GI Bleed in Children

GI Bleed could result from bleeding from esophagus to anus. It has been divided into upper, middle and lower depending on the site of bleeding.

“Upper GI bleeding is defined as bleeding from a source in the esophagus, stomach or duodenum above the ampulla of vater. Middle GI bleeding is defined as small intestinal bleeding from ampulla of vater to terminal ileum and lower GI bleeding is colonic bleeding”

GI Bleeding may be overt or obscure. Overt bleeding may present as hematemesis, melena or hematochezia. Obscure GI bleed is defined as bleeding from GIT that persists or recurs without any obvious etiology after a diagnostic endoscopy. It accounts for around 5% of all GI bleeds. It is of 2 types- occult and overt. Occult bleeding may present as positive fecal occult blood test (e.g. guaiac testing), laboratory evidence of anemia and iron deficiency, or symptoms of anemia or blood loss (e.g. fatigue, lightheadedness, syncope, dyspnea, angina)

Hematemesis is used to describe vomiting of either bright red or coffee-ground-like material and suggests that the source of bleeding is located above the ligament of Treitz. Melena, the passing of dark black, tarry stools, occurs in patients bleeding from a site located above the ileocecal value. The jet black color of melanotic stool results from the action of bacteria on hemoglobin that has been converted to hematin or other hemochromes. However an upper GI source may occasionally bleed so briskly that the blood does not remain in the GIT long enough for melena to occur. UGI and Middle GI bleed should be atleast 50 ml in volume and should stay for more than 6 hours to lead to melena. Hematochezia, the passage of bright red or maroon blood from the rectum, is most commonly associated with a colonic source of bleeding. If hematochezia is from an upper GI site, it reflect major bleeding with rapid transit which presents with hemodynamic instability and increased mortality.

Epidemiology

The annual incidence of hospitalization for upper GI bleeding is approximately 1 per 1000 adults and mortality is around 7 -10%. UGI bleed is an indication for UGI endoscopy in 5% of children.

Etiology

Table 1: Causes of UGI Bleed in Children

<table>
<thead>
<tr>
<th>Newborn</th>
<th>Infant</th>
<th>Children</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Swallowed maternal blood</td>
<td>• Stress ulcer / gastritis</td>
<td>• Mallory – Weiss tear</td>
</tr>
<tr>
<td>• Hemorrhagic disease of</td>
<td>• Acid-peptic disease</td>
<td>• Acid-peptic disease</td>
</tr>
</tbody>
</table>

(1)  
(2)  
(3)  
(4)
newborn  
- Stress gastritis  
- Acid peptic disease  
- Vascular anomaly  
- Coagulopathy  
- CMPI  

Mallory –Weiss tear  
- Vascular anomaly  
- Duplication cyst  
- Varices  
- Webs  
- Intestinal obstruction  

Varices  
- Stress ulcer / gastritis  
- Caustic injury  
- Foreign body  
- Vasculitis  
- Corhn’s disease  
- Intestinal obstruction  
- Dieulafoy lesion  
- Hemobilia  
- Pancreatic Pseudoaneurysm

Table 2: Common causes of rectal bleeding in children

<table>
<thead>
<tr>
<th>Infant</th>
<th>Older child</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Anal fissure</td>
<td>• Anal fissure</td>
</tr>
<tr>
<td>• Milk protein intolerance</td>
<td>• Intussusception</td>
</tr>
<tr>
<td>• Necrotizing enterocolitis</td>
<td>• Infectious enterocolitis, Amebiasis</td>
</tr>
<tr>
<td>• Swallowed maternal blood</td>
<td>• Meckel's diverticulum</td>
</tr>
<tr>
<td>• Vitamin K deficiency</td>
<td>• Juvenile polyp</td>
</tr>
</tbody>
</table>

Table 3: Less common causes of rectal bleeding in children

<table>
<thead>
<tr>
<th>Infant</th>
<th>Older Child</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Vascular lesions</td>
<td>• Inflammatory bowel disease</td>
</tr>
<tr>
<td>• Bleeding diathesis</td>
<td>• Ischaemic/collagenous colitis</td>
</tr>
<tr>
<td>• Hirschprung enterocolitis</td>
<td>• Vascular malformations</td>
</tr>
<tr>
<td>• Meckel diverticulum</td>
<td>• Intestinal duplication</td>
</tr>
<tr>
<td>• Malrotation with volvulus</td>
<td>• Bleeding diathesis</td>
</tr>
<tr>
<td>• Intestinal duplication</td>
<td>• Henoch-Schonlein purpura</td>
</tr>
<tr>
<td>• Intussusception</td>
<td>• Hemolytic-uremic syndrome</td>
</tr>
</tbody>
</table>

Initial evaluation

Is it G.I Bleed?

