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Managing acute upper gastrointestinal bleeding in the acute assessment unit

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Acute upper gastrointestinal bleeding (UGIB) accounts for approximately 50,000–70,000 hospital admissions annually in the UK.[1–3] It is twice as common in men than in women and increases with age.[2] Although most patients in the UK are managed in hospital, the mortality rate remains around 10%,[1] highest in the elderly and in patients with significant comorbidity.

Peptic ulcer disease and oesophagogastric varices secondary to chronic liver disease are the commonest causes of severe haemorrhage in acute UGIB (Table 1). Other pathologies usually cause self-limiting bleeding.

Patients present with haematemesis (vomiting of either fresh blood or 'coffee-ground') and/or melaena (passage of black, tarry stools). As blood has a cathartic effect, the latter tends to appear within 2–6 hours of the acute bleed and may persist for up to 48 hours in the absence of further haemorrhage. Haematochezia (passage of minimally altered blood) occurs in approximately 30% of patients with massive UGIB. Acute blood loss can lead to hypovolaemic shock (weakness, sweating, postural dizziness, tachycardia and hypotension). All patients with severe bleeding are at risk of developing acute renal failure, whilst those with coexistent cardiovascular disease carry the additional risk of myocardial infarction and/or stroke.

When a patient is admitted to the acute admissions unit (AAU) with a significant GI bleed, the on-call gastroenterologist should be contacted and GI surgical colleagues informed. It is imperative to contact them early before an endoscopy or operation might be necessary, rather than when it becomes an inevitable intervention.

The priorities in the early management of the patient are:

- resuscitation and initial medical management
- assessment of severity of the bleed
- identifying the site of bleeding
- establishing haemostasis.

Resuscitation and initial medical management

Resuscitation

The principles of airway, breathing and circulation should be observed.

Intravenous (iv) access should be secured with the placement of two large (eg 14–16G) cannulae. Initially, central venous cannulation should be reserved for patients in whom peripheral access is limited. In the presence of hypovolaemic shock (blood pressure <100 mmHg, pulse >100 bpm) 1–2 litre crystalloid (eg 0.9% saline) or 0.5–1 litre colloid (eg Gelofusin) 'stat' should be provided whilst awaiting blood. There are no data to suggest any particular type of colloid solution is safer or more effective,[4] or to dictate the rate of infusion. In general, a slower infusion rate should be used in elderly patients (>70 years of age) and/or those with pre-existing cardiac disease to minimise the risk of pulmonary oedema. However, this needs to be tempered by the severity of the hypotension. It is important to remember that aggressive fluid resuscitation causes haemodilution (approximately 10% decrease in haemoglobin level for each litre of iv fluid given).

All patients in whom a significant bleed is suspected should receive supplemental nasal oxygen and be kept nil by mouth, with cardiac monitoring provided unless the patient is at very low risk.

Blood transfusion

Urgent blood transfusion can be lifesaving in patients with haemodynamic instability. All acute trusts have written protocols for the management of massive blood loss (including the use of O-negative blood, platelets and clotting factors) which should be followed in this scenario.[5]

The benefits of blood transfusion are not so clearly defined with less severe bleeding.[6] Blood transfusion is itself not without risk – it has has been shown to be associated with a higher rebleeding rate and a trend towards higher mortality.[2,5] Therefore, transfusion should be restricted to patients with a haemoglobin 10 g/dl or below, with 10 ml (4.5 mEq) of 10% calcium gluconate given after every 3–4 units of blood transfused.

Correction of coagulopathy

Thrombocytopenia. There is no clinical benefit in correcting thrombocytopenia unless the patient is actively bleeding and

Table 1. Causes of acute upper gastrointestinal bleeding.

Cause	Cases (%)
Peptic ulcer	35–50
Mallory-Weiss tear	5–15
Gastroduodenal erosions	8–15
Oesophagitis	5–15
Gastro-oesophageal varices	7–10
Vascular malformations:	5
• hereditary haemorrhagic telangectasia	
• GAVE	
• portal hypertensive gastropathy	
• angiodysplasia	
• Dieulafoy lesion	
• Miscellaneous:	5
• hiatus hernia	
• Meckel's diverticulum	
• Crohn's disease	
• aorto-enteric fistula	
• tuberculosis	
• herpes viridiae (HSV, CMV)	
Upper GI malignancy	1

CMV = cytomegalovirus; GAVE = gastric antral vascular ectasia; GI = gastrointestinal; HSV = herpes simplex virus.

