REVIEW

Upper gastrointestinal haemorrhage: an update

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ABSTRACT

Upper gastrointestinal (GI) haemorrhage is a common cause for admission to hospital and is associated with a mortality of around 10%. Prompt assessment and resuscitation are vital, as are risk stratification of the severity of bleeding, early involvement of the multidisciplinary team and timely access to endoscopy, preferably within 24 h. The majority of bleeds are due to peptic ulcers for which *Helicobacter pylori* and non-steroidal anti-inflammatory agents are the main risk factors. Although proton pump inhibitors (PPIs) are widely used before endoscopy, this is controversial. Pre-endoscopic risk stratification with the Glasgow Blatchford score is recommended as is the use of the Rockall score postendoscopy. Endoscopic therapy, with at least two haemostatic modalities, remains the mainstay of treating high-risk lesions and reduces rebleeding rates and mortality. High-dose PPI therapy after endoscopic haemostasis also reduces rebleeding rates and mortality. Variceal oesophageal haemorrhage is associated with a higher rebleeding rate and risk of death. Antibiotics and vasopressin analogues are advised in suspected variceal bleeding; however, endoscopic variceal band ligation remains the haemostatic treatment of choice. Balloon tamponade remains useful in the presence of torrential variceal haemorrhage or when endoscopy fails to secure haemostasis, and can be a bridge to further endoscopic attempts or placement of a transjugular intrahepatic portosystemic shunt. This review aims to provide an update on the latest evidence-based recommendations for the management of acute upper GI haemorrhage.

haemorrhage to be around 10%, but as high as 26% in inpatients.¹ The main reason for death remains poorly tolerated shock, with destabilisation of underlying cardiopulmonary, liver or renal disease. Thus the risk of mortality is highest in patients of advanced age or those with severe coexistent comorbidities. This