Haematemesis and melaena

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Abstract

Upper gastrointestinal haemorrhage is common and carries a significant mortality. Peptic ulcer disease remains the most common aetiology, but varices are an important cause. The patient's history, physiology and blood results guide the timing of endoscopy and can disclose underlying liver disease. Resuscitation and risk assessment scoring are the main priorities in acute presentations. Patients who are haemodynamically unstable or have suspected bleeding varices should undergo endoscopy immediately after resuscitation. Endoscopy allows diagnosis and treatment as well as prognostic information. Peptic ulcers that are bleeding or show stigmata of recent haemorrhage are treated with dual endoscopic therapy and an intravenous proton pump inhibitor for 72 hours. Oesophageal varices are treated with endoscopic variceal band ligation and terlipressin. Gastric varices are treated with thrombin, glue injection or transjugular intrahepatic portosystemic shunt placement. Cirrhotic patients with acute upper gastrointestinal bleeding should also be given broad-spectrum antibiotics.

Keywords Gastrointestinal haemorrhage; haematemesis; melaena; oesophageal and gastric varices; peptic ulcer haemorrhage; proton pump inhibitors; terlipressin

Key points

- Rapid, focused clinical assessment and resuscitation is critical in all patients with upper gastrointestinal bleeding
- Risk scores are useful in identifying patients at higher risk of mortality

Peptic ulcers:

- Patients who have ulcers with high-risk stigmata or active bleeding should be given dual endoscopic therapy, followed by intravenous proton pump inhibitor infusion for 72 hours
- Peptic ulcers are usually caused by *Helicobacter pylori* and/or non-steroidal anti-inflammatory drugs (NSAIDs). Treating *H. pylori* and minimizing NSAIDs are key in allowing peptic ulcers to heal and reducing rebleeding
- To exclude malignancy, patients with a gastric ulcer should undergo repeat endoscopy with biopsies in the event they fail to heal
- Rebleeding from ulcers may require mesenteric angiography and embolization, with surgical management if this is unsuccessful

Varices:

- Evidence of chronic liver disease means that bleeding may be variceal in origin. These patients benefit from broad-spectrum antibiotics and consideration of terlipressin before endoscopy. They are likely to require urgent endoscopic intervention with variceal band ligation for oesophageal varices (or thrombin/glue injection for gastric varices)
- Rebleeding is managed with repeat endoscopy, and consideration of Sengstaken–Blakemore tube insertion and transjugular intrahepatic portosystemic shunting

Introduction

Acute upper gastrointestinal (UGI) haemorrhage is the most common GI emergency, with an estimated incidence of 50–190 per 100,000 population per year. Patients admitted to hospital with UGI bleeding have a 7% mortality rate. Patients who develop acute UGI haemorrhage during hospitalization for another condition have a substantially higher mortality (26%). The overall mortality has been largely unchanged over the last few decades, despite evidence-based improvements in management. This may be because of the increasing prevalence of liver disease and variceal haemorrhage, and the increasing age of the population, with a consequent effect on associated comorbidity.

In the UK, the National Institute for Health and Care (formerly Clinical) Excellence (NICE) has produced a guideline for the management of acute UGI bleeding.[1,2]

Definitions

Haematemesis (vomiting blood) and melaena (black, tarry liquid stool representing altered blood) are the typical presenting symptoms of UGI bleeding. Bleeding from the oesophagus, stomach and duodenum is classed as 'upper gastrointestinal'; the demarcation point is the ligament of Treitz/jejunum. Melaena (but not haematemesis) occasionally occurs with bleeding from a jejunal, ileal or right-sided colonic source. Fresh red haematemesis, as opposed to more altered 'coffee ground' vomitus, is suggestive of more significant, active bleeding.

Causes

Peptic ulcer disease is the most frequent cause of major, life-threatening acute GI bleeding, accounting for 36% of cases. Significant haemorrhage results from erosion of an underlying artery. Branches of the gastroduodenal (posterior wall duodenal ulcers) and

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left gastric (high, lesser curve gastric ulcers) arteries are typically involved in the most severe bleeds. For further discussion of peptic ulcers and *Helicobacter pylori*, see *MEDICINE* 2015; **43**(4):215–222.

Variceal haemorrhage is less common than peptic ulcer bleeding (11% of cases), but associated with a greater mortality (15% versus 9% at 30 days). Mortality is directly related to severity of the underlying liver disease. The portal vein pressure is usually greater than 12 mmHg, as a consequence of cirrhosis. Varices can also form in non-cirrhotic states, such as splenic vein occlusion caused by pancreatitis. Gastro-oesophageal varices are present in 50% of patients with cirrhosis; 8% of cirrhotic patients without varices develop them within a year. Similarly, patients with small varices progress to having large varices at a rate of 8% per year. For a more detailed discussion of portal hypertension and varices, see *MEDICINE* 2015; **43**(11):669–673.