Table 2. The Blatchford score is a cumulative index based on simple clinical observations, which predict the need for (urgent) endoscopy.[10] A score of 0 predicts a low risk of rebleeding, enabling early discharge with outpatient endoscopy. A score <4 indicates a low probability for the need for urgent endoscopy.

Admission risk marker	Range	Score
Urea (mmol/l)	<6.5	0
	6.5–8.0	2
	8.0–10.0	3
	10.0–25.0	4
	≥25.0	6
Haemoglobin (men) (g/dl)	>13.0	0
	12.0–13.0	1
	10.0–12.0	3
	<10.0	6
Haemoglobin (women) (g/dl)	>12.0	0
	10.0–12.0	1
	<10.0	6
Systolic BP (mmHg)	>109	0
	100–109	1
	90–99	2
	<90	3
Other markers	Pulse >100	1
	Melaena	1
	Syncope	2
	Hepatic disease	2
	Cardiac failure	2

BP = blood pressure.

the platelet count below 50 x 10^9/l. In this situation, 1–2 pools (5–10 units) should be given.

Warfarin reversal. If the patient is actively bleeding, the anticoagulant effect of warfarin requires immediate reversal with vitamin K-dependent clotting factors (II, VII, IX, X). Although present in fresh frozen plasma (FFP), the most rapid and effective way of normalising the international normalised ratio (INR) is to prescribe prothrombin complex concentrate (eg Beriplex) together with iv vitamin K. If, the patient is haemodynamically stable, low-dose vitamin K (0.5–1 mg iv) should be given. This will correct the INR within 6–12 hours. Large doses of vitamin K should be avoided once the bleeding is controlled, particularly in patients who will require re-anticoagulation (eg those with a prosthetic heart valve).

Heparin reversal. Protamine (1 mg/100 anti-Xa units) can be used to neutralise unfractionated or low-molecular weight heparin (LMWH), reducing the dose by 50% if LMWH has been given more than eight hours beforehand.

Other coagulopathies. A raised INR not due to warfarin administration can be corrected with FFP (10–15 ml/kg). Cryoprecipitate is indicated for those with low fibrinogen (eg in association with disseminated intravascular coagulation).

Intravenous proton pump inhibitor therapy

Proton pump inhibitor (PPI) therapy raises intragastric pH reducing acid-dependent protease clot lysis. There is a growing tendency for iv PPI therapy to be prescribed in all patients presenting with acute UGIB. However, a clinical benefit (ie reduction in rebleeding rate and surgery) has been demonstrated only in patients with high-risk endoscopic stigmata, namely adherent clot and/or visible vessel. Therefore, if it is possible to arrange for urgent gastroscopy, the prescription of PPI therapy should be guided by endoscopic findings. It is prudent to commence PPI therapy if endoscopy is delayed, although this will be without significant clinical benefit in approximately 70%.[6,7]

Terlipressin

Variceal bleeding is associated with high mortality (25–50% with an index bleed). Pharmacological reduction of the portal pressure can reduce the magnitude of haemorrhage and help stabilise the patient prior to endoscopic therapy, and thereby improve outcome. All patients with suspected variceal bleeding should be started on terlipressin (2 mg qds) at presentation, continued for 3–5 days or until definitive endoscopic haemostasis has been established.

Antibiotic prophylaxis

Acute variceal bleeding is associated with a risk of Gram-negative bacteraemia and/or spontaneous bacterial peritonitis. All patients with possible variceal bleeding should be treated with a prophylactic broad-spectrum antibiotic at presentation for 5–7 days.

Non-steroidal anti-inflammatory drugs (NSAIDs)

These should be stopped at the time of presentation. If considered essential, this medication can be re-introduced, with maintenance PPI cover, once any mucosal ulceration has healed (usually 4–6 weeks).

