Gastrointestinal Bleeding

Objectives :

- ★ List the causes of Upper GI bleeding (UGIB).
- ★ Explain the pathophysiology of shock from upper gastrointestinal bleeding.
- ★ Identify the symptoms for patients presenting with GI bleeding.
- ★ Discuss the risk stratification and initial assessment for patient with UGIB.
- ★ Illustrate important physical signs in patients presenting with UGIB
- ★ Outline the investigations required.
- ★ Plan the management of patients with UGIB
- ★ Recognize the clinical manifestations of upper gastrointestinal bleeding.
- ★ Understand the principles of managing patients with upper gastrointestinal bleeding.
- ★ Understand the principles of pharmacological therapy of patients with upper gastrointestinal bleeding.
- ★ Recognize the differences between variceal and non-variceal hemorrhage.

Editing file

Color index

Original text
Females slides
Males slides
Doctor's notes [438]
Doctor's notes [439]
Text book
Important
Golden notes
Extra

Outline

- **Acute upper GI bleeding (hematemesis, melena ..etc)**

What the whole lecture is about

- Risk factors
- Pathophysiology
- Causes of upper GI bleeding (variceal or non variceal)
- Clinical features (type of bleeding, signs of volume depletion, signs and symptoms of anemia)
- Management :
  - Management of non-variceal hemorrhage:
    - Pre-endoscopic : initial resuscitation, transfusion requirements, risk stratification (scales), pre-endoscopic therapy
    - Endoscopic management: endoscopic findings & hemostasis
    - Pharmacological therapy : hospitalization, admission to monitored settings, treatment of ulcers (H.pylori, NSAIDs or idiopathic ulcers)
  - Management of Variceal hemorrhage :
    - Patients with moderate/large ulcers that have NOT bled
    - Patients with acute esophageal variceal hemorrhage

- **Acute lower GI bleeding**

NOT an objective

- Massive bleeding from the lower gastrointestinal tract.

Introduction

◀ Anatomical landmarks and location of GI bleeding:

Upper GI Bleeding

A source of bleeding above the ligament of Treitz (suspensory muscle of duodenum).

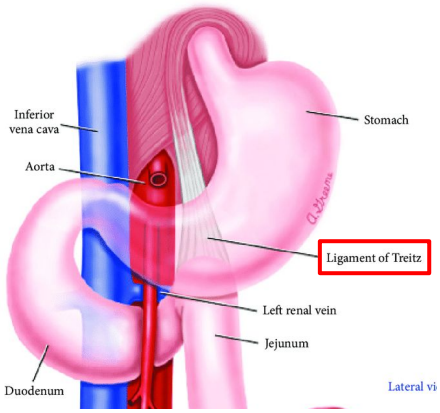
➔ **Including:** Esophagus, stomach and duodenum

Lower GI Bleeding

A source of bleeding Bleeding below the ligament of Treitz.

➔ **Including**: Small & large bowel and the rectum

From 437 medicine team



Acute upper gastrointestinal bleeding:

- This is the most common gastrointestinal emergency, with approximately 10% mortality rate
- The cardinal features are[1]:
  - **Haematemesis**
  - **Melaena**
- Could present with unaltered blood can appear per rectum (hematochezia) , but the bleeding must be **massive** and is almost always accompanied by **shock**.

lower gastrointestinal bleeding:

EXTRA

- **Acute:** Massive bleeding from the lower gastrointestinal tract is **rare** and presents with profuse **red or maroon diarrhoea** and with shock. usually from
  - diverticular disease
  - ischaemic colitis.
- **subacute or chronic:** small bleeds. commonly caused by:
  - **Haemorrhoids.** [2]
  - **Anal fissures**.[3]

Chronic gastrointestinal bleeding:[4]

- Patients usually present with **iron-deficiency anaemia**
- can occur with any lesion of the GI tract that produces acute bleeding
- The primary concern is to exclude cancer. particularly of the stomach or right colon and coeliac disease

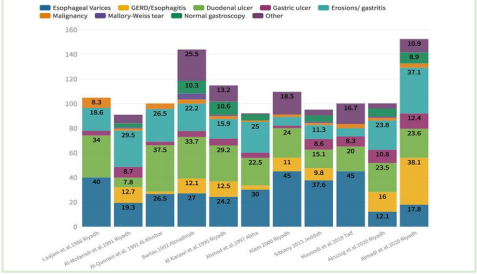
1- symptoms are explained in details in coming slides.
2- Haemorrhoidal bleeding is bright red occur during or after defecation. (On toilet paper)
3-present with fresh rectal bleeding and **anal pain** occur during defecation.
4-hookworm is the most common worldwide cause of chronic GI blood loss.

GI Bleeding

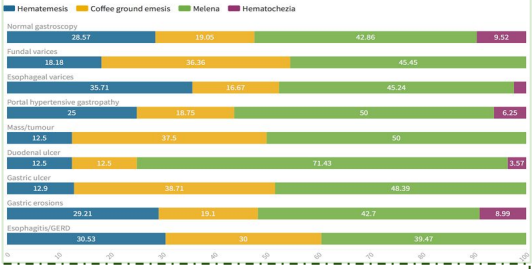
Time trends comparing variceal and non-variceal source of upper gastrointestinal bleeding

A study conducted at alshumaisi hospital, during 13 years , the vast majority of GI bleeding were due to non variceal causes. Non-vericeal is more than vericeal.

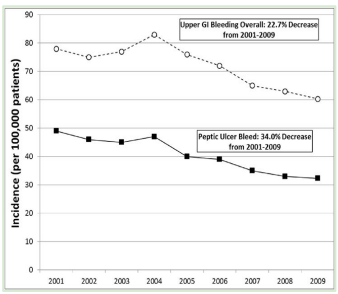
Proportion of Causes of Upper Gastrointestinal Bleeding Based on Different Studies in KSA



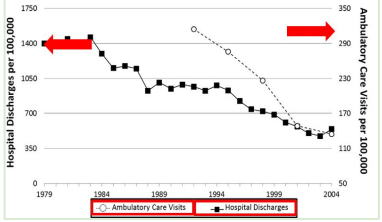
Causes of Upper Gastrointestinal Bleeding in a Tertiary Care Center in Riyadh

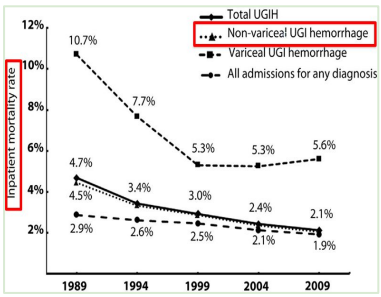


this study shows the different presentations of pt presenting with the same disease. Having hematochezia is not always due to lower GI bleed , symptoms can vary.



PUD with all listed diagnoses in the USA 1979 to 2004





Changes in incidence rates in different regions of the world

Location	Decrease in Incidence in UGIB
United States	108 per 100,000 in 1994 to 78 per 100,000 in 2009
Spain	54.6 per 100,000 in 1996 to 25.8 per 100,000 in 2005
Veneto region (Italy)	64.4 per 100,000 in 2001 to 35.9 per 100,000 in 2010
Netherlands	61.7 per 100,000 in 1993 per 1994 to 47.7 per 100,000 in 2000
New Zealand	53.6 per 100,000 in 2001–2005 to 45.8 per 100,000 in 2006–2010
Sweden	63.9 per 100,000 in 1987 to 35.3 per 100,000 in 2005

PUD is decreasing globally , could be due to better treatment of heartburn and PUD with PPI and h.pylori eradication also, the use of NSAIDs is not encouraged and usually given with a medications to prevent PUD.