Most research into GI bleeding has focused on peptic ulcer and variceal haemorrhage, as these are the most frequent causes of life-threatening bleeding. Other causes of bleeding often settle with conservative management or are rare:

- A **Mallory–Weiss tear** is a superficial mucosal tear at the gastro-oesophageal junction, related to vomiting; endoscopic therapy is occasionally required.
- **Oesophagitis** can present with 'coffee ground' vomiting and can occur in the context of systemic illness or drug toxicity. Proton pump inhibitors (PPIs) are the mainstay of treatment.
- **Gastritis, duodenitis and erosions** are usually caused by non-steroidal anti-inflammatory drugs (NSAIDs), *H. pylori* or both. PPI therapy, stopping NSAIDs and *H. pylori* eradication are recommended.
- **Arteriovenous malformations** (AVMs) are often an incidental finding, without anaemia or bleeding, and in this situation can be safely ignored. Large or multiple AVMs can cause chronic GI blood loss, presenting with iron deficiency anaemia, particularly in patients taking long-term anticoagulation. They occasionally cause significant acute haemorrhage. Hereditary haemorrhagic telangiectasia (Osler–Weber–Rendu syndrome) is characterized by AVMs distributed throughout the GI tract. If the AVM is thought to be the source of bleeding, superficial thermal ablation using argon plasma coagulation (APC) is effective.
- **Gastric antral vascular ectasia** (GAVE, 'watermelon stomach') has a characteristic appearance of red, linear streaks in the stomach radiating longitudinally from the pylorus. GAVE usually presents with iron deficiency but occasionally as an acute bleed. Treatment involves multiple APC sessions.
- **Dieulafoy's lesion** results from erosion of a superficial submucosal artery, usually within 6 cm of the gastro-oesophageal junction in the fundus of the stomach, but occasionally in other parts of the UGI tract. Bleeding can be recurrent and significant. The lesion is not associated with ulceration, and the diagnosis is frequently made only when active bleeding is present. Endoscopic therapy is usually required.
- **Oesophagogastric malignancy** (carcinoma of the stomach or oesophagus, GI stromal tumour (GIST)) occasionally presents as an acute UGI bleed. Tumours typically ooze blood, but erosion of an artery can cause major bleeding. APC can be helpful to reduce blood loss. Ulcerated GISTs causing bleeding often require surgical excision.
- **Aortoduodenal fistula** should be suspected in all patients with previous aortic graft surgery. Bleeding typically occurs from 2 weeks to 10 years after initial surgery, and can present with one or more minor 'herald' bleeds before a major haemorrhage. If suspected, computed tomography (CT) and endoscopy are the first-line investigations. Patients should be referred to a vascular surgical unit immediately after initial resuscitation.
- Melaena can occasionally be caused by **small bowel or right-sided colonic lesions.** If initial endoscopy does not identify a bleeding source, colonoscopy, CT angiography or capsule endoscopy may be indicated. Consider **Meckel's diverticulum** in younger patients.

Initial assessment

Rapid, focused initial assessment serves several purposes. Clues in the history can suggest the source of bleeding and potentially avoidable adverse sequelae. Clinical examination findings help to guide fluid resuscitation, and provide information regarding the likelihood of chronic liver disease (and variceal bleeding). Blood tests guide resuscitation and blood product administration. Risk scores are designed to provide prognostic information and guide timing of endoscopy, using data from the history, examination, laboratory results and endoscopic findings.

History

Shocked or obtunded patients require rapid assessment and resuscitation. The history should include:

- a description of the haematemesis and/or melaena, including colour, volume, timing and whether it was preceded by non-bloody vomitus
- syncope/presyncopal symptoms
- abdominal pain, dyspepsia, acid reflux symptoms, dysphagia and weight loss
- medication – aspirin and other NSAIDs, antiplatelet drugs (e.g. clopidogrel) and anticoagulants (e.g. warfarin) worsen bleeding. Iron supplementation typically turns stools black but not usually loose or 'tarry'
- previous GI bleeding
- liver disease – cirrhosis with consequent portal hypertension increases the likelihood that bleeding has arisen from varices. Non-variceal bleeds in cirrhotic patients can be worsened by coagulopathy and thrombocytopenia
- underlying medical conditions – particularly cardiovascular and respiratory disease, which can decompensate if the patient is shocked and/or anaemic. Renal disease and cardiac failure increase the risk of volume overload during fluid resuscitation.

Examination

Assess fluid balance including blood pressure (BP), preferably erect and supine, and heart rate. BP and pulse correlate with the degree of shock. Younger patients with good physiological reserve often maintain normal cardiovascular parameters despite significant blood loss.

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