Antiplatelet medication

Aspirin. The data concerning the use of antiplatelet medication in patients with acute UGIB are not clear. Low-dose aspirin, prescribed for secondary prevention of vascular events, should be continued. Stopping this medication is associated with

Key points

Acute upper gastrointestinal bleeding (UBIB) is a common medical problem encountered on the acute admissions unit

Early assessment of severity is important to stratify risk

High risk, unstable patients should be admitted to the high dependency unit (HDU) and considered for early endoscopic intervention

Low risk patients may be discharged home for early outpatient endoscopy

Appropriate use of PPI, terlipressin and antibiotics in specific clinical situations improve outcome

KEY WORDS: acute upper gastrointestinal bleeding, acute admissions unit, endoscopy, medical management, re-bleeding

increased mortality due to vascular events, with no benefit on the rate of rebleeding.[8]

Clopidogrel. There are no data to advise whether clopidogrel should be discontinued (ie the risk of rebleeding vs the risk of coronary stent occlusion). A decision should be made on an individual patient basis after discussion with the cardiology team, taking into consideration the rebleeding risk (see below).

Assessment of severity

It is important to stratify patients with respect to their mortality risk on admission. Those at high risk should be admitted to a high dependency unit (HDU) and considered for urgent endoscopy, while those in the low-risk group can be managed safely in an AAU setting.[8] Two validated scoring systems have been developed in the UK, both of which can also be used to quantify the risk of rebleeding:[8]

1 The Blatchford score (Table 2) predicts the need for endoscopy.[10]
2 The Rockall score (Table 3, Fig 1), calculated postendoscopy, defines the mortality risk after both acute variceal and non-variceal haemorrhage.[10]

Although both scoring systems convey some objectivity and repeatability to the assessment of patient risk, their usefulness in clinical practice is limited. The experienced clinician will appreciate that a patient with active bleeding or a large bleed, with shock, high urea and melaena is at high risk and requires urgent diagnostic and therapeutic intervention.

Table 3. Rockall score predicts mortality risk (low ≤2, moderate 3-5, high ≥6) and can be calculated only postendoscopy.[11] A score ≤2 indicates a low risk of rebleeding, with consideration for early discharge. A modified Rockall score can be derived using the clinical variables (light blue shading), similar to the Blatchford score in predicting the need for endoscopy.

	Score			
Variable	**0**	**1**	**2**	**3**
Age (years)	<60	60–79	⩾80	
Shock		Tachycardia Pulse >100 bpm	Hypotension sys BP <100 mmHg	
Comorbidity			Cardiovascular IHD	Renal hepatic failure Disseminated malignancy
Endoscopic diagnosis	Mallory-Weiss tear	Other diagnoses	UGI tract malignancy	
Endoscopic SRH	None or dark spots		Blood in upper GI tract, clot, visible or spurting vessel	

bpm = beats per minute; IHD = ischaemic heart disease; SRH = stigmata of recent haemorrhage; sys BP = systolic blood pressure (mmHg); UGI = upper gastrointestinal.

Endoscopy

Gastroscopy is the primary intervention in patients with acute UGIB. It establishes a cause in over 80% of cases and provides information that defines management and prognosis (Table 3), enabling haemostatic therapies when there is active bleeding and/or stigmata associated with a high risk of rebleeding.

Timing

Patients with evidence of a large haemorrhage should undergo urgent gastroscopy as soon as they have been stabilised. Because of the inherent risk of rebleeding, ideally all other patients should undergo gastroscopy within 24 hours of admission. Young patients at low risk of rebleeding (Blatchford score 0) can be discharged from AAU or A&E, with provision for outpatient gastroscopy within the following 2–3 days.

During normal working hours, the endoscopy unit should be contacted to arrange for an urgent gastroscopy. Out-of-hours, discussion with the on-call endoscopist is recommended.

Endoscopic therapy

Non-variceal bleeding. The clinical data regarding endoscopic therapy are based on studies involving peptic ulcer bleeding (accounting for 30–50% of cases). The same principles pertain to the endoscopic management of the other causes, in particular Dieulafoy lesions and Mallory-Weiss tears.