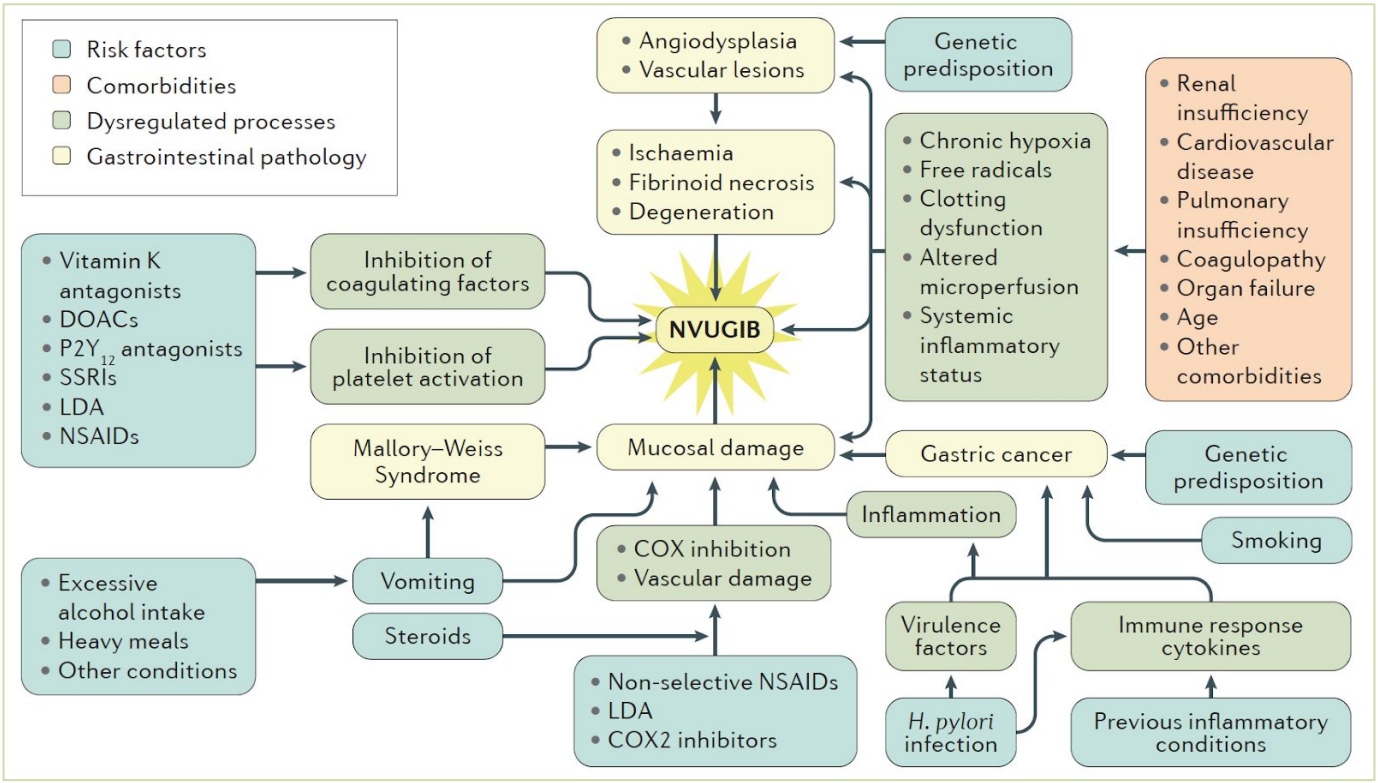
Risk Factors

Females slides

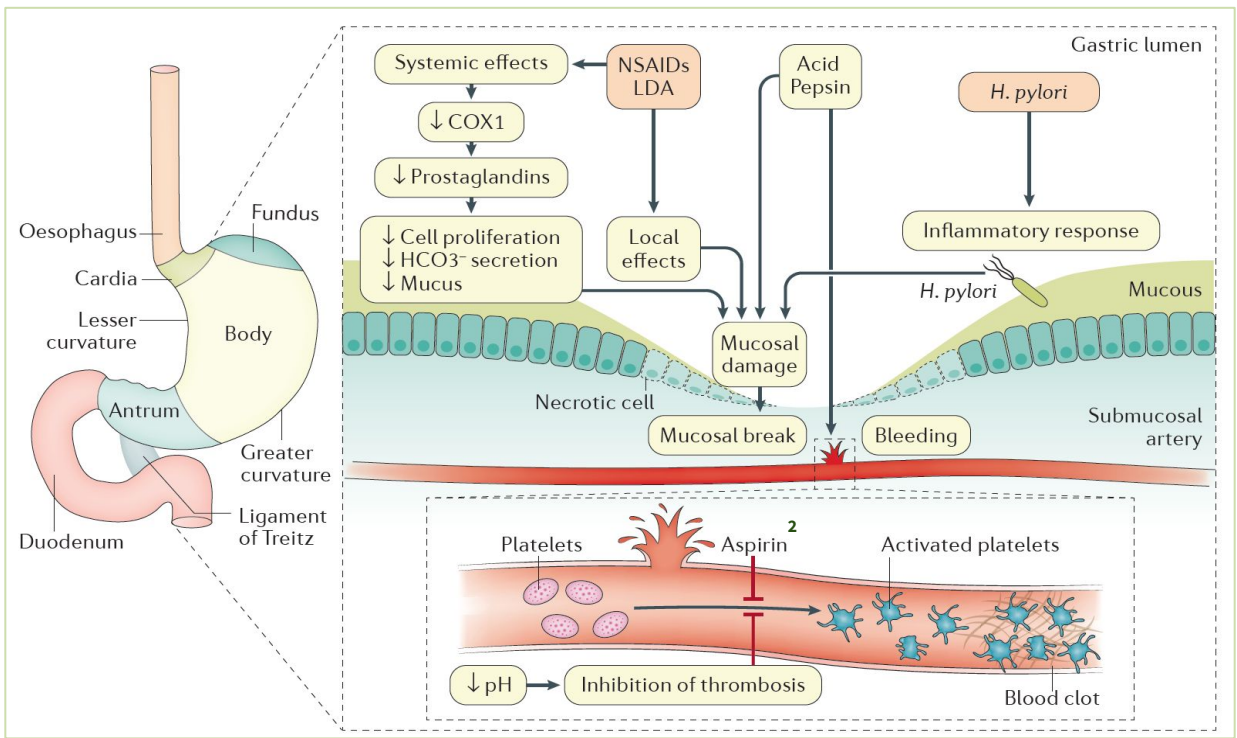
- Age > 65
- **Previous peptic ulcer** or ulcer-related upper GI complication
- **Multiple or High-dose NSAIDs**
- Selection of NSAID (eg. COX1 vs COX2 inhibitors)
- NSAID related dyspepsia
- **Aspirin** (including cardioprotective dosage)
- **H.pylori infection**
- Cigarette smoking and Alcohol consumption
- Chronic debilitating disorders (eg. Cardiovascular disease, rheumatoid arthritis), liver cirrhosis and renal disease, respiratory disease (COPD)

- Concomitant use of:
  - NSAID & low dose aspirin can produce ulcers and erosions
  - Oral bisphosphonates (eg. Alendronate)
  - Corticosteroids in the usual therapeutic doses have no influence on gastrointestinal haemorrhage. However, the combination of glucocorticoids and NSAIDs results in a synergistic increase in the incidence of gastrointestinal haemorrhage.
  - Anticoagulant or coagulopathy
  - Antiplatelets (eg. Clopidogrel)
  - Anticoagulants and antiplatelet agents do not cause acute gastrointestinal haemorrhage per se, but bleeding from any cause is greater if the patient is anticoagulated.
  - SSRI

◀ Complex pathophysiology of NVUGIB [1] :



◀ Mechanisms of upper GI bleeding induced by NSAIDs, Low dose Aspirin or H.pylori :



1- Non variceal upper gastrointestinal bleeding.

2- Effects of aspirin and other NSAIDs: causes ulcer and prevents an ulcer from healing (by preventing platelet aggregation)

Upper GI Bleeding

★ UGIB Incidence; 57-78 cases per 100,000 population

◄ Etiology:

01 Peptic ulcer disease (duodenal and gastric ulcers)

The **most common** cause of **upper GI bleeding** (36-50%). Aspirin is a risk factor for peptic ulcer.

02 Variceal bleeding

2nd most common cause. Caused by liver cirrhosis (secondary to portal hypertension) and chronic liver disease. common in egyptians due to the high prevalence of hepatitis C and Schistosomiasis.

03 Dieulafoy's lesion

Rare, the vessel below the surface of the mucosa opens up and bleeds then disappears (no abnormality found in endoscopy) can be picked up when the pt is actively bleeding

04 Mallory weiss tear

A linear mucosal tear occurs at the oesophagogastric junction and produced by a sudden increase in intra-abdominal pressure. It often occurs after a bout of coughing or retching and is classically seen after **alcoholic** 'dry heaves'. **Recurrent/frequent vomiting** which can be due to medications especially chemotherapy, or pregnancy and classically after alcohol intake. They can have hematemesis or coffee ground emesis (the patient doesn't see blood in the first episodes of vomiting).

05 Malignancy

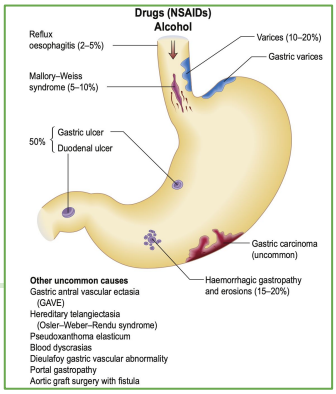
The patient comes with weight loss and loss of appetite.

06 Mucosal Erosive disease (Esophagitis, Gastritis, Duodenitis)

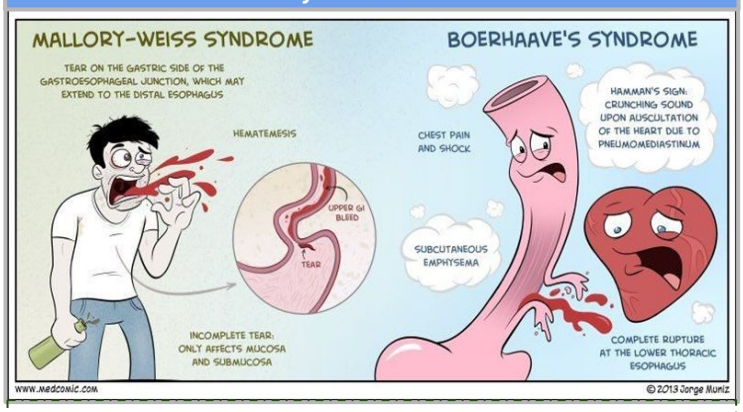
07 Arteriovenous malformation

08 Gastric antral vascular ectasia (GAVE)

Also known as "Watermelon Stomach", is a condition in which the blood vessels in the lining of the stomach become fragile and become prone to rupture and bleeding.



Only in males' slides



When the tear goes to the full thickness causing complete muscle tear (esophageal rupture) and not only limited to the surface then it is called **boerhaave's syndrome** and people die from it. The pt will have mediastinal bleeding and mediastinitis which has a high fatality rate.

Only in males' slides

Table 1. Estimated Hemorrhage-Related Deaths per Year and Years of Life Lost in the United States and Worldwide, According to the Cause of Hemorrhage.

Cause of Hemorrhage	Deaths from Hemorrhage*	U.S. Cases of Hemorrhage		Global Cases of Hemorrhage	
		No. of Deaths per Yr	Yr of Life Lost	No. of Deaths per Yr	Yr of Life Lost
	percent				
Abdominal aortic aneurysm	100	9,988†	65,273‡	191,700§	2,881,760¶
Maternal disorder	23§	138§	7,572**	69,690§	4,298,240**
Peptic ulcer disease	60††	1,860§	38,597**	141,000§	3,903,600**
Trauma	30‡‡	49,440§	1,951,786**	1,481,700§	74,368,000**
Total		61,426	2,043,228	1,884,090	85,651,600

it shows the risk of PUD, 4 millions years were lost due to PUD only

Clinical Features

1. Types of bleeding:

1

Hematemesis:

- Vomiting fresh, red blood. **suggests upper GI bleeding (stomach, esophagus or duodenum).** Occur when bleeding is **rapid** and **profuse** (moderate to sever)

2

"Coffee grounds" emesis :

- Suggests upper GI bleeding. occur when bleeding is less severe and at lower rate.vomitus has enough time to be oxidised and transformed into coffee grounds

3

Melena: [1]

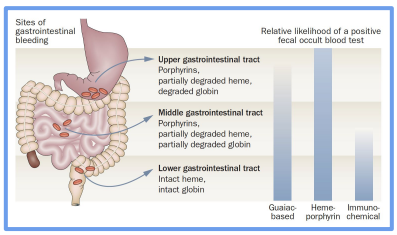
- (This dark discoloration of the stool is due to hematin, a dark pigment that forms when heme is oxidized by gastric acid in the upper GI tract)
- **black, tarry, foul-smelling stool containing altered blood**
- The characteristic colour and smell result by the action of digestive enzymes and bacteria on haemoglobin
- indicates that blood has remained in GI tract for several hours.
- **Melena suggests upper GI bleeding 90% of the time (stomach, esophagus or duodenum).** Or middle GI bleeding (small bowel). Or lower GI bleeding (the left side : cecum or ascending colon).
- Note that dark stools can also result from bismuth[2], iron[3], spinach, charcoal, and licorice.

4

Hematochezia:

- **bright red blood per rectum** (with or without stool) In LGIB. Oxidation of heme occurs with the help of intestinal bacteria. The blood is bright red because the degree of bacterial degradation in the final portion of the intestine is limited.
- usually represents a **lower GI source** (typically left colon or rectum).

  Colon: Marron, Jelly-like trace of blood in the stool | **Rectum**: Streak of fresh blood in the stool.
  - Consider diverticulosis, AV malformations, hemorrhoids and colon cancers.
- It may result from **massive upper GI bleeding** (5-10% of hematochezia) that is **very heavy and quick** (so that blood does not remain in colon to turn into melena).
  - patient often has some degree of hemodynamic instability.



5

Occult blood in stool: Guaiac +ve stool

- **Invisible blood** or its breakdown products in stool.
- presents with iron deficiency anaemia.
- Source of bleeding may be anywhere along GI tract.
  - the most important cause is colorectal cancer.

