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Approach to Bright Red Blood Per Rectum

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Consultant: Salix & Ironwood, Clinical Area- IBS-D, IBS-C, CIC

Speakers Bureau: Salix, Clinical Area – CIC, IBS-C, IBS-D; Takeda, Clinical Area - CIC

Rectal Bleeding – Definitions

- **Hematochezia:** passage of fresh blood usually in or with stool. Mostly with lower GI bleeds, but can also occur with a brisk upper GI bleed. Bright red or maroon in color
- **Acute Lower GI bleed (LGIB):** recent onset of hematochezia from colon, rectum or anus with risk for hemodynamic instability, anemia or the need for a blood transfusion
- **Minor Bright Red Blood Per Rectum (BRBPR):** passage of scant fresh blood which may appear as drops in the toilet bowel, a streak on toilet paper or coating the stool

Taking a History

Onset? Acute or Chronic?

Color? Black, maroon, bright red?

With or without BMs? Blood mixed in the stool, coating stool, after defecation

Perianal pain? Burning, throbbing, glass cutting, itchy

Quantify blood? Streak on toilet paper, drops in toilet bowl, filling toilet bowl, soiling underwear

Associated symptoms: fevers/chills, weight loss, N/V, abdominal pain, constipation diarrhea, tenesmus?

Red flags: IDA, unexplained weight loss, onset after 50 yrs of age, severe or progressively worsening symptoms, family hx of CRC or IBD

Medications:

- NSAIDs COX 1 and 2 inhibitors
- Anti-platelets, anticoagulation
- Constipating or diarrhea inducing medications
- Immunosuppressants

PSH: hx of colorectal surgery, hemorrhoidectomy, banding, fissurectomy

Soc Hx: anal intercourse, tobacco, alcohol

PMH:

- CRC, premalignant polyps, hereditary syndrome (familial polyposis, Lynch)
- IBD or Celiac
- Hx of pelvic radiation
- Connective tissue dz
- Cardiac surgery
- Hx of STDs (rectal HPV, HIV)
- Severe endometriosis

Physical Exam and Labs

Vitals: Hemodynamic instability: elevated HR, low BP, low O_2 sats → send to ER

PE:

GEN: acute distress? Pale/weak?

Cardiac: systolic murmur, tachycardia

Abd exam: point or diffuse TTP, guarding, rebound TTP

Perianal exam: Active bleeding, excoriations, abscess, anal fissure, fistula, malignancy, external hemorrhoids,

Digital Rectal Exam: palpate for IHs, mass, fecal impaction, blood

Anoscopy exam: can have higher sensitivity for detection of hemorrhoids than a flex sig

The presence of hemorrhoids does not exclude a more proximal etiology

Labs:

- CBC
- If anemic order iron studies +/- B12/folate

Acute LGIB:

- Cr, BUN, electrolytes
- PT/PTT
- Type and crossmatch

Bloody diarrhea

- Infectious work-up
- CRP, fecal calprotectin

Indications for Scoping

No Scope

- < 35-40 yrs with no other alarm symptoms or RFs and DRE/anoscopy reveals a source (large hemorrhoids, anal fissure)

