



UPPER GASTROINTESTINAL Bleeding

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INTRODUCTION

- UGIB is considered medical emergency and require admission to hospital for urgent diagnosis and management. It is a common cause of emergency admission to hospital.
- UGIB vs LGIB= 5:1. Incidence: 170/100,000/yr. (US data). More common in males. Over 350,000 US hospitalization/yr.; cost \$ I billion/yr.
- Despite a decrease incidence of ulcer disease and improvements in the management of UGIB, mortality remains high at 6-10%, which increase in the elderly. Patients die rarely from exsanguinations but from complications of an underlying disease. Self-limited in 80% of patients.
- Patients are usually stratified into having either Variceal or Non-variceal sources of UGIB, as the two
 have different treatment algorithms and prognosis: Variceal: Look for evidence of chronic liver
 disease such as jaundice, spider naevi, ascites and so on. Non-variceal: Any previous history of PUD,
 NSAIDs, anticoagulants, dyspeptic symptoms.

UpperGI Bleeding

Non variceal Upper GI bleeding 90%

Variceal bleeding 10%

CAUSES

Pathology	Risk factor	Frequency (%)
Peptic Ulcer: Duodenal Ulcer; Gastric ulcer	H. pylori; NSAIDs	35-50
Gastric erosions; hemorrhagic gastropathy	Alcohol: NSAIDs	10-20
Esophageal varices; gastric varices	Liver disease; portal vein thrombosis	2-9
Erosive Esophagitis; esophageal ulcer	Usually with hiatus hernia	10
Mallory-Weis tear	Retching	5
Vascular malformation: Dieulafoy lesion; Hereditary telangiectasia (Osler-Weber-Rendu syndrome; angiodysplasia; gastric antral vascular ectasia (watermelon stomach).	Hereditary	5
Esophageal; gastric cancer		2
Aorto-enteric fistula	Aortic graft; primary	0.2
Portal gastropathy; Blood dyscrasia; coagulopathies; alcohol & drugs (anti-platelets; anticoagulants; aspirin; NSAIDs; SSRIs; steroids); Hemobilia; Hemosuccus pancreaticus; Pseudoxanthoma elasticum; trauma; foreign body; peri-ampullary ca; duodenitis; anastomotic ulcer; Crohn; severe superior mesenteric artery syndrome; Cameron lesions; Non-Gl source (epistaxis), Factitious bleeding; unknown		10
Castric Ulcer Varices Varices Varices Varices Varices Varices Varices	Ormophagitis Number Num	Liver Portal vein staamer tincrobain Varices Varices Varices Varices (2.5%) Ret:ting Maliory-Works Lear (3.5%) Gancer of stamach or oscillation (25%) Nichols Alcohol Gastrie crossions (3.5%)

OTHER CAUSES (1)

- Drugs: NSAIDs: 50% of patients > 60 yr. presented with UGIB has history of NSAIDs; Steroid therapy: rarely may cause Cushing's ulcer; Poorly controlled Anticoagulant therapy; SSRIs (Selective serotonin reuptake inhibitors): double the rate of UGIB.
- **Stress ulcer**: fortunately uncommon, associated with high mortality.
- Acute mucosal ulceration (stress gastritis): often multiple and not extend through muscularis mucosa. Diffuse and typically involve the gastric body and fundus. More frequently seen in the following conditions (shock, sepsis, surgery, trauma, burn, renal or respiratory failure, and jaundice). It is due to imbalance between aggressive and protective mucosal factors. Both H2-blockers and antacid are effective in prevention. Stress gastritis and mucosal ulceration are historically associated with (1) head injuries with associated elevations in intracranial pressure and (2) burn injuries. These stress ulcers are called Cushing ulcer and Curling ulcer, respectively.
- Mallory-Weiss syndrome: Account for 5-15% of all cases of UGIB, and are relatively common in alcoholics. Bleeding from a laceration in the mucosa at the gastric cardia or GEJ. This is usually caused by severe vomiting because of alcoholism or bulimia, but can be caused by any condition which causes violent vomiting and retching such as food poisoning. Forceful vomiting, retching, coughing or straining may create a rapid increase in the gradient between intragastric and intrathoracic pressures → gastric mucosal tear from the forceful distension of the GEJ. The tear involves the mucosa and submucosa but not the muscular layer (contrast to Boerhaave syndrome which involves all the layers). It often presents as an episode of hematemesis after violent retching or vomiting, but may also be noticed as melena, and a history of retching may be absent. In most cases, the bleeding stops spontaneously after 24–48 hours, but endoscopic or surgical treatment is sometimes required. The condition is rarely fatal.
- Hemobilia: is usually associated with intraductal neoplasm, trauma, or iatrogenic injury such as percutaneous liver biopsy and cystic artery pseudoaneurysm. Suggested by jaundice, RUQ pain & UGIB. May be confirmed at endoscopy but often require angiography. Angiographic therapy is the treatment of choice, although occasionally surgical therapy is necessary.
- Hemosuccus pancreaticus: most commonly due to a splenic artery pseudoaneurysm in patients with CP, pseudocyst, but rarely due to pancreatic duct malignancy.