- Melena and hematochezia can be caused by either UGIB or LGIB
- Melena: UGIB: Due to oxidation process | LGIB: When blood moves slowly it will get more time to oxidate
- Hematochezia: UGIB: When the blood moves fast | LGIB: Because there is no time to oxidize the blood

Only in males' slides

Sources of GI Bleeding

	Esophagus	Stomach	Duodenum	Small Intestine[a]	Right Colon	Left Colon
Hematemesis	X	X	X	—	—	—
Coffee-ground emesis	X	X	X	—	—	—
Melena	X	X	X	X	X	—
Guaiac-positive stool[4]	X	X	X	X	X	X
BRBPR	(If severe)	(If severe)	(If severe)	(If severe)	X	X

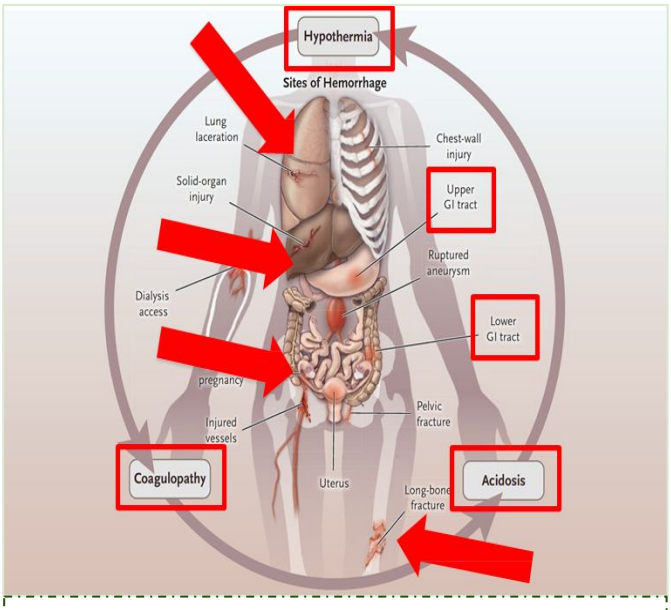
Bright Red Blood Per Rectum aka **hematochezia**

1- soft stool not hard
2- used to treat heartburn but we don't use it here. causes black discoloration of the stool
3- how to know if it is melena or due to iron supplements ? if the patient is taking iron the stool won't be loose and it will not have foul smell. So not every black stool is melena!!
4- Guaiac-positive stool: old method to detect blood on stool, used if the patient complains of abdominal pain and you suspect bleeding . However, it has been replaced by a a higher sensitivity method called **Fecal Immunochemical Testing (FIT).**

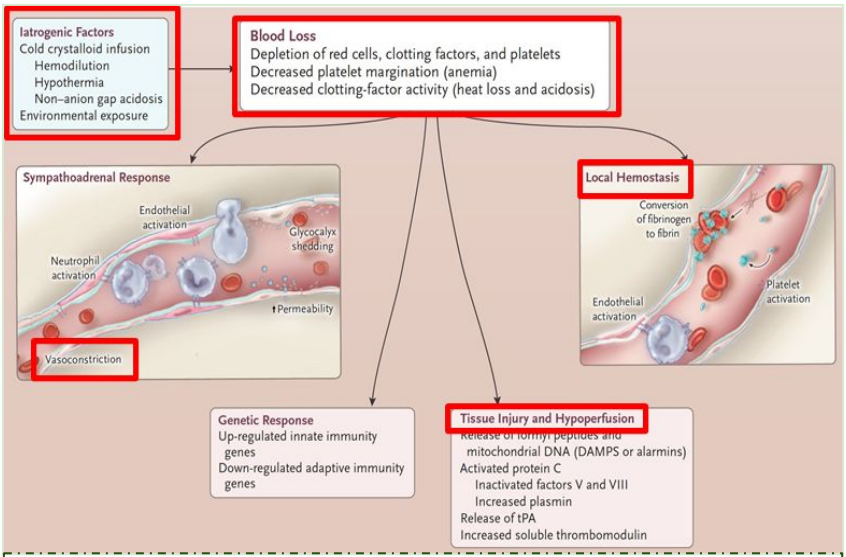
2. Signs of volume depletion

➢ Pathophysiology of shock :

Following a bleed from the upper gastrointestinal tract, unaltered blood can appear per rectum, but the bleeding must be massive and is almost always **accompanied by shock**. The passage of dark blood and clots without shock is always due to lower gastrointestinal bleeding.



- all the causes in red can manifest as shock which can cause hypothermia ,coagulopathy and acidosis. It is a vicious cycle.
- Same as trauma : GI bleeding → blood loss → the body tries to compensate → vasoconstriction → ↓blood flow → hypothermia, coagulopathy and acidosis.



- Managing GI bleeding with IV fluids can make it physiologically worse.
- In this figure, we can see that the blood is diluted due to the loss of it's components that accompanies bleeding.
- when the pt is bleeding and you give them IV fluids you will dilute the blood products even more.
- The fluids can be cold which can decrease body temperature. Sometimes we warm up the fluids before introducing them to the pt.

➢ clinical features based on the amount of blood loss:

Blood loss (mL)	<750	750–1500	1500–2000	>2000
Blood loss (%) ↑	<15	15–30	30–40	>40
Pulse rate ↑	<100	>100	>120	>140
Blood pressure ↓	Normal	Normal	Decreased	Decreased
Pulse pressure ↑↓	Normal or increased	Decreased	Decreased	Decreased
Respiratory rate ↑	14–20	20–30	30–40	>35
Urine output (mL) ↓	>30	20–30	5–15	Negligible
Mental status ↓	Slightly anxious	Mildly anxious	Anxious and confused	Confused and lethargic
Fluid replacement	Crystalloid	Crystalloid	Crystalloid and blood	Crystalloid and blood

Normally we have 4.5-5 L of blood .

In the beginning of bleeding there is (small amounts of blood are lost):
- No change in pH (no acidosis) thus,
- no change in pulse rate or respiratory rate

However, ↑bleeding → acidosis → ↑RR → ↑CO_2 → Respiratory alkalosis (Patients hyperventilate trying to compensate).

Also, ↑bleeding → ↓ urine output because the kidneys are trying to preserve volume.

Table 2. Classification of Hemorrhagic Shock.*

Shock Class	Blood Loss† ml (%)	Heart Rate beats/min	Blood Pressure	Pulse Pressure	Respiratory Rate breaths/min	Mental Status
I	<750 (15)	<100	Normal	Normal	14–20	Slightly anxious
II	750–1500 (15–30)	100–120	Normal	Narrowed	20–30	Mildly anxious
III	1500–2000 (30–40)	120–140	Decreased	Narrowed	30–40	Anxious, confused
IV	>2000 (>40)	>140	Decreased	Narrowed	>35	Confused, lethargic

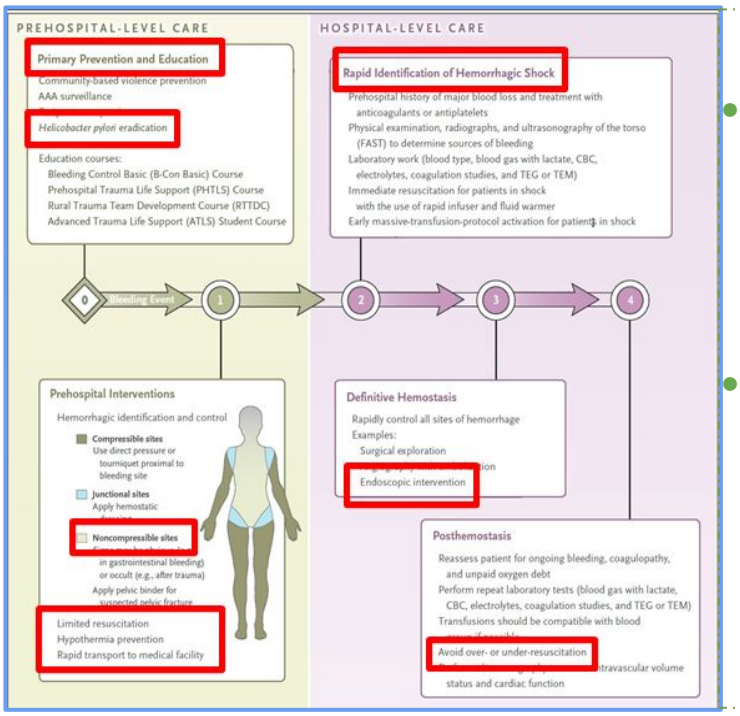
* Data are from the American College of Surgeons Committee on Trauma.[42]
† Blood-loss volume and percentage of total blood volume are for a male patient with a body weight of 70 kg.

3. Signs and Symptoms of anemia

- (e.g., fatigue, pallor, exertional dyspnea)

Essentials of diagnosis	Females slides
Symptoms	Coffee ground vomiting, hematemesis, melena, hematochezia, anemic symptoms
PMH[1]	Liver cirrhosis, use of NSAIDs
Signs[2,3]	Hypotension, tachycardia, pallor, altered mental status, melena or blood per rectum, decreased urine output
Bloods	Anemia, raised urea, high urea or creatinine ratio
Endoscopy	Ulcers, varices, Mallory-Weiss tera, erosive disease, neoplasms, vascular ectasia, and vascular malformations

Management



- **Pre hospital care :** mainly primary prevention. the red box on the left lower corner is what you want to achieve before reaching the hospital
- **Hospital level :** Avoid over or under resuscitation (Nobody knows what is the cutoff of each), this is important to avoid permissive hypotension, it means that our aim is not to return the pt to 100% normal, we try to achieve 70-80% because we don't want to give too much fluids causing the disruption we have discussed before .

1- Risk Factors for GI bleeding
2- All these signs are important in determining the degree of GI bleeding.
3- First early signs of active bleeding is tachycardia >>> postural hypotension >>> hypotension >>> tachypnoea >>> altered mental status (in advance stage when there is more than 25% loss of intravascular volume).

Management

1 Pre endoscopic managem

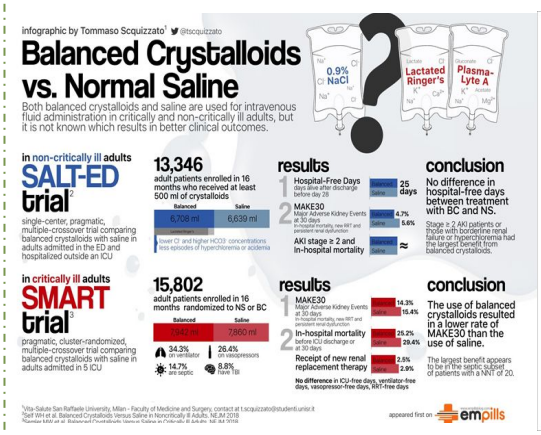
◄ Initial Resuscitation:

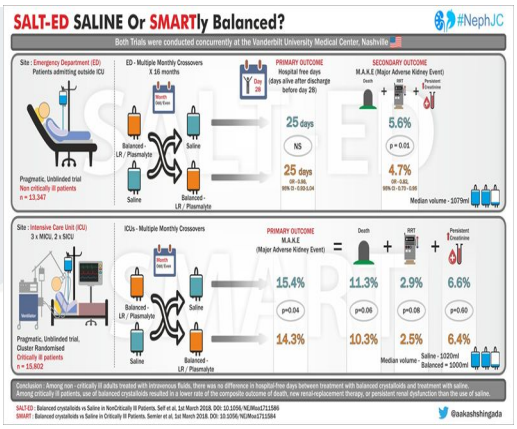
- **ABCs:** Maintain airway, breathing and circulation
- Ensure two large-bore peripheral I.V access and consider monitored setting
- **Resuscitate initially with crystalloid solutions [1] & permissive hypotension**
- Send blood work including CBC, coagulation studies and type and cross- matching
- **Full blood count.** Chronic or subacute bleeding leads to anaemia. Thrombocytopenia may be a clue to the presence of hypersplenism in chronic liver disease.
- **Hemoglobin/hematocrit level** (may not be decreased in acute bleeds): A hemoglobin level >7 to 8 g/dL is generally acceptable in young, healthy patients without active bleeding. However, most elderly patients (especially those with cardiac disease) should have a hemoglobin level >10 g/dL.
- A **low MCV** is suggestive of iron deficiency anemia (chronic blood loss). Patients with acute bleeding have normocytic red blood cells.
- **Urea and electrolytes.** may show evidence of renal failure. The blood urea rises in upper GI bleeding because the blood will be digested to protein then absorbed from the small intestine and converted to urea in the liver ; an elevated blood urea with normal creatinine concentration implies severe bleeding.
- **Liver function tests.** may show evidence of chronic liver disease.
- **Coagulation profile** (platelet count, PT: Check with clinical suggestion of liver disease or in anticoagulated patients, PTT, INR).
- **Cross-matching.** At least 2 (Davidson's) units of blood should be cross-matched if a significant bleed is suspected. cross-match at least 4 (Kumar) units of blood if there is evidence of a large bleed (BP <100 mmHg, pulse >100 beats/min, cool or cold extremities with slow capillary refill, Hb <100 g/L).

