Acute upper gastrointestinal haemorrhage

Heather D Lafferty

John Morris

Abstract

Acute upper gastrointestinal (GI) haemorrhage is one of the most common GI emergencies. Bleeding from peptic ulcer disease and variceal haemorrhage are the most frequently observed causes of haemorrhage. Mortality is increased in the elderly, patients with liver disease and those who present with shock. The use of risk stratification scores such as the Glasgow Blatchford or Rockall scores is recommended in all patients to predict the need for endoscopic intervention or death. Appropriate resuscitation of the patient is the first priority.

Early access to upper GI endoscopy for diagnosis and therapy is essential for patients with acute bleeding. Peptic ulcers with active bleeding should undergo dual endoscopic therapy followed by 72-hour infusion of intravenous proton pump inhibitor. For oesophageal varices that are bleeding, band ligation therapy is the endoscopic treatment of choice. Patients with chronic liver disease should be given prophylactic antibiotic therapy following endoscopy to reduce the incidence of complications (sepsis, re-bleeding) and reduce risk of death. Transjugular intrahepatic portosystemic shunt is an appropriate rescue therapy when endoscopic techniques fail or re-bleeding occurs.

New endoscopic haemostatic methods, including Hemospray®, a novel powder haemostat, offer the prospect of improved haemostasis but need further evaluation.

Keywords blood transfusion; endoscopy; oesophageal and gastric varices; gastrointestinal; gastrointestinal haemorrhage; peptic ulcer

What's new?

- In the most recent large UK audit, acute upper gastrointestinal bleeding remained a common emergency with a significant mortality
- More evidence is available favouring restrictive blood transfusion and highlighting the increased risk of rebleeding in transfusing to a target above 7–9 g/dl
- Evidence favours combined endoscopic therapy with injection and mechanical modalities for peptic ulcer bleeding
- New endoscopic modalities such as Hemospray and pharmacological treatments including tranexamic acid show promise and require further investigation by randomized controlled trial

Introduction

Acute upper gastrointestinal (GI) haemorrhage is a common GI emergency requiring admission to hospital. It accounts for up to 9000 hospital deaths per year in the UK.[1]

Mortality: in a UK audit in 2007,[1] overall mortality was 10% but this was substantially higher (26%) in those who were already inpatients at the time of haemorrhage compared to those admitted with bleeding (6.8%). This reflects the higher burden of co-morbidity and increased age in this group. Patients with the highest risk of death from GI bleeding include those with severe liver disease, the elderly, and those presenting with shock.

Heather D Lafferty *MBChB MRCP is a Specialty Trainee in Gastroenterology at Glasgow Royal Infirmary, UK. Competing interests: none declared.*

John Morris *MB FRCP is Consultant Gastroenterologist, Clinical Lead for Gastroenterology at Glasgow Royal Infirmary and Director of the West of Scotland Endoscopy Training Centre, UK. Competing interests: Dr Morris has received speaker and consulting fees from Cook Medical, Ferring, Falk, Vifor, MSD, Abvie and Takeda Pharmaceuticals.*

Aetiology: among those presenting with upper GI bleeding, peptic ulcer disease accounted for 36% of cases and variceal haemorrhage for 11%. Other recognized causes include oesophageal or gastric malignancy, oesophagitis, gastritis, duodenitis, Mallory–Weiss tear, arteriovenous malformation, and Dieulafoy lesion (a large tortuous arteriole that erodes and bleeds). Risk factors for upper GI bleeding include the use of non-steroidal anti-inflammatory drugs and *Helicobacter pylori* infection, as well as treatment with aspirin, clopidogrel, warfarin and other antiplatelet and anticoagulant agents.

Resuscitation and risk stratification

The aims of management in acute upper GI bleeding are to assess and resuscitate the patient, and stratify risk, followed by upper GI endoscopy for diagnosis and definitive treatment.

Patients should be assessed for evidence of active bleeding, shock, and underlying pathology such as liver disease.

Resuscitation

Intravenous fluid, usually isotonic crystalloid is administered to re-expand circulating volume. Red cell transfusion is usually required when around 30% of circulating volume has been lost (1500–2000 ml). Clinical features will include reduced systolic and diastolic blood pressure, tachycardia (in the region of 120 beats per minute or more), increased respiratory rate, altered cognition, agitation and, in severe cases, drowsiness.[2] In the case of massive haemorrhage, local protocols should exist for rapidly obtaining and administering blood products.