OTHER CAUSES (2)

- Dieulafoy lesion: is characterized by a large tortuous arteriole most commonly in the gastric submucosa that erodes and bleeds. 75-95% occur in the proximal stomach, usually on the lesser curvature and within 6 cm of GEJ, although they have been reported to occur throughout the GIT. It can cause UGIB, but is relatively uncommon. It is thought to cause < 5% of all GI bleeds in adults. It is also called "caliber-persistent artery" or "aneurysm" of gastric vessels. However, unlike most other aneurysms, these are thought to be developmental malformations rather than degenerative changes.
- **Cameron lesion:** is a linear erosion or ulceration of the mucosal folds lining the stomach where it is constricted by the thoracic diaphragm in persons with large hiatal hernias. The lesions may cause chronic blood loss resulting in iron deficiency anemia; less often they cause acute bleeding. Treatment of anemia includes iron supplements and PPI acid suppression. Surgical hernia repair is sometimes needed.
- Angiodysplasia/ectasia: whether sporadic or secondary, is the most common vascular anomaly seen in the GIT. They are dilated tortuous vessels in the mucosa & submucosa. May be due to intermittent obstruction of the submucosal veins because of the colonic wall tension, which is highest in the cecum. May be sporadic, usually developing in the elderly or may be found in association with a number of disorders including renal failure, cirrhosis, the CREST syndrome, radiation injuries, von Willebrand's disease and aortic stenosis. May occur anywhere in the GIT, but are more commonly found in the colon (most common in the cecum & ascending colon), followed by the small intestine and the stomach. These lesions usually lead to occult blood loss, but can also cause overt GI bleeding.
- Gastric antral vascular ectasia (GAVE, watermelon stomach) : characterized by rows or stripes of ectatic (distensible) mucosal blood vessels that emanate from the pylorus and extend proximally into the antrum. It is an uncommon cause of chronic UGIB or iron deficiency anemia. Occasionally may present as acute UGIB (melena and/or hematochezia). The condition is associated with dilated small blood vessels in the pyloric antrum. The dilated vessels result in intestinal bleeding. It is also called watermelon stomach because streaky long red areas that are present in the stomach may resemble the markings on watermelon. GAVE is associated with a number of conditions, including portal hypertension, chronic kidney failure, and collagen vascular diseases. It also occurs particularly with scleroderma, and especially the subtype known as systemic sclerosis. GAVE is treated commonly by means of an endoscope, including argon plasma coagulation and electrocautery. Since endoscopy with argon photocoagulation is "usually effective", surgery (antrectomy) is usually not required.

RISK FOR UGIB

Acute Illness: •

- Shock
- 2. **Respiratory** failure
- 3 Head trauma
- Thermal injury 4.

Chronic Conditions: •

- Renal dysfunction I.
- 2 Liver diseases
- 3. Coagulopathy
- Helicobacter pylori 4.

Drugs: ٠

- Anticoagulants Ι.
- 2. Antiplatelets agents
- 3. **NSAIDs**
- **SSRIs** 4

Devices: •

- Mechanical ventilation
- 2. Renal –replacement therapy
- 3. Extracorporeal life support

Upper GI Bleed

History

- PUD, prior bleeds, EtOH, prior surgical/endoscopic interventions (marginal ulcers), liver disease (varices), tumor, prior radiation
- Meds NSAIDs, anti-platelets, anticoagulation
- ROS epigastric pain (PUD), retching (Mallory-Weiss tear), odynophagia/dysphagia (esophageal ulcer)

Physical Exam

- Look for evidence of hypovolemia (tachycardia/hypotension)
- Abdominal exam
- Rectal exam
- Guninc?!

Accurate H&P allows for proper assessment of bleeding severity, volume status, risk factors, and triage decision

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Low-doin aspro-	0.46	2.94	144
Storette -	1000	-0.00	2.28

18.0 Figure 5, Heat map of interaction of reiNSAIDs COX-2 inhibitors, and low 20 4.80 1.0 ed on the REP. and from evence and increa ergth of interaction.

