhea
Bleeding	 Uncommon but can be present Indicating severe mucosal slough 	
Diagnosis	 History Stool culture Lower gastrointestinal endoscopy will show scattered inflammatory plaques between edematous mucosa (pseudomembranous colitis). 	 History Stool culture Endoscopy with biopsy confirms the diagnosis.
Treatment	 Treatment includes oral or transrectal enema with with high dose metronidazole or vancomycin with supportive care. Emergency subtotal colectomy with end ileostomy is indicated if the colitis does not settle or if serious complications (bleeding or perforation) has developed. 	



Infectious

Radiation Proctitis



Proctitis: An inflammation of the lining of the rectum caused by sexually transmitted infections (e.g., gonorrhea, genital herpes, candidiasis, chlamydia), radiation therapy, and inflammatory bowel disease.

Radiation proctitis is a complication of radiation therapy. Those who have had radiation directed at their rectum or at areas around their rectum (e.g. after treatment for pelvic malignant neoplasms) have increased risk of developing radiation proctitis.

Patients with proctitis usually pass fresh blood or bloodstained mucus either mixed with stool or streaked onto the surface of normal or hard stool

Treatment: Resection

- $E.coli, Shigella, Salmonella \ (food\ poisoning)\ are\ the\ most\ common, I\ will\ not\ talk\ about\ them, only\ "treat\ by\ antibiotics" and the most\ common, I\ will\ not\ talk\ about\ them, only\ "treat\ by\ antibiotics" and the most\ common, I\ will\ not\ talk\ about\ them, only\ "treat\ by\ antibiotics" and the most\ common, I\ will\ not\ talk\ about\ them, only\ "treat\ by\ antibiotics" and the most\ common, I\ will\ not\ talk\ about\ them, only\ "treat\ by\ antibiotics" and the most\ common, I\ will\ not\ talk\ about\ them, only\ "treat\ by\ antibiotics" and the most\ common, I\ will\ not\ talk\ about\ them, only\ "treat\ by\ antibiotics" and the most\ common, I\ will\ not\ talk\ about\ them, only\ "treat\ by\ antibiotics" and the most\ common, I\ will\ not\ talk\ about\ them, only\ "treat\ by\ antibiotics" and the most\ common, I\ will\ not\ talk\ about\ them, only\ "treat\ by\ antibiotics" and the most\ common, I\ will\ not\ talk\ about\ them, only\ "treat\ by\ antibiotics" and the most\ common, I\ will\ not\ talk\ about\ them, only\ "treat\ by\ antibiotics" and the most\ common, I\ will\ not\ talk\ about\ them, only\ "treat\ by\ antibiotics" and the most\ common, I\ will\ not\ talk\ about\ them, only\ "treat\ by\ antibiotics" and the most\ common, I\ will\ not\ talk\ about\ them, only\ "treat\ by\ antibiotics" and the most\ common, I\ will\ not\ talk\ about\ them, only\ "treat\ by\ antibiotics" and the most\ common, I\ will\ not\ talk\ about\ them, only\ "treat\ by\ antibiotics" and the most\ common, I\ will\ not\ talk\ about\ them, only\ "treat\ by\ antibiotics" and the most\ about\ them, only\ "treat\ by\ antibiotics" and the most\ about\ them, only\ the most\ about\ them, only\ them$
- Any GI infection can lead to bleeding so you treat them like any other infection based on the cause.
- You must wash your hands after examining the patient. Sanitizing is not enough.



Causes of Colitis:









Ischemic Colitis (Colonic ischemia

Hypoperfusion of the large bowel, which is mostly transient and self-limiting (non gangrenous form), but can also lead to severe acute ischemia with bowel infarction (gangrenous form). Most common form of intestinal ischemia

It is a diagnosis of an older patient.

Caused by transient hypoperfusion

- Occlusive: due to acute cut of the blood supply to a major arterial supply of the colon. This occurs due to thromboembolism, particularly as a result of atrial fibrillation or aortic reconstructive surgery that occlude the inferior mesenteric artery.
- Non-occlusive: age older than 65 years, cardiac arrhythmias, vasculitis, medications, long-distance running, sickle cell disease, hypotension with low blood flow and profound shock.

Pathophysiology

Intestinal blood flow of the superior mesenteric artery (SMA) and/or inferior mesenteric artery (IMA) is suddenly compromised \rightarrow intestinal hypoxia \rightarrow intestinal wall damage \rightarrow mucosal inflammation and possibly bleeding \rightarrow may progress to infarction and necrosis (gangrenous type) \rightarrow disruption of mucosal barrier and perforation \rightarrow release of bacteria, toxins, vasoactive substances \rightarrow life-threatening sepsis

The intestines can tolerate a state of ischemia for approx. 6 hours.

