APPROACH TO UPPER GASTROINTESTINAL BLEED

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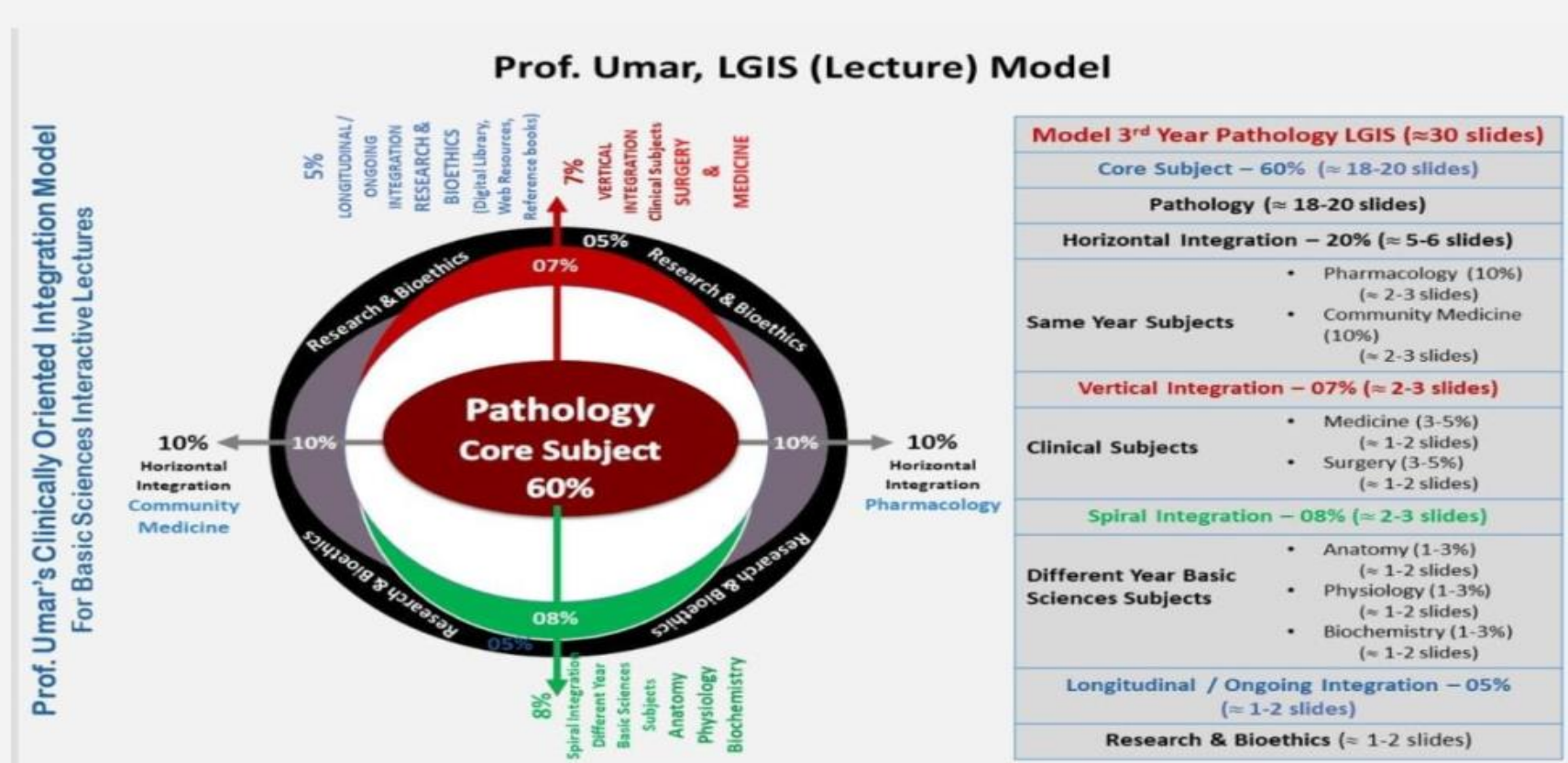
Upper GI bleed



Learning Outcomes

At the end of this lecture students of 3rd year MBBS should be able to:

- Define UGIB.
- Enlist its causes.
- Explain relevant points of history and examination
- Recall risk stratification score.
- Describe its management.

CASE SCENERIO

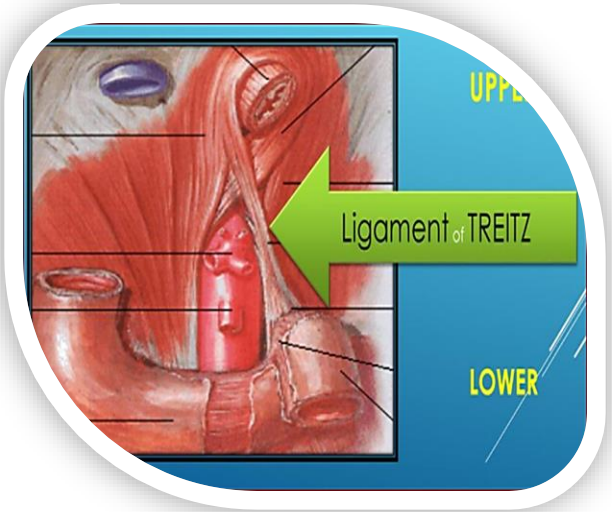
A 35 years old lady presents to ER. She has multiple episodes of vomiting fresh blood effortlessly in last 6 hours. She was diagnosed as a case of Rheumatoid Arthritis about one month back and was prescribed steroids(Deltacortil) and analgesics (NSAIDS) which she is taking on regular basis.

CASE SCENERIO

A 50 years House wife brought to Medical Emergency with two days' history of passage of black colored stools. She told that she got interferon therapy for HCV infection 15 years back. She never followed up with her doctor after that.

Examination revealed, pulse:130/min, BP110/70 mm Hg with postural drop and splenomegaly.

CORE SUBJECT



UGI Bleed proximal to ligament of Treitz.

Hematemesis : vomiting of blood.

1. Fresh blood, suggests rapid bleeding
2. Altered blood (Coffee grounds), suggest slower rate of bleeding

Melena : passage of black tarry stools (semi solid /strong odour).