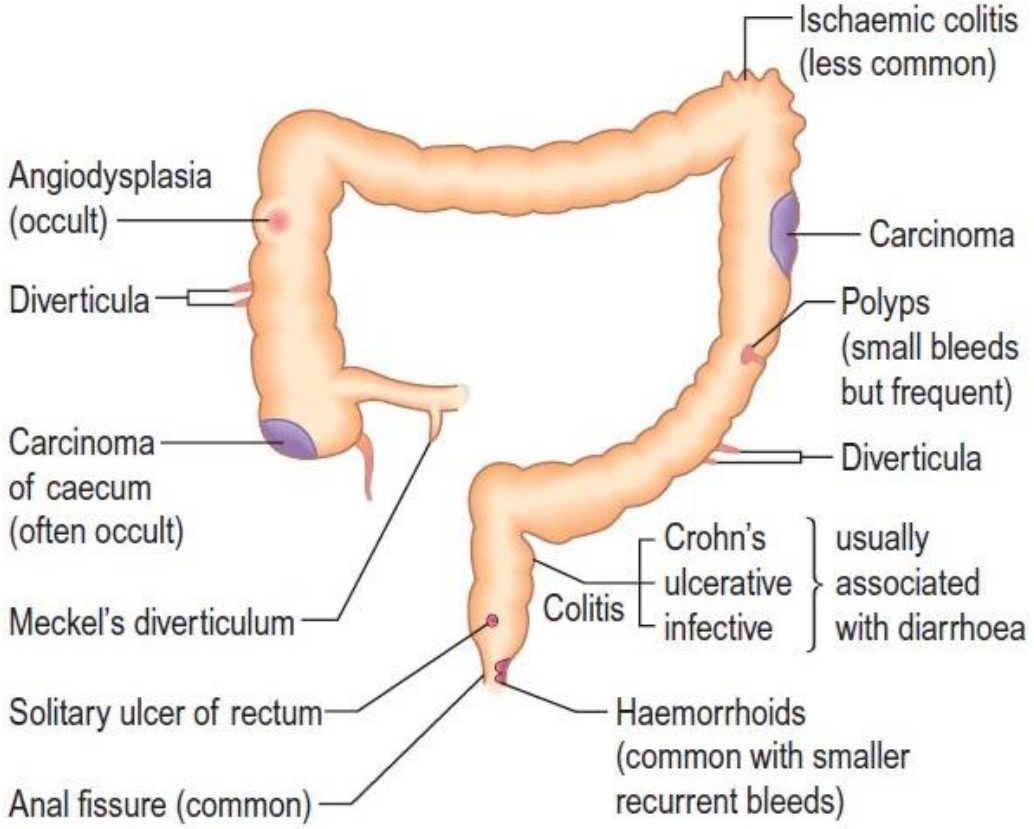
Sigmoidoscopy

- <35-40 yrs with no other alarm symptoms or RFs with equivocal findings on DRE/anoscopy
- <35-40 with persistent symptoms despite treatment
- >35-40 yr with a normal colonoscopy in the past 2-3 yrs and low risk for CRC with equivocal findings on DRE/anoscopy

Colonoscopy

- Red flags at any age
- Age 45-50 yrs with an absence of red flags and who are due for CRC screening
- Age 35-40 with equivocal findings on DRE/anoscopy
- For patients who proceed with a flex sig and a source is not found

Causes of Hematochezia and BRBPR



Causes of Acute LGIB

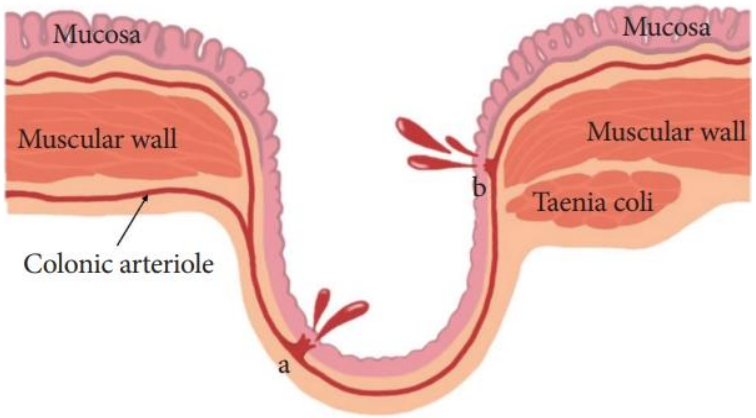
Colon (80%)	Diverticular disease	17-40%
	Angiodysplasia	0-3%
	Neoplasm (polyps and CRC)	3-11%
	Colitis (ischemic, infectious, IBD, radiation)	9-21%
	Post-polypectomy/post anastomotic bleeding	0-13%
Anorectal	Hemorrhoids, anal fissure, rectal varices, rectal ulcers	4-10%
SB	AVMs, Meckel diverticulum, IBD, Neoplasia	2-9%
UGI	Vasculitis, peptic ulcer, neoplasia, Dieulafoy lesion	4-10%

- Stops spontaneously in 80-85% of patients
- Acute bleeding = normocytic +/- anemia
- Chronic bleeding = microcytic + IDA
- Timing of colonoscopy determined on hemodynamic stability, resuscitation and ongoing bleeding
- Unstable hematochezia: exclude UGI source with nasogastric lavage and/or EGD
- If no source found, consider CT angiogram/CT with bleeding protocol, Meckel's Scan or video capsule endoscopy

Zuccaro G et al. *Am J Gastroenterol*. 1998;93(8):1202-1208.