Sites of compromise

- Superior mesenteric artery (SMA): supplies the distal duodenum, jejunum, ileum, and the right colon from the cecum to the splenic flexure.
- Inferior mesenteric artery (IMA): supplies the left colon from the splenic flexure to the rectum.
- Watershed areas (These areas receive dual blood supply from the most distal branches of two large arteries (i.e., SMA and IMA). In the case of severe hypoperfusion, blood supply through these end arteries becomes insufficient.)
 - Splenic flexure
 - Rectosigmoid colon
 - Cecum

Classified into

- Gangrenous: A complete loss of arterial flow causes bowel wall infarction and gangrene.
- Stricturing: A gross impairment of the arterial supply, leading to hemorrhagic infarction of the mucosa which ulcerates, heals by fibrosis and finally leads to stopped.
- Transient: A transient reversible impairment of the arterial supply, which causes a partial mucosal slough that heals by mucosal regeneration in a few days (most common)

Symptoms

Typically presents with 3 clinical stages:

Hyperactive phase: (most patients recover and don't progress beyond this phase)

- Bloody loose stool (60% of patients)
- Sudden onset of cramps abdominal pain (usually LLQ)

- Paralytic phase:
- Pain more diffuse
 Absent bowel sound due to decreased bowel mortality
- Bloating
- Bloody stool cease

Shock

- Acute abdomen with abdominal guarding and rebound tenderness due to perforation and peritonitis
- Signs of septic shock

A classic case of ischemic colitis is a patient who presents with bloody diarrhea and severe abdominal pain after an abdominal aortic aneurysm repair.

Diagnosis

- Plain abdominal radiograph: insensitive, unspecific (air-filled, distended bowel), but helps exclude other disorders
- CT scan: wall thickening
 - Thumbprint sign: an edematous thickening of the mucosa causes indentations in the large bowel wall.
 Appears like thumbprints in CT or plain radiograph. (unspecific sign for colitis)
- Colonoscopy: Procedure of choice in mild to moderate cases of ischemic colitis. Findings include edema, cyanosis, and/or ulceration of mucosa
- Exploratory laparotomy: in severe cases (possibly with resection
- Angiography is not indicated. If it is performed it is often normal.

Treatment

- Mild: Supportive care (IV fluid, bowel rest & nasogastric tube), antiplatelet drugs & reduce the risk of atherosclerosis
- Severe (signs of peritonitis & sepsis): laparotomy & bowel resection





Causes of Colitis:









- Surgical emergency related to vascular compromise of the midgut, including the small bowel and proximal colon. The major blood vessel supplying this portion of the intestine is the SMA, and the venous drainage is through the superior mesenteric vein (SMV).
- Unfortunately it's difficult diagnosis to make, and delays often result in significant bowel necrosis and an overall mortality that remains high.

Risk factors

- Hypercoagulable states. E.g. malignancy, esp. Adenocarcinoma, autoimmune, pregnancy and trauma patients.
- Vasculitis
- Cardiovascular disease (Atrial fibrillation Congestive heart failure Acute myocardial infarction)
- Recent abdominal vascular surgery
- Medications (Vasopressors Digoxin)
- Middle-aged male smoker

Arterial insufficiency



Classified into

- Arterial embolus (the most common cause), embolisms usually originate from the heart can become dislodged in the mesenteric artery and are frequently associated with atrial fibrillation. Patients typically will have evidence of severe atherosclerotic disease in other organ systems (i.e. associated coronary artery disease)
- Arterial thrombosis (due to preexisting visceral atherosclerosis, arteritis, aortic aneurysms or dissection)

- Low flow state (AMI / Shock), Usually has clinical evidence of a low flow state (acute cardiac disease).
- Generally diagnosed in patients in shock, blood may be shunted away from the GI tract. Initially, this process may be beneficial, because the body shunts blood to maintain cerebral blood flow. prolonged ischemia (e.g., >12 hours) is likely to progress to bowel necrosis.
- Potential causes include: hypotension, vasopressors, digitalis, ergotamine & cocaine.
- Occurs in hypercoagulable states.

Usually is found in younger patients. Venous insufficiency / venous thrombosis Has a lower mortality. Can be treated with immediate anticoagulation. Chronic mesenteric ischemia Acute mesenteric ischemia (most common) • Periumbilical abdominal pain that is sudden, severe, diffused constant and disproportionate to physical findings • No rebound tenderness Caused by subacute occlusion or atherosclerotic narrowing of the mesenteric arteries Nausea and vomiting Abdominal / intestinal angina Diarrhea: bloody in later stages (currant jelly stools) Recurrent, dull, postprandial epigastric pain usually within Gangrenous bowel: rectal bleeding, acidosis and signs of sepsis (e.g., Clinical features the first hour after eating tachycardia, hypotension) Can lead to a fear of eating → weight loss and malabsorption In advanced case, the patient will be dehydrated, Tachycardiac, pyrexic and hypotensive Bloating, nausea, occasional diarrhea Abdominal bruit caused by stenosis of mesenteric vessels • On initial PE (mild abdominal tenderness and exaggerated bowel sounds) but late clinical signs are abdominal distention, tenderness, guarding, rigidity, and sluggish bowel sound. Made based on clinical index of suspicion