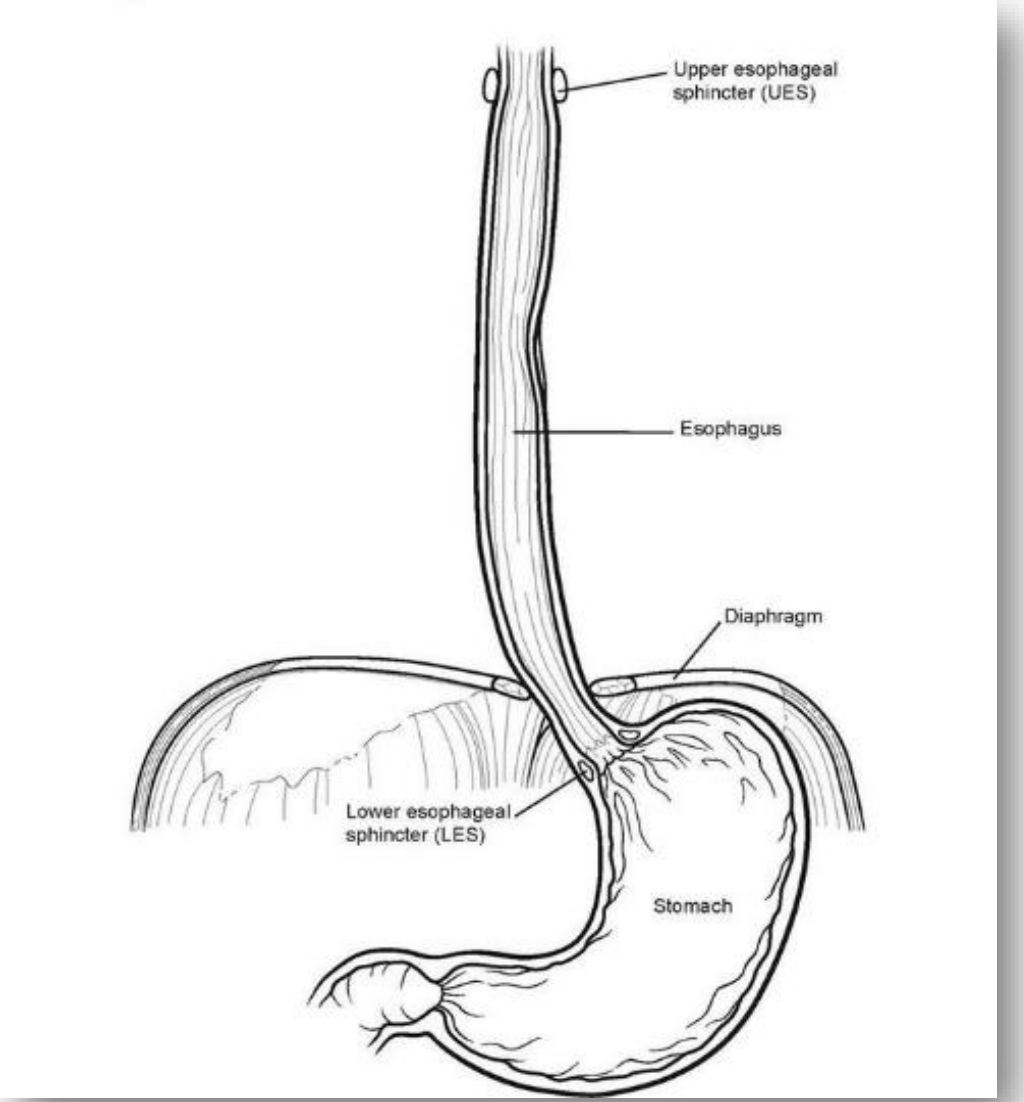
Action of digestive enzymes and bacteria on Hb

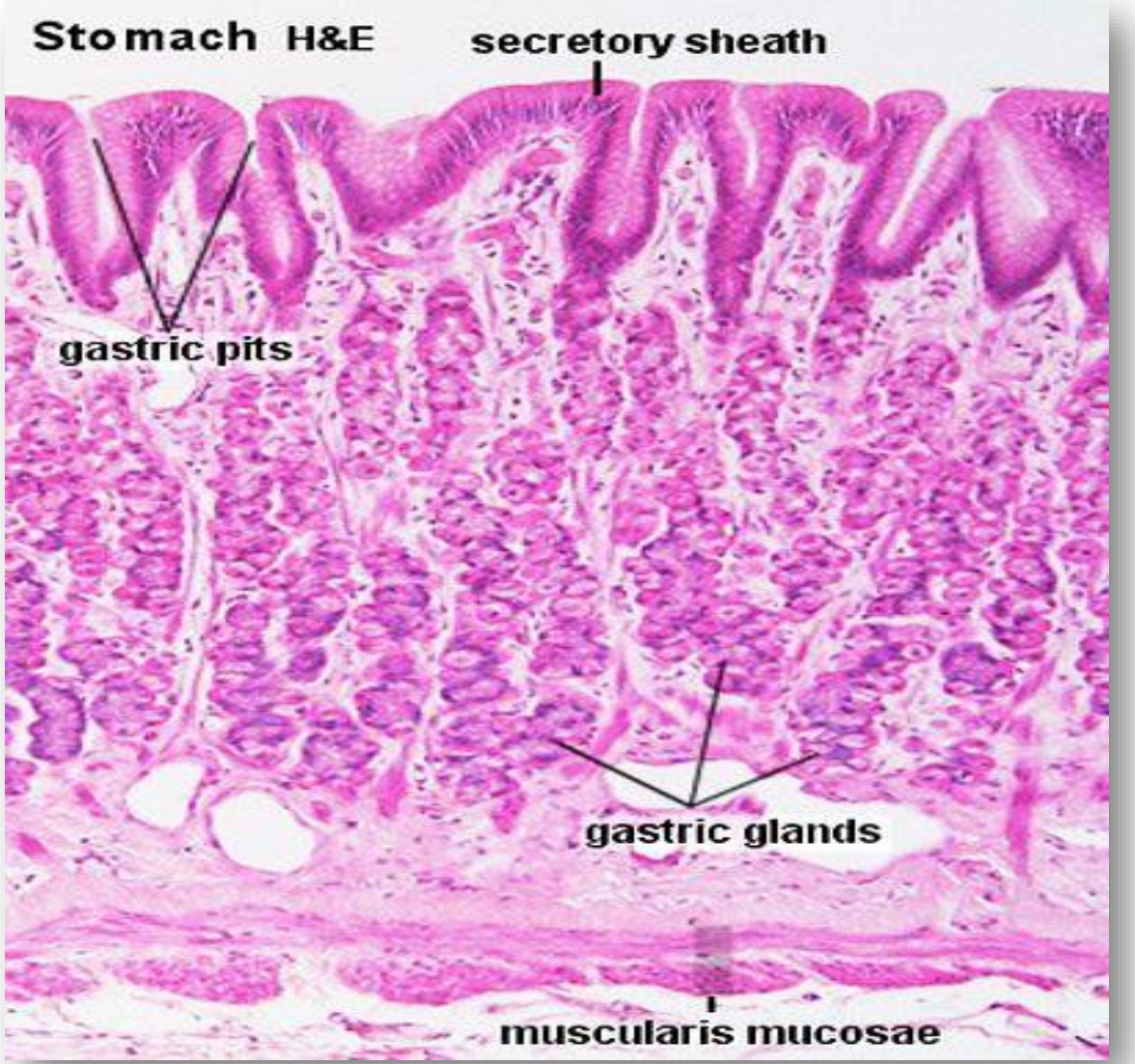
Hematochezia : Bright red blood per rectum usually represent LGIB

(although a brisk upper GI source of bleed can also cause hemotochezia as the blood doesn't remain in the bowel long enough for melena to develop)

