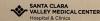


Etiology UGIB Comment & Treatment		
PUD (20–67%) (NEJM 2016:374:2367) See "PUD"	Treatment: PPI: 80 mg IV bolus + 8 mg/h drip = 40 mg IV BID boluse Endoscopic therapy: epi inj + bipolar cautery or hemoclip. Biopsies for ? H. pylori and treat if ⊕.  High-risk (for rebleeding) ulcer: arterial spurting, adherent clot, visible vessel. Endo Rx, IV PPI × 72 h post EGD, then Δ to high-dose oral PPI. Arteriography w/ embolization; surgery (last resort). Intermediate-risk ulcer: oozing, in o/w stable Pt. Endo Rx, can Δ to oral PPI after EGD and observe 24–48 h.  Low-risk ulcer: clean-based or flat. Oral PPI and ? discharge. Hold anticoag & antiplatelet Rx until hemostasis; can resume after hemostasis & PPI on board (8M) 2012;344:e3412).	
Erosive Precipitants: NSAIDs, ASA, EtOH, cocaine, gut ischemia, XRT Stress-related mucosal injury in ICU Pts. Risk factors include s coagulopathy, mech vent >48 h, high dose glucocorticoids Treatment: high-dose PPI		
Erosive esophagitis (5–18%)	Risk factors: cirrhosis, anticoagulation, critical illness. Rx offending cause + high dose PPI; repeat EGD later to r/o underling Barrett's.	

Esophageal or gastric varices (4–20%) (Hep 2007;46:922; NEJM 2010;362:823) See "Cirrhosis"		$2^{\circ}$ to portal HTN. If isolated gastric $\rightarrow$ r/o splenic vein thrombosis.
		Octreotide 50 µg IV bolus → 50 µg/h infusion (84% success).  Usually × 5 d, but most benefit w/in 24–48 h.  Abx: 20% cirrhotics p/w GIB have infxn, & ~50% develop infxn during hospitalization; Ppx w/ IV CTX, cipro, or levoflox × 7 d  Nonpharmacologic
		Endoscopic band ligation (>90% success) or sclerotherapy Arteriography w/ coiling, or if available, endoscopic injection of cyanoacrylate (glue) for gastric varices
		Covered esophageal stent placement or balloon tamponade used for bleeding refractory to ligation as bridge to TIPS (consider early if persistent bleed on EGD or Child-Pugh C; NEJM 2010;362:2370) For persistent gastric variceal bleed: TIPS or balloon-retrograde transvenous obliteration
	tal HTN ropathy	$\uparrow$ portal venous pressure $\rightarrow$ ectatic vessels, hyperemia in prox. gastric body. No endoscopic option; Rx portal HTN (octreotide), $\beta B.$
(9	Angioectasia AVMs, HHT (see below)	AVMs congenital. Angioectasia (ectatic submucosal vessels) a/w ↑ age, CKD, cirrhosis, CTD, severe CV dis. Heyde syndrome: GIB d/t angioectasias + aortic stenosis. Endo Rx.
Vascular (2-8%)	Dieulafoy's lesion	Large (1–3 mm) submucosal artery protruding through fundal mucosa → sudden, massive UGIB. Difficult to identify. Endo Rx.
	Gastric antral vasc. ectasia (GAVE)	"Watermelon stomach"; ectatic gastric vessels, often a/w cirrhosis, CTD, typically older 9. Rx w/ thermal hemostasis, repeat q4–8wk to eradicate lesions.TIPS does not improve outcomes.
	Aortoenteric fistula	AAA or aortic graft erodes into 3 <sup>rd</sup> portion of duodenum. P/w "herald bleed"; if suspected, diagnose by endoscopy or CT.
Mal	ignancy (2-8%)	Endoscopic hemostasis of mass temporizing measure till cancer Rx
	lory-Weiss tear 12%)	GE jxn lacerations due to vomiting $\to \uparrow$ intraabd pressure & shearing effect. Can self-resolve w/o endo Rx. Rx w/ antiemetics, PPI.
Car	meron's lesions	Linear erosions in hiatal hernia due to mech trauma of diaphragm
	t-sphincter- my bleeding	Occurs in ~2% of cases, ↑ risk w/ more complicated procedure. Bleeding into duodenum. Rx w/ endo hemostasis.