- **Labs:** ↑ LDH. ↑lactate (metabolic acidosis). ↑creatine kinase, leukocytosis.
- CT angiography: confirmatory. Detects disrupted flow and vascular stenosis. Also detects distended intestinal loops and air-fluid levels, wall thickening and pneumatosis intestinalis which suggests transmural ischemia or infarction.
- MR angiography: alternative to CT

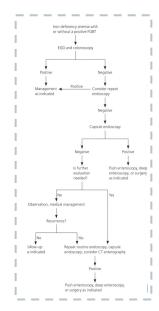
Diagnosis

- The ischemia is self-limited in 85% of cases, and resolves without complications.
- Supportive which include bowel rest, intravenous antibiotics, cardiovascular support, and correction of the low-flow state.
- Surgery is indicated in 15% of cases when the patient develops progressive ischemia and gangrene. Resection of the ischemic intestine and creation of an end stoma is the recommended procedure. Failure of the ischemia to resolve and the need for surgical intervention is indicated by: Fever, Tachycardia, Peritonitis, Acidosis, Marked leukocytosis & Fluid requirement
- Emergent laparotomy (advanced ischemia or hemodynamically unstable): SMA embolectomy (if embolus) or SMA bypass (thrombosis) or immediate heparin (venous thrombosis) and resection of necrotic bowel.
- Revascularization (hemodynamically <u>stable</u> patients without ischemia): Balloon angioplasty and stenting, catheter-based thrombolytics and/or mechanical thrombectomy

Treatment

This is the bleeding which is not apparent to the patients or physician, and the patient present with symptoms of iron deficiency

anemia with or without a positive fecal occult blood test. Bleeding in quantities too small too small to be macroscopically observable (requires chemical tests or microscopic examination to be detected)		
Diagnosis	If -ve UGI and LGI endoscopy: 1. Push endoscopy 2. Capsule endoscopy 3. Occult Blood Test (OBT) It is a guaiac slide test (benzidine, phenolphthalein, Ortho toluidine) Normal stool has 2.5 cc of blood daily. Melena has 50-100 cc of blood per day. Positive OBT: is 10-50 cc of blood/day in the stool. Iron does not give positive OBT. False-positive results are high.	
Treatment	 Management generally depends on the identification of the source of bleeding. Iron replacement combined with intermittent blood transfusion is occasionally necessary. 	





Extra Notes:

Patient resuscitation:

- ABCD + IV cannula Packed RBCs. (Always start with ABC with any patient complains of bleeding, and make sure is he/she stable or not. Give blood if available but because sometimes it's hard to get it fast we give the patient lactate fluids or whatever other fluids.)
- How do you know if the patient has adequate tissue perfusion? Normal Urine output.

Risk assessment & severity of bleeding:

- Volume loss:
 - <10% blood volume loss = Minor bleeding</p>
 - 10-20% blood volume loss + hemodynamically stable = Moderate.
 - >20-25% blood volume loss = Massive bleeding + shock.
- Type of bleeding;
 - Melena = not massive
 - Clot = massive
 - Fresh red blood = more massive
- Low risk \rightarrow medication.
- High risk \rightarrow endoscopy.

Approach:

- Hx & PE (suspect GI bleeding?) 1.
- 2. NG tube.
- 3. Endoscopy (best modality).
- 4. If the endoscopy failed? Targeted RBC scan.
- Active massive bleeding & the pt is $\underline{stable} \rightarrow Angiography$.
 - More accurate but only with active bleeding to identify the source of bleeding.
- Active massive bleeding & the pt is $\underline{unstable} \rightarrow surgery$.

Most the cases of lower GI bleeding we do colonoscopy and find the source then deal with it.

In the lower Gi bleeding the problem is you wanna look at the lumen and the colon is usually full of stool, so if the patient is:

- **Stable** we can give them bowel prep to clean their gastrointestinal tract and scope them.
- Stable and proximal bleeding site (most of the time): blood is a strong laxative so the blood is gonna take everything out and you can give the patient a short or fast prep and try to scope them.
- Completely unstable: you can get an CT angiogram and based on the location of bleeding you can go ahead with surgery but it's very rare we go

The patient is crashing and we don't know the cause of bleeding:

- We sometimes go in and we take the whole colon out but it's very rare
- Some doctors will clamp the colon to see which segment is the source.
- Nowadays we go for angio and locate the problem then resect the exact segment.
- Very rare cases where the patient with maroon stool (not black nor red) or the patient has anemia and we can't find the source we can do capsule or angiography and most of the times we find out the patient has AV malformation or GIST somewhere in the small bowel.