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CLINICAL FEATURE

- Past history: dyspepsia, alcohol or NSAIDs ingestion, weight loss may suggest the cause.
- Hematemesis: Vomiting of blood, could be: digested blood in the stomach (coffee-ground vomitus that indicate slower rate of bleeding) or fresh/unaltered blood (gross blood and clots, indicates rapid bleeding). More common with lesions in the esophagus and stomach.
- Melena: Stool consisting of partially digested blood (black tarry, semi solid, shiny and has a distinctive odor, when present it indicated that blood has been present in the GIT for at least 14 hr. The more proximal the bleeding site, the more likely melena will occur. More common with lesions distal to the pylorus. The black color of melena stool is caused by hematin, the product of enzymatic degradation and oxidation of heme (Fe in hemoglobin) by intestinal and bacterial enzymes during passage through ileum and colon. Foul smelling, black (not dark) (make sure patient is not on iron or bismuth medication).
- Hematochezia: It is defined as passage of bright-red blood or maroon stools from the rectum. Bright red blood may come out unchanged in the stool. Usually represents a lower GI source of bleeding, however, an upper GI lesion may bleed so briskly (> IL blood loss) that blood does not remain in the bowel long enough for melena to develop. When hematochezia is the presenting symptom of UGIB, it is associated with hemodynamic instability and low hemoglobin. Present if profuse UGIB.
- Signs of severe acute blood loss: Pallor, clammy skin, tachycardia, and hypotension.
- Signs & symptoms of the underlying disease (e.g. liver disease, malignancy)
- BUN/Cr: usually > 30:1 ratio. Secondary to blood protein absorption or pre-renal azotemia.
- Positive Guaiac.
- The diagnosis of UGIB is assumed when hematemesis is documented. If absent, an upper source for bleeding is likely if there is melena or positive gastroccult test or endoscopic image of gastric or duodenal ulcer with stigmata of recent hemorrhage (e.g., visible vessel).

MANAGEMENT

- Four steps:
 - I. Resuscitation.
 - 2. Initial Assessment & Triage.
 - 3. Establishment of a diagnosis (Endoscopy).
 - 4. Management of specific conditions.
- Initial assessment, resuscitation and triage:
 - UGIB may have different clinical presentations:
 - > Hematemesis or hematochezia with hemodynamic instability,
 - > Melena or rectal bleeding without hemodynamic compromise.
 - Patients may have chronic GI bleeding with asymptomatic iron-deficiency anemia, or hemoccultpositive stool on screening for colorectal cancer.
 - Patients presenting to the ER with hemodynamic instability require rapid clinical assessment:
 - Intravenous access with at least two large-bore lines.
 - Nasogastric tube placement (controversial).
 - > Determination of hematocrit and coagulation studies, and type and cross for blood products.
 - Patients with altered mental status should undergo endotracheal intubation for airway protection.
 - Emergent evaluation by a gastroenterologist should be requested.
 - > The patients should be stabilized before proceeding to urgent endoscopy.



RESUSCITATION

- I. Measure BP and HR & repeat measurement hourly.
- 2. Admission (if SBP < 100 or HR > 100 \rightarrow ICU admission).
- **3**. NPO: for 24 hr.
- 4. Complete bed rest.
- 5. IV access with two large bore cannula (IV lines).
- 6. Draw blood samples for Basic investigations:
 - A. CBC (Hg level).
 - **B**. U & E.
 - C. LFT.
 - D. PT, PTT.
 - E. Blood group and cross match.



Controversial pre-endoscopy actions

- Naso-gastric tube placement with aspiration

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- Use of prokinetic agents (erythromycin or metoclopramide) intravenously 20 to 120 minutes before endoscopy in patients with severe bleeding
 Indexed serving Swall - brite which storg endoscer
- 7. IV colloids or crystalloids. Plasma expanders: e.g. Hess solution.
- 8. Start blood transfusion.
- 9. CVP line
- 10. Insert Foley's catheter and monitor the urine output hourly.
- 11. Insert NG tube (controversial). (NG aspirate may be negative in 10% of bleeding DU due to edema or pylorospasm; the sensitivity of NG aspirate of assessing active bleeding is 79%; NG may cause trauma or dislodge a clot!).
- 12. Endoscopy after stabilization for diagnosis & treatment.
- **I3.** IV PPI therapy for bleeding ulcer.





