
GASTRO-INTESTINAL BLEEDING IN CHILDREN AND ADOLESCENTS

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Introduction

There are many causes of gastro-intestinal bleeding in children. Some span the whole paediatric age range, while others reflect congenital malformations and present with bleeding in early childhood. Advances in paediatric endoscopy have allowed us to determine the cause in most children who present with GI bleeding, provided the clinical evaluation and management are timely.

1. Determine the severity of the bleeding
2. Determine the site of bleeding
 - Upper GI (proximal to the ligament of Treitz, the 2nd part of the duodenum) or
 - Lower GI (distal to the ligament of Treitz).
 - Exclude bleeding that is not gastro-intestinal e.g. epistaxis, maternal blood, dental work, haemoptysis. Substances such as iron, bismuth, beets, spinach and blueberries can mimic melaena.
 - Colour of the bleeding.
 - Haematemesis (vomited blood) can be either red or the colour of coffee grounds. It is most commonly associated with an upper gastro-intestinal bleed. Bright red blood suggests active bleeding which has not come into contact with the gastric acid secretions. Coffee grounds result when the gastric secretions have the chance to interact with blood.
 - Melaena (black tarry stools) generally indicates significant blood loss proximal to the ileocaecal valve. The black colour results from bacterial break down of haemoglobin.
 - Haematochezia (bright red blood per rectum) generally indicates a colonic site of bleeding. Occasionally this type of bleeding may originate from the small intestine as a result of a fast gut transit time.
3. Consider other factors:
 - Age is a major determinant for determining the likely cause (see Tables)
 - The presence or absence of significant pain is important
 - Signs of a surgical abnormality
 - The presence or absence of diarrhoea.

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Differential Diagnosis

Upper Gastro-Intestinal Bleeding.

Gastritis and duodenitis occurs in all age groups. Gastric ulcer is more common in early childhood while duodenal ulcers are, in general, more common in the older age groups.

Oesophageal varices should be considered if there is any evidence of chronic liver disease and this can occur from as early as a few months of life but usually from after 12 months of age.

Consider a Mallory-Weiss tear in any patient with protracted vomiting.

Rectal bleeding.

In infants, consider anal fissures, swallowed maternal blood, necrotising enterocolitis, mid gut volvulus, intussusception, infectious diarrhoea and milk protein allergy (allergic colitis). Allergic colitis is seen in infants up to four months of age exposed to cows' milk or Soya milk protein formula. These infants are generally well and have normal slightly mucousy loose stool streaked with blood. Bleeding from juvenile polyps may occur in the first year of life but usually presents from 1 to 12 years. Nodular lymphoid hyperplasia may also cause lower GI bleeding in the first years of life. Ulcerative colitis can present in the first year.

Bright red blood per rectum (with otherwise normal stool), or spots of red blood on the toilet paper implies bleeding from anal or rectal lesions. Possibilities include anal fissures, juvenile polyp or allergic proctitis.

Bright red blood mixed with mucus and associated with diarrhoea, abdominal cramps and tenesmus suggests a colitis. The cause could be:

Infection: Salmonella, Campylobacter, Shigella, C-difficile as well as entero-invasive E.coli and occasionally Yersinia. In patients who are immuno-compromised, consider CMV, HSV and candida as well.

Pseudomembranous colitis

Ulcerative colitis.

Painless rectal bleeding suggests a Meckel's diverticulum, duplication, polyp or angio dysplasia. Rarely, painless rectal bleeding may be due to a deep ulcer in the right colon or terminal ileum from Crohn's disease. Copious amounts of red blood are seen with Meckel's diverticulum or a colonic arteriovenous malformation.

Massive G-I bleeding can be due to oesophageal varices, peptic ulcers, Meckel's, AV malformation.

Abdominal pain. If present, a surgical abnormality should be ruled out.

Peritonitis could indicate a perforated ulcer, necrotising enterocolitis in an infant or a perforated viscus secondary to Crohn's disease in an older patient.