Intra-abdominal Bleeding

Ruptured visceral aneurys	rm (<u>splenic</u> artery aneurysm)
Etiology	Clinical Features
 This may occur primarily as a complication of atherosclerosis in elderly patients where the calcified wall of the aneurysm may be visible on x-ray or secondary to intra-abdominal sepsis and acute or chronic pancreatitis. Rupture occurs more commonly during pregnancy (3rd trimester) or at labor. 	 It ruptures into the peritoneal cavity with similar symptoms to those of splenic rupture: Abdominal pain Distention Drowsiness Pain in the tip of the left shoulder Hypovolemic shock.
Ruptured visceral aneurys	m (<u>hepatic</u> artery aneurysm)
Etiology	Clinical Features
 Intrahepatic (20%) In the past, infections associated with IVDU were the most common cause. Nowadays, the majority are due to trauma or interventional biliary and hepatic procedures. Extrahepatic (80%) Due to degenerative and atherosclerotic changes Aneurysms due to liver transplant are usually extrahepatic and associated with infections. 	 Asymptomatic (Found incidentally). If symptomatic, it usually presents with RUQ or Epigastric pain. Jaundice can also occur if the aneurysm compresses the bile duct. Rupture: Patients present with abdominal pain and shock. Intrahepatic: Rupture into biliary tree causing Quincke triad (Abdominal pain, Hemobilia, Obstructive jaundice). Extrahepatic: Intraperitoneal rupture.
Ruptured visceral aneurysm	(mesenteric artery aneurysm)
Etiology	Clinical Features
 The primary etiology now is atherosclerotic. Up to one-third of superior mesenteric artery aneurysms have historically been described as mycotic or septic, with septic emboli being a known cause. Streptococcus from left-sided cardiac valvular endocarditis has been reported. When excluding pseudoaneurysms, infection is currently an etiologic factor in less than 5% of cases. Medial degeneration, also seen in splenic and hepatic aneurysms, often with secondary atherosclerosis, accounts for 25% of SMA aneurysms. Other reported causes are inflammatory processes in the abdomen or retroperitoneum (cholecystitis, pancreatitis) and trauma. 	 Most patients (90%) are symptomatic with abdominal pain and a pulsatile mass. The pulsatile mass may be notably mobile, differentiating it from an abdominal aortic aneurysm. Patients also may present with frank intraperitoneal hemorrhage or symptoms and signs of mesenteric ischemia. 50% of patients present with rupture, and the mortality rate is 30%.

Intra-abdominal Bleeding

Retroperitoneal hemorrhage (over anticoagulation)		
Etiology	Clinical Features	
Retroperitoneal bleeding has been shown in patients on systemic anticoagulation with warfarin, unfractionated heparin, or low-molecular-weight heparin.	Clinically present as pain in the lower abdominal, flank, or inguinal area with radiation to the thigh and lumbar region. Hypotension and nerve-compression effects characterized by motor or sensory deficits in the groin and thigh may be additional suggestive signs.	
Ruptured Abdominal Aortic Aneurysm		
Etiology	Clinical Features	
Ruptured AAA: is the most common emergency	The pain from a ruptured abdominal aortic aneurysm begins in	

atherosclerotic aneurysms. It's a surgical emergency; it should be suspected in a patient with the triad of severe abdominal and/or back pain, hypotension and a pulsatile abdominal mass.

Patients with abdominal aortic aneurysms invariably are,

or have been, smokers and may have a family history of

presentation of AAA.

Cullen's sign **Grev Turner's sig**

- the centre of the abdomen but commonly radiates to the back and may radiate to the groin along the course of the genitofemoral nerve.
- Cullen's sign and Grey Turner's sign bruising around the umbilicus and in the flank respectively – are late (3–4 days) indicators of a long-standing rupture.
- Expansile pulsatile tender mass may be confirmed. It consists of the aneurysm and the surrounding haematoma.
- Large amounts of free blood in the abdomen, obesity, marked guarding and hypotension may all conspire to render a leaking aneurysm impalpable.
- Severe abdominal pain and collapse with clear evidence of hypovolaemia are strongly suggestive of a leaking aneurysm in an elderly male who is known to be hypertensive and a smoker.
- The bowel sounds may be diminished as a consequence of the irritation caused by intraperitoneal blood.
- Vascular bruits may be heard.

Etiology **Clinical Features** The spleen may be ruptured by blunt or penetrating When the spleen is ruptured by an external injury, the left lower ribs are often fractured. If the ribs are broken, there will be local pain and tenderness Delayed presentation, due to an unusual mechanism and sharp pain on inspiration. (e.g., postcolonoscopy). Splenic haemorrhage usually causes pain in the left hypochondrium and upper abdomen. It may be associated with left shoulder-tip pain if blood or a haematoma is irritating the left hemidiaphragm. Shifting dullness and flank dullness may be detected. Occasionally, a ruptured pathologically enlarged spleen is palpable in the right hypochondrium.

Etiology	Clinical Features
HCC is relatively uncommon in the developed world but is common in Africa and the Far East. In the West, about two-thirds of patients have preexisting cirrhosis and many others have evidence of hepatitis B or C infection. In Africa, 'aflatoxin' (derived from the fungus, Aspergillus flavus, which contaminates maize and nuts) is an important hepatocarcinogen.	 Progression of the existing liver disease symptoms. Abdominal pain and distention. Hepatosplenomegaly or palpable abdominal mass. Loss of appetite and weight loss. Fever. Spontaneous rupture with intraperitoneal hemorrhage.