Diverticular Bleeds

- **Etiology:** Diverticula develop at sites of weakness in the colonic wall where vasa recta penetrate the circular muscle layer. As a diverticulum herniates, the vasa recta drape over the dome of the diverticulum and become susceptible to trauma and disruption
- **CP:** acute painless BRBPR in the ABSENCE of stool
- MOST COMMON cause of acute LGIB
- **Amount of Bleeding**: moderate to severe
- **Facts:** 90% of diverticula in sigmoid colon, but 60% of diverticular bleeds from the thinner-walled right colon
- Not associated with diverticulitis
- **RF:** advanced age, low fiber diet, obesity, inherited connective tissue disorders (Marfan's syndrome, Ehlers-Danlos)
- **Rx**: supportive care, IVFs, resolved spontaneous in approx. 80+% of patients, 20% require endoscopic hemostasis



Wilkins T et al. *Am Fam Physician.* 2009;80(9):977-83; Image: *Clin Endosc.* 2019;52 (5):419-425.

Nomenclature of Vascular Lesions in the GI tract

Term	Definition	Facts
Angiodysplasia, angiectasia, vascular ectasia	Small vascular lesion of arterioles, capillaries, venules that has a supplying artery and a draining vein	Most common source of vascular bleeding from SB and Colon in elderly patients
Telangiectasia	Tiny vascular lesion from dilation of the terminal end of a vessel. Similar to angiodysplasia	Lesions of hereditary hemorrhagic telangiectasia – HHT, scleroderma, CREST, cutaneous and mucosal (GI tract, nose, mouth, lip, skin). Lesions are widespread in GI tract
Haemangiomas	A tumor produced by enlargement or new formation of blood vessels	Benign vascular tumor
Arteriorvenous malformation (AVM)	A congenital disorder of blood vessels consisting of abnormal connection between arteries and veins without intervening capillaries	Use only for a congenital vascular lesion that has direct connection between arteries and veins. Should not be used when referring to vascular ectasias
Dieulafoy lesion	Arterial in origin. Abnormally large submucosal end arteries	Can cause massive bleeding, located in upper stomach
GAVE (gastric antral vascular ectasia)	Capillary type vascular lesion Dilated tortuous mucosal capillaries in the antrum of the stomach	Scleroderma/portal HTN. Melena or IDA

Brandt LJ. *Gastrointest Endosc*. 2018;87(6):1595-1596.

Vascular Ectasia
(Aka Angiodysplasia, Angiectasia)

- **Terminology:**
  - Angiodysplasia typically describes colonic lesions
  - Angiectasia or vascular ectasia: typically used as a generic term
- **Etiology:** venous in origin. focal submucosal areas of thin, weak and dilated vessels. 2/3 are R-sided (cecum or ascending colon)
- **Facts:** Most common vascular abnormality in the GI tract. Typically more than one
- **CP:** painless, IDA, bleeding is small in quantity because origin in venous. Recurrent and chronic
- **Endoscopy findings:** 5-10 mm, flat, cherry-red lesions with a fern-like pattern radiation from a central vessel, typically more than one
- **RF:** advanced age, von Willebrand's disease, CKD, aortic stenosis (Heyde Syndrome), LVAD (acquired VWD)
- **Rx:** APC, electrocoagulation, mechanical hemostasis. Incidentally found, without occult or acute bleeding, should NOT be treated

Colitis

Infectious colitis

- **RF:** international travel, immunocompromised, eating suspected contaminated food, cirrhosis (Vibrio)
- **Inflammatory diarrhea pathogens:** Salmonella, Campylobacter, Shigella, enterohemorrhagic *E. coli*, Yersinia, *Vibrio parahemolyticus*, *Entamoeba histolytica*

Inflammatory Bowel Disease

- **CP:** Hematochezia, abdominal pain, tenesmus
- **Labs:** Elevated CRP and fecal calprotectin
- Refer to GHAPP IBD presentations

NSAIDs

- **CP:** mostly subclinical. IDA, hematochezia, BRB, stricture
- Can cause microscopic colitis which presents with secretary diarrhea (w/o bleeding)
- Intestinal diaphragms (circumferential small bowel strictures) are pathognomonic
- Erosions, ulcers, colitis are non-specific, and should improve with stopping the NSAID
- PPIs provide no protection
- COX-2 inhibitors provide minimal prevention

Ischemic Colitis

Radiation proctitis

Laine L et al. *Gastroenterology*. 2008;135(5):1517-1525.
Image: http://www.pathologyoutlines.com/topic/smallboweldiaphragmdisease.html.