Mimickers of Gastrointestinal Bleeding

- Bleeding from the nose and oropharynx
- Bright red blood may be mimicked by red colored candies, juices, and foods
• Black colored stools may be caused by:
  – Bismuth
  – Iron preparations
  – Spinach
  – Blueberries

Table 4: Causes of false positive and false negative stool guaiac test (to detect occult blood)

<table>
<thead>
<tr>
<th>False-Positive</th>
<th>False – Negative</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Meat</td>
<td>• Vitamin- C</td>
</tr>
<tr>
<td>• Horseradish</td>
<td>• Storage of specimen &gt; 4 days</td>
</tr>
<tr>
<td>• Turnips</td>
<td>• Outdated reagent or card</td>
</tr>
<tr>
<td>• Ferrous sulfate (stool pH &lt; 6.0)</td>
<td></td>
</tr>
<tr>
<td>• Tomatoes</td>
<td></td>
</tr>
<tr>
<td>• Fresh red cherries</td>
<td></td>
</tr>
</tbody>
</table>

Causes of Upper G.I Bleeding

Mallory –Weiss Tears: It occurs at the GE junction primary on the gastric side, resulting, retching or coughing is classically reported prior to hematemesis. It is more common in adults and is less frequently seen with children.

Hemorrhagic and erosive gastropathy: It refers to subepithelial hemorrhage and erosions. These are restricted to the mucosa, where no large blood vessels are present and therefore do not cause major bleeding. Erosions are reported in around 2-10% of patients with UGIB. The most common causes are NSAID use and stress. Prevalence in NICU is around 44% and but out of these only 5% are clinically significant.\(^5\)

Peptic ulcer: Incidence of bleeding ulcers appears to have decreased over past decades. The decrease is due to the decreasing H. pylori prevalence and possibly the increased use of acid suppressive medications. Increasing use of NSAID is an important cause of peptic ulcer disease in children. Approximately 1/3\(^{rd}\) of patients found to have an ulcer with active bleeding or a non-bleeding visible vessel will have further bleeding requiring surgery if treated expectantly.\(^6\) These patients should receive endoscopic therapy and IV infusion of PPI. In less than 10 years of age, around 77% of the PUD is in duodenum. Hematemesis and perforation are more in secondary PUD. Factors associated with PUD:
  • Primary: Related to H.pylori, Bile reflux
• Secondary: Related to
  - NSAID intake
  - Stress – Shock, ischemia
  - Drugs
  - Corrosives
  - Menetrier’s disease
  - Zollinger-Ellison syndrome

Table 5: Classification of peptic ulcer disease\(^{(7)}\)
Esophageal varices and Portal Hypertension:

Portal hypertension has been defined as Hepatic Venous Pressure gradient (HVPG) of more than 5 mm of Hg. Unlike adults where cirrhosis is the most common cause of portal hypertension, extrahepatic portal venous thrombosis (EHPVO) is the most common cause in children.

<table>
<thead>
<tr>
<th>Endoscopic findings</th>
<th>Forrest classification</th>
<th>% of total</th>
<th>Rebleeding (%)</th>
<th>Surgery (%)</th>
<th>Mortality (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clean base</td>
<td>III</td>
<td>42</td>
<td>5</td>
<td>0.5</td>
<td>2</td>
</tr>
<tr>
<td>Pigmented flat spot</td>
<td>IIc</td>
<td>20</td>
<td>10</td>
<td>6</td>
<td>3</td>
</tr>
<tr>
<td>Adherent clot</td>
<td>IIB</td>
<td>17</td>
<td>22</td>
<td>10</td>
<td>7</td>
</tr>
<tr>
<td>NBVV</td>
<td>IIA</td>
<td>17</td>
<td>43</td>
<td>34</td>
<td>11</td>
</tr>
<tr>
<td>Active bleed</td>
<td>I</td>
<td>18</td>
<td>55</td>
<td>35</td>
<td>11</td>
</tr>
</tbody>
</table>
Table 6: Portosystemic Shunts (natural)