Pain accompanies the vasculitis of Henoch-Schonlein purpura and sometimes gastro-intestinal bleeding may precede the characteristic rash by several days.

If there are signs of bowel obstruction, exclude intussusception. While 'redcurrent jelly' stools are a classic presentation, this sign is not always present.

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History

Exclude chronic lung disease, renal disease, bleeding disorders or liver disease.

Cystic fibrosis patients are at risk of oesophageal varices due to biliary cirrhosis, and vitamin K deficiency may be a factor secondary to fat soluble vitamin absorption.

Medications including NSAIDs and prior antibiotic exposure.

Overseas travel.

Family medical history: peptic ulcer disease, bleeding disorders, inflammatory bowel disease, polyposis syndrome or early colon cancer. Other sick contacts may indicate an infectious cause e.g. contaminated food.

Physical Examination

Look for:

Chronic constipation. Possible anal fissure. Rectal exam to exclude faecal retention.

If bleeding is severe, look for tachycardia and orthostatic hypertension (a rise in the pulse rate by 20 beats per minute or a fall in the systolic blood pressure of more than 10mmHg indicates significant volume depletion, usually > 20%).

Cutaneous haemangiomas may indicate the presence of GI mucosal haemangiomas.

Pigmentation of the lips and buccal mucosa may suggest Peutz-Jeghers syndrome.

Purpura on the buttocks and lower extremities are characteristic of HSP.

Portal hypertension. Signs include hepatosplenomegaly, as well as other stigmata of chronic liver disease (clubbing, spider naevi, jaundice and ascites).

Laboratory Tests

FBC. A recent bleed may not alter the haemoglobin or haematocrit but the MCV can be low in chronic low grade bleeding. Raised eosinophils may signify an allergic colitis.

ESR. An elevated ESR may indicate inflammatory bowel disease.

Coagulation profile to rule out a bleeding disorder.

Liver function tests if there are signs of portal hypertension or chronic liver disease.

If there are loose stools, stool cultures and a C-difficile toxin assay.

Renal function tests. A high urea may be a clue for haemolytic uraemic syndrome or may indicate the presence of dehydration.

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Imaging Studies

Upper GI bleeding. Contrast studies should not be the initial study to rule out oesophagitis, gastritis or peptic ulcers because of the lack of sensitivity. Endoscopy is far more sensitive. Contrast studies may be indicated in patients with dysphagia or odynophagia. Ultrasound should be requested if there is evidence of liver disease or splenomegoly.

Haematochezia. Contrast studies should not be the initial evaluation. Flexible endoscopy is better. The exception would be suspected intussusception, where ultrasound should be requested (and if confirmed, an enema for reduction).

Massive painless bleeding. A Meckel scan is the procedure of choice. False negative results have been reported because of insufficient gastric tissue mass, down stream washout of isotope, impaired blood supply or suboptimal techniques. Repeat Meckel scans may therefore be necessary to identify the type of gastric tissue.

Obscure bleeding in the upper or lower GI tract. Technetium labelled RBC scans may aid localisation, but require active bleeding of $> 0.5\text{ml/min}$.

Endoscopy

Fibreoptic endoscopy and biopsy has increased the rate of positive diagnosis. The yield decreases if endoscopy is delayed, so it is important that endoscopy occurs promptly. Preparation of the patient is critically important. In emergency situations where bleeding is severe, resuscitation of the patient is paramount. Endoscopy should not be performed hastily if the patient is unstable. For colonoscopy, the patient requires adequate bowel preparation and this varies with the age and compliance of the child. Urgent endoscopy does allow prompt diagnosis and the ability to perform therapeutic interventions such as sclerotherapy.

For patients with melaena or haematemesis, upper endoscopy is usually done first. Patients with haematochezia should at least receive a flexible sigmoidoscopy. Many would recommend colonoscopy as the first examination, particularly if the procedure is being performed under general anaesthetic. A significant number of polyps are found proximally, beyond the reach of rigid sigmoidoscopes.