Comparison of flow rates through IV catheters

Type and Diameter of Venous Catheter	Maximum Flow Rate
20-gauge	60 mL/min
18-gauge	105 mL/min
16-gauge	220 mL/min
Triple lumen catheter	
Medial (blue)/proximal (white) lumen (18-gauge)	26 mL/min
Distal (brown) lumen (16-gauge)	52 mL/min
Cordis: 8.5 French (100 mm)	126 mL/min 333 mL/min under pressure[a]
Intraosseous line	80 mL/min 150 mL/min under pressure[a]

- The larger the number, the smaller the catheter.
- 16 gauge is the largest, you can give 1L in 5 minutes, and if we use it in both side it will give 1L in 2.5 minutes.
- Triple lumen is very small, it is a long catheter with 3 openings.
- **Intraosseous line :** used usually in children and can be used in trauma.





you can use either crystalloids or normal saline . the outcomes with crystalloids are better . two studies were done, smart trial for critical and severe cases which revealed better outcomes with crytalloids, salted trial in less severe cases showed no difference between the two interventions.

1-Resuscitate the pt with **IV fluids, IV fluids, IV fluids, IV fluids, IV fluids, IV fluids→ then blood**. **(SUPER IMPORTANT!)**
It takes time to wait for blood bank so you save the pt with **IV fluids** . If you don't have time and you need to give the pt blood, you can use unmatched blood "O-" , but if you have time you match them because they still could have adverse reactions..

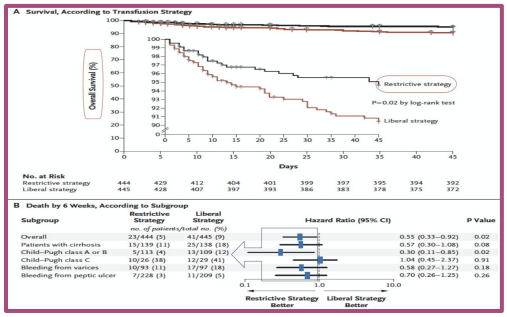
Management

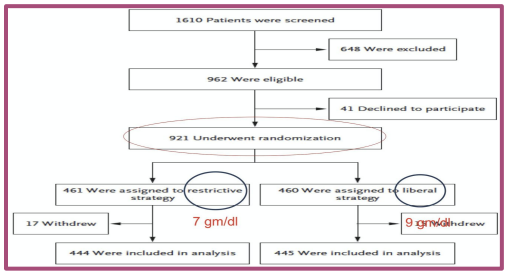
1 pre endoscopic management *cont'*

◄ Transfusion Requirements:

- The role of transfusion in clinically stable patients with mild GI bleeding remains controversial, with uncertainty at which hemoglobin level transfusion should be initiated. Literature suggesting poor outcomes in patients managed with a liberal transfusion.
- The restrictive RBC transfusion had significantly improved survival and reduced rebleeding.
- **Packed red blood cells:** If the hemoglobin level < 7 g/dL or If hemoglobin < 10 g/dL in patients with preexisting cardiovascular disease or patients with symptoms.
- Transfuse platelets only if platelet count < 50x109/L or <100x109/L with suspected platelet dysfunction
- **Fresh frozen plasma:** if INR is elevated.

(It contains different types of clotting factors like factor 2,7,9,10 and protein c and s)





◄ Risk stratification[3]:

Low vs. high risk :
Inform the surgeon that this pt has a high risk and s/he might need to be taken to the OR.

Glasgow - blatchford score [2]

Early identification :
Even if you suspect that melena is caused by iron intake, manage as bleeding.

Rockall score [2]

Appropriate intervention

AIMS65 score [2]

Minimizes morbidity and mortality [1]

AIMS56 score	438 slides
Risk Factor	**Score**
Albumin < 3 g\dL	1
INR > 1.5	1
Altered mental status	1
SBP≤ 90 mmHg	1
Age > 65	1

The following factors affect the risk of rebleeding and death:

- Age.
- Evidence of comorbidity, e.g. cardiac failure, ischaemic heart disease, chronic kidney disease and malignant disease.
- Presence of the classical clinical features of shock (pallor, cold peripheries, tachycardia and low blood pressure).
- Endoscopic diagnosis, e.g. Mallory–Weiss tear, peptic ulceration.
- Endoscopic stigmata of recent bleeding, e.g. adherent blood clot, spurting vessel.
- Clinical signs of chronic liver disease.

1- Avoid surgery due to high morbidity and mortality. consult interventional radiology, gastroenterology and admit to ICU to avoid surgery.
2- There's no need to memorize them.
3- Very important in ER as it direct your management.

Management

1 pre endoscopic management *cont'*

		Glasgow - blatchford score [1] (GBS)		
		Risk factor at presentation	**Threshold**	**Score**
3	**Urea**	Blood Urea Nitrogen (mmol\L)	6.5 - 7.9 8 - 9.9 10 - 24.9 ≥ 25	2 3 4 6
	CBC	Hb for men (g\L)	120 - 130 100 - 119 < 100	1 3 6
		Hb for women (g\L)	100 - 120 < 100	1 6
2	**Physical examination**	Systolic BP (mmHg)	100 - 109 90 - 99 < 90	1 2 3
		HR (bpm)	>100	1
1	**History**	Melena	Present	1
		Syncope	Present	2
		Hepatic disease	Present	2
		Cardiac failure	Present	2

Total score (0-23)[2]. **Patient with score >0 are considered to be at high risk and requires admission**

1-It is considered old but the **best** score so far. Scoring systems have been developed to assess the risk of rebleeding or death. The Blatchford score uses the level of plasma urea, haemoglobin and clinical markers but not endoscopic findings to determine the need for intervention such as blood transfusion or endoscopy in GI bleeding.

2- Zero score indicates lower risk patients and we can manage them outpatient and do endoscopy at next day or two days later.

Management

1 pre endoscopic management *cont'*

[Rockall score[1]]

Females' slides

		Variable		Score
Complete Rockall Score	Clinical Rockall score	**Age (History)**	< 60 60 - 79 ≥80	0 1 2
		Shock (Physical exm)	HR >100 Systolic BP < 100	1 2
		Coexisting illness (History)	Ischemic heart disease, congestive HF, other major illness Renal or hepatic failure, metastatic cancer	2 3
	-	**Endoscopic diagnosis**	No lesion observed, Mallory weiss tear Peptic ulcer, erosive disease, esophagitis Cancer of upper GI	0 1 2
		Endoscopic stigmata of recent hemorrhage	Clean base ulcer, flat pigmented spot Blood in upper GI, active bleeding, visible vessel, clot	0 2

◄ **Pre-endoscopic therapy:**

- Provide erythromycin I.V 30 minutes prior to endoscopy
- High-dose I.V PPI should initiated
- The routine use of nasogastric lavage and/or tranexamix acid is not recommended
- **Patients receiving anticoagulants:**
  - correction of coagulopathy is recommended
  - Endoscopy should not be delayed for a high INR unless the INR is supratherapeutic [2]
- **Aspirin, NSAIDs and warfarin are stopped** and the INR reversed if necessary.

2 Endoscopic management

- **Definition of early endoscopy:**
  - Ranges from 2 to 24 hours AFTER INITIAL PRESENTATION [3]
- **May need to be delayed or deferred:**
  - Active acute coronary syndromes
  - Suspected perforation [4]
- **Different modalities for patient with GI bleeding:**
  - Endoscopy: The gold standard for UGIB
  - Colonoscopy: The gold standard for LGIB
  - Arteriography; Best initial test for unstable patient

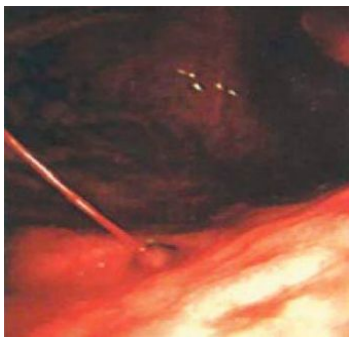
1- Not used anymore, Rockall score is based on clinical and endoscopic findings.
2- Anything **less than 2-2.5 you can do your intervention**.Supratherapeutic: **6-9** . you need to **reduce INR** with either vit K in pt taking warfarin, the antidote for heparin or fresh frozen plasma AKA cryoprecipitate.
3-Non variceal within 24h, **Variceal within 12 hours.**
4- Surgery is a better choice.

Management

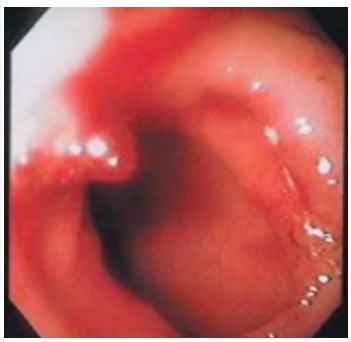
2 Endoscopic management

◄ Endoscopic findings:

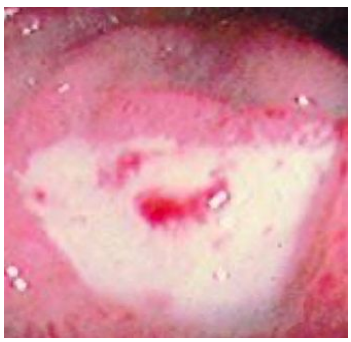
Spurting Blood
Arterial bleeding



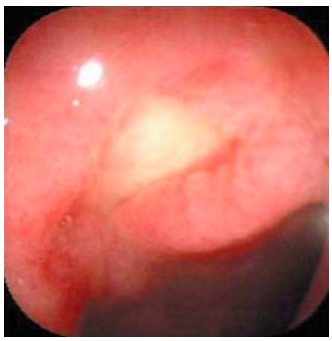
Non-bleeding Visible Vessel



Flat, Pigmented Spot

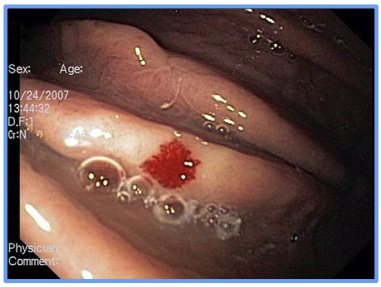


Clean Base[3]

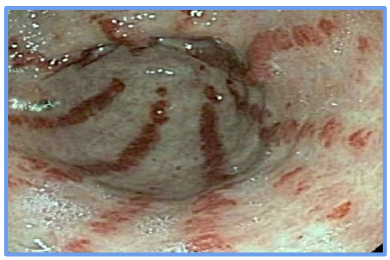


- **High-risks lesions** are those that spurt blood, ooze blood , contain a non bleeding visible vessel , or have an adherent clot . Need for endoscopy treatment
- **Low-risk lesions** are those that have a flat, pigmented spot or a clean base . Managed by PPI

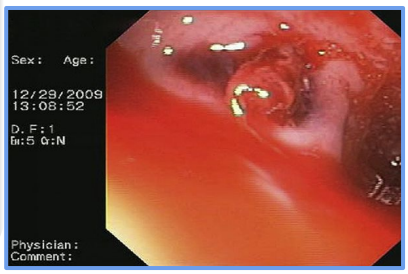
AV malformation [1]