<table>
<thead>
<tr>
<th>Site of communication</th>
<th>Systemic vein</th>
<th>Portal vein</th>
<th>Manifestation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Esophagus and stomach</td>
<td>Azygos vein</td>
<td>Left gastric and short gastric</td>
<td>GE varices hematemesis, malaena</td>
</tr>
<tr>
<td>Ano rectum</td>
<td>Middle and on inferior hemorrhoid</td>
<td>Superior haemorrhoid</td>
<td>Rectal varices Bleed per rectum</td>
</tr>
<tr>
<td>Around umblicus</td>
<td>Superficial vein of anterior abd vein( sup and inferior epigatsric)</td>
<td>Paraumbilical veins accompanying theround ligament</td>
<td>Caput medusa</td>
</tr>
<tr>
<td>Veins of Retizius</td>
<td>Retroperitoneal</td>
<td>Inferior mesenteric vein</td>
<td>Bleeding from stoma site/silent</td>
</tr>
<tr>
<td>Extraperitoneal surfaces of abdominal organs</td>
<td>Subdiaphragmatic (veins of Sappey and retroperitoneal vein)</td>
<td>Tributaries of superior and inferior mesenteric veins</td>
<td>Silent</td>
</tr>
</tbody>
</table>

In country like India, portal hypertension is caused by EHPVO (68-76%), cirrhosis (24-28%); and infrequently due to congenital hepatic fibrosis (3%), non cirrhotic portal fibrosis and Budd Chiari Syndrome. EHPVO is also the most common and important cause of GI bleeding in children. It accounts for almost 70% of pediatric patients with portal hypertension. [8,9]

Oesophageal varices can be classified as small, medium and large (<5mm, 5-10mm and >10mm) or can be divided into 4 grades endoscopically according to Conns classification:

Table 7: Conns Classification of esophageal varices [10]

<table>
<thead>
<tr>
<th>Grade 1</th>
<th>On inspiration only</th>
<th>Can be effaced</th>
<th>Straight</th>
<th>Red</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade 2</td>
<td>Both on</td>
<td>Cannot be</td>
<td>Straight</td>
<td>Red</td>
</tr>
</tbody>
</table>
Predictors of variceal bleed include large varices and presence of red colour signs (Red wale marking, diffuse redness, haematocystic malformation).

**Gastric varices** are found most commonly with splenic vein thrombosis or after EST of oesophageal varices. They have been classified endoscopically according to location (Sarin’s Classification). (11)

- **Gastro-esophageal varices (GOV)** – Gastric varices continuing with esophageal varices
  - GOV1 - Extending towards lesser curvature
  - GOV2 - Extending towards fundus

- **Isolated gastric varices (IGV)** – Gastric varices discontinuous with esophageal varices or present in absence of esophageal varices
  - IGV1 - Fundal varices, tortuous, nodular
  - IGV2 – Ectopic varices (body, antral, pylorus)

**Ectopic varices** account for 5% of all variceal bleed. Duodenum is a common site. (12)

**Extra-hepatic Portal Vein Obstruction**: The mean age of presentation is 5 to 6 years. (13) In India 83% of patients with Extrahepatic Portal Vein Obstruction (EHPVO) present with upper gastrointestinal bleeding before the age of 20 years, compared to the data of the western world where more than 43% may present after this age. (13) Hematemesis with or without melena is the commonest presentation; only 8-10% patients may not bleed. (13) UGI bleeding is massive and recurrent but risk of rebleeding after major episode is less than cirrhosis (14) but is fairly uniform and occurs once in every
2 years. The average number of bleed is 2.5-5 episodes per patient. Splenomegaly is almost universal in patient with EHPVO. It can be present as early as 1 month of age and is usually seen before 3 years of age. Splenomegaly is mild (<6cm) in 42%, moderate (6-10 cm) in 40% and massive (>10cm) in 18%. Children range with EHPVO do not grow as do their healthy sibs. Their mental function is normal. They usually do not develop encephalopathy even with massive GI bleed. They have normal liver functions. They may develop transient ascites following major bleeding episode. Persistent or massive ascites in children with EHPVO should doubt about the diagnosis or suggest the possibility of presence of coexistent cirrhosis. It has been observed that the frequency and severity of upper GI bleed in children with EHPVO decreases with age. It is usually diagnosed by ultrasonography. At the time when occlusion of portal vein by a thrombus develops, patient may remain asymptomatic, the thrombus become organized and tortuous collaterals develop around the blocked portal vein a process termed as cavernous transformation. However, sometimes acute portal vein thrombosis is associated with development of progressive ascites, abdominal pain secondary to small bowel ischemia and intestinal infarction leading to acute abdomen and melena if thrombus extends into SMV.