Needle Gauges for Injections Size Chart

	14 GAUGE CRIDICIOUSE OUTER DIAMETER: 072IN (1.63MM)
	COLOR: AMBER OUTER DIAMETER: . 065IN (1.85MM)
And the second s	OUTER DIAMETER: .064IN (1.63MM)
	18 GAUGE OUTER DIAMETER: .050IN (1.27MM)
	20 GAUGE COLORI, PINK OUTER DIAMETER: .036IN (.9IMM)
	21 GAUGE COLORE FURTHER OUTER DIAMETER: 033IN (.63MM)
	22 GAUGE CONTRETENCE OUTER DIAMETER: ,027(N (.70MM)
	23 GAUGE COLOR: ORANGE OUTEN DIAMETER: . 025IN (.63MM)
	25 GAUGE COLONI HID OUTER DIAMETER: .020IN (.53MM)
() · · · ·	27 GAUGE Color: WHITE OUTER DIAMETER: .0(6in (.42MM)

	SPECIFICATIONS									
	Gauge	Colour Code	Ext. Dia. mm	Length mm	Flow Rate ml/min					
_	14G	Orange	2.1	45	240					
	16G	Grey	1.8	45	180					
	18G	Green	1.3	32/45	90					
_	20G	Pink	1.1	32	60					
-	22G	Blue	0.9	25	36					
	24G	Yellow	0.7	19	20					
	26G	Violet	0.6	19	13					

Types of Needles for Injection

Color	Gauge Size	External Diameter (mm)*	Length (mm)*	Water Flow Rate (mL/min)*	Recommended Uses
Orange	14G	2.1 mm	45mm	~240 mL/min	Trauma, Rapid blood transfusion, Surgery ¹
Gray	16G	1.8 mm	45mm	~180 mL/min	Rapid fluid replacement, Trauma, Rapid blood transfusion ¹
Green	18G	1.3 mm	32mm	~90 mL/min	Rapid fluid replacement, Trauma, Rapid blood transfusion ¹
Pink	20G	1.1 mm	32mm	~60 mL/min	Most infusions, Rapid fluid replacement, Trauma, Routine blood transfusion ¹
Blue	226	0.9 mm	25mm	~36 mL/min	Most infusions Neonate, pediatric, older adults Routine blood transfusion ¹
Yellow	24G	0.7mm	19mm	~20 mL/min	Most infusions Neonate, pediatric, older adults, Routine blood transfusion, Neonate or Pediatric blood transfusion ¹
Purple	26G	0.6 mm	19mm	-13 mL/min	Pediatrics, Neonate ¹

BRITISH SOCIETY OF GASTROENTEROLOGY RECOMMENDATIONS

- We recommend that patients with hematemesis, melena, or coffee ground vomiting in the absence of an alternate diagnosis (e.g., bowel obstruction) trigger the acute upper gastrointestinal bleeding (AUGIB) bundle.
- We recommend that patients with suspected AUGIB should have urgent observations performed using a validated early warning score such as the National Early Warning Score (NEWS).
- We recommend all patients with AUGIB be commenced on intravenous fluids. We recommend in hemodynamically unstable patients a crystalloid solution as a bolus of 500 mL in less than 15 min.
- We recommend that red blood cell transfusion should follow a restrictive protocol (trigger: Hb <70 g/L; target: 70–100 g/L). A higher trigger should be considered in patients with ischemic heart disease or hemodynamic instability.
- We recommend that patients with AUGIB with ongoing hemodynamic instability are referred for critical care review.
- We suggest that **platelets** should be given in active acute upper GI bleeding with a platelet count ≤50×109/L, as per major hemorrhage protocols.
- We recommend the **Glasgow-Blatchford Score** (GBS) is calculated at presentation with AUGIB.
- We recommend that patients with GBS ≤ 1 at presentation are considered for outpatient management.
- We recommend **intravenous terlipressin** is given to all patients with suspected **cirrhosis/variceal bleeding**. However, caution should be exercised in patients with ischemic heart disease or peripheral vascular disease.
- We recommend giving intravenous antibiotics as per local protocol to patients with suspected cirrhosis/variceal bleeding.
- We recommend continuing aspirin at presentation.
- We recommend interrupting P2Y12 inhibitors until hemostasis is achieved unless the patient has coronary artery stents, in which case, a decision should be undertaken after discussion with a cardiologist. (P2Y12 receptor blockers are a group of antiplatelet drugs. This group of drugs includes: Clopidogrel, Ticlopidine, Ticagrelor, Prasugrel, and Cangrelor).
- We recommend interrupting warfarin therapy at presentation.
- We recommend interrupting direct oral anticoagulant therapy at presentation.
- We recommend **endoscopy** is offered to patients admitted with suspected AUGIB within 24 hours of presentation. Patients with ongoing hemodynamic instability will require more urgent endoscopy after resuscitation.
- We suggest that the endoscopy report should be reviewed by the ward team.
- We suggest that all patients with varices or those requiring endoscopic therapy are referred to a specialist gastroenterology service.
- We recommend patients with bleeding from ulcers with high-risk stigmata at endoscopy receive high-dose intravenous proton pump inhibitor (PPI) therapy; high-dose oral PPIs may be considered as an alternative.
- We recommend patients with AUGIB in whom antithrombotic therapy is interrupted have a clear plan for resumption.