SPIRAL INTEGRATION

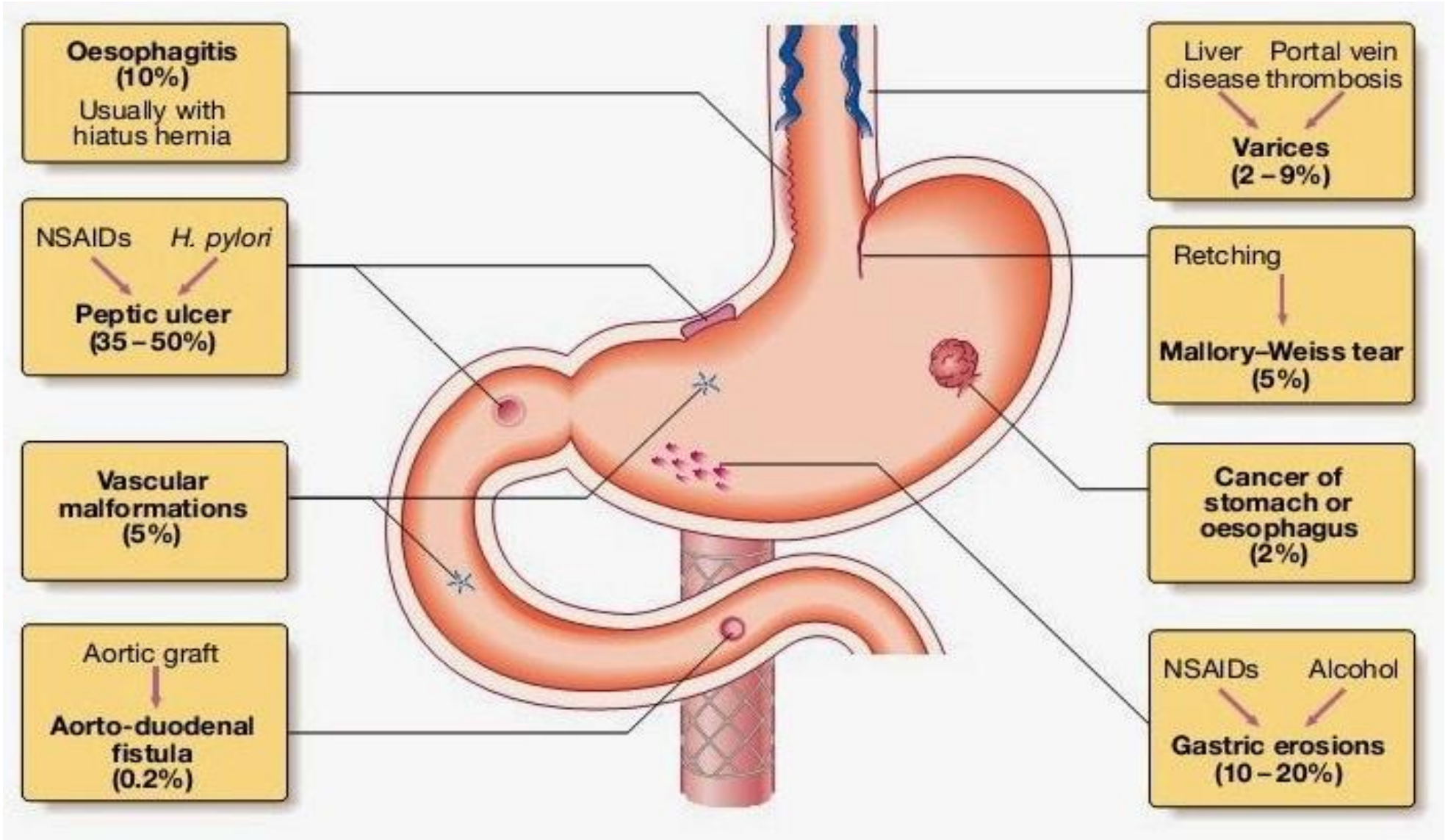
ANATOMY OF UPPER GI TRACT





HORIZONTAL INTEGRATION

PATHOPHYSIOLOGY OF UPPER GI BLEED



CAUSES OF UPPER GI BLEED

Common causes	Other causes
Peptic ulcer disease (gastric or duodenal)	Hemosuccus pancreaticus
Gastric or esophageal varices	Cameron lesions
Erosive esophagitis	Hemobilia
Upper gastrointestinal tumors	Aortoenteric fistula
Upper gastrointestinal angioectasias	Anastomotic bleeding
Mallory-Weiss tear	Arteriovenous malformation
Gastric or duodenal erosions	Acute esophageal necrosis
Dieulafoy lesion	Atrial-esophageal fistula
	Gastric antral vascular ectasia

CORE SUBJECT

HISTORY QUESTIONS

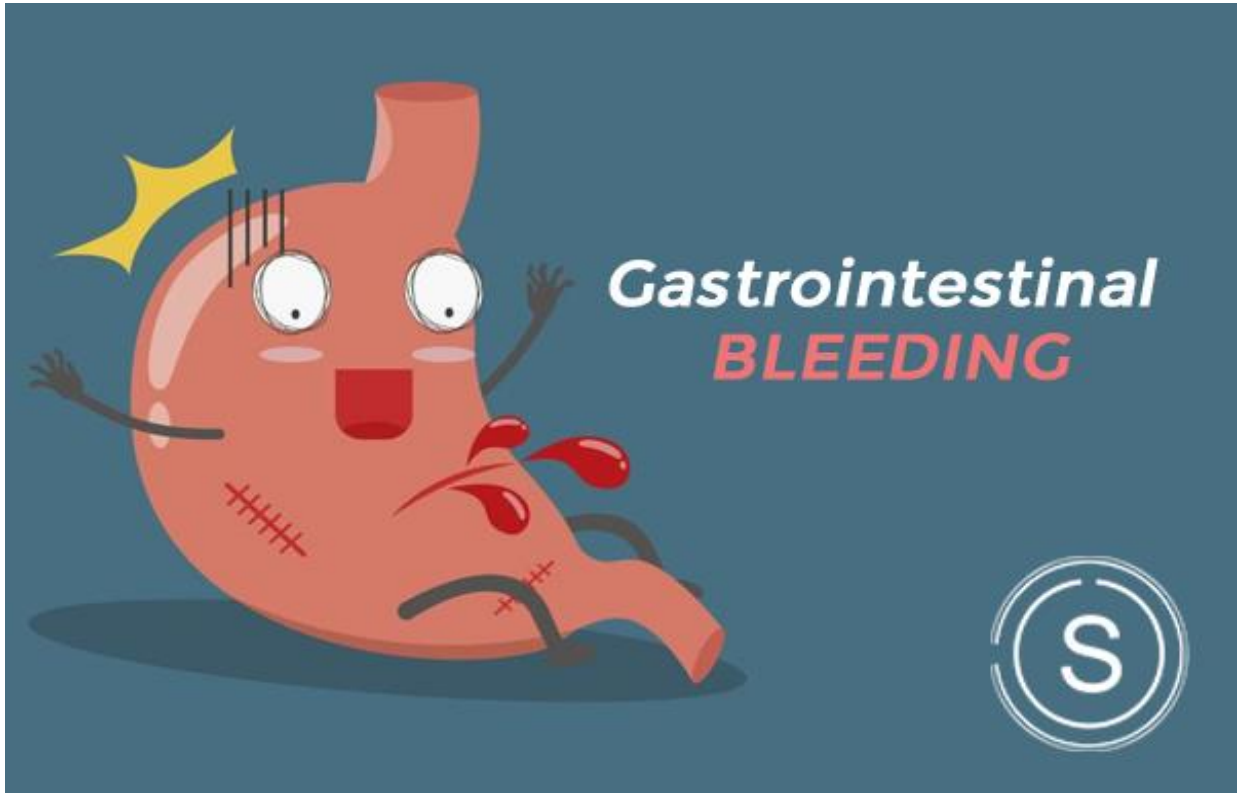
1. Was there fresh blood or coffee ground vomitus?
2. Have you passed any black or bloody stools?
3. Intense retching or coughing before bloody vomitus?
4. Have you been taking any drugs like aspirin, NSAIDS, anticoagulants or steroids?
5. Do you drink alcohol? Do you have liver disease- jaundice, ascites?
6. Have you ever had a peptic ulcer-upper abd pain/dyspepsia?
7. Have you lost weight?
8. Family history of GI malignancy?
9. History of abdominal aortic aneurysm repair or graft?
10. Comorbid illnesses; coronary artey disease, renal failure.