Intra-abdominal Bleeding

Ruptured Liver Adenoma			
Etiology		Clinical Features	
Liver cell adenoma can develop in young women taking the contraceptive pill containing high levels of estrogen and may rupture spontaneously.		 They may be asymptomatic but generally present with right hypochondrial pain as a result of haemorrhage within the tumor. Superficial tumors may bleed spontaneously and present with symptoms of hemoperitoneum. Hypovolemic collapse is common. 	
	Ruptured E	ctopic Pregnancy	
Etiology	Clinical Features	Diagnosis	Treatment
A fertilised ovum implants at an abnormal site (outside the uterus); usually the fallopian tube. The ectopic fetus might escape through the fallopian tubes into the abdominopelvic cavity.	 The patient may know she is pregnant; morning sickness, amenorrhoea, breast swelling, frequent urination or a positive pregnancy test are present before the rupture happens. Patient c/o of severe RLQ or LLQ pain with radiation. The patient experiences bouts of cramping iliac fossa pain that may be associated with fainting and vaginal bleeding. Growth causes rupture and can lead to massive bleeding. The trophoblasts might penetrate the wall of the involved organ in an attempt to manifest a feto-maternal circulation → wall lysis → rupture. (often ruptures after about 6 weeks) Rupture produces sudden severe pain, bleeding and circulatory collapse, with the abdominal pain often becoming generalised. Episodes of iliac fossa pain prior to rupture → rupture → severe pain, bleeding or even fainting. 	Consider a pregnancy test in all females in childbearing age • Transvaginal US: Best initial test and confirms the diagnosis. • β-HCG: Supportive. • Blood type and screen: ABO and Rh testing to identify patients who might need Rho immunization • Exploratory laparotomy • Unstable patients suspected of having an ectopic pregnancy • In pregnancy of unknown location if the location is still uncertain after 7–10 days (earlier for high risk patients e.g. previous ectopic pregnancy) • Endometrial biopsy Consider only in cases of pregnancy of unknown location where non viability is certain.	 Pain management Hemodynamically Stable: Methotrexate (drug of choice), do not use it in cases of rupture. Hemodynamically Unstable: Ruptured: Salpingectomy (removal of the fallopian tube) Unruptured: Salpingostomy (opening of the fallopian tube and extraction of the pregnancy tissue) These patients are subsequently followed up with the monitoring of β-HCG levels until negative results are obtained to exclude the presence of residual trophoblastic tissue. Anti-D immunoglobulin for Rhnegative patients who presents with bleeding to prevent maternal alloimmunization and reduce the risk of hemolytic disease of the fetus and newborn in future pregnancies. Prenatal and contraceptive counseling once treatment is complete. Patients who have received methotrexate should typically not attempt to conceive again for at least three months after completing treatment.
	Ruptured Ovarian Cyst		
Etiology		Clinic	cal Features

Large pathological ovarian cysts can rupture and bleed

Patients may collapse from the associated hypovolaemic shock or present with sudden and severe lower abdominal pain and then develop the signs of internal bleeding.

profusely.

Benign ovarian cysts are a common cause of torsion, rupture and

bleeding.

Causes of chronic lower GI bleed

- 1- Haemorrhoids (the commonest)
- 2- Anal fissure
- 3- Colon cancer
- 4- IBC
- 5- Others: colitis, diverticulitis, polyps and endometriosis

Causes of massive lower GI bleed

- 1- Diverticular disease
- 2- Angiodysplasia
- 3- Polyps
- 4- Ulceration of hemorrhoids
- 5- Post-operative bleeding
- 6- Coagulopathy

Causes of intra-abdominal bleed

- 1- Most common cause is abdominal **trauma** (especially RTA) could be blunt or penetrating (trauma could most commonly cause rupture of spleen, Liver or Mesentery)
- 2- **Ruptured ectopic pregnancy** (presents 4-6 weeks after pregnancy. The fertilized ovum is not going to the uterus and its stuck somewhere in the fallopian tube then the tube will rupture resulting in a bleed)
- 3- Ruptured ovarian cyst
- 4- Ruptured aneurysm (these patient usually already have cardiac problems and the presentation is severe chest or abdominal pain)
- 5- **Post-operative bleeding** (Ex. Post cholecystectomy the patient shifted to the ward, the clips on the cystic artery slips and the patient start bleeding)
- 6- Ruptured liver lesion (either Hepatoma or Hepatocellular carcinoma) (very rare)
- 7- Perforated ulcer

Features of presence of intra abdominal blood

- Peritonism
- Peritonitis

Treatment of intraabdominal bleeding:

- If <u>unstable</u> then go for surgery or angio
- If <u>stable</u> then treat the cause.



Case 1

20 YO male comes to the clinic with hematochezia what does he have?

Hemorrhoids

Do we have to rule out cancer?