Treatment

If there is significant bleeding, re-establish blood volume and O₂ carrying capacity (rapid infusion of normal saline followed if appropriate by red cells)

Determine the site of blood loss

Known oesophageal varices and severe bleeding.

Octreotide infusion, starting with 1mcg/kg IV bolus followed by a continuous infusion of 1 mcg/kg/hour, increasing every eight hours if there is no reduction in the bleeding up to 4-5 mcg/kg/hour as a continuous infusion. When there is no active bleeding after 24 hours the dose could be halved every 12 hours. Side effects of Octreotide include nausea, abdominal cramps, diarrhoea, bradycardia and hyperglycemia which usually resolve spontaneously.

As a last resort, a Sengstaken tube. This has significant complications including aspiration and oesophageal rupture.

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Sclerotherapy can be successful in obliterating oesophageal varices after multiple sessions in up to 92% of paediatric patients. It is used in infants.

Oesophageal band ligation has also been successful, with fewer complications compared with sclerotherapy.

Peptic disease such as gastritis or oesophagitis.

H₂ antagonists such as Ranitidine (2-4mg/kg/dose, maximum 150 mg/dose, twice a day).

Proton pump inhibitors such as Omeprazole (0.7-1.4mg/kg/dose, max 40mg, once a day) are indicated in selected cases. A liquid preparation is available where doses do not relate to capsule sizes.

Sucralfate (250mg q.i.d. for children <2 years old, 0.5-1g q.i.d. for children over 2 years) as a barrier agent is used for peptic ulcers and after sclerotherapy. Doses should be given on an empty stomach (1 hour before, or two hours after food). Tablets can be crushed and dispersed in water.

Liquid antacids such as Gaviscon and Mylanta P (0.5-1ml/kg/dose, maximum 20 ml per dose every 4 hours) are a useful adjunct for breakthrough symptoms in older children. These should be given after meals and two hours apart from other medicines.

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TABLE 1 DIFFERENTIAL DIAGNOSIS NEONATES:

Haematemesis and Melaena	Haematochezia
Swallowed maternal blood. Stress ulcers. Gastritis duplication. Vascular malformation. Vitamin K deficiency. Haemophilia. Maternal idiopathic thrombocytopenic purpura. Maternal NSAIDS.	Swallowed maternal blood. Poor protein intolerance. Infectious colitis. Necrotising enterocolitis. Hirschsprung's disease with enterocolitis. Duplication. Vascular malformation. Vitamin K deficiency. Haemophilia. Maternal idiopathic thrombocytopenic purpura. Maternal NSAIDS use.

TABLE 2 DIFFERENTIAL DIAGNOSIS IN INFANTS:

Haematemesis, Melaena	Haematochezia
Oesophagitis. Gastritis.	Anal fissures. Intussusception. Infectious colitis. Milk protein intolerance. Meckel's diverticulum. Duplication. Vascular malformation.

TABLE 3 DIFFERENTIAL DIAGNOSIS IN CHILDREN:

Haematemesis, Melaena	Haematochezia
Oesophagitis. Gastritis. Peptic ulcer disease. Mallory-Weiss tears. Oesophageal varices.	Anal fissure. Infectious colitis. Polyps. Lymphoid nodular hyperplasia. Inflammatory bowel disease. Henoch-Schonlein purpura. Intussusception. Meckel's diverticulum. Haemolytic uraemic syndrome.

TABLE 4 DIFFERENTIAL DIAGNOSIS ADOLESCENTS:

Haematemesis, Melaena	Haematochezia
Oesophagitis. Gastritis. Peptic ulcer disease. Mallory-Weiss tears. Oesophageal varices.	Infectious colitis. Inflammatory bowel disease. Anal fissures. Polyps.

TABLE 5 THERAPY:

Supportive Care	Specific Care	Endoscopic Therapy
Blood products. Pressors.	Barrier agents - (Sucralfate). H2 antagonists -(Ranitidine). Proton pump inhibitors (Omeprazole). Octreotide.	Variceal injection or ligation. Polypectomy.