CORE SUBJECT

EXAMINATION

- Vital signs, Orthostatic Hypotension
- Abdominal tenderness/ guarding.
- Skin, oral examination
- Stigmata of liver disease
- Rectal examination

ACUTE UPPER GI BLEED



MANAGEMENT

Intravenous Access:

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

Initial Clinical Assessment:

- *Define circulatory status:*

Severe bleeding causes tachycardia, hypotension and oliguria. The patient is cold and sweating, and may be agitated.

- *Seek evidence of liver disease:*

Jaundice, cutaneous stigmata, hepatosplenomegaly and ascites may be present in decompensated cirrhosis.

- *Identify comorbidity:*

The presence of cardiorespiratory, cerebrovascular or renal disease is important, both because these may be worsened by acute bleeding and because they increase the hazards of endoscopy and surgical operations

VERTICAL INTEGRATION

INITIAL ASSESSMENT

- Always remember to assess A,B,C.
- Assess degree of hypovolemic shock

	Class I	Class II	Class III	Class IV
Blood loss (mL)	750	750-1500	1500-2000	>2000
Blood volume loss (%)	< 15%	15-30%	30-40%	>40%
Heart rate	<100	>100	>120	>140
SBP	No change	Orthostatic change	Reduced	Very low, supine
Urine output (mL/hr)	>30	20-30	10-20	<10
Mental status	Alert	Anxious	Aggressive/drowsy	Confused/unconscious

VERTICAL INTEGRATION

Basic Investigations:

Full blood count. Chronic or subacute bleeding leads to anaemia but the haemoglobin concentration may be normal after sudden, major bleeding until haemodilution occurs.

Thrombocytopenia may be a clue to the presence of hypersplenism in chronic liver disease.

Urea and electrolytes. The blood urea rises as the absorbed products of luminal blood are metabolised by the liver; an elevated blood urea with normal creatinine concentration implies severe bleeding.

Liver function tests. These may show evidence of chronic liver disease.

Prothrombin time. Check when there is a clinical suggestion of liver disease or patients are anticoagulated.

Cross-matching. At least 2 units of blood should be cross-matched if a significant bleed is suspected.

VERTICAL INTEGRATION

4. Resuscitation

- Use crystalloids first.

- Anticipate need for blood transfusion

    1. Threshold should be based on underlying comorbid condition, hemodynamic status etc.
    2. Should be administered if Hb ≤ 7 g/dL
    3. 1pint of RCC should raise Hb by 1g/dl (HCT by 3%)
    4. Remember that initial Hct can be misleading.

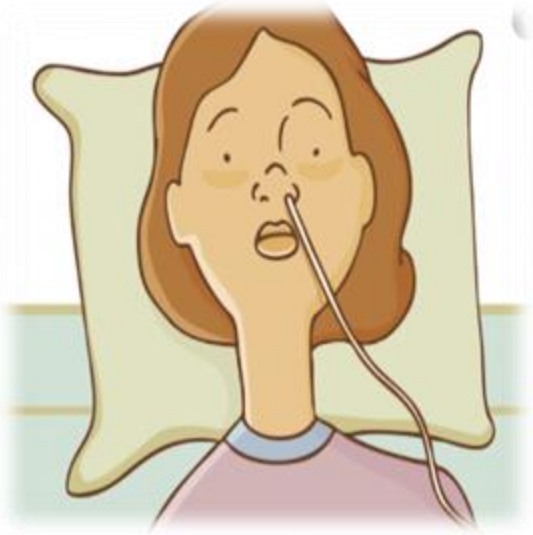
- Correct coagulopathy

- Patients with chronic liver disease should be given broad spectrum antibiotics

VERTICAL INTEGRATION

UTILITY OF NG TUBE

- Most useful situation: patients with severe hematochezia or uncertainty about source of bleed (UGIB vs. LGIB)
  - Red blood per NGT – predictive of high risk endoscopic lesion
  - Coffee grounds – less severe/inactive bleeding



RISK STRATIFICATION

- Identify patients at high risk for adverse outcomes
- Helps determine disposition (ICU vs. Ward vs. discharge)
- May help guide appropriate timing of endoscopy

These factors can be combined using the Blatchford score

- (Box 21.16), which can be calculated at the bedside. A score of 2 or less is associated with a good prognosis, while progressively higher scores are associated with poorer outcomes

21.16 Modified Blatchford score: risk stratification in acute upper gastrointestinal bleeding

Admission risk marker	Score component value
Blood urea	
≥25 mmol/L (70 mg/dL)	6
10–25 mmol/L (28–70 mg/dL)	4
8–10 mmol/L (21.4–28 mg/dL)	3
6.5–8 mmol/L (18.2–22.4 mg/dL)	2
<6.5 mmol/L (18.2 mg/dL)	0
Haemoglobin for men	
<100 g/L (10 g/dL)	6
100–119 g/L (10–11.9 g/dL)	3
120–129 g/L (12–12.9 g/dL)	1
≥130 g/L (13 g/dL)	0
Haemoglobin for women	
<100 g/L (10 g/dL)	6
100–119 g/L (10–11.9 g/dL)	1
≥120 g/L (12 g/dL)	0
Systolic blood pressure	
<90 mmHg	3
90–99 mmHg	2
100–109 mmHg	1
>109 mmHg	0
Other markers	
Presentation with syncope	2
Hepatic disease	2
Cardiac failure	2
Pulse ≥100 beats/min	1
Presentation with melaena	1
None of the above	0

ROCKALL SCORING SYSTEM

- Validated predictor of mortality in patients with UGIB
- 2 components: clinical + endoscopic

ROCKALL SCORE

- For Risk of Rebleeding and Death After Admission to the Hospital for Acute GI Bleeding

Variable	Score 0	1	2	3
Age (yrs)	< 60	60-79	≥ 80	
Comorbidity	No or mild coexisting	Moderate coexisting (e.g., hypertension)	Severe coexisting (e.g., CHF)	Life threatening (e.g., RF)
Hemodynamic status	No shock P < 100 Syst BP ≥ 100	P ≥ 100 plus Sys BP ≥ 100	Hypotension	
Diagnosis	MW tear, normal endoscopy with no blood seen	All other diagnosis	Malignancy of UGI tract	
Major stigmata of recent hemorrhage	None or dark spot		Blood in UGI tract Adherent clot, visible or spurting vessel	

Interpretation: A score less than 3 carries good prognosis but total score more than 8 carries high risk of mortality.