Yes but it's less likely the cause (not your number 1 differential)

Case 2

90 YO lost a lot of weight with constipation and has blood per rectum?

Cancer is number 1 differential

Summary

Additional medical history		
Painless bleeding	Varices, angiodysplasia, diverticulosis or cancer	
Epigastric pain	Ulcer disease, gastritis, or esophagitis	
Cramping abdominal pain	Inflammatory bowel disease, colitis, or partially obstructing cancer	
Cramping abdominal pain, abdominal guarding, tenderness, weight loss & constipation	Diverticulitis	
Severe, acute, sudden onset of pain	Perforated viscus	
Pain out of proportion to examination	Hallmark for bowel ischemia	
Prior surgical interventions	Marginal ulcers or arterio enteric fistula	
Chronic alcohol or drug use	Risk factor for liver disease precipitating gastric varices	
Fever and chills	Infectious or inflammatory causative agent	
Weight loss, anorexia, and fatigue	Malignancy	
Dizziness, orthostatic symptoms	Indicates large, acute volume loss or severe anemia	

Recall

Q1: What is the definition of lower GI bleed?

Answer: Bleeding distal to the ligament of Treitz; vast majority occurs in the colon.

Q2: What are the symptoms of lower GI bleeding?

Answer: Hematochezia (Bright red blood per rectum [BRBPR]), with or without abdominal pain, melena, anorexia, fatigue, syncope, shortness of breath and shock.

Q3: What are the DDx of lower GI bleeding?

Answer: Hemorrhoids, Angiodysplasia, ischemic, IBD and neoplasm.

Q4: What are the most common causes of massive lower GI bleeding?

Answer: Diverticulosis and vascular ectasia

Q5: What is the initial treatment?

Answer: IVFs: Lactated Ringer's; packed red blood cells as needed, IV x 2, Foley catheter to follow urine output, discontinue aspirin and nasogastric tube (NGT).

Q6: What diagnostic tests should be performed for all lower GI bleeds?

Answer: History, physical examination, NGT aspiration (to rule out upper GI bleeding; bile or blood must be seen; otherwise, perform EGD), anoscopy/proctoscopic exam.

Q7: What must be ruled out in patients with lower GI bleeding?

Answer: Upper GI bleeding! Remember NGT aspiration is not 100% accurate (even if you get bile without blood)

Q8: What is the diagnostic test of choice for localizing a slow to moderate lower GI bleeding source?

Answer: Colonoscopy

Summary

	Etiology	Clinical features	Work up
Diverticular Disease	Chronic constipation	Only 3% to 15% experience any bleeding, 75-80% resolves spontaneously and 10% rebleed within a year	 Colonoscopy: best Initial, diagnostic test and treatment Angioembolization (risk for perforation & ischemia) Surgery when other modalities have failed
Angiodysplasia	Acquired arteriovenous malformations Secondary to progressive dilation of normal blood vessels within the submucosa	 Found in patients > 50 Associated with aortic stenosis and renal failure 	 Diagnosis: colonoscopy or angiography Treatment: Colonoscopy: sclerotherapy injection Angiography: vasopressin, selective gel-foam embolization Surgery: if these therapies fail or bleeding recurs
Neoplasia	 Colorectal carcinoma Polyps Juvenile polyps Gastrointestinal Stromal Tumor (GIST) 	 Painless Intermittent Slow → iron deficiency anemia 	 Colonoscopy is the best diagnostic tool Treatment: Endoscopy: If the polyp is the source of bleeding Surgery: emergency procedure or elective after control of bleeding
Anorectal diseases	 Internal hemorrhoid Anal fissures Colorectal neoplasia 	 Low-volume bleeding Bright red blood Tenesmus Chronically cause anemia 	 Exclude other diagnoses. (e.g. colorectal cancer, Crohn disease) Anal fissures: stool-bulking agents, softeners & topical nitroglycerin ointment or diltiazem Hemorrhoids: Grade I: conservative Grade III: rubber band ligation Grade III: rubber band ligation, consider surgical management Grade IV: hemorrhoidectomy
C. difficile infection	Prior antibiotic useHospitalization	 Abdominal pain Diarrhea: explosive, foul-smelling Bleeding Uncommon 	 Diagnosis: history, stool culture Treatment: vancomycin and high dose metronidazole Surgery: if the colitis does not settle or if serious complications (bleeding or perforation) developed
Cytomegalovirus (CMV)	Immunocompromised patient	Bloody Diarrhea	Diagnosis: history, stool culture