Endoscopic therapy is indicated for active arterial bleeding, or either a non-bleeding visible vessel or an adherent blood clot as both findings are associated with a high risk of rebleeding (50% and 25–30%, respectively). Endoscopic therapy involves dual therapy with one of the following combinations:[1,2,11]

- injection of adrenaline (1 in 10,000) and placement of metal clips
- injection of adrenaline (1 in 10,000) and thermal coagulation
- injection of adrenaline (1 in 10,000) and fibrin/thrombin injection.

All these techniques are equally effective in reducing rebleeding rates.

Variceal bleeding. The aim of endoscopic therapy for oesophageal varices is obliteration by inducing thrombosis at the level where the varices perforate through the oesophageal wall. Band ligation has replaced intravariceal injection of sclerosant as the treatment modality of choice because of superior efficacy and safety. Gastric variceal bleeding is usually treated by intravariceal injection of N-butyl-2-cyanoacrylate glue.

Management following endoscopy

All patients with variceal bleeding or non-variceal patients with high-risk lesions should

be kept nil by mouth overnight in case further endoscopy is required, especially if the endoscopist is concerned about the adequacy of the haemostasis achieved at initial endoscopy. If the patient remains stable, oral feeding can be reintroduced the following day.

Non-variceal bleeding. A patient with a high-risk lesion should be started on iv PPI therapy (eg pantoprazole 40 mg bd) for 48–72 hours before being considered for discharge. Those with a low-risk lesion (eg Mallory-Weiss tear or ulcer without stigmata of bleeding) can be considered for discharge on the same day as the endoscopy.

Variceal bleeding. It is not uncommon for these patients to develop other problems related to the underlying chronic liver disease. Their postendoscopy management should at least be shared-care with the gastroenterology/hepatology team.

Rebleeding

Rebleeding is associated with a high mortality (Fig 1). The patient should be transferred to the HDU and management undertaken by a multidisciplinary team, including gastroenterologists/hepatologists, surgeons and interventional radiologists.

Non-variceal bleeding. In patients with non-variceal bleeding, rebleeding is usually limited to those with peptic ulcer disease.[8] A repeat gastroscopy should be performed to provide additional haemostatic therapy. If this fails, percutaneous angiography with embolisation or surgery should be considered. Both have a similar efficacy, but the radiological approach is preferable due to a lower associated morbidity. Whichever modality is chosen, it is crucial that it is undertaken promptly.

Variceal bleeding. In the event of variceal rebleeding, further endoscopy is warranted. If this fails, it is usually necessary to place a Sengstaken tube to balloon-tamponade the varices for temporary haemostasis, whilst considering the patient's suitability for a 'rescue' transjugular intrahepatic portosystemic shunt.[2,8]

Postdischarge management

Non-variceal

All patients with erosive mucosal disease should be prescribed high-dose PPI therapy (eg omeprazole 40 mg od or 20 mg bd) for 6–8 weeks to effect mucosal healing. Subsequently, low-dose maintenance PPI therapy should be considered for patients who experienced a significant bleed, especially the elderly, those with comorbidity and in those taking long-term ulcerogenic (ie steroids and NSAIDs) or antiplatelet (ie aspirin and clopidogrel) therapy.

Patients with *Helicobacter pylori*-related peptic ulcer disease should be prescribed eradication therapy. The success of this therapy should be confirmed with a urea breath test at le ast two weeks after the 6–8 week course of PPI has also been completed.

All patients with a gastric ulcer require a repeat gastroscopy in 6–8 weeks to confirm complete healing, excluding underlying carcinoma.

Variceal

High-dose PPI therapy should be prescribed for 6–8 weeks. Patients should be started on low-dose propranolol (20–40 mg tds) to reduce portal pressure in order to decrease the risk of repeat bleeding, titrating the dose until the heart rate is about 60 bpm. Repeat endoscopy with band ligation is required at 1–2 weekly intervals until the varices have been obliterated. Thereafter, patients should remain on an endoscopic surveillance programme.