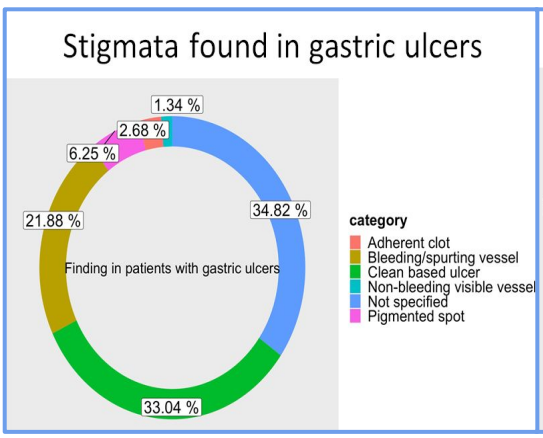


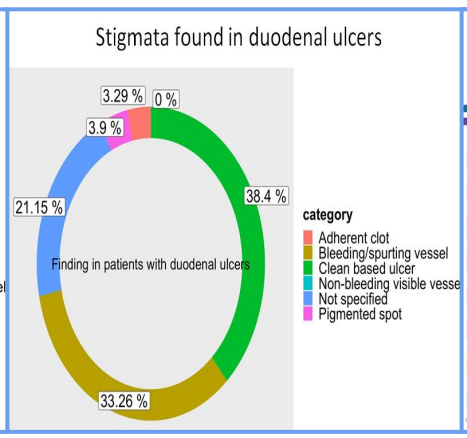
GAVE [2]

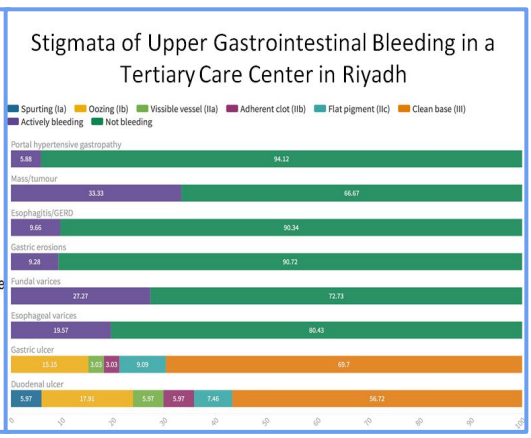


Visible vessel
Bleeding blood vessel







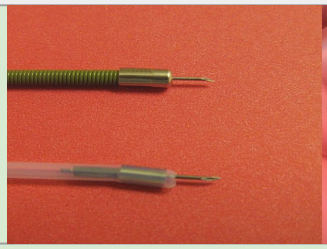


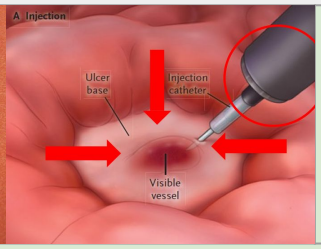
1- looks like a tree, can be seen in liver cirrhosis, pregnancy (high estrogen state) and hereditary hemorrhagic telangiectasia (HHT). Common presentation: a boy with abnormal small spots around mouth (you will see more spots in the GIT using endoscopy).
2- Gastric antral vascular ectasia AKA **watermelon stomach**, the red lines are vascular malformations . Happens usually in elderly who do renal dialysis and sometimes without known reason. Treated by argon plasma laser .
3- Give only PPI. It is one of the indications to look for H.pylori and treat it.

2 Endoscopic management

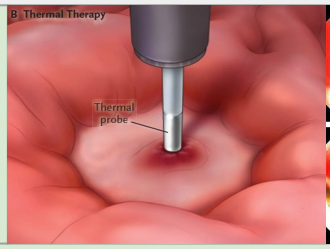
Endoscopic hemostasis

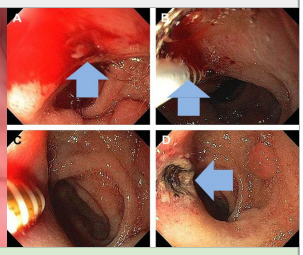
epinephrine injection [1]



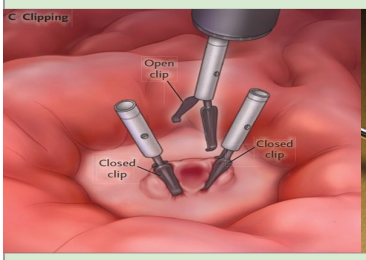


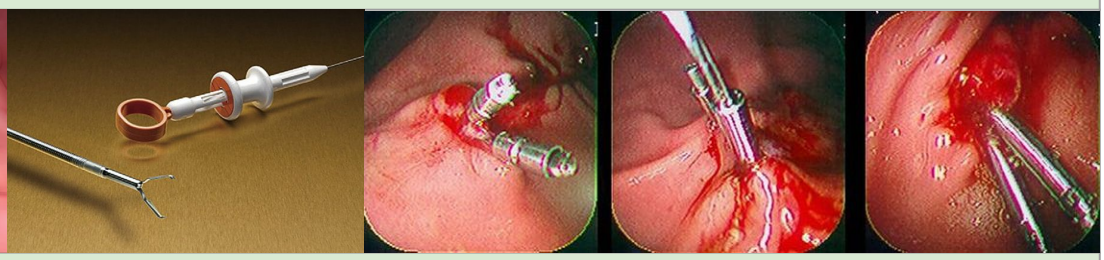
Thermal therapy [2]



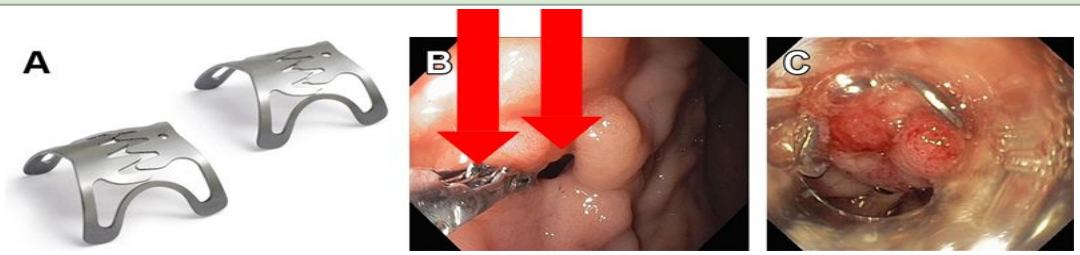


Mechanical clips

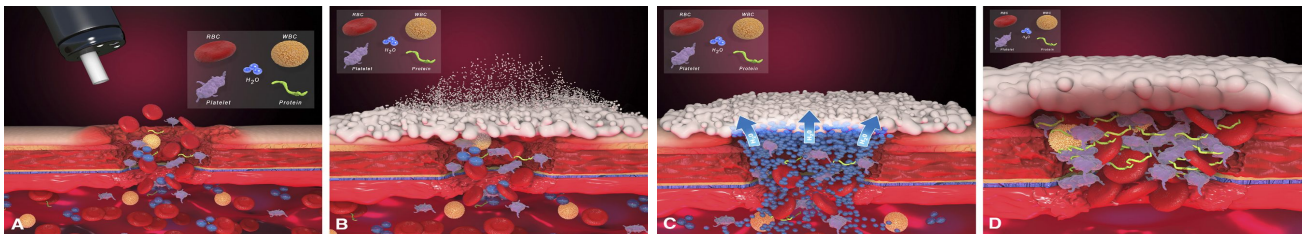




Over the scope clip (OTSC)

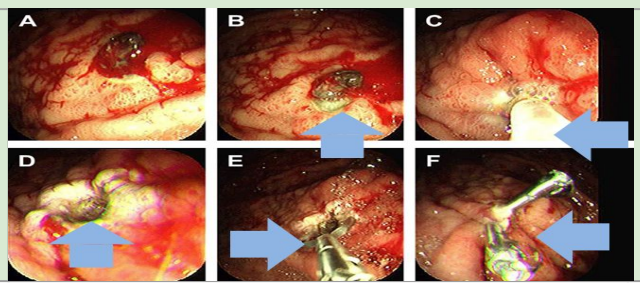


Hemostatic powder spray [4]

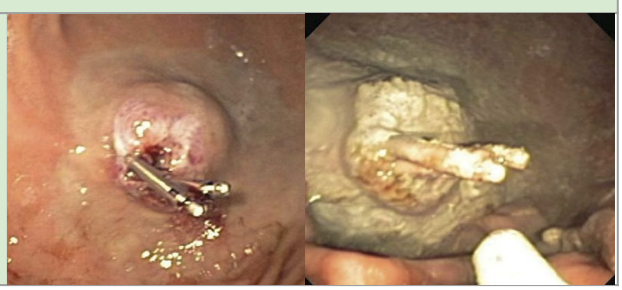


combination of 2 techniques : [3]

thermal therapy and clips



clips and powder spray



1-What do we do when we find a visible vessel ? inject a fluid composed of epinephrine diluted by normal saline to cause vasoconstriction around the vessel using a needle .The whole idea is to create pressure to stop bleeding .

2- We burn the area to stop the bleeding (الكوي).

3-In real life we usually use more than one modality.

4- يستعمل بالجيش لانه سريع, اذا بخيته يسحب الماء ويصير زي العجين

Management

3 pharmacological therapy

◄ Hospitalization :

- It takes 72 hours for most high-risk lesions to become low-risk lesions AFTER endoscopic therapy.
- **Why 3 days ?** 60% -76% of patients who had **rebleeding** within 30 days **AFTER endoscopic hemostasis PLUS high-dose PPI therapy did so within the first 72 hours.**
- In-hospital mortality 1.7−3.7%
- 30-day mortality 6−11% , When you discharge the patient that doesn't mean there is no risk anymore, there is still a high risk of mortality

Estimated risk for post endoscopic bleeding risk

Endoscopic Procedure	Low-risk Bleeding (<1.5%)	High-risk Bleeding (>1.5%)
Diagnostic EGD or colonoscopy (with or without biopsy)	X	—
Nonthermal removal of small polyps (<1 cm)	X	—
Coagulation or ablation of tumors or vascular lesions (includes APC, bipolar cautery, and laser ablation)	—	X
Large (>1 cm) polypectomy	—	X
Variceal band ligation	—	X
Hemostatic clip placement	X (unknown risk)	—
Injection therapy	X (unknown risk)	—
Bipolar cautery	—	X

438 slides

◄ Admission to a monitored setting: (ICU)

- **For at least the first 24 hours on the basis of risk or clinical condition**

Hemodynamic instability [1] | **Increasing age** | **Severe comorbidity** | **Active bleeding at endoscopy** | **Large ulcer size (>2 cm)**

EDITORIAL | Annals of Internal Medicine

Aspirin Withdrawal in Acute Peptic Ulcer Bleeding: Are We Harming Patients?

Most of the mortality cases before was caused by stroke, because they were stopping antiplatelets . The patient leaves the hospital "stopped aspirin after GI bleeding" → gets thrombosis → dies.

The pt has taken the aspirin for a reason, so its withdrawal will increase the risk that he had before within 3 days.

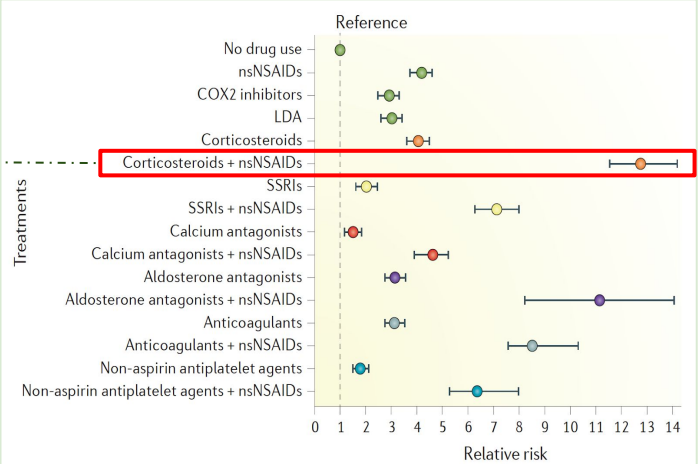
So, Do NOT stop the antiplatelet !!!