Summary

	Etiology	Clinical features	Work up
Ulcerative colitis	-	 Starts distally in the rectum Progresses proximally to involve the entire colon Mucosal disease Crampy abdominal pain Tenesmus. More likely to bleed 	Diagnosis: endoscopy with biopsy Surgical management: Colectomy can be curative Emergency: Fulminant colitis Life-threatening hemorrhage Free perforation Toxic megacolon Acute complications of medical management Elective: Failure or of medical management Complications of medical management Concerns about long term immunosuppression Onset of colorectal adenocarcinoma Severe dysplasia Relapsing on discontinuation of medical management & steroid dependency Debilitating extracolonic manifestations
Crohn's disease	-	 Can affect the entire GI tract Skip lesions Pattern Transmural thickening Diarrhea Mucus-filled bowel movements Rarely bleed 	Diagnosis: endoscopy with biopsy & Contrast studies Surgical management: Colectomy can't be curative Emergency: Fulminant colitis Life-threatening haemorrhage Abscess or free perforation Acute bowel obstruction Perianal abscess Elective: Failure or of medical management Complications of medical management Concerns about long term immunosuppression Onset of malignancy Subacute obstruction Perianal sepsis and fistula Enterocutaneous fistula debilitating extracolonic manifestations
Radiation Proctitis	Radiation directed at their rectum or at areas around	-	Treatment: resection
Ischemia	 Cardiovascular disease AF HF Acute MI Hypercoagulable states Recent abdominal vascular surgery Vasculitis Medications (Vasopressors, Digoxin) 	-	 Diagnosis: CT scan (thickened bowel wall), endoscopy (confirmatory) Treatment: Supportive Surgery: indicated by Fever, Tachycardia, Peritonitis, Acidosism, Marked leukocytosis & Fluid requirement
Occult Bleed	<u>-</u>	Characterized by iron deficiency anemia	 Diagnosis: If -ve UGI and LGI endoscopy Push endoscopy Capsule endoscopy Treatment: depends on the identification of the source of bleeding & Iron replacement

Q1: Which of the following statements is true regarding H.pylori?

- A) The organism is round shaped.
- B) It has the ability to hydrolase urea with production of ammonia.
- C) Treatment is recommended in asymptomatic patients.

Q2: Common sites of peptic ulcer includes:

- A) First part of the duodenum.
- B) Esophagus.
- C) Fundus of the stomach.

Q3: Which of the following is a predisposing factor for gastric ulcers?

- A) Gastric acid.
- B) NSAIDs.
- C) Smoking.

Q4: A 50 Y/O man presents to the outpatients clinic with an 8 - week history of bleeding from his back end. This is typically bright red and copious during or following stool. The patient has had no change in his bowel habits, no weight loss or family Hx of bowel cancer, what is the most appropriate course of action?

- A) Preform rubber band and ligation of hemorrhoids.
- B) Blood tests including CEA.
- C) Flexible sigmoidoscopy.

Q5: A Patient is rushed to the ER following a large amount of hematemesis, what is the best initial step?

- A) Resuscitate with crystalloid solution.
- B) Cross match and blood transfusion.
- C) Maintain ABC.

Answers

Q1		Q4	
Q2	А	Q5	
Q3	B&C		



Explanations

Q1 Explanation: Helicobacter pylori is spiral shaped and resides in the mucous layer of the stomach. Its ability to produce ammonia, an alkali, results in increased gastrin release due to a negative feedback. H. pylori infection is very common and population infection rates of 80–90 percent are not unusual. It appears that most infection is acquired in childhood and the probability of infection is inversely related to socioeconomic group. At present, eradication therapy is recommended for patients with duodenal ulcer disease, but not for non-ulcer dyspepsia or in asymptomatic patients who are infected. H. pylori is now classified by the WHO as a class 1 carcinogen.

Q2 Explanation: The common sites for peptic ulcers are the first part of duodenum and the lesser curve of stomach, but they may occur in the oesophagus, the stoma following gastric surgery and Meckel's diverticulum. It is now widely accepted that H. pylori is the most important factor in the development of peptic ulceration. The other major factor is the ingestion of NSAIDs. Patients with gastric ulcers have relatively normal levels of gastric acid secretion. Kissing ulcers refer to two adjacent ulcers on the anterior and posterior walls of the duodenum.

Q3 Explanation: The most common causes of haematemesis are bleeding peptic ulcer (60%), erosions (26 %), Mallory–Weiss tear (4 %) and esophageal varices (4 %). Whatever the cause, the principles of management are identical – initial resuscitation followed by urgent investigations to determine the cause of bleeding and definitive therapy. There are numerous endoscopic measures to control peptic ulcer bleeding, such as injections and argon diathermy. The criteria for surgical intervention – patients continuing to bleed, rebleed, visible vessel at ulcer base, a spurting vessel or an ulcer with a clot on the base – are statistically likely to require surgical treatment. Elderly and unfit patients are more likely to die from the bleed than younger patients and hence should have early surgery. In general, anyone needing more than 6 units of blood needs surgery.

Q5 Explanation: The most likely diagnosis here is haemorrhoids, however, a left-sided colorectal adenocarcinoma must be excluded, despite the low index of suspicion. Such patients should be reassured, but advised to undergo a flexible sigmoidoscopy to exclude cancer or colonic polyps. Patients with change in bowel habit would need a colonoscopy. Treating the patient for haemorrhoids without excluding malignancy is not acceptable. An examination under anaesthetic will not allow sufficient visualisation of the left colon unless a flexible sigmoidoscopy was also done as part of the procedure. Blood tests and CEA levels should not be used to diagnose or exclude colonic malignancy.