5. PRE-ENDOSCOPIC PHARMACOTHERAPY

For Non-Variceal UGIB:

IV PPI: 80 mg bolus followed by 8 mg/hr infusion.

Rationale: suppress acid and prevents clot dissolution.

VERTICAL INTEGRATION

VARICEAL BLEED MANAGEMENT

- **V**asoconstrictor therapy (Sandostatin , Terlipressin)
- Octreotide (Sandostatin) :

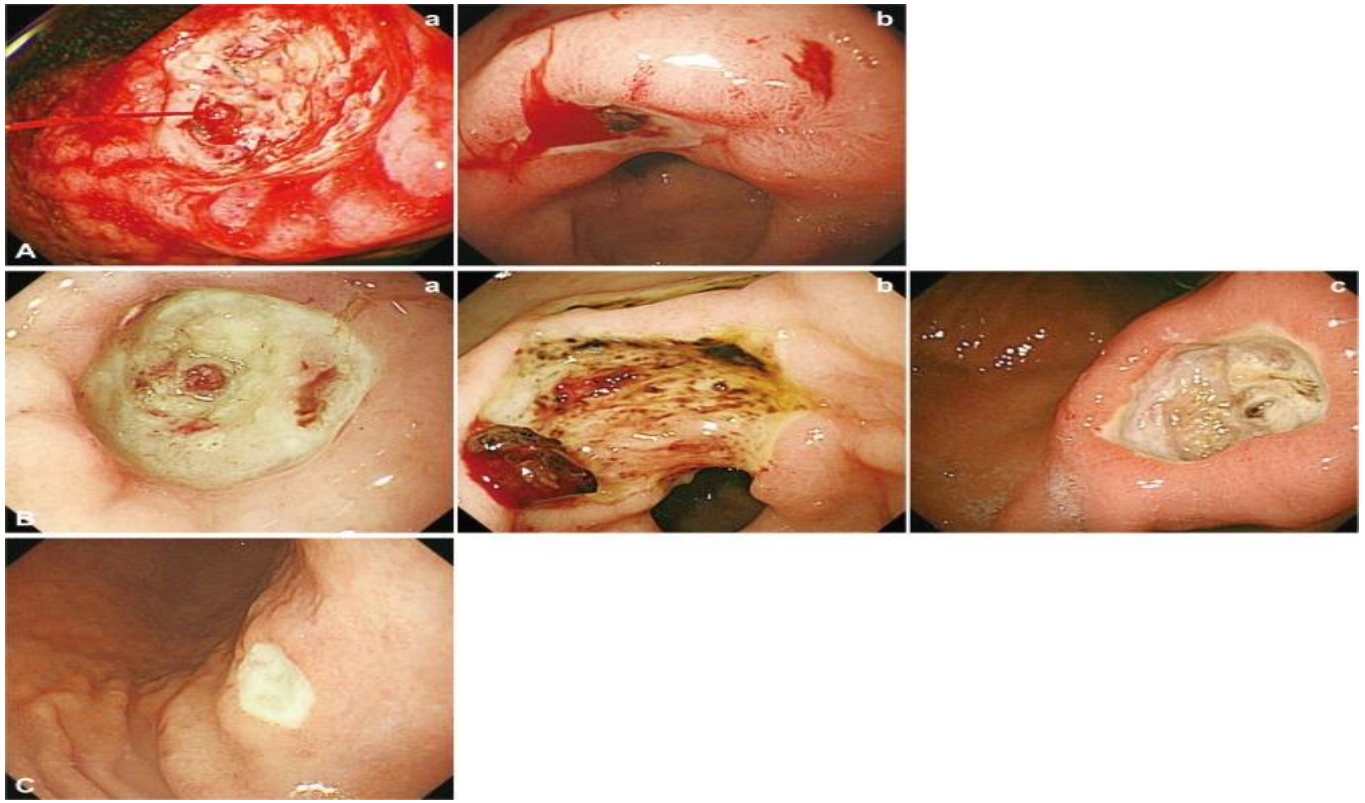
splanchnic vasoconstrictor

Dose: 50 ug IV/SC stat, then @25-50ug/hr

- **A**ntibiotics
- **R**esuscitation
- **ICU** level care
- **E**ndoscopy
- **AL**ternative/Rescue therapies
- **B**eta blockade

6. ENDOSCOPY

Early endoscopy (within 24 hours) is recommended for most patients with acute UGIB.



FORREST CLASSIFICATION OF PEPTIC ULCER BLEED

ENDOSCOPIC HEMOSTASIS THERAPY

- Epinephrine injection

- Thermal electrocoagulation

- Mechanical (hemoclips)

Combination therapy is superior to monotherapy

VERTICAL INTEGRATION

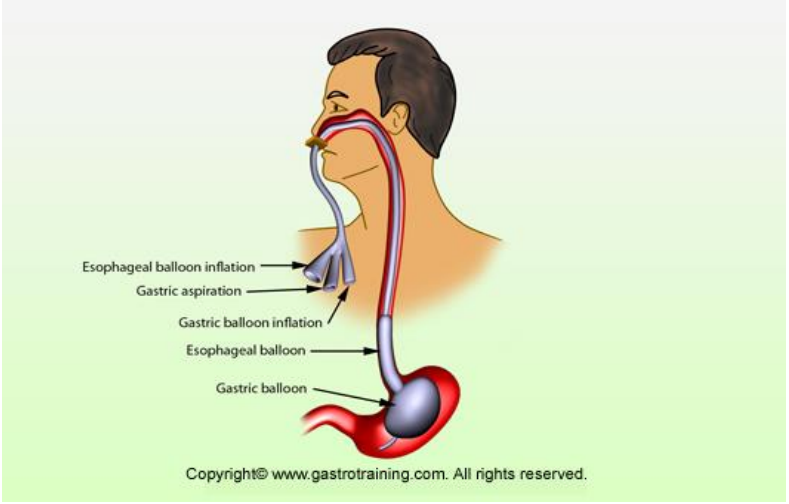
Endoscopic Therapy for varices

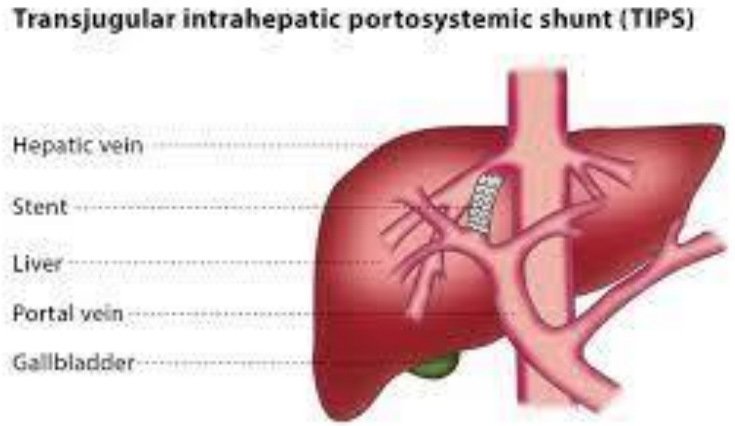
- Should be performed as soon as possible after resuscitation (within 12 hours)
- Endotracheal intubation frequently needed
- Band ligation is preferred method