TERLIPRESSIN

- Synthetic Vasopressin (Anti-diuretic hormone), used in variceal bleeds and hepatorenal syndrome.
- Mechanism of action: Slowly cleaved to vasopressin + intrinsic vasoconstrictor effect of its own.
- Dose: 2 mg IV followed by 1-2 mg every 4-6 hours, until bleeding is controlled, for up to 72 hours.
- Contraindications: Vascular disease (esp. coronaries), chronic nephritis. (Caution in asthma, epilepsy, migraine, renal impairment, pregnancy)
- Side Effects: Fluid retention, pallor, tremor, headache, nausea, vomiting, coronary artery constriction, peripheral ischemia, hypersensitivity reactions.
- Alternative/additional therapies: Vasopressin, Octreotide, Sclerotherapy, Baloon tamponade, Band ligation, TIPS.



VASOPRESSIN AND TERLIPRESSIN

- **Vasopressin** (Pitressin) is a nonselective vasoconstricting agent that causes a reduction of splanchnic blood flow and thereby a reduced portal pressure. Vasopressin, which is associated with severe vascular complications, has been largely replaced by other vasoconstrictors such as its synthetic analogue, triglycyl-lysine vasopressin (terlipressin).
- **Terlipressin** has fewer side effects and a longer biological half-life, allowing its use as a bolus intravenous injection (2 mg every 4 hours for the initial 24 hours, then 1 mg every 4 hours for the next 24–48 hours). Terlipressin has been shown to control bleeding in about 80% of cases and is the only pharmacologic therapy proven, to reduce mortality from acute variceal hemorrhage. In patients with esophageal variceal bleeding, a 24-hour course of terlipressin was shown to be as effective as a 72-hour course when used as adjunct therapy to successful variceal band ligation.
- In a randomized study, terlipressin was compared to norepinephrine in regard to effects on creatinine clearance and urine flow in septic patients; the authors concluded that renal function was improved with both drugs. Terlipressin decreases oxygen consumption. Because it has been speculated that terlipressin might exhibit anti-inflammatory effects that decrease oxygen demand of the tissues, this reduction of oxygen consumption may be interpreted as a positive consequence of terlipressin action. Terlipressin is the drug of choice in hepatorenal syndrome: It reverses hepatorenal syndrome in half of the treated patients and appears to be safe and well tolerated
- **Terlipressin** is an analogue of vasopressin used as a vasoactive drug in the management of low blood pressure. It has been found to be effective when norepinephrine does not help.
- Indications for use include norepinephrine-resistant septic shock and hepatorenal syndrome. In addition, it is used to treat bleeding esophageal varices.

BMP, CMP, TEG

A **basic metabolic panel (BMP**) is a test that measures **eight** different substances in the blood. Other names: chemistry panel, chemistry screen, chem 7, electrolyte panel.

It provides important information about the body's chemical balance and metabolism. A BMP includes tests for the following:

- I. Glucose.
- 2. Calcium.
- **3. Sodium**, **potassium**, **carbon dioxide**, and **chloride**.
- 4. BUN and creatinine.

A comprehensive metabolic panel (CMP): includes the same eight tests as a BMP, plus six more tests, which measure certain proteins and liver enzymes. The additional tests are:

- I. Albumin.
- 2. Total protein.
- **3. ALP** (alkaline phosphatase).
- 4. ALT (alanine transaminase).
- 5. AST (aspartate aminotransferase).
- 6. Bilirubin.

Thromboelastography (**TEG**) is a method of testing the efficiency of blood coagulation. It is a test mainly used in surgery and anesthesiology, although increasingly used in resuscitations in Emergency Departments, intensive care units, and labor and delivery suites. More common tests of blood coagulation include prothrombin time (**PT**) and partial thromboplastin time (**aPTT**) which measure coagulation factor function, but TEG also can assess **platelet function**, **clot strength**, and **fibrinolysis**, which these other tests cannot.