**Chronic liver disease:** In portal hypertension occurring secondary to chronic liver disease the presentation is usually dominated by manifestations of primary disease. The major causes in children are viral hepatitis, neonatal cholestasis, autoimmune liver disease, metabolic liver disease like Wilson’s disease, glycogen storage disease etc.

Children with EHPVO in comparison to CLD have
- More chances of UGI bleed (61.6% vs 14.7%)
- More previous bleeding episodes (2.7 vs 1.2)
- Long duration of symptoms (26 vs 12 mth)
- Absence of jaundice
- Preservation of liver function
- Less Hb value.

**Pointers to diagnosis are:**
- If significant painless bleed - variceal bleed.
- Splenomegaly ~ 95% PHTN
- Jaundice, ascites, stigmata – CLD
- H/o NSAID – PUD/ gastritis,
- Pain – PUD, Mallory Weiss (crystalloid and colloids).
Table 8: Clinical clues to etiology in a GI bleed patient

<table>
<thead>
<tr>
<th>Bleeding etiology</th>
<th>Clinical clues</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mallory – Weiss tear</td>
<td>Emesis before hematemesis, pain+</td>
</tr>
<tr>
<td>Esophageal ulcer</td>
<td>Odynophagia, GERD, H/O pill ingestion</td>
</tr>
<tr>
<td>Stress gastritis</td>
<td>Sick patient in ICU, respiratory failure</td>
</tr>
<tr>
<td>Angiodysplasia</td>
<td>Renal failure, hereditary haemorrhagic telangiectasia</td>
</tr>
<tr>
<td>Aortoenteric fistula</td>
<td>H/O aortic aneurysm or surgery</td>
</tr>
</tbody>
</table>

Fig 2: Approach to Etiology:

The heart rate and B.P. provide the most important information is the initial assessment of a patient with UGI as major bleeding leads to postural changes in H.R. or B.P., tachycardia and eventually hypotension. Vital signs, an essential part of this evaluation should include the pulse rate and the blood pressure in lying, sitting and if possible, in standing position. An increase in the pulse rate of more than 20 beats per minute or a decrease in diastolic blood pressure of more than 10mm Hg or a decrease in systolic of more than 20mmHg within 3min of standing, indicates a greater than 20% loss of intravascular volume. As blood loss increases to 20-25%, the pulse rate increases more than 150 beats per minute. When the intravascular volume deficit exceeds 25%, the capillary refill is prolonged as blood is shunted from the skin to the brain and kidneys. Urine output decreases with further compromise and metabolic results. In short, all
features of hypovolemic shock develop. Urine output and fluid intake must be carefully monitored throughout fluid resuscitation of patients in shock. Urine output in children is normally 2 to 3 ml per kg per hour, and urine output less than 1 ml per kg per hour is indicative of renal hypoperfusion. As blood loss exceeds 40%, compensation fails, pulses are lost, cerebral perfusion decreases, and the patient passes from lethargy to coma. The respiratory rate should also be carefully monitored. Hyperventilation is an early sign of a developing acidosis induced by a decrease in central nervous systems pH.

Physical examination by an experienced observer to evaluate capillary perfusion, skin color for the presence of cyanosis, pulse, blood pressure, respiratory pattern, and level of consciousness should continue on an ongoing basis throughout the hemorrhage to enable rapid recognition of recurring shock. To enhance the ability to anticipate shock, it is helpful if the rate of bleeding is assessed periodically. In upper gastrointestinal bleeding this is best accomplished through lavage of the stomach by means of a nasogastric tube placed in the stomach. Nasogastric aspiration that are grossly bleeding confirm upper G. I. sources but a negative aspiration does not rule out. Upto 15-18% of patients with UGIB have non – bloody nasogastric aspirate. The amount of blood lost in the stool should also be monitored, estimated, and recorded and any change in color from melena to bright red blood should be noted as a possible sign of increased blood loss and hypovolemic shock. It is prudent that all patients with significant gastrointestinal bleeding be monitored in a pediatric intensive care unit for changes in vital signs and urine output until they are stable and bleeding has ceased.

Table 9: Treatment Goals

- Immediate assessment of severity and causes
- Establish and maintain the intravascular volume.
- Packed red blood cell transfusion: Hb > 8gm/dl
- FFP transfusion with INR > 1.5 , platelets transfusion if platelet < 50,000/mm^3.
- Determine source and site of blood loss
- Stop gastrointestinal bleeding.
- Lab Hemogram, LFT, BUN, Creatinine, PT, APTT, USG (abdomen or doppler), Endoscopy.