Ischemic Colitis

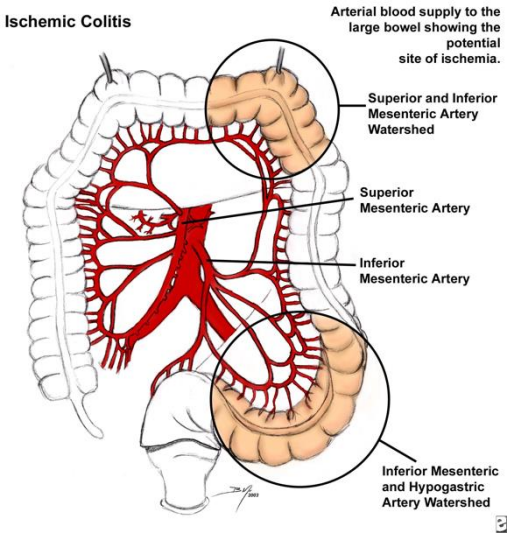
Pathophysiology: low-flow state, **"watershed"** areas with poor perfusion. Between areas of colonic blood supply (R colon supplied by SMA, L colon by IMA, splenic flexure poorly supplied by both). Mucosal → transmural

RF: dehydration, HF, shock, aortoiliac procedures, hypercoagulable states, extreme exercise/long distance running, HD, certain vasoconstricting drugs (digitalis, vasopressors, cocaine)

CP: acute abdominal pain (not always), bloody diarrhea, chronic ischemia can result in stricture formation

Endoscopy: L sided. Mucosal friability and findings that resemble UC, rectal sparing, single longitudinal ulcer

Rx: self limited, 85-90% resolve with correction of underlying cause and volume repletion



Acute Mesenteric Ischemia

AMI is arterial embolus or thrombosis leading to bowel necrosis. high mortality. affects the SB and is often transmural. Severe pain

UpToDate

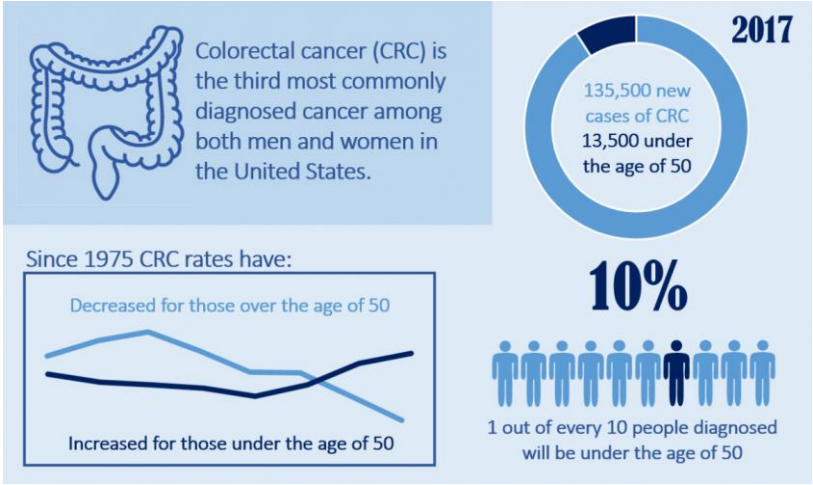
Anorectal Causes of BRBPR

- Neoplasia (malignancy and polyps)
- Hemorrhoids
- Anal fissure
- Radiation and ulcerative proctitis
- Stercoral ulcer and solitary ulcer syndrome
- Rectal Varices
- Trauma
- Rectal hemangioma
- Intestinal endometriosis: less like to present with BRBPR and more likely pelvic pain/rectal pain, constipation/diarrhea, pain with intercourse. Can mimic IBS

Colon and Rectal Cancer in Young Adults

From 1992-2016:

- **Rectal cancer increased by >90%**
- **Colon cancer increased by 40%**
- Across all racial/ethnic groups and similar rates in Asia, Europe and Australia
- 1 in 10 new diagnosis of CRC are 50 yrs or younger
- 3 of 4 patients with early-onset CRC have no family hx
- Most are rectum or distal colon compared to proximal colon in older patients
- Etiology: ? Diet, environmental exposures, lifestyle factors
- Double the prevalence of pathogenic germline variants (MSH6, PMS2, MMR – Lynch; APC – FAP)
- Recommend genetic testing with early-onset CRC
- American Cancer Society 2019: start screening for average risk at age 45



Stoffel EM, Murphy CC. *Gastroenterology*. 2020;158:341–353; Image: https://ruesch.georgetown.edu/youngadultcrc/.

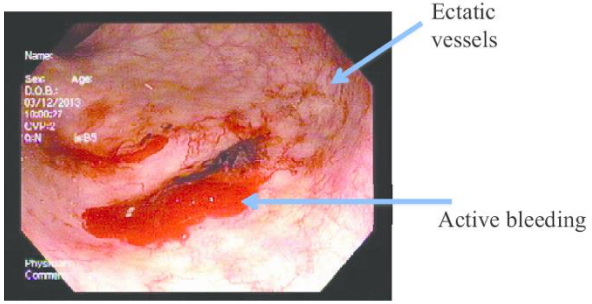
Radiation Proctitis

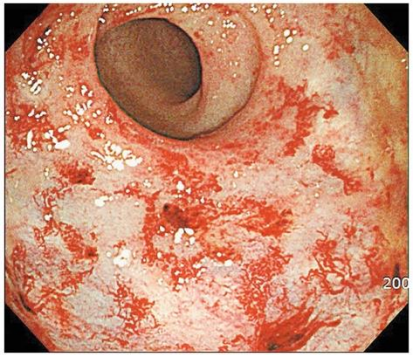
CP: bloody diarrhea, tenesmus, mucus, recurrent, risk for stricture

RF: pelvic radiation (prostate, rectal, cervical cancer).

External beam radiation = greater exposure

Brachytherapy = less exposure





Acute:

- Onset: 6 weeks of radiation
- direct mucosal damage from radiation exposure

Chronic

- Onset: 9-14 mo after exposure
- progressive epithelial atrophy and fibrosis, endarteritis and chronic mucosal ischemia

Rx: Supportive treatment: hydration, antidiarrheals

Endoscopic therapy: APC (argon plasma radiation), RFA (radiofrequency ablation). May require more than 1 session. Stricture dilation

At risk for secondary malignancies, the majority are CRC

Sarin A, Safar B. *Gastroenterol Clin North Am*. 2013;42(4):913-925.

Internal Hemorrhoids

Etiology: cushion (plexus) of dilated AV channels and connective tissues.