Thromboelastometry (TEM), previously named rotational thromboelastography (ROTEG) or rotational thromboelastometry (ROTEM), is another version of TEG in which it is the sensor shaft, rather than the cup, that rotates.

UK ACUTE UPPER GI BLEEDING BUNDLE



Haemodynamic instability? Think Major Haemorrhage Protocol +/critical care review



INITIAL ASSESSMENT & TRIAGE:

- To identify patients with non-variceal UGIB at greatest risk for mortality and rebleeding. Patients may be categorized as low, intermediate and high risk.
- Pre-endoscopy scoring systems:
 - I. Blatchford Score: BP, BUN level, Hemoglobir disease, heart failure.
 - 2. Clinical Rockall score: patient's age, shock & cc
- Post-endoscopy scoring system:
 - Complete Rockall score: Clinical Rockall score well with mortality & risk of rebleeding.



 Risk factors associated with increased mortality, recurrent bleeding, the need for endoscopic hemostasis, or surgery: Age > 60; severe comorbidity; active bleeding (e.g. witnessed hematemesis, red blood per NG tube, fresh blood per rectum); hemodynamic instability (hypotension); red blood transfusion > 6 units; severe coagulopathy.

ROCKALL RISK SCORE (1)

• Rockwall's score:

Variable	Score
Age	0-2
Presence of Shock	0-2
Comorbidity	0-3
Diagnosis	0-2
Endoscopic Stigmata of Recent Hemorrhage	0-2

Rockall Risk Score for rebleeding & death after admission to The hospital for Acute UGIB (2)

Variable	Score					
	0	1	2	3		
Age (years)	<60	60-79	≥ 80			
Shock Pulse rate SBP (mmHg)	"No shock" < 100 ≥ 100	"Tachycardia", ≥ 100 ≥ 100	"Hypotension" < 100			
Comorbidity	No Major comorbidity		Cardiac failure, ischemic heart disease, any major comorbidity	Renal or liver failure, disseminated malignancy		
Diagnosis	Mallory-Weiss tear or no lesion identified and no SRH/blood	All other diagnosis	Malignant lesion of Upper GIT			
Major SRH	None or dark spot only		Blood in the UGIT, adherent clot, visible or spurting vessel			

Rockwall Risk Score (3)

Score	% of Total	Rebleeding	Death
0	5.6	4.9	0
1	11	3.2	0
2	12.8	5	0.3
3	15.9	12.2	2
4	17.8	13.8	4.2
5	14.5	16.9	7.9
6	9.4	29.4	15.1
7	8	39.6	19.8
>8	5.1	47.7	39.1

Rockall Risk Score (4)

Score	Mean Hospital Stay (days)
0	3.7
1	4.1
2	6.1
3	7.6
4	9.3
5	10.8
б	10.6
7	12.7
>8	15.3
Total	8.6

Risk-Stratification Tools for Upper Gastrointestinal Hemorrhage

A Blatchford Score		B Rockall S	core		
At Presentation	Points	(T) 1003000.7		Variable	Points
Systolic blood pressure				- Age	
100–109 mm Hg	1			Age	
9099 mm Hg	2			<60 yr	0
<90 mm Hg	3		I	6079 yr	1
Blood urea nitrogen				⊒80 yr	2
6.5-7.9 mmol/liter	2		etter to all	Shock	
8.0-9.9 mmol/liter	3		Clinical	Heart rate >100 beats/min	1
10.0-24.9 mmol/liter	4		Rockall	Systolic blood pressure <100 mm Hg	2
≥25 mmol/liter	6		Score	Coexisting illness	
Hemoglobin for men		2 8		Ischemic heart disease, congestive	2
12.0-12.9 g/dl	1	Complete	I	heart failure, other major illness	
10.0-11.9 g/dl	3	Rockall	I	Benel follows benetic follows metastatic	
<10.0 g/dl	6	Score	I	Renal failure, nepatic failure, metastatic	
Hemoglabin for women		120023433	. L.	cancer	
10.0-11.9 g/dl	1		1.52	Endoscopic diagnosis	
<10.0 g/dl	6			No lesion observed, Mallory–Weiss tear	0
Other variables at presentation				Peptic ulcer, erosive disease, esophagitis	1
Pulse ≥100	1			Cancer of upper GI tract	2
Melena	1			Endoscopic stigmata of recent hemorrhage	
Syncope	2			Clean base ulcer, flat pigmented spot	0
Hepatic disease	2			Blood in upper GI tract, active bleeding, visible	2
Cardiac failure	2			vessel, clot	

Blatchford scores from 0 to 23, with higher scores indicating higher risk

Blatchfurd O, Murtay WB, Blatchford M. Langet 2000; 318 1338-31

The Rockall score :

Leicester

-Used clinical and endoscopic criteria

-The scale ranges from 0 to 11 points, with higher scores indicating higher risk.