Periprocedual management of NOACs

Creatinine Clearance (mL/min)	Half-life (h)	Moderate Procedural Bleeding Risk; Discontinue Drug for 2–3 Half-lives (d)	High Procedural Bleeding Risk; Discontinue the Drug for 4–5 Half-lives (d)
>80	13 (11–22)	1–1.5	2–3
>50 to ≤80	15 (12–34)	1–2	2–3
>30 to ≤50	18 (13–23)	1.5–2	3–4
≤30	27 (22–35)	2–3	4–6

438 slides

They have increased risk so add PPI (proton pump inhibitor). Don't wait for bleeding !



1- If the patient is unstable "in shock"→ ICU

Management

3 pharmacological therapy

[Initial treatment of ulcer bleeding, according to the endoscopic feature of the ulcer]

Males slides

Endoscopic Feature	Active bleeding or visible vessels	Adherent clot	Flat pigment spot	Clean base
Endoscopic Therapy	Endoscopic therapy	May consider endoscopic therapy	No endoscopic therapy	No endoscopic therapy
Medical Therapy	Intensive PPI therapy	Intensive PPI therapy	Once daily PPI therapy	Once daily PPI therapy
Diet	Clear liquids for ~2 days	Clear liquids for~2 days	Clear liquids for ~1 day	Regular diet
Hospital Stay	3 days	3 days	1-2 days	Discharge after endoscopy

★ Intensive proton-pump inhibitor (PPI) therapy is an intravenous bolus (80 mg) followed by an infusion (8 mg per hour) for 72 hours or an oral or intravenous bolus (e.g, 80 mg) followed by intermittent high-dose PPI therapy (e.g. 40 to 80 mg twice daily) for 3 days."

★ The diets shown are diets after endoscopy in patients who do not have nausea or vomiting.

★ The duration of hospital stay after endoscopy is shown in patients who are in stable condition and do not have further bleeding or concurrent medical conditions requiring hospitalization.

H.pylori ulcer

- Patients with bleeding peptic ulcers should be tested for H. pylori
  - Receive eradication therapy if present
  - Confirmation of eradication **urea breath test or faecal antigen testing.**
- Negative H. pylori diagnostic tests obtained in the acute setting should be repeated[2].
- No need for continuing PPI therapy after eradication of the H.pylori

NSAID- induced ulcer

- No need for continuing PPI therapy after discontinuation of NSAID.
- If NSAID required, consider COX-2 inhibitor with PPI therapy.
- Use PPI with low dose Aspirin if needed for secondary prevention [1]

Idiopathic ulcers

- PPI therapy should be prescribed indefinitely

1-usually we will tell them to stop for less than 3 days, return it ASAP. you might **stop NSAIDs** or **anticoagulants** and they will die from MI or stroke. stop NSAIDs and anticoagulants if they are not highly indicated . if you can't then continue with PPI.

2- due to active bleeding

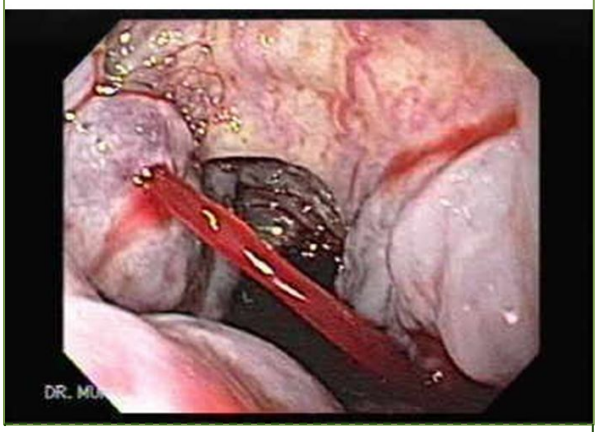
Management of variceal hemorrhage

Definition

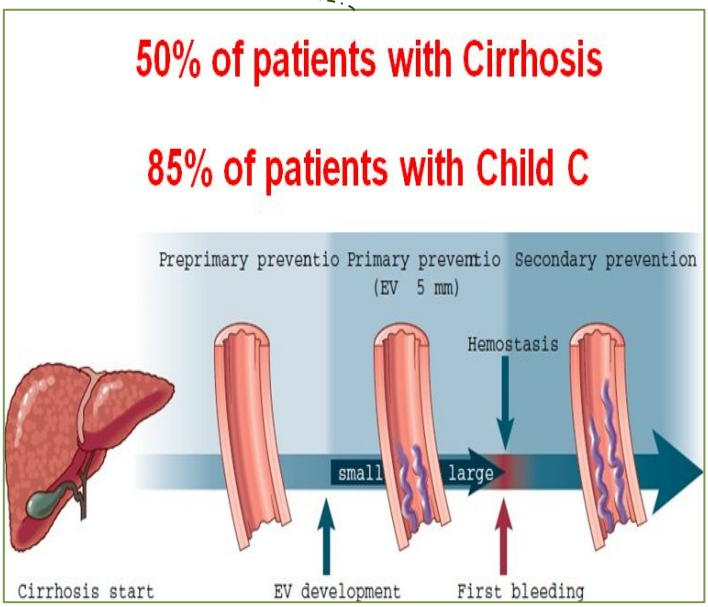
Acute upper GI hemorrhage from gastro-esophageal varices is usually associated with portal HTN, that is caused by pre-hepatic causes (portal vein thrombosis) hepatic causes (e.g cirrhosis) or post-hepatic (Rt sided HF).

- **Primary prevention :** for patients with hepatitis B, C or fatty liver, treat the cause (**before bleeding**)
- **Secondary prevention : bleeding occurred,** so we treat it by banding .

Variceal Hemorrhage



Venous bleeding with a high flow rate



◄ Patients with Moderate / Large Varices that have NOT Bled:

Therapy	Dose	Therapy goals	Maintenance/follow-up evaluation
Propranolol [1]	- 20 mg orally twice a days. Adjust every 2-3 days until the goal is achieved. - Maximum daily dose is 320mg	**Aiming for resting HR of 50-55 beats\minutes**	- Every outpatient visit make sure patient on therapy. - Continue indefinitely - No need for follow-up EGD
Nadolol	- 40 mg orally once a days. Adjust every 2-3 days until the goal is achieved. - Maximum daily dose is 160 mg		
Carvedilol	- Start with 6.25mg once a day. After 3 days increase to 12.5 mg. - maximum dose is 12.5 mg\day (except arterial hypertension patients)	**Systolic BP shouldn't be< 90 mmHG**	
EVL	Every 1-4 weeks until the obliteration of varices.	-Obliterate varices - Eradicate new varices after initial obliteration	- First EGD performed 1-3 months after obliteration and every 6-12 months thereafter

1- **As a primary or secondary prevention**. Patients who recently diagnosed with cirrhosis should start primary prevention by giving them propranolol to decrease portal pressure, thus, decreasing the risk of varices progression and bleeding.

Management of variceal hemorrhage

◄ Most Commonly Used Vasoactive Agents in the Management of Acute Hemorrhage: [1]

Drug	Somatostatin	Octreotide (Somatostatin analogue)	Terlipressin (Vasopressin analogue)
Dose	- Initial **IV bolus** 250mcg (can be repeated in the first hour if ongoing bleeding) - Continuous IV infusion of 250-500 mcg\h	- Initial **IV bolus** 50mcg (can be repeated in the first hour if ongoing bleeding) - Continuous IV infusion of 50 mcg\h	- Initial 48 hrs: 2mg IV every 4 hrs until control bleeding - Maintenance: 1mg IV every 4hrs to prevent re-bleeing
Duration	Up to 5 days		
MOA	- Inhibits vasodilator hormone similar to glucagon, causing splanchnic vasoconstriction and reduces portal blood flow - Facilitates adrenergic vasoconstriction		- Splanchnic vasoconstriction. The active metabolite "lysin-vasopressin" is released gradually over several hrs in tissue, thus decreasing typical systemic vasopressin side effect

◄ Pharmacological therapy in the management of acute esophageal variceal hemorrhage : [2,3]

Regimen	Dose	Duration	Follow-up
Vasoconstrictor			
Octreotide	Intravenous 50-μg bolus, followed by infusion of 50 μg/h	2–5 d	Bolus can be repeated in first hour if variceal hemorrhage uncontrolled; if rebleeding occurs during therapy, consider TIPS
Terlipressin	2 mg given intravenously every 4 h for first 48 h, followed by 1 mg given intravenously every 4 h	2–5 d	If rebleeding occurs during therapy, consider TIPS
Somatostatin	Intravenous 250-μg bolus, followed by infusion of 250–500 μg/h	2–5 d	Bolus can be repeated in first hour if variceal hemorrhage uncontrolled; if rebleeding occurs during therapy, consider TIPS
Antibiotic			
Ceftriaxone	Intravenous ceftriaxone at a dose of 1 g once a day	5–7 d or until discharge	No long-term antibiotics unless spontaneous bacterial peritonitis develops
Norfloxacin	400 mg given orally twice a day	5–7 d or until discharge	No long-term antibiotics unless spontaneous bacterial peritonitis develops

1- pt should be admitted for 5 days and remains on IV infusion with .vasoactive agent after performing endoscopy , DON'T DISCHARGE.
2- we don't use norfloxacin in KSA , we use ciprofloxacin instead
3- Antibiotics are given as prophylaxis to prevent SBP with ascites "spontaneous bacterial peritonitis".

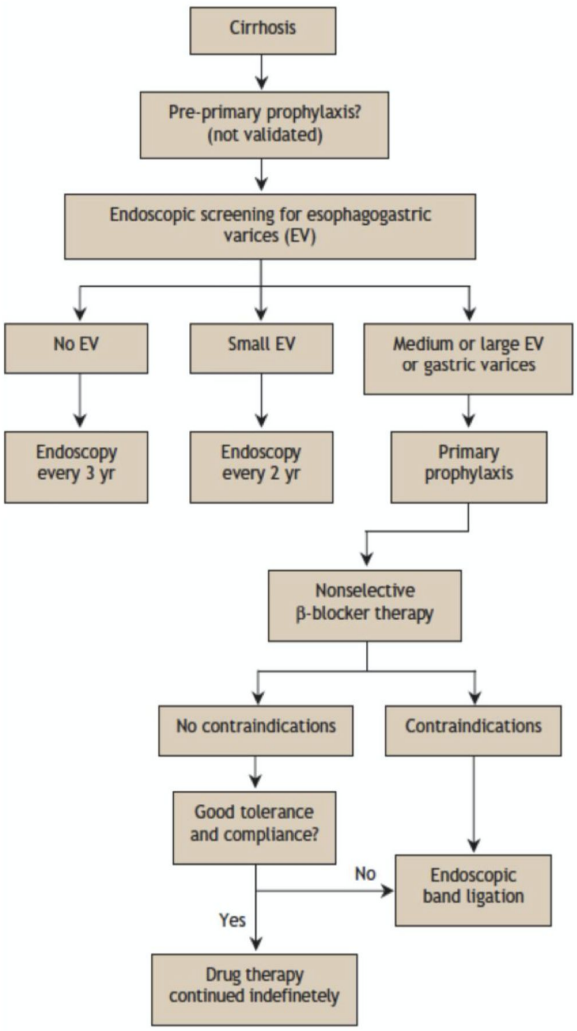
The 4 differences between variceal non-variceal:
1- octreotide 2- antibiotics 3- beta blocker 4- timing of endoscopy "12h for variceal"