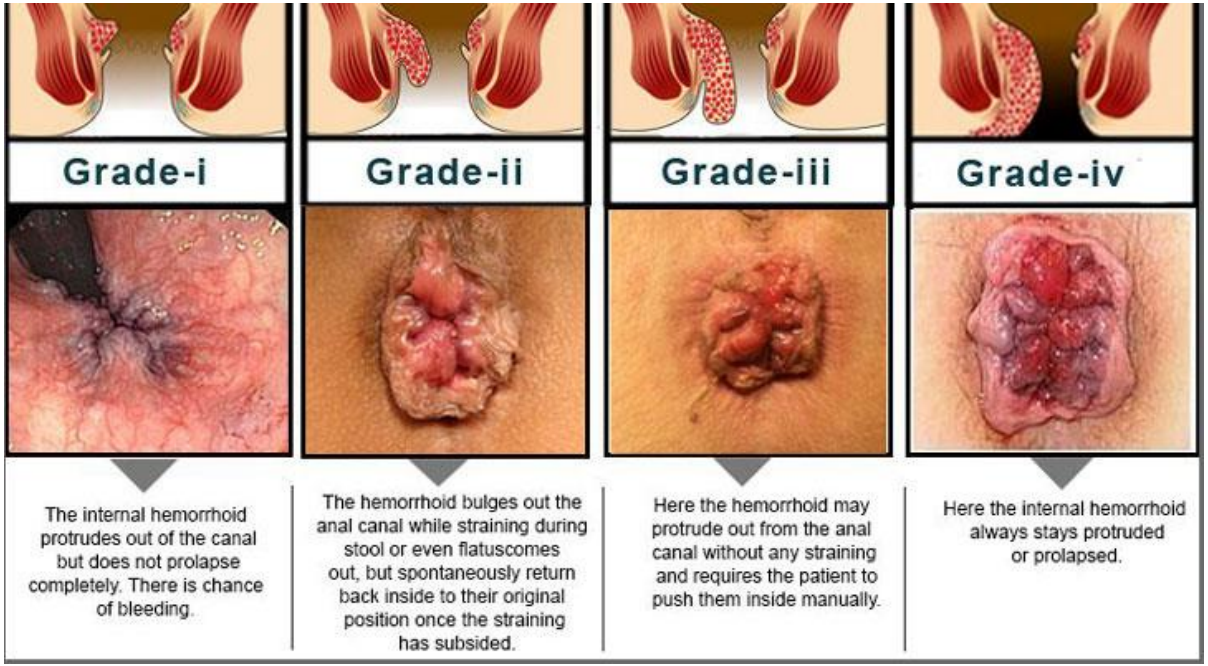
IH = superior hemorrhoid plexus

EH = inferior hemorrhoid plexus

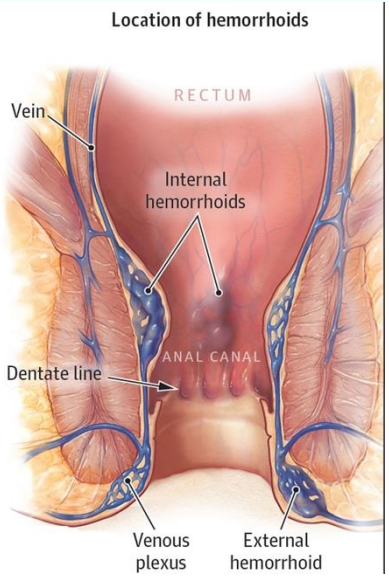
Location: proximal to the dentate line

CP: Painless BRBPR w/BMs, blood coats stools at end of defecation. Blood may drip into the toilet or stain toilet paper, or appear in underwear following defecation

Rx: treat constipation, avoid straining, banding, hemorrhoidectomy



External Hemorrhoids



CP: Scant BRB with wiping, burning pain

Thrombosed:

- Acute pain
- Can spontaneously release
- <48-72 hrs - can excise
- >48-72 hrs - conservative treatment

Non-thrombosed:

- Conservative Rx: treat constipation, Sitz baths, non alcohol based rectal wipes, hydrocortisone cream (up to 10-14 days)

RF: pregnancy, obesity, chronic diarrhea/constipation, heavy lifting

Stercoral Ulcer vs Solitary Rectal Syndrome

Stercoral Ulcer[1]

- **Etiology**: stagnate impacted feces, eventually eroding and ulcerating rectosigmoid mucosa
- Mortality rate may exceed 50% if ulceration leads to perforation
- **RF**: elderly or bedridden patient, hx of constipation and HTN, possibly dialysis.
- **Clinical features:** acute GI bleeding
- **Gross description:** sharply demarcated ulcer(s), perforation may be present
- **Treatment:** possibly surgery, laxatives/disimpaction

Solitary Rectal syndrome[2]

- **Etiology:** rectal mucosal prolapse or intussusception resulting in localized ulceration
- **Clinical features:** constipation, blood and mucus in rectum, rectal pain
- Uncommon, 3rd and 4th decade, more common in women
- **Gross description:** well demarcated irregular ulcer(s) on rectal wall 4-10 cm from anal margin. Also polypoid, rough, erythematous lesions. Mucosal thickening.
- **Treatment:** laxatives, possible resection, pelvic floor therapy or surgical correction or prolapse

1.Gonzalez R. PathologyOutlines.com website. http://www.pathologyoutlines.com/topic/colonstercoralulcer.html. Accessed June 3, 2020; 2. Gonzalez R. PathologyOutlines.com website. http://www.pathologyoutlines.com/topic/colonsolitaryrectalulcer.html. Accessed June 3, 2020.

Thank You!