Variceal bleeding is a manifestation of chronic liver disease. This patient group has a variety of other medical issues, requiring active follow-up in a specialist gastroenterology/hepatology clinic.

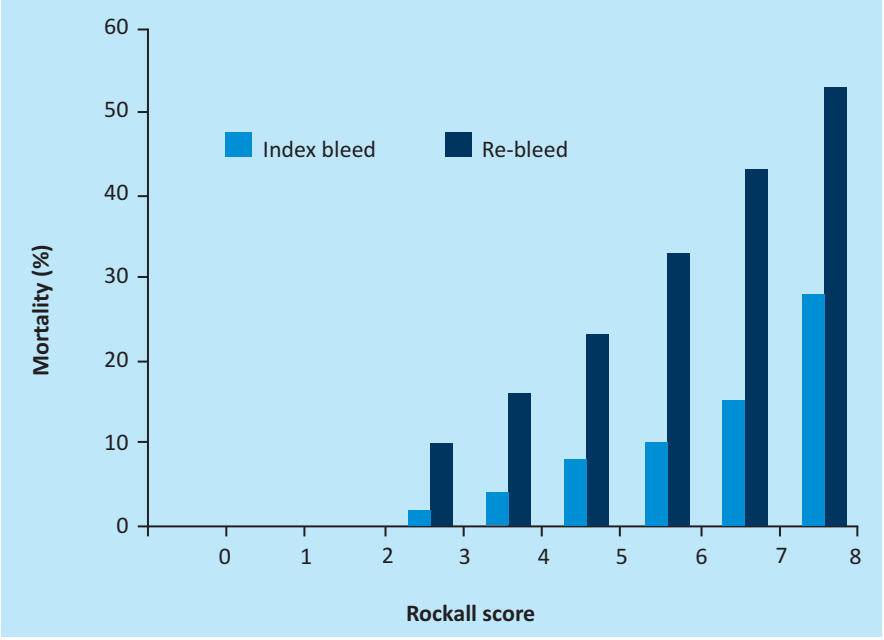

Fig 1. Rockall score and mortality rate. There is a marked and incremental increase in the mortality rate in both medium- and high-risk groups in the event of rebleeding.

Summary

AAUs should develop and review protocols and local guidelines for the multidisciplinary team management of upper GI bleeding, for example with respect to:

- early and appropriate resuscitation
- use of the tools for assessing severity
- timing of endoscopy, including recognising low-risk patients who could be discharged for outpatient investigation
- postendoscopy drug treatment, including appropriate and limited use of iv PPIs and *Helicobacter pylori* eradication therapy
- follow up, including referral for repeat endoscopy and/or urea breath testing.

Regular review and audit of local guidance of the management of UGIB should become an integral part of an acute trust's clinical governance programme.

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CME Gastroenterology SAQs (71574)

Self-assessment questionnaire

Nick Bosanko

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1 A 28-year-old woman with anorexia nervosa was admitted for enteral feeding. She had no other medical history. She was taking fluoxetine 20 mg once daily. On assessment, she was found to be severely malnourished with a body mass index of 13 kg/m^2 (18–25). Continuous nasogastric feeding was initiated with her consent. Her baseline electrolytes were normal.

Three days after admission she was found on the floor of her room and was observed by the nursing staff to have a tonic–clonic seizure.

What is the most likely cause for this event?

(a) hypercalcaemia
(b) hyperkalaemia
(c) hypernatraemia
(d) hypoglycaemia
(e) hypophosphataemia

2 A 64-year-old man was admitted to hospital complaining of diarrhoea and a rash. He had been receiving home total parenteral nutrition for six months following a small bowel resection for bowel ischaemia.

On examination, he was afebrile, with a pulse of 72 beats per minute and blood pressure of 178/87 mmHg. His abdomen was soft. He had a widespread crusting erythematous rash on his trunk and limbs.

Investigations:

haemoglobin	127 g/l (130–180)
MCV	79 fl (80–96)

What is the most likely diagnosis?

(a) amyloidosis
(b) coeliac disease
(c) pellagra
(d) tuberculosis
(e) zinc deficiency