Resuscitation: hemodynamic stability. Treatment of Hypovolemic Shock secondary to GI Hemorrhage

- Oxygen is given to counter hypoxia due to acute blood loss
• Nasogastric aspiration is done to check ongoing losses
• Nasogastric lavage is done to clear the stomach for endoscopy, to define bleeding site, to check ongoing losses, and to prevent blood going down the intestine. This avoids the rise in blood urea nitrogen and prevents hepatic encephalopathy particularly if there is underlying liver disease.
• Establish adequate intravenous access.
• Rapidly infuse saline, lactated Ringer. Carefully monitor pulse, blood pressure and central venous pressure
• Hematocrit should be kept at around 24 and overtranfusion should be avoided.
• Monitor urine output and skin perfusion and orthostatic changes in pulse.
• Carefully record all fluids transfused and estimate and record all recognized fluids lost

**Calculating Transfusion Requirement**

• *Quantity of Packed red blood cells* - \[
\frac{[(70 \text{ml/kg} \times \text{weight in kg}) - (\text{desired hemoglobin} - \text{present hemoglobin})]}{23 \text{ gm hemoglobin per 100 dl of packed red blood cells}}\]

• *Rough guideline* - 5 ml of packed RBC/kg will raise the Hct. 3 points and Hb. 1gm/dl (5:3:1)

**Control of active bleeding( Variceal/nonvariceal)**

This can be done by:
• Control of ongoing bleeding
• Prevention of 1st bleeding
• Prevention of recurrent bleeding

**Control of Acute variceal bleeding can be by the following modalities:**
1. Pharmacotherapy
2. Balloon tamponade
3. Endotherapy
4. TIPS/surgery

1. Pharmacotherapy: The most widely used agents to stop variceal bleed are:
   a. Intravenous vasopressin
   b. Terlipressin
   c. Nitroglycerine
   d. Somatostatin
   e. Octreotide-synthetic analogue of somatostatin
a) Vasopressin: (VP) It is a potent non-selective vasoconstricctor. It lowers the portal pressure by causing splanchnic arterial vasoconstriction and reducing the splanchnic blood flow. It is given in a bolus of 1 unit per 3 Kg of body weight diluted with 2ml/Kg of 5% dextrose given over a period of 15-20 minutes. This agent causes bleeding control in 50%, vasospastic side effects in 50% and treatment had to be discontinued in 20% of cases. There is a high risk of myocardial infarction. To reduce this risk, nitroglycerine which is a vasodilator is used with it.

b) Nitroglycerine: This increases the local concentration of NO, so it produces vasodilation by decreasing the venous return and thereby reducing cardiac output. It also causes arterial vasodilatation.

c) Terlipressin: This is a synthetic analog of VP - the triglycyl-lysine VP (Terlipressin) which undergoes cleavage of glycy1 residues to allow a slow release of lysine-vasopressin. It acts by immediate intrinsic vasoconstriction. There is a limited experience in children of the use of this drug. However, it is found to be more effective in controlling bleeding (upto 79%) than vasopressin without any adverse side effects. It can be given as intravenous injections (2mg) every 4 hours till bleeding free interval of 24-48 hours is achieved.

d) Somatostatin: It has growth hormone inhibitory property. It acts by inhibiting release of several vasodilatory hormones such as glucagon. It induces selective splanchnic vasoconstriction. The recommended dosage is one to three bolus injections (250 microgram/bolus) during first hour of therapy followed by infusion of 250 microgram/hour of continuous infusion for 2-5 days. There is lower failure rate and complications in comparison to vasopressin but the disadvantage is its short half life.

e) Octreotide: It is a synthetic analog of somatostatin with half life of 90 minutes. In children the dose is 1 to 2 mcg/kg over 2 to 5 min, then 1 to 2 mcg/kg per hour for 5 days. Side effects are uncommon.

Effectiveness of somatostatin and octreotide for controlling acute variceal bleeding is comparable to that of vasopressin and EST.