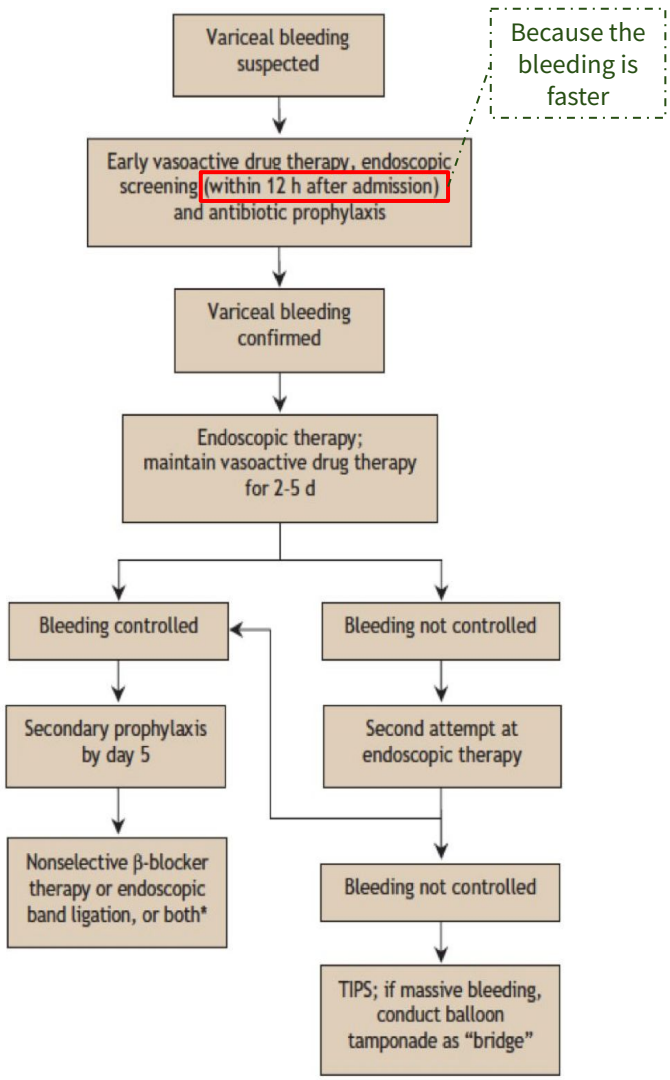
Management of variceal hemorrhage

◄ **management of patients who have bled from varices and in whom the goal is to prevent recurrence of hemorrhage :**

Therapy	Starting dose	Therapy goals	Maintenance/follow-up evaluation
Propranolol	20 mg orally twice a day Adjust every 2–3 days until treatment goal is achieved Maximal daily dose should not exceed 320 mg	Maximum tolerated dose Aim for resting heart rate of 50–55 beats per minute	At every outpatient visit make sure that patient is appropriately β-blocked Continue indefinitely In patients with refractory ascites reduce dose or discontinue if SBP < 90 mm Hg, serum sodium <130, or with acute kidney injury
Nadolol	40 mg orally once a day Adjust every 2–3 days until treatment goal is achieved Maximal daily dose should not exceed 160 mg		
EVL	Every 2–4 weeks until the obliteration of varices	Obliteration varices Eradication of new varices after initial obliteration	First EGD performed 1–3 months after obliteration and every 6–12 months thereafter

NOTE. Combination of 1 nonselective β-blocker (propranolol or nadolol) plus EVL is recommended.
SBP, spontaneous bacterial peritonitis.



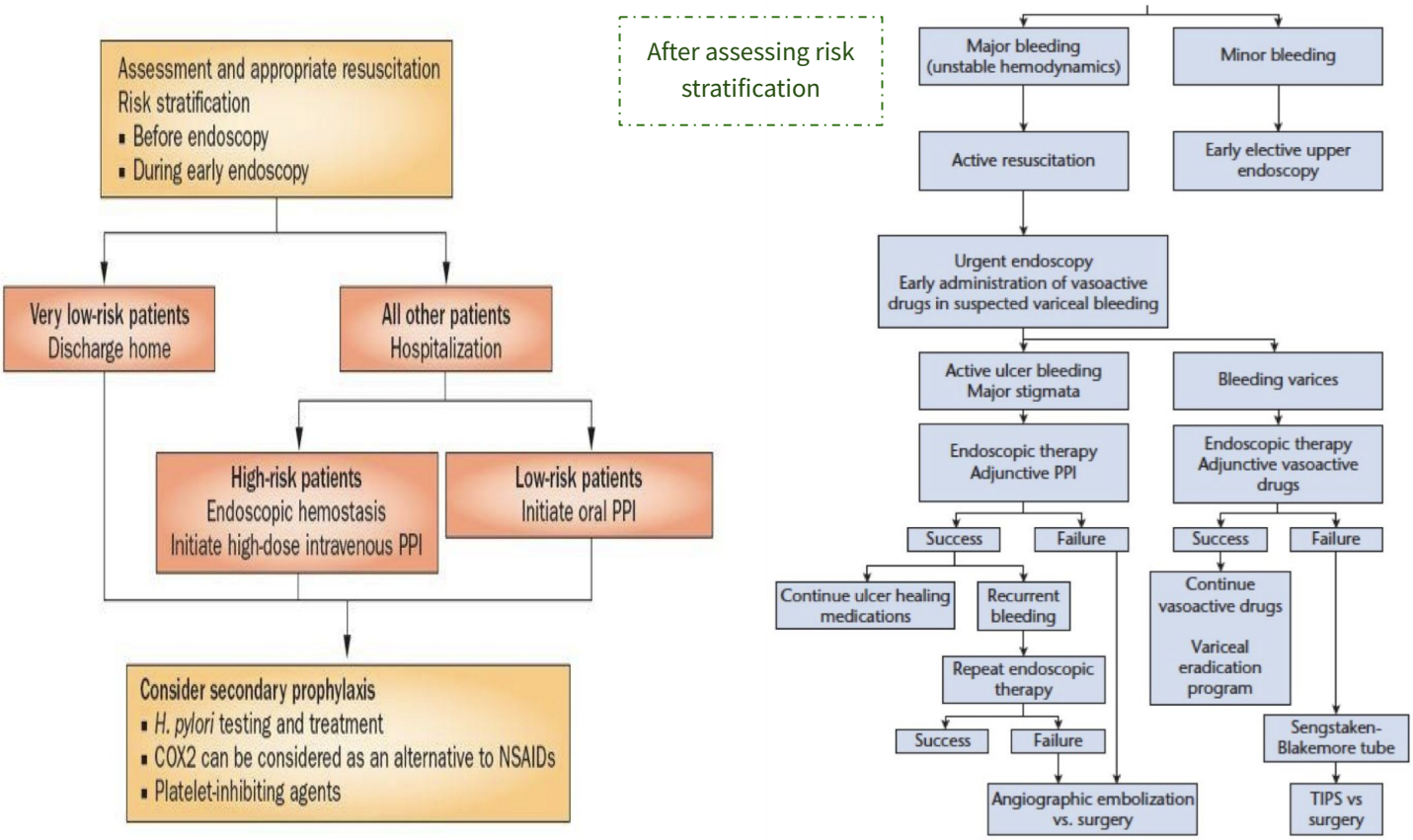


GI bleeding

◄ Summary of GI bleeding approach:

1. **Initial assessment detailed history and check vital signs.**
2. Hemodynamic status and resuscitation physical examination
   1-Tachycardic 2- Hypotensive.
3. Blood transfusion
4. Risk assessment and stratification Anemia Hb<7 transfer blood + Uremia.
5. Pre-endoscopic medical therapy PPI (will stop the bleeding ulcer) + Octreotide (vasoconstriction the bleeding vessels) (both IV), so we can see clearly while scoping.
6. Timing of endoscopy
7. Endoscopic therapy clipping, banding, cauterizing.
8. Post-endoscopy (H pylori eradication, PPI, stop using aspirin)

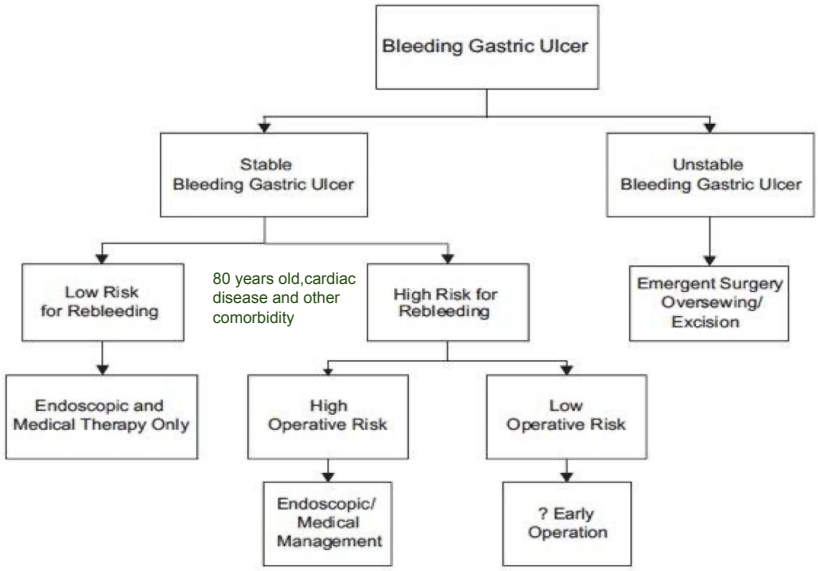
◄ Algorithm for the management of acute GI bleeding:



GI bleeding

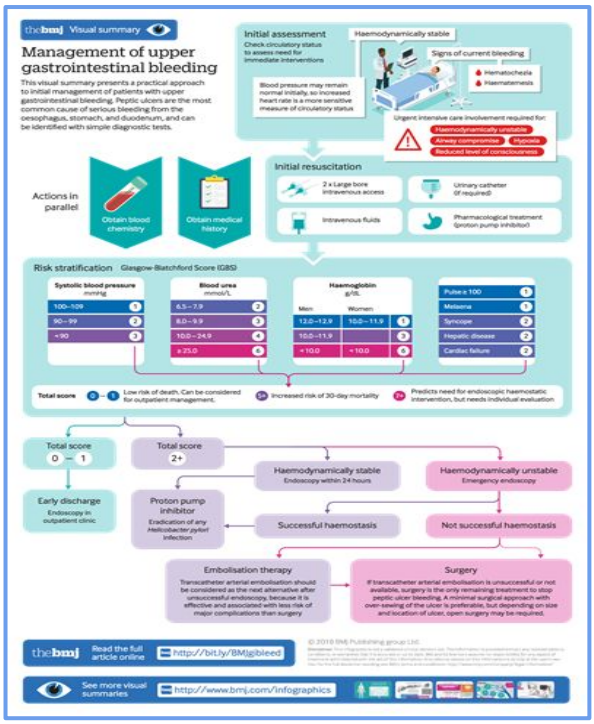
◀ When to go to surgery?

Surgery is indicated when endoscopic haemostasis fails to stop active bleeding and if rebleeding occurs on one occasion in an elderly or frail patient, or twice in a younger, fitter patient. If available, angiographic embolisation is an effective alternative to surgery in frail patients.



Conclusion

- Resuscitation should be initiated prior to any diagnostic procedure.
- Gastrointestinal endoscopy allows visualization of the stigmata, accurate assessment of the level of risk and treatment of the underlying lesion.
- Intravenous PPI therapy after endoscopy is crucial to decrease the risk of cardiovascular complications and to prevent recurrence of bleeding.
- Helicobacter pylori testing should be performed in the acute setting.