Etiology LGIB Comment & Treatment (Am J Gastro 2015;110:1265 & 2016;111:755	
Diverticular bleed (30%)	Pathophysiology: Intimal thickening and medial thinning of vasa recta as they course over dome of diverticulum → weakening of vascular wall → arterial rupture. Diverticula more common in left colon; but bleeding diverticula more often in right colon.  Clinical: older, ASA/NSAIDs, painless hematochezia, ± abd cramping Treatment: Usually stops spont. (~75%) but may take hrs—days; ~20% recur. Can perform endo hemostasis w/ epi injections ± electrocautery (NEJM 2000;342:78), hemoclip, banding. Intra-arterial vasopressin or embo. Surgery (partial colectomy) last resort.
Polyp/Tumor (20%) Typically slow ooze, p/w fatigue, weight loss, iron deficiency and	
Colitis (20%) Infectious (see "Acute Diarrhea"), IBD, ischemic colitis, XRT	
Anorectal Internal, external hemorrhoids; anal fissures, rectal ulcers, rectal disorders (20%) varices (Rx by ↓ portal venous pressure in cirrhotics), XRT	
Vascular (<10%)  Angioectasia & AVMs (see above). Hereditary Hemorrhagic  Telangiectasia (Weber-Osler-Rendu): diffuse AVMs, telangiectasias throughout GI mucosa (also involve lips, oral mucosa, fingertips)	
Meckel's diverticulum	Congenital blind intestinal pouch due to incomplete obliteration of vitelline duct. 2% of pop, w/in 2' of IC valve, 2" long, 3:2 2:1, often present age 2 (but can cause obscure GIB in adults). Dx w/ 99mTc-pertechnetate scintigraphy. Rx w/ angioembo, surgical resection.







## Patient PHI

nead:

#### TRANSFUSION OF BLOOD/COMPONENTS ADVISORY AND CONSENT (Medical Record)

TRANSFUSION ADVISORY (physician check boxes that apply)

I have discussed the possible risks/benefits and alternatives of blood and/or blood components transfusion with the patient or legal representative, including:

- · Any adverse reactions that may reasonably be expected to occur;
- Any alternative methods of treatment which may be medically viable;
- The potential problems that may occur during recuperation;
- Any research or economic interest he/she may have regarding this treatment and
- Techniques for blood conservation directed donation and processing, such as hemodilution, use of a cell saver, autologous banked blood, plasmapheresis, hemodialysis, cardiopulmonary bypass, chest tube drainage, autotransfusion, etc.

of ac dii pa m	Health Service: dvantages, disar- rected homologo attient or other p edical contraindi Patient or legal	ent's legal representative has been provided with a sinformation sheet, A Patient's Guide to Blood dvantages, risks, and benefits of autologous blood bus blood products from volunteers. I have also a erson to pre-donate blood for transfusion purpocations or the patient waives the right of pre-donarepresentative consents to transfusion of all blood epresentative consents to receive some blood componentative Red Blood Cells Plasma Platelets Crambuman Derived Coagulation Factors	Transfusions, concerning the content of the content
	(Patient must s	representative refuses transfusions of blood or bign form #5193ESV).	plood components
Date	Time (AM/PM)	Signature MD Physician signs Pr	ovider Stamp (or MD Print Name /Number
5. Sp A signed transfusion nature of, each tran	pod component to the patient of the patient has the pati	pable throughout the patient's hospitalization. For outpatients with a stable chroni d consent form is applicable for one year or if during that one-year period circum sion and/or the atternative to treatment. For outpatients without such a chronic of eleption to withdraw consent at any time.	risks and benefits.  uding pre-donation.  and/or blood component  c medical condition for which ongoing  nstances change so as to materially affect the condition, a consent form must be signed for
Date	Time (AM/PM)	Signature patient/parent/conservator/guardian//legal representative Pt/DPOA signs	Relationship to patient
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I have the pat	tient's or legal repre	ppletely read this document to (patient or representative) _	ir y language). He/She understood
Date_	and the second second second second	Signature of Interpreter (if applicable)	Interpreter ID
5193B-EE	(Rev. 11/20)	DISTRIBUTION: ORIGINAL: Medical Record, Administrative COPY: Patie	ent

#### **Published Estimates of Transfusion Risks**

Reference: A Compendium of Transfusion Practice Guidelines 2019, Edition 3a by American Red Cross

The reported incidence of adverse reactions after transfusion varies widely among studies. Published rates depend on a number of factors, including but not limited to: the patient population and the presence of underlying disease, concurrent medication, or immunosuppression; blood component type and preparation method; and the surveillance methods used for reporting and characterizing transfusion reactions or suspected infections. Therefore, it is important to consider the many factors that affect the estimates of incidence in different clinical

Before blood transfusion, the clinician should explain to the patient the potential risks, possible benefits and alternatives, when available, before transfusion. The Summary Table provides broad-based estimates from a variety of current sources which could be used to develop general information for patients. However, risk depends on patient-related factors, type and characteristics of the blood components, geographically-defined and other variables which should be periodically evaluated, as warranted.