Table 10: Mechanism of action of pharmaco and endotherapy

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Portal flow</th>
<th>Portal resistance</th>
<th>Portal pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vasoconstrictor (β₁ Blockers)</td>
<td>↓↓</td>
<td>↑</td>
<td>↓</td>
</tr>
<tr>
<td>Splanchnic vasoconstrictor</td>
<td>↓↓</td>
<td>-</td>
<td>↓↓</td>
</tr>
<tr>
<td>Venodilator</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>Endotherapy</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>TIPS / Shunt</td>
<td>↑</td>
<td>↓↓</td>
<td>↓↓</td>
</tr>
</tbody>
</table>

2) Balloon tamponade: The balloon tube tamponade may be life saving in patients with active variceal bleeding if emergency sclerotherapy or banding is unavailable or not technically possible.
because visibility is obscured. In patients with active bleeding, an endotracheal tube should be inserted to protect the airway before attempting to place the oesophageal balloon tube.

The Minnesota balloon tube has four lumens, one for gastric aspiration, two to inflate the gastric and oesophageal balloons, and one above the oesophageal balloon for suction of secretions to prevent aspiration. The tube is inserted through the mouth, and correct sitting within the stomach is checked by auscultation while injecting air through the gastric lumen. The gastric balloon is then inflated with 200 ml of air. Once fully inflated, the gastric balloon is pulled up against the oesophago gastric junction, compressing the submucosal varices. The tension is maintained by strapping a split tennis ball to the tube at the patient's mouth.

The oesophageal balloon is rarely required. The main complications are gastric and oesophageal ulceration, aspiration pneumonia, and oesophageal perforation. Continued bleeding during balloon tamponade indicates an incorrectly positioned tube or bleeding from another source. After resuscitation, and within 12 hours, the tube is removed and endoscopic treatment repeated.

3) Endotherapy: various methods are:

- EVL (Endoscopic Variceal Ligation): Using multiband ligator
- EST (Endoscopic Sclerotherapy): Injection into (intravariceal) or around (perivariceal) of sclerosant. Various sclerosants used are:
  - Absolute alcohol
  - Polidocanol
  - Sodium morrhuate
  - Sodium tetradecylsulfate
- For bleeding Gastric Varices:
  - Injection of Glue (N-butyl-2-Cyanoacrylate)

Sclerotherapy with sclerosant like ethanolamine oleate or band ligation is used for controlling bleed from oesophageal varices. Glue injection with N-butyl-2-cyanoacrylate is used to obliterate fundal varices. Bleeding from an ulcer is controlled using injection with adrenaline and recently hemoclips are also available for clipping at the site of vessel bleed at the base of the ulcer.

Table 11: Comparison of EVL and EST

<table>
<thead>
<tr>
<th></th>
<th>EVL</th>
<th>EST</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Indications</strong></td>
<td>Large Varices, difficult in small children</td>
<td>Any size</td>
</tr>
<tr>
<td><strong>Complications</strong></td>
<td>Less (4%)</td>
<td>More (25%)</td>
</tr>
<tr>
<td><strong>Rebleeding rate</strong></td>
<td>Low (4%)</td>
<td>More (25%)</td>
</tr>
<tr>
<td><strong>Sessions needed</strong></td>
<td>More (17%)</td>
<td>Less (10%)</td>
</tr>
<tr>
<td><strong>Recurrence of Varices</strong></td>
<td>Increase</td>
<td>IGV &amp; GOV</td>
</tr>
<tr>
<td><strong>Gastric Varices</strong></td>
<td>Increase</td>
<td>Increase</td>
</tr>
</tbody>
</table>
Secondary prophylaxis: Beta blockers has also been compared directly with EST on 10 RCT in adults. EST was associated with a lower rate of rebleeding but no survival advantage. The actual probability of rebleeding at 1 year was 33% in the EST group and 53% in propranolol. The benefits of EST in preventing rebleeding may be balanced by an increased rate of complications. (17)

Primary prophylaxis: Non selective beta blockers (propranolol and nadolol) are used for the primary prophylaxis. By Beta 1 blockage they reduce the cardiac output and thereby lower the portal pressure and by beta 2 blocking action they produce splanchnic vasoconstriction due to unopposed adrenergic activity and reduce portal pressure and variceal flow. With beta blocker therapy 25% reduction of sleeping pulse rate from baseline is often used as a surrogate marker of efficacy. ‘R’ they any trials of B Blockers in children. (27,23)

Complications of endotherapy (24)
- Fever
- Chest pain
- Dysphagia
- Superficial mucosal ulcerations (6-70%)
- Esophageal perforation
• Pulmonary complications
• Esophageal stricture

4) TIPS (Transjugular Intrahepatic Portosystemic Shunt):

Transjugular intrahepatic portosystemic shunt is the best procedure for patients whose bleeding is not controlled by endoscopy. It is effective only in portal hypertension of hepatic origin. The procedure is performed via the internal jugular vein under local anaesthesia with sedation. The hepatic vein is cannulated and a tract created through the liver parenchyma from the hepatic to the portal vein, with a needle under ultrasonographic and fluoroscopic guidance. The tract is dilated and an expandable metal stent inserted to create an intrahepatic portosystemic shunt. The success rate is excellent. Haemodynamic effects are similar to those found with surgical shunts, with a lower procedural morbidity and mortality.