VERTICAL INTEGRATION

ALTERNATE/RESCUE THERAPIES

- Sengstaken Blakemore tube.
- TIPS – Transjugular Intrahepatic Portosystemic Shunt.
- Liver Transplant.





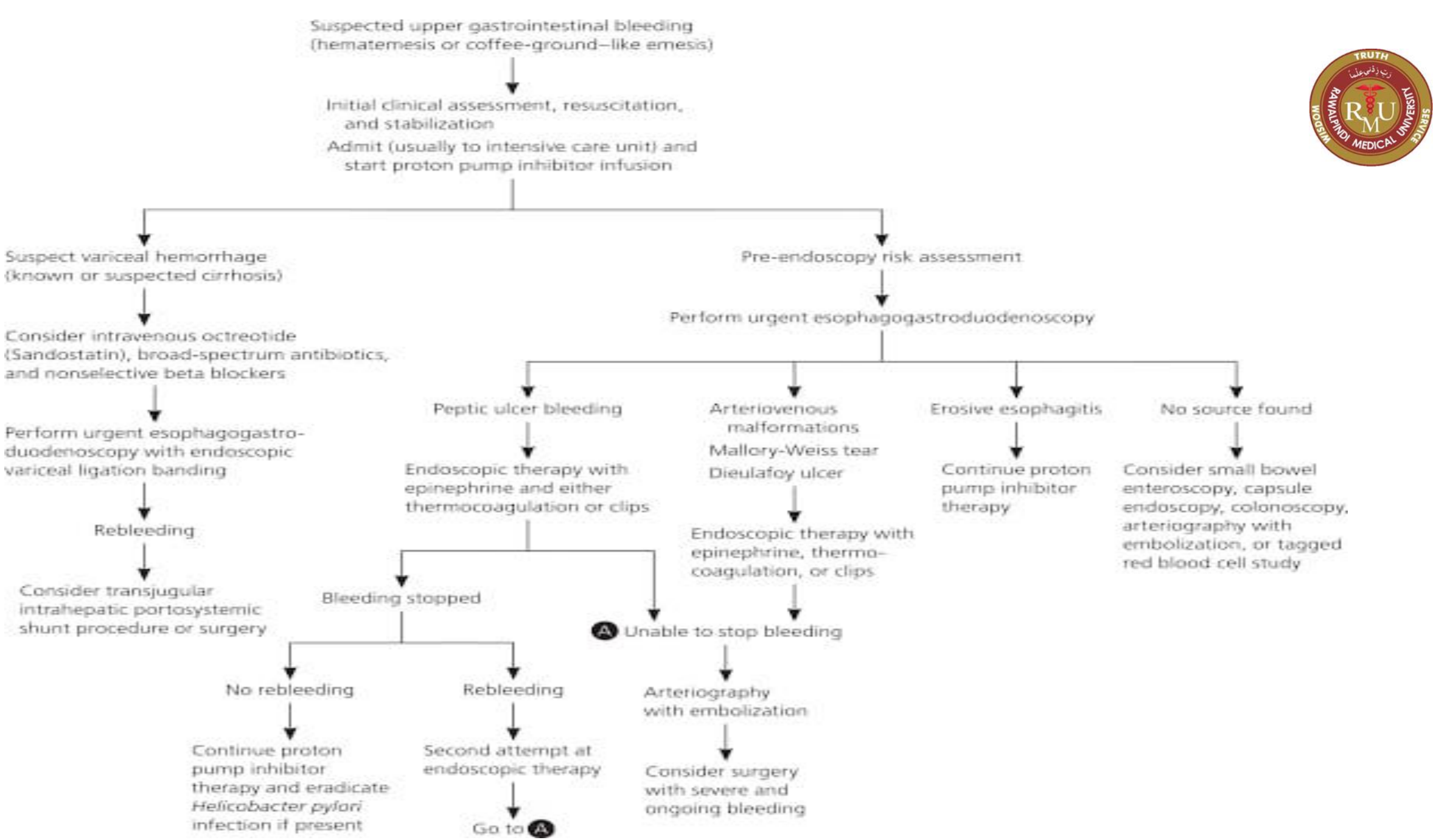
Role of Surgery

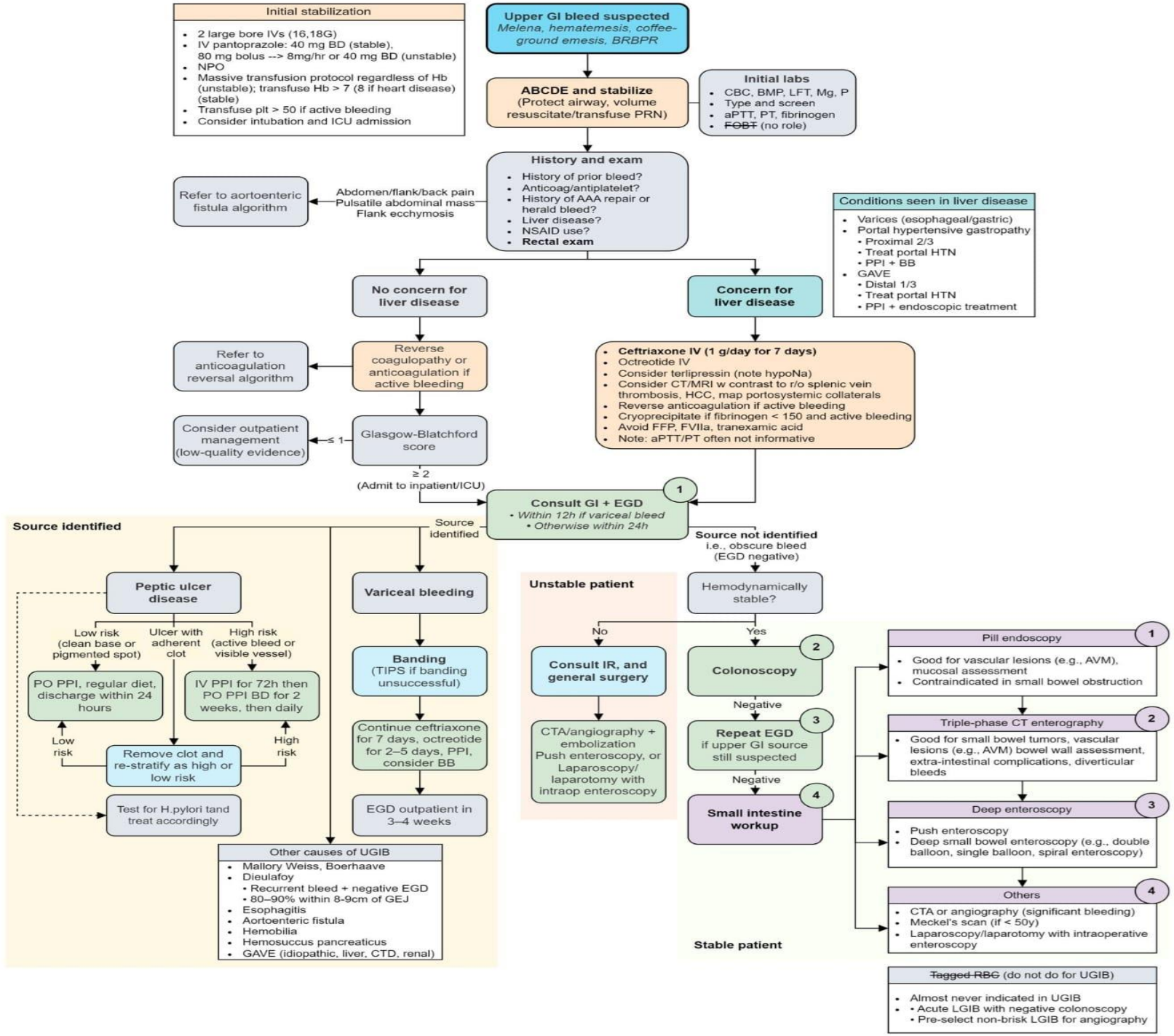
If there is failure of bleeding control with medical and endoscopic therapy, involve:

- Interventional radiology.
- Surgery.

8. Post Endoscopic Management

- Patients with low risk ulcers can be fed promptly, put on oral PPI therapy.
- Patients with ulcers requiring endoscopic therapy should receive **PPI inf x 72 hours.**
- Determine *H. pylori* status in all ulcer patients and eradicate if positive
- Discharge patients on PPI.
- In patients with cardiovascular disease on low dose aspirin: **restart as soon as bleeding has resolved**


Suspected upper gastrointestinal bleeding (hematemesis or coffee-ground–like emesis)
Initial clinical assessment, resuscitation, and stabilization
Admit (usually to intensive care unit) and start proton pump inhibitor infusion
Suspect variceal hemorrhage (known or suspected cirrhosis)
Consider intravenous octreotide (Sandostatin), broad-spectrum antibiotics, and nonselective beta blockers
Perform urgent esophagogastro-duodenoscopy with endoscopic variceal ligation banding
Rebleeding
Consider transjugular intrahepatic portosystemic shunt procedure or surgery
Pre-endoscopy risk assessment
Perform urgent esophagogastroduodenoscopy
Peptic ulcer bleeding
Endoscopic therapy with epinephrine and either thermocoagulation or clips
Bleeding stopped
No rebleeding
Continue proton pump inhibitor therapy and eradicate Helicobacter pylori infection if present
Rebleeding
Second attempt at endoscopic therapy
Go to A
Arteriovenous malformations
Mallory-Weiss tear
Dieulafoy ulcer
Endoscopic therapy with epinephrine, thermo-coagulation, or clips
A Unable to stop bleeding
Arteriography with embolization
Consider surgery with severe and ongoing bleeding
Erosive esophagitis
Continue proton pump inhibitor therapy
No source found
Consider small bowel enteroscopy, capsule endoscopy, colonoscopy, arteriography with embolization, or tagged red blood cell study


Initial stabilization
2 large bore IVs (16,18G)
IV pantoprazole: 40 mg BD (stable), 80 mg bolus --> 8mg/hr or 40 mg BD (unstable)
NPO
Massive transfusion protocol regardless of Hb (unstable); transfuse Hb > 7 (8 if heart disease) (stable)
Transfuse plt > 50 if active bleeding
Consider intubation and ICU admission
Upper GI bleed suspected
Melena, hematemesis, coffee-ground emesis, BRBPR
ABCDE and stabilize
(Protect airway, volume resuscitate/transfuse PRN)
Initial labs
CBC, BMP, LFT, Mg, P
Type and screen
aPTT, PT, fibrinogen
FOBT (no role)
History and exam
History of prior bleed?
Anticoag/antiplatelet?
History of AAA repair or herald bleed?
Liver disease?
NSAID use?
Rectal exam
Abdomen/flank/back pain
Pulsatile abdominal mass
Flank ecchymosis
Refer to aortoenteric fistula algorithm
Conditions seen in liver disease
Varices (esophageal/gastric)
Portal hypertensive gastropathy
Proximal 2/3
Treat portal HTN
PPI + BB
GAVE
Distal 1/3
Treat portal HTN
PPI + endoscopic treatment
No concern for liver disease
Concern for liver disease
Reverse coagulopathy or anticoagulation if active bleeding
Refer to anticoagulation reversal algorithm
Glasgow-Blatchford score
≤ 1
Consider outpatient management (low-quality evidence)
≥ 2
(Admit to inpatient/ICU)
Ceftriaxone IV (1 g/day for 7 days)
Octreotide IV
Consider terlipressin (note hypoNa)
Consider CT/MRI w contrast to r/o splenic vein thrombosis, HCC, map portosystemic collaterals
Reverse anticoagulation if active bleeding
Cryoprecipitate if fibrinogen < 150 and active bleeding
Avoid FFP, FVIIa, tranexamic acid
Note: aPTT/PT often not informative
1
Consult GI + EGD
Within 12h if variceal bleed
Otherwise within 24h
Source identified
Source not identified
i.e., obscure bleed (EGD negative)
Peptic ulcer disease
Low risk (clean base or pigmented spot)
Ulcer with adherent clot
High risk (active bleed or visible vessel)
PO PPI, regular diet, discharge within 24 hours
IV PPI for 72h then PO PPI BD for 2 weeks, then daily
Low risk
High risk
Remove clot and re-stratify as high or low risk
Test for H.pylori tand treat accordingly
Variceal bleeding
Banding
(TIPS if banding unsuccessful)
Continue ceftriaxone for 7 days, octreotide for 2–5 days, PPI, consider BB
EGD outpatient in 3–4 weeks
Other causes of UGIB
Mallory Weiss, Boerhaave
Dieulafoy
Recurrent bleed + negative EGD
80–90% within 8-9cm of GEJ
Esophagitis
Aortoenteric fistula
Hemobilia
Hemosuccus pancreaticus
GAVE (idiopathic, liver, CTD, renal)
Unstable patient
Hemodynamically stable?
No
Yes
Consult IR, and general surgery
CTA/angiography + embolization
Push enteroscopy, or Laparoscopy/ laparotomy with intraop enteroscopy
2
Colonoscopy
Negative
3
Repeat EGD
if upper GI source still suspected
Negative
4
Small intestine workup
Stable patient
Pill endoscopy
1
Good for vascular lesions (e.g., AVM), mucosal assessment
Contraindicated in small bowel obstruction
Triple-phase CT enterography
2
Good for small bowel tumors, vascular lesions (e.g., AVM) bowel wall assessment, extra-intestinal complications, diverticular bleeds
Deep enteroscopy
3
Push enteroscopy
Deep small bowel enteroscopy (e.g., double balloon, single balloon, spiral enteroscopy)
Others
4
CTA or angiography (significant bleeding)
Meckel's scan (if < 50y)
Laparoscopy/laparotomy with intraoperative enteroscopy
Tagged RBC (do not do for UGIB)
Almost never indicated in UGIB
Acute LGIB with negative colonoscopy
Pre-select non-brisk LGIB for angiography