Transfusion Reaction or Infection	Estimated Rate Among Transfused Patients
Allergic (mild)	1:20
Fever/chills (nonhemolytic)	1:50
Transfusion-associated circulatory overload (TACO)	1:100
TRALI	1:12,000
Acute hemolytic (mistransfusion)	1:40,000
Acute hemolytic (incompatible plasma)	1:50,000
Delayed hemolytic	1:50,000
Septic reaction (apheresis platelets)	1:100,000
Anaphylaxis	1:500,000
HIV, HBV, HCV	Approximately 1:1,000,000

Risks of Autologous Transfusion: bacterial contamination, air/particle embolism, immunomodulation, wrong unit transfusion, volume overload, fainting at time of blood donation, bruising at time of blood donation, not getting own unit of blood, still needing additional blood from donor pool, autologous blood may not be suitable for transfusion, and unknown.

#### **Expected Benefits of Transfusion:**

- RBC: Restoration of O<sub>2</sub> carrying capacity in those patients who have preexisting cardiac and cerebral vascular disease and who are anemic, by increasing the circulating red blood cell mass.
- Platelets: Improves hemostasis; associated with cessation of bleeding, correction of prolonged bleeding time and a rise in the platelet count.
- Plasma: Contains plasma proteins, including nonlabile clotting factors such as fibrinogen and Factor IX; treats clotting factors deficiencies.

#### Alternatives - Proceed with planned treatment, but consider:

- No transfusion
- Use of other pharmacologic agents; metabolic supplements, drugs
- Cessation of offending drugs, electrolytes, or colloid solutions, hespan
- Transfusion, but only with autologous and/or directed donations

The greatest threat to blood safety is donation by seronegative individuals during the infectious window period when they are undergoing seroconversion and infection cannot be detected by available laboratory test.

## **CC**: Coffee-ground emesis & melena

**HPI**: 57 y/o man.

1 day prior to admission: 1x coffee-ground emesis. On day of admission: 2x melena, prompting him to go ED.

No prior history of EGD / colo.

ROS negative.

## **PHYSICAL EXAM:**

Tmax: 36.3C, BP: 105/64, HR: 88, RR: 16, SpO2: 100% on RA

General: in NAD, resting in bed

HEENT: PERRL, EOMI, oropharynx clear, anicteric sclera

CV: irregular rhythm, no M/R/G

Pulm: unlabored breathing on room air, CTAB

**GI:** BS+, nondistended, soft, nontender to palpation, no rebound

tenderness, no guarding

MSK: no edema, warm extremities with palpable distal pulses

Neuro: AOx3, answering questions appropriately, moving all extremities

against gravity

PMH: T2DM, HTN, afib

**SH:** Denied current or past use of ETOH, tobacco, or drugs.

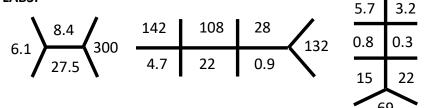
**FH:** noncontributory

**Allergies:** NKDA

Meds:

- Xarelto 20mg daily
- Metformin 1000mg BID
- Lisinopril 5mg daily
- Metoprolol succinate 25mg daily

## LABS:



PT: 16.6 INR: 1.4

11VIV. 1.4

Fibrinogen: 326

H. Pylori IgG Ab: 0.21 (not detected)

#### FGD:

- Salmon-colored mucosa was present in distal esophagus, suspect barrett's.
- Four non-bleeding gastric ulcers with a clean ulcer base (Forrest Class III) were found in the gastric antrum and in the prepyloric region of the stomach.
- One non-bleeding duodenal ulcer with a clean ulcer base (Forrest Class III) was found in the duodenal bulb.

#### PROBLEM REPRESENTATION:

Middle-aged man w/ PMH T2DM, HTN, afib (on Xarelto), who presented with acute coffee-ground emesis and melena.

# <u>Diagnosis:</u> UGIB due to gastric and duodenal ulcers

## **LEARNING POINTS:**

- 1. Practical skills when managing GIB
  - IV Access! Two large-bore peripheral IVs
  - Blood transfusion consent
  - Type & Screen

## 2. Favors UGIB

- Melena (also due to R-sided colonic bleed w/ slow transit)
- Hematemesis, coffee-ground emesis
- BUN/Cr > 30
- Hx UGIB

### 3. Favors LGIB

- Hematochezia (can also be massive UGIB w/ rapid transit through GI tract)
- Clots per rectum
- Hx of LGIB

## 4. Worrisome features:

PMH of cirrhosis / AAA repair / comorbidities that reduce hemodynamic reserve, active bleeding, syncope/presyncope, nml Hgb w/ unstable VS, anemia unresponsive to transfusion(s)