**Control of non variceal bleeding:**
Treatment depends on the cause of the bleeding. Various treatment modalities are:
• H2 Receptor Antagonists/PPIs
• Vasoactive agents
• Endotherapy - Injection
  - Sclerosants
  - Adrenaline
  - Alcohol
  - Thrombin/ Fibrin glue
• Mechanical
  - Clips
  - Suturing
• Electro - thermal
  - Electro - thermal cautery
  - Laser Photo coagulation
  - Bipolar coagulation
  - Argon plasma coagulation

**Identifying the other sources of GI Hemorrhage:**
• Oesophago-gastro-duodenoscopy
• Colonoscopy
• Meckel’s scans
• Bleeding Scan
• Angiography
• Capsule Imaging
• Upper GI radiography
• Barium enema

**Endoscopy:** Oesphago-gastro-duodenoscopy or colonoscopy is very useful to detect the cause of GI bleed in most cases. With the availability of newer fibreoptic scopes for neonatal and pediatric procedures, it is now possible to identify the cause of bleeding in 85-90% of patients. Oesophageal gastroduodenoscopy provides information like oesophagitis, M.W. tears, varices, gastritis, ulcer, vascular malformations etc. Similarly, colonoscopy provides a lot of information in the colonic causes of bleeding like colitis, polyps, inflammatory bowel disease, ulcer, vascular malformations etc. Endoscopy is also therapeutic in most cases.

Colonic polyps are an important cause of bleeding in children from 2-7 years. Polypectomy using snare polypectomy with coagulation current is now a routinely used endotherapeutic procedure to remove colonic polyps. This should be preferably carried out with flexible scopes and one must evaluate the entire colon because of the possibility of more than one polyp in a patient and polyps higher up in the colon. Disadvantage with rigid scopes (mostly used by surgeons) is that they can evaluate only the rectum and sigmoid. Colonoscopy also allows access for direct coagulation of bleeding lesions using electrocoagulation, thermocoagulation, laser therapy or argon plasma coagulation.

**Meckel’s Scan:** Because technetium (Te) 99m pertechnetate is actively accumulated by cells in gastric mucosa, it can be used to identify the presence of ectopic gastric mucosa in a Meckel’s diverticulum or intestinal duplications. More than 90% of bleeding Meckel’s diverticula do contain gastric mucosa. A Meckel’s scan should be considered whenever significant lower gastrointestinal bleeding has occurred.

**Bleeding scan:** It’s a noninvasive study with a low radiation exposure. Tc sulfur or Tc pertechnetate labelled red blood cell scan are used. Tc sulfur red cell scan can detect site of bleeding even when the rate of bleeding is as low as 0.1ml/mt. However it’s half life is only 2 minutes. Tc pertechnetate labeled red blood cell scan can visualize the site of bleeding when the bleeding rate is 0.5 ml/mt or higher. However, the longer half life of the labeled red blood cells enables the images of the gastrointestinal system to continue for up to 24 hours. The Tc pertechnetate- labelled blood cell scan should be considered when a small intestinal source of bleeding is suspected. In most institutions, the Tc-pertechnetate labelled red blood cell scan is used to direct subsequent localized angiography. The nuclear scan can be readily repeated in patients with intermittent bleeding.

**Angiography:** The usefulness of angiography as a diagnostic test is usually limited to patients with active gastrointestinal bleeding and the rate of bleeding must be at least 0.5 ml/minute. When used in the appropriate situation, angiography has an accuracy of 50% to 75% but is associated with a significant complication rate of approximately 2%. The diagnostic yield of emergency arteriography is low because most hindgut lesion bleeding is intermittent and not from large calibre artery or vein. In a comparative study of emergency arteriography versus colonoscopy for diagnosis of severe ongoing
hematochezia, the diagnostic yield from colonic lesion by emergency arteriography was $\approx 10\%$ and for colonoscopy was $80\%$.