RESEARCH

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Characteristics of peptic ulcer bleeding in cirrhotic patients with esophageal and gastric varices

Zheng Lu[1,3], Xiaotian Sun[2,3], Jingjing Han[1], Bo Jin[1], Wenhui Zhang[1], Jun Han[1], Xuemei Ma[1], Bo Liu[1], Xiaoli Yu[1], Qin Wu[1], Yanling Wang[1] & Hanwei Li[1]✉

Upper gastrointestinal bleeding (UGIB) is common in liver cirrhosis. Although esophageal and gastric varices (EGV) is the main bleeding source, there were still a proportion of patients with peptic ulcer bleeding. Thus, this study aimed to analyze the characteristic of variceal bleeding and peptic ulcer bleeding in liver cirrhosis. Cirrhotic patients with confirmed UGIB by urgent endoscopy from July 2012 to June 2018 were enrolled, and classified into peptic ulcer bleeding group (n = 248) and variceal bleeding group (n = 402). Clinical and endoscopic characteristics, therapeutic efficacy and prognosis were evaluated, and independent risk factors for 42-day morality were determined. The mean age and gender ratio of peptic ulcer bleeding group were higher than those in variceal bleeding group (55.58 ± 11.37 vs. 52.87 ± 11.57, $P < 0.01$; 4.51:1 vs. 2.87:1, $P = 0.023$). Variceal bleeding group most commonly presented as red blood emesis and coffee grounds (67.16%), while peptic ulcer group primarily manifested as melena (62.10%). Hepatocellular carcinoma was more prevalent in peptic ulcer group (141 vs. 119, $P < 0.01$). Albumin level in variceal bleeding group was lower higher ($P < 0.01$), but serum bilirubin, creatinine and prothrombin time were significantly higher (all $P < 0.01$). Success rate of endoscopic hemostasis for variceal bleeding and peptic ulcer bleeding was 89.05% and 94.35% ($P = 0.021$). Univariate and multivariate analysis identified prothrombin time ($P = 0.041$, OR [95% CI] 0.884 [0.786–0.995]), MELD score ($P = 0.000$, OR [95% CI] 1.153 [1.073–1.240]), emergency intervention ($P = 0.002$, OR [95% CI] 8.656 [2.219–33.764]), hepatic encephalopathy before bleeding ($P = 0.003$, OR [95% CI] 8.119 [2.084–31.637]) and hepatic renal syndrome before bleeding ($P = 0.029$, OR [95% CI] 3.877 [1.152–13.045]) as the independent predictors for 42-day mortality. Peptic ulcer bleeding should be distinguished from variceal bleeding by clinical and endoscopic characteristics.

Upper gastrointestinal bleeding (UGIB) is an important public health issue with a prevalence of 150 per 100 thousand each year[1,2], and the inpatient mortality can be up to 10%[3]. In clinical practice, acute UGIB is a critical condition, and liver cirrhosis with UGIB is one of the deadliest complications in such patients. The main reasons included portal hypertension associated diseases like esophageal and gastric varices (EGV), portal hypertensive gastropathy (PHG) and other diseases observed in general population like peptic ulcer, acute gastric mucosal lesions (AGML), Mallory–Weiss syndrome and tumor. Among them, EGV is the most common cause for liver cirrhosis with UGIB. EGV accounts for 60–65% of cirrhotic patients with bleeding, a 6-week mortality can reach 20%[4]. The prognosis is closely correlated with the severity of liver diseases, and the effective and rapid treatment of acute UGIB can obviously reduce the mortality.

Although UGIB in cirrhotic patients with EGV is mostly caused by EGV, UGIB from peptic ulcer can also be detected in a relatively small proportion. Bleeding is one of the common and severe complications of peptic ulcer, and liver dysfunction and coagulation disorders will further increase the risk for UGIB in peptic ulcer patients. Previous studies have examined the prognostic effect of chronic liver diseases on the mortality of peptic ulcer with UGIB[5], which reported that the 90-day mortality risk after admission in patients with chronic liver diseases, especially liver cirrhosis, was greatly higher than that in those without chronic liver diseases. The incidence and prevalence of peptic ulcer in patients with liver cirrhosis was increased[6,7], but the possible pathogenesis has not been fully understood[8,9]. Siringoet al.[6] prospectively evaluated the epidemiological and clinical features of 324 cirrhotic patients with peptic ulcer, and found that the prevalence and annual incidence of peptic ulcer was 11.7%

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Lets recall the topic that we have just studied

CASE SCENERIO

A 35 years old lady presents to ER. She has multiple episodes of vomiting fresh blood effortlessly in last 6 hours. She was diagnosed as a case of Rheumatoid Arthritis about one month back and was prescribed steroids(Deltacortil) and analgesics (NSAIDS) which she is taking on regular basis.

Questions:

- What is the cause of bleed?
- How will you do risk stratification?
- how will you investigate?
- What is the medical management?
- What is the endoscopic management (name only)

CASE SCENERIO

A 50 years House wife brought to Medical Emergency Dept. with two days history of passage of black colored stools. She told that she got interferon therapy for HCV infection 15 years back. She never followed up with her doctor after that.

Examination revealed, pulse:130/min, BP110/70 mm Hg with postural drop and splenomegaly.

1. What is the likely cause of bleed in this patient?
2. What is the endoscopic management?

MCQ

A 50 years old patient came to you with 3 episodes of coffee ground vomiting. He is taking aspirin 300mg for his generalized body aches and pains. After initial resuscitation , the Gastro Resident has done his Endoscopy which showed an ulcer in deudenal blub. Being an internee in ER dept, you are also seeing this patient how will you do risk stratification of this patient?

1. Apply child pugh score.
2. Apply Meld score.
3. Apply rockall score.
4. Apply BISAP score.

MCQ

You are attending Gastro ward where your consultant sends you to accompany a patient who had an episode of hematemesis1 day back to Endoscopy Dept. The patient was taking NSAIDS for his body aches and pains. The resident on call does the endoscopy and found an oozing ulcer in antrum .In your opinion what should he do to secure hemostatis?

1. Adrenaline injection.
2. Band ligation.
3. Adrenaline plus gold probe coagulation.
4. Refer for surgery urgently.


Thank You