Q1: A 50-year-old man presents to the outpatient clinic with an 8-week history of bleeding from his back end. This is typically bright red and copious during or following a stool. The patient has had no change in his bowel habit, no weight loss, and has no family history of bowel cancer. What is the most appropriate course of action?

- A) Perform rubber band ligation of haemorrhoids
- B) Perform injection sclerotherapy of haemorrhoids
- C) Blood tests including CEA
- D) Examination under anaesthetic +/- proceed to treat haemorrhoids
- E) Flexible sigmoidoscopy

Q2: An 85-year-old patient is admitted to the emergency department in shock with a short history of large-volume fresh bleeding per rectum. You resuscitate the patient with blood and fluids. There is no identifiable source on rectal examination. However, the patient continues to be unstable and you suspect continued bleeding. Her bowels open and pass an additional large volume of blood. Your next stage of management is

- A) Laparotomy
- B) Radionucleotide red cell scanning
- C) Esophagogastroduodenoscopy
- D) Mesenteric angiography
- E) Colonoscopy

Q3: Which of the following features is not characteristic of Crohn's disease?

- A) Rose thorn abscesses
- B) Cobblestoning
- C) Skip lesions
- D) Lead piping
- E) Serosal involvement

Answers

Q1	Е
Q2	
Q3	



Explanations

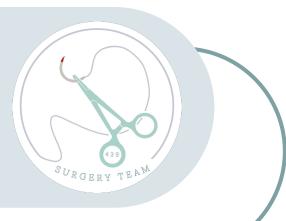
Q1 Explanation: The most likely diagnosis here is hemorrhoids, however, a left-sided colorectal adenocarcinoma must be excluded, despite the low index of suspicion. Such patients should be reassured, but advised to undergo a flexible sigmoidoscopy to exclude cancer or colonic polyps. Patients with change in bowel habit would need a colonoscopy. Treating the patient for haemorrhoids without excluding malignancy is not acceptable. An examination under anaesthetic will not allow sufficient visualisation of the left colon unless a flexible sigmoidoscopy was also done as part of the procedure. Blood tests and CEA levels should not be used to diagnose or exclude colonic malignancy.

Q2 Explanation: Gastrointestinal haemorrhage is a common clinical problem, account- ing for 1%–2% of acute hospital admissions. It is broadly divided into upper and lower gastrointestinal bleeding, according to whether haemorrhage occurs proximal or distal to the ligament of Treitz. Upper gastrointestinal bleeding accounts for 80% of all gastrointestinal haemorrhage. Ninety-five per cent of lower gastrointestinal haemorrhage bleeding is from the colon, the small intestine, accounts for less than 5%. The management of lower gastrointestinal haemorrhage relies on the accurate localization of a source; blind emergency hemicolectomy must be avoided as it results in a re-bleeding rate of up to 50%. Initial assessment should aim to exclude anorectal causes as a possible diagnosis by careful rectal examination and rigid proctoscopy/sigmoidoscopy. If no source is found the next stage is to exclude an upper gastrointestinal cause as these are readily treatable at endoscopy; 15% of per-rectal bleeds in haemodynamically compromised patients originate proximal to the ligament of Treitz. Following a negative OGD further management will depend on the haemodynamic status of the patient. Colonoscopy is possible in mild bleeding, but it's diagnostic yield is severely restricted in moderate to severe haemorrhage as the view is compromised. Major haemorrhage in patients who are hemodynamically unstable indicates a need to localise the source of bleeding. CT mesenteric angiography is effective but requires a reasonable rate of blood loss to identify a source. Angiography can localize the lesion to a greater degree of accuracy and has the option of therapeutic intervention, but it takes longer, is more invasive and is more technically demanding. Both techniques require an actively bleeding lesion and a reasonable cardiac output. In severely compromised patients refractory to resuscitation, the decision may be made to perform a laparotomy and isolate the lesion through serial clamping and in-theatre colonoscopy, but this is

Q3 Explanation: .Comparing and contrasting the histopathological features of ulcerative colitis and Crohn's disease is a classic exam question all candidates should be prepared for. In schools with viva examinations, barium studies may be shown and candidates will be expected to distinguish between the two diseases radiographically. Ulcerative colitis is a superficial disease and therefore its features are consistent with superficial fibrosis; the colon loses its normal haustra and appears rigid on barium studies, so-called lead piping. Its lesions are confluent and always affect the rectum. Stricturing is rare and fistulae are very rare. Histologically, crypt abscesses are identifiable but the serosa is normal. Crohn's disease, however, involves all layers of the bowel wall down to the serosa. The mucosal surface is pocketed with deep abscesses, which have a characteristic appearance of rose thorns on barium studies. The mucosa is said to be cobblestoned, strictures are common, as are fistula. The mucosal involvement is not confluent, that is 'skip lesions', and the rectum is only involved in 50% of cases.

Good Luck!





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