**UGI Radiology:** Contrast studies of the upper gastrointestinal tract have taken a secondary role in the evaluation of patients since the introduction of endoscopy. Radiographic studies are particularly useful in defining anatomic deficits such as esophageal strictures, malrotation of the bowel, or deep ulcerations.

**Barium Enema:** Barium enema is effective in identifying the anatomic location of the large bowel and is particularly important in neonates and infants presenting with signs of obstruction associated with gastrointestinal bleeding. In these patients, malrotation with secondary intestinal volvulus must be rapidly identified. Intussusception, a common problem in infants particularly between the ages of 6 months and 24 months, can be diagnosed and in many cases treated by barium enema. The barium enema is also effective in identifying the presence of polyps.

**Surgery**

- Surgery of Meckel’s Divertculum, Duplication of small bowel, Hirschsprung’s disease
- Portosystemic shunt, Esophageal transection, TIPS, Liver transplantation
  (Rarely necessary in the upper GI hemorrhage refractory to medical, endoscopic and radiologic intervention)
- In lower GI hemorrhage, hemicolecetomy or subtotal colectomy is occasionally required.
Approach to a case of Upper GI Bleed/Massive Lower GI Bleed
Hemodynamically stable Patients

**OesophagoGastro duodenoscopy**

- Diagnostic
  - Varices
    - Band Ligation
    - Sclerotherapy
    - Glue injection (fundal varices)
  - Ulcer
    - Adrenalin injection
    - Clipping
    - Suturing
  - Vascular malformation
    - Electro coagulation
    - Oesophagitis
    - M.W. tears
    - Gastritis etc.
  - Others
  - Colonoscopy/Enteroscopy
    - Negative
    - Positive
      - Bleeding Scan
        - Meckel’s
          - Negative
          - Positive
        - Capsule Endoscopy
        - Surgery
      - Specific therapy
      - Angiography
        - Embolisation
Approach to a case of Lower GI Bleed
(Hemodynamically Stable Patient)

Colonoscopy

Diagnostic

Specific Therapy

Nondiagnostic

Negative

Positive

UGI Tract Evaluation

Meckel’s Scan

Bleeding Scan

CT Scan Angiography

Positive

Negative

Positive

Surgery

Angiography

Surgery +/- Embolization
Key learning points

- **Hematemesis** is vomiting of bright red or coffee-ground-like material and suggests that the source of bleeding is located above the ligament of Treitz.
- **Melena**, the passing of dark black, tarry stools, occurs in patients bleeding from a site located above the ileocecal valve.
- **Hematochezia**, the passage of bright red or maroon blood from the rectum, is most commonly associated with a colonic source of bleeding.
- **Upper GI bleeding** is defined as bleeding from a source in the esophagus, stomach or duodenum above the ampulla of vater. **Middle GI bleeding** is defined as small intestinal bleeding from ampulla of vater to terminal ileum and **lower GI bleeding** is colonic bleeding.
- Bright red blood may be mimicked by red colored candies, juices, and foods.
- Portal hypertension is defined as Hepatic Venous Pressure gradient (HVPG) > 5 mm of Hg. It is the commonest cause of GI bleed in children in India.
- Extrahepatic portal venous thrombosis (EHPVO) is the most common cause GI Bleed in children in India.
- **Oesophageal varices** can be classified as small, medium and large (<5mm, 5-10mm and >10mm).
- **Gastric varices** are found most commonly with splenic vein thrombosis or after EST of oesophageal varices.
- UGI bleeding is massive and recurrent in EHPVO but risk of rebleeding after major episode is less.
- UGI Endoscopic therapy is recommended in any patient presenting with documented UGI bleed & esophageal varices.
- Hematocrit should be kept at around 24 and overtransfusion should be avoided.
- Effectiveness of somatostatin and octreotide for controlling acute variceal bleeding is comparable to that of vasopressin and Endoscopic Sclerotherapy (EST).
- The balloon tube tamponade may be life saving in patients with active variceal bleeding if emergency sclerotherapy or banding is unavailable.
- Endoscopic variceal ligation (EVL) is recommended modality of choice, although EST may be used if EVL is technically difficult as in small babies. Choice may depend on local expertise and technical considerations.
- For secondary prophylaxis, EVL is effective in children & may be superior to EST when feasible.
- Transjugular intrahepatic portosystemic shunt (TIPS) is the best procedure for patients whose bleeding is not controlled by endoscopy.
References