Annals of Internal Medicine — CLINICAL GUIDELINE

Management of Nonvariceal Upper Gastrointestinal Bleeding: Guideline Recommendations From the International Consensus Group

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Description: This update of the 2010 International Consensus Recommendations on the Management of Patients With Nonvariceal Upper Gastrointestinal Bleeding (UGIB) refines previous important statements and presents new clinically relevant recommendations.

Methods: An international multidisciplinary group of experts developed the recommendations. Data sources included evidence summarized in previous recommendations, as well as systematic reviews and trials identified from a series of literature searches of several electronic bibliographic databases from inception to April 2018. Using an iterative process, group members formulated key questions. Two methodologists prepared evidence profiles and assessed quality (certainty) of evidence relevant to the key questions according to the GRADE (Grading of Recommendations Assessment, Development and Evaluation) approach. Group members reviewed the evidence profiles and, using a consensus process, voted on recommendations and determined the strength of recommendations as strong or conditional.

Recommendations: *Preendoscopic management:* The group suggests using a Glasgow Blatchford score of 1 or less to identify patients at very low risk for rebleeding, who may not require hospitalization. In patients without cardiovascular disease, the suggested hemoglobin threshold for blood transfusion is less than 80 g/L, with a higher threshold for those with cardiovascular disease. *Endoscopic management:* The group suggests that patients with acute UGIB undergo endoscopy within 24 hours of presentation. Thermocoagulation and sclerosant injection are recommended, and clips are suggested, for endoscopic therapy in patients with high-risk stigmata. Use of TC-325 (hemostatic powder) was suggested as temporizing therapy, but not as sole treatment, in patients with actively bleeding ulcers. *Pharmacologic management:* The group recommends that patients with bleeding ulcers with high-risk stigmata who have had successful endoscopic therapy receive high-dose proton-pump inhibitor (PPI) therapy (intravenous loading dose followed by continuous infusion) for 3 days. For these high-risk patients, continued oral PPI therapy is suggested twice daily through 14 days, then once daily for a total duration that depends on the nature of the bleeding lesion. *Secondary prophylaxis:* The group suggests PPI therapy for patients with previous ulcer bleeding who require antiplatelet or anticoagulant therapy for cardiovascular prophylaxis.

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For author affiliations, see end of text.
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Click for a summary in males' slides

◀ Case study 1:

❖ A **65 y/o** male referred for evaluation of 4 months HX of **weight loss, fatigue** and weakness. He also gave history of passing **dark stool intermittently** for the last 3 months. He is known DM on insulin, hyperlipidemia on statin and occasionally **aspirin**.

- **What other information you would like to ask?**
  - **start by detailed abdominal history**
  - abdominal pain
  - Hematemesis
  - vomiting (is it coffee ground)
  - Hematochezia
  - heartburn
  - dysphagia
  - Details of weight loss
  - Other symptoms like odynophagia or dysphagia (with solids or fluids) for esophageal pathology. Anemic symptoms: fatigue, SOB, dizziness, palpitation,weakness, syncope or near syncope.
  - Hypotension: in severe presentation not like this case (3 months).
  - Trauma (abdominal aortic aneurysm) but not suitable with Hx of 3 months.
  - P.M/ cirrhosis , jaundice , any liver disease , IBD, cardiac disease, renal disease, history of peptic ulcer, reflux
  - Medication history (NSAIDS, aspirin, Anticoagulant)
- **What is the likely diagnosis?** In this case it is upper GI bleeding (Gastric cancer)
- **What will be the next step?** IV fluid resuscitation then endoscopy

◀ Case study 2:

❖ A **42** years old male complaining o**f chronic recurrent epigastric pain** which worsen recently especially when he is **fasting**[2]
For the last **2 days** he started to have frequent **vomiting associated with blood** (Red flag)**.**
He is not known to have any chronic medical problems and not on any medications.

- **What is the best next step in the approach of such patient?**
  - Detailed HX, Full Physical examination: Vital signs, look for clubbing, spider nevi, fluid thrill, splenomegaly, lymph nodes, etc..
- **How would you assess the bleeding severity?**
- **By Risk Stratification[1] :**
  - Glasgow- Blatchford Score (GBS)
  - Rockall Score
  - Modified-GBS
  - AIMS65
- **What is the diagnosis and its associated risk factors?**
  - Duodenal ulcer.

1- Direct you toward admission:
1. send home and perform endoscopy the next day?
2. admit?
3. admit to ICU?

2-May indicate duodenal ulcer (as it's worsened with fasting).

◀ Case study 3:

❖ A **52** years old lady presented to ER with **one day** history of **vomiting of fresh blood**. She also notices passing **black tarry stool**. She is feeling **dizzy and unwell** .
Past HX of jaundice no other medical problems and not on any medications.
Clinically **jaundiced and pale**.
Vital signs **BP 100/70 pulse 110/min** (tachycardia)
Abdomen examination showed **liver span of 7 cm** (normally 9-12 in male, 8-12 in female) and **spleen felt 3 fingers below costal margin**[1] **with few spider nevi seen over chest.**

- **What is the likely diagnosis of this case and list 4 common aetiology ?**
  - **Diagnosis** → Liver Cirrhosis with portal hypertension[1](varicella bleeding).
  - **Aetiology** → Drug induced hepatitis (alcohol, acetaminophen), Viral hepatitis B, C, Autoimmune hepatitis, NASH, hemolysis disease (Sickle cell..).
- **What is the priority in the management of this patient?**
- **stabilize the pt**
  - IV Fluid Resuscitation. 2 large bore cannula
  - endoscopy can be done after 6 hours after stabilizing the patient[2]
- **What is the target Hb and INR prior to the endoscopy for this case?**
  - Target Hb is 7 g/dL and above.

◀ Case study 4:

❖ A 47 years old male known to have **alcoholic liver disease** presented with **hematemesis of large amount and dizziness**
after resuscitation an upper GI endoscopy done which showed **multiple large esophageal varix which was banded**, however 12 hrs post endoscopy he continued to have melena with drop of Hb and hypotension.

- **What is the next step in the patient management?**
- consult gastroenterology, interventional radiology, surgery and admit to ICU.
  - since it's persistent we can perform a surgery.

1- Increased pressure in the portal vein by a blockage in the blood flow through the liver causes large veins (varices) to develop across the esophagus and stomach to get around the blockage and that lead to massive bleeding.

2- Do not do endoscopy early as it associated with high mortality

Summary

Gastrointestinal Bleeding

Upper Vs. Lower GI Bleeding

Upper GI Bleeding:

1. Peptic ulcer disease (most common cause)
2. Variceal bleeding (2nd most common cause)

Lower GI Bleeding:

1. Diverticular disease (most common cause)

Clinical Features

- **Type of bleeding:**
  - Hematemesis
  - "Coffee grounds" emesis
  - Melena
  - Hematochezia
  - Occult blood in stool
- **Signs of volume depletion**
- **Signs and symptoms of anemia**

Diagnosis & Management

- **Intravenous access**
  - At least one large-bore cannula.
- **Initial clinical assessment**
  - Define circulatory status
  - Seek evidence of liver disease.
  - Identify comorbidity
- **Basic investigations**
  - Full blood count
  - Urea and electrolytes (elevated blood urea with normal creatinine concentration implies severe bleeding)
  - Liver function tests
  - Prothrombin time
  - Cross-matching
- **Resuscitation**
  - **Intravenous crystalloid fluids:** should be given to raise the blood pressure
  - **Packed red blood cells:** If the hemoglobin level < 7 g/dL or If hemoglobin < 10 g/dL in patients with preexisting cardiovascular disease or patients with symptoms.
  - **Fresh frozen plasma:** if PT or INR is elevated.
- **Oxygen**
- **Endoscopy**
  - Diagnostic and potentially therapeutic. This should be carried out after adequate resuscitation, ideally within 24 hours. Treating endoscopically using a thermal or mechanical modality combined with intravenous proton pump inhibitor (PPI) therapy, prevent rebleeding, thus avoiding the need for surgery.
- **Monitoring**
- **Surgery**
- **Eradication**
  - All patients should avoid NSAIDs and those who test positive for H. pylori infection should receive eradication therapy.

Lecture Quiz

Q1: Which of the following presentations suggests variceal hemorrhage?
A- Unexplained iron deficiency anemia
B- Dysphagia
C- Abdominal bloating
D- Abdominal pain
E- Hematemesis

Q2: The effect of H. pylori eradication therapy always needs to be assessed in patients with which of the following?
A- A bleeding peptic ulcer
B- Reflux esophagitis
C- Non Ulcer dyspepsia
D- Uncomplicated peptic ulcer
E- Chronic active gastritis

Q3: A 80-year-old woman presents with melena, hematemesis, and syncope. Examination reveals hypotension and tachycardia. What is the first step in management?
A- Emergent endoscopy
B- Nasogastric lavage
C- Intravenous proton pump inhibitor
D- Tagged red blood cell scan
E- Intravenous access and intravascular volume repletion

Q4: A 50-year-old man without any prior medical problems began taking ibuprofen 800 mg three times daily for lower back pain after a work-related injury. He subsequently developed nausea followed by hematemesis and melena. He now presents to the emergency department for further evaluation. On the basis of this presentation and epidemiologic studies, what is the most likely cause of the suspected upper gastrointestinal (GI) hemorrhage?
A- Peptic ulcer
B- Mallory-Weiss tear
C- Esophagitis
D- Esophageal varices
E- Dieulafoy lesion

Q5: A 70-year-old woman presents to the emergency department with dizziness and five episodes of bright red blood per rectum in the last 24 hours. Nasogastric tube lavage yields bilious fluid without blood. What is the most common cause of severe hematochezia?
A- Diverticulosis
B- Colonic angiodysplasia
C- Internal hemorrhoids
D- Ulcerative colitis
E- Ischemic colitis

Q6: A 45-year-old man is brought to the emergency department after an episode of hematemesis. The patient had spent the night at a bar drinking with his colleagues. After leaving the bar, he vomited multiple times and noticed bright-red blood mixed with the vomitus, and he called an ambulance. Past medical history is notable for hypertension, for which he is taking lisinopril. Vital signs are within normal limits. Physical examination shows a patient in no acute distress. Cardiac, pulmonary, and abdominal exams are non-contributory. Which of the following additional findings will most likely develop in this patient?
A- Abdominal pain exacerbated with eating
B- Black tarry stools
C- Sloughed mucosa mixed with stool
D- Passage of bright red blood from the anus
E- Foul smelling oily stool

Answers: Q1:E | Q2:A | Q3:E | Q4:A | Q5:A | Q6:B

Answers Explanation File!

GOOD LUCK !

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اللهمّ إنّا نسألك لفقيدتنا الغالية أن تصب على قبرها الضياء والنور والفسحة والسرور

اللهم ارضَ عنها وجازها بالحسنات إحسانا، وبالسيئات عفوًا وغفرانًا

اللهم اجعل قبرها روضة من رياض الجنة

اللهم إنها في ضيافتك فأكرمها يا أكرم الأكرمين، واجمعنا بها في الفردوس الأعلى يا أرحم الراحمين

ربي أسألك أن تظلها تحت ظلك، وأسألك أن تطيب ثراها وأن تكرم منزلتها ومثواها، وأن تسكنها الجنة وتجعلها سكنًا لها ومأواها

اللهم كما طيبت ذكرها في أرضك بين خلقك ،طيب ذكرها في سمائك بين ملائكتك، وارحمها واغفر لها وانظر إليها بعين لطفك وكرمك يا أرحم الراحمين