Blood Urea	Nitrogen(mmol/L)	-Other markers	
6.5 - 7.9	2	Pulse ≥100 (per min)	1
8 - 9.9	3	Presentation with melena	1
10 - 24.9	4	Presentation with syncope	2
>25	6	Hepatic disease	2
Haemoglob	oin (g/dL) for men	Cardiac failure	2
12-12.9	1	•Score from 0 to 23	
10-11.9	3	·Scores ≥ 6 – 50% risk of needle	ng an
<10	6	intervention.	0.0000
Haemoglob	oin (g/dL) for		
women		Score is "0" if :	
10-11.9	1	 Hemoglobin level 	
<10	6	>12.9 g/dl(men) or	
Systolic BP	(mm Ha)	>11.9 g/dl(women)	and Mar
100-109	1	*Systolic blood pressure >109 P	nm rig
- 90-99	2	•BUN level <6.5 mmol/L	
- <90	3	•No melena or syncope	
Carlo Col Marcana		 No liver disease or heart failure 	

		Contract of Providence of Contract	
AMPR C	-	Typics, M (month)	
H9-84	7.	1. 210-328	114
100-100	1	* 89-89	13
318-0-036	4	+ +90	
125	1.6		
State States	1	Caral Market	
110.0 (10.0	1.	+ False 2180 (per wild)	
888-928	2	Presentation with melastra	1
100.0		* Presentation with sprapp	
		+ Hepatic diverse	12
augusta States		+ Kantactoline	1.1
	1000 2010-020 200-020 200-020 200-020 200-020 200-020 200-020 200-020 200-020 200-020 200-000-0	11100 241-000 2 3150-024 3 2150-024 2 2150-025 6 7150-026 1 1150-026 1 1150 0 1150 0 1	Hype Specific AF (Home) (

ESTABLISHMENT OF A DIAGNOSIS

- The foundation of diagnosis and management of patients with an UGIB is an endoscopy. After stabilization, upper GI endoscopy: under minimum sedation with wide pore suction channel endoscope.
- Advantage of early endoscopy (within 24 hours):
 - I. Identify the bleeding site.
 - 2. Assess the rate of bleeding.
 - 3. Therapeutic hemostatic procedures "adrenaline, laser diathermy, heater probe..."
 - 4. Reduction in blood transfusion requirement.
 - 5. Identify patients who are not suitable for surgery.
 - 6. Decrease in the need for surgery.
 - 7. Shorter length of hospital stay

Endoscopic finding



Stigmata of recent hemorrhage



ENDOSCOPIC TREATMENT MODALITIES:

- The nature of the visible vessel could be: a vessel; a pseudoaneurysm; or a clot.
- Topical treatment include: tissue adhesives (cyanoacrylate), blood clotting factors (thrombin, fibrinogen), vasoconstriction drugs (epinephrine), collagen, Ferromagnetic tamponade.

Endoscopic treatment modalities				
Injection	 Adrenaline (1:10000) Sclerosants (ethanolamine, ethanol, polidocanol) Pro-coagulants (thrombin, fibrin) Cyanoacrylate 	 Most commonly used for variceal UGIB		
Thermal Devices	 Heater probes Electrocautery probes Argon plasma coagulation Lasers photocoagulation 			
Mechanical Therapy	 Clips Band ligation 	 Modality of choice for variceal UGIB		

Meta-analyses have found that combination therapy (adrenaline + 2nd modality) is superior to adrenaline alone in treating high risk stigmata lesions (reducing risk of rebleeding, mortality and surgery).

Endoscopic therapy















Angiographic therapy: Embolization



Contraued & Repeat colors: anterlogram revealed active extraoration of indivated contrast from the superior particulated anterp R-Col entrolization of the pathoticational anterp



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I. BLEEDING PEPTIC ULCER:

• Medical treatment:

• Proton pump inhibitor

• Endoscopic therapy (hemostasis):

- I. Injection therapy: Adrenaline (1/10,000) or sclerosant injection.
- 2. Heat probes.
- 3. Bipolar diathermy.
- 4. Laser photocoagulation: using the Nd-YAG laser.
- 5. Metallic clips application.

• Angiographic therapy:

• Angiographic Embolization.