Blood transfusion: in a randomized controlled trial published in 2013,[3] restrictive transfusion (target haemoglobin (Hb) 7–9 g/dl) was associated with better outcomes with less re-bleeding and lower mortality than patients who were transfused more liberally (target Hb 9–11 g/dl). This effect was most marked in patients with bleeding due to portal hypertension, in whom transfusion has been shown to raise portal pressure further, leading to increased bleeding. A target Hb of 7 g/dl following transfusion has therefore been suggested, but it is important to assess each patient individually as other factors, such as cardiovascular disease, may affect the decision to transfuse. These results were replicated in a UK observational study of early

transfusion (within 12 hours), in which re-bleeding rates were higher in the early transfusion group especially in those with Hb above 8 g/dl[4]

Correction of anticoagulation: in some cases it is appropriate to administer other blood products targeted at the reversal of iatrogenic or pathologic anticoagulation. Platelets should be administered where there is active bleeding and platelet count of less than 50×10^9/litre. Fresh frozen plasma should be given to patients with fibrinogen less than 1 g/l or prothrombin time/activated partial thromboplastin time ratio more than 1.5. In patients who have been taking warfarin and who are actively bleeding, dried prothrombin complex may be administered.[2,5]

Risk stratification

Patients presenting with GI bleeding should be assessed for risk of further bleeding, need for early endoscopic intervention or death. Two well-validated scoring systems in use are the Rockall score and the Glasgow Blatchford score (GBS).[6,7]

Rockall score: the Rockall pre-endoscopy and full Rockall (post-endoscopy) scores use factors identified from multivariate analysis that predict outcome (Table 1). Patients with full Rockall scores of 0–2 have a rate of re-bleeding of less than 5% and minimal mortality (0–0.9%) whereas those with a full score of 8–11 have a mortality of up to 40%.[6]

Glasgow Blatchford score: the Glasgow Blatchford score (GBS) was designed to predict requirement for endoscopic treatment and uses only clinical and laboratory variables that are readily measured and available soon after initial hospital assessment (Table 2).[7] A low GBS identifies those at low risk of further bleeding or death and can identify those who may be safe to be discharged.[8] The GBS also correctly identifies patients with variceal haemorrhage as being at high risk.[9]

Comparison of the two scoring systems has shown superiority of the GBS and full Rockall score to the pre-endoscopic component of the Rockall score in predicting the need for endoscopic intervention and re-bleeding; the GBS was superior to full Rockall score in predicting the need for blood transfusion.[10]

Endoscopy

The timing of endoscopy depends on the clinical condition of the patient as well as availability of local services. Patients who are unstable and actively bleeding after initial resuscitation should undergo endoscopy. In most patients this should be done within the first 24 hours after admission,[2,5] although some are at sufficiently low risk to be suitable for discharge and a scheduled outpatient endoscopy.[5,8]

Endoscopic treatment

Peptic ulcer disease

Endoscopic features of bleeding duodenal and gastric ulcers can be classified by the Forrest classification system[11] (Table 3). This can also be used to determine which lesions have a higher benefit from endoscopic therapy. Endotherapy is appropriate in lesions associated with acute haemorrhage (Forrest Ia Ib) (Figure 1) or non-bleeding with a visible vessel (IIa). Controversy still exists about the appropriateness of treating ulcers with visible adherent clot overlying the ulcer (IIb).

Combination (dual) therapy with injection solution (adrenaline (epinephrine) 1:10000 solution ≥13 ml injected around the lesion)[2] and a mechanical method (heater probe, clip) are optimal. The latter has the effect of tamponade around the bleeding vessel, complementing the potent vasoconstrictive effect of adrenaline. The use of two modalities has been shown to be significantly superior to the use of injection alone in preventing re-bleeding (Figure 2).[2,5]

Variceal haemorrhage: varices in the GI tract develop as a result of portal hypertension, usually due to chronic liver disease. Eighty per cent of bleeding varices are located in the oesophagus (Figure 3). Gastric varices can be classified according to site though are less common. Oesophageal varices are treated by endoscopic band ligation (Figure 4), gastric varices by injection with cyanoacrylate glue or thrombin.[2,5]

Other bleeding lesions and endoscopic modalities: bleeding lesions other than peptic ulcer and varices include arteriovenous malformations (AVM). These can treated with dual endotherapy in the same way as bleeding from ulcer disease (Figures 5 and 6).

Rockall score[6]

Variable		Score 1	Score 2	Score 3
Age	<60	60–79	≥80	
Shock	No shock SBP>100, pulse <100	Tachycardia SBP >100, pulse >100	Hypotension SBP <100, pulse >100	
Co-morbidity	No major co-morbidity		Ischaemic heart disease, cardiac failure, any major co-morbidity	Renal failure, liver failure, disseminated malignancy
Diagnosis	Mallory–Weiss tear, no lesion and no SRH	All other diagnoses	Malignancy of upper GI tract	
Stigmata of recent haemorrhage (SRH)	None or dark spot		Blood in upper GI tract, adherent clot, visible or spurting vessel	

Table 1