• Surgical treatment; indications:

- Continued bleeding.
- Recurrence of bleeding after endoscopic therapy.
- Patient > 60 yr. who need > 6 units of blood for stabilization.



PEPTIC ULCER BLEEDING: CLASSIFICATION

Peptic ulcer bleeding:

- Good prognosis: 80% stop spontaneously.
- Low mortality (2%) unless rebleeding occurs (25% of patients, 10% mortality).
- Endoscopic predictors of rebleeding (Forrest Classification): Spurt or ooze, visible vessel, fibrin clot.
- Patient can be sent home, if clinically stable, bleed is minor, no comorbidities, endoscopy shows clean ulcer with no high risk predictors of rebleeding.
- Esophageal varices have a high rebleeding rate (55%) and mortality (29%).

Forrest Class	Type of Lesion	Risk of Rebleed (%)
L	Arteria bleeding (oozing / spurting)	55-100
lla	Visible vessel	43
llb	Sentinel clot	22
llc	Hematin covered flat clot	10
III	No stigmata of hemorrhage	05

Forrest Prognostic Classification of Bleeding Peptic Ulcers

SURGICAL ASPECTS IN TREATMENT

- Due to advances in medications and therapeutic endoscopy techniques and angiographic therapy, UGIB is now usually treated without surgery.
- Surgery is indicated if endoscopic therapy or embolization failed.
- DU is treated by vagotomy + pyloroplasty + undersewing of the bleeding ulcer.
- If DU is large and pyloroplasty is difficult, polya gastrectomy should be done.
- For bleeding GU \rightarrow Billroth I gastrectomy.
- If the bleeding site is not recognized at endoscopy → the operation should be initiated by gastrotomy.





2. ESOPHAGEAIVARICES

- High mortality and morbidity and high recurrence rate.
- Initial treatment: Rubber band ligation or Injection sclerotherapy "ethanolamine or polidocanol." And: vasopressin infusion "vasoconstrictor" should be tried.
- If failed \rightarrow Blakemore-Sengstaken tube should be tried.
- If failed \rightarrow TIPS
- If failed \rightarrow Esophageal transection + gastric devascularization "Sugiura procedure".















3. GASTRIC EROSIONS

- IV proton pump inhibitor.
- Endoscopic hemostasis procedures.
- Total gastrectomy for persistent bleeding "high mortality"

4. MALLORY-WEISS TEAR

• Treatment is usually supportive as persistent bleeding is uncommon because in most cases the bleeding stops spontaneously. However cauterization (Endoscopic application of thermal probes) or local injection of adrenaline to stop the bleeding may be undertaken during the index endoscopy procedure. If all other methods fail, high gastrostomy can be used to ligate the bleeding vessel (direct suturing). Wide wedge resection of the artery and bleeding site is preferable to oversewing the artery in the area of the mucosal defect. Patients who are poor surgical candidates may respond to angiographic embolization. Very rarely embolization of the arteries supplying the region may be required to stop the bleeding. The tube will not be able to stop bleeding as here the bleeding is arterial and the pressure in the balloon is not sufficient to overcome the arterial pressure.



Mallory–Weiss tear affecting the esophageal side of the gastroesophageal junction

5. **ESOPHAGITIS**

- IV proton pump inhibitor in severe cases.
- 6. Tumors

•

• Hemostasis then elective surgery.

7. Vascular malformations "Dieulafoy lesion"

• Endoscopic hemostasis.

8. Angiodysplasia:

 radiologic imaging

 • Mesentric Angiography

 • Rationuclide scanning

 • CT enterography and CT engrography

Usually apparent at endoscopy, at which time therapy with laser or thermal probes may be applied. Bleeding that is refractory to endoscopic or medical therapy is an indication for surgical resection.

9. Aortoenteric fistula:





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Source: Longo DL, Fauci AS, Kasper DL, Hauser SL, Jameson JL, Loscalzo J: Harrison's Principles of Internal Medicine, 18th Edition: www.accessmedicine.com

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PREVENTION

- Stop smoking, avoid exposure to secondary smoke
- Avoid alcohol, caffeine
- Avoid Aspirin, heavy or regular use of medications.
- The approach for primary prevention of NSAIDs related mucosal injury has included avoiding the agent, using NSAIDs that are theoretically less injurious, and/or the use of concomitant medical therapy to prevent NSAID-induced injury. Prophylactic therapy may include: Misoprostol and PPI. Several nonselective NSAIDs that are associated with a lower likelihood of GI toxicity include diclofenac, aceclofenac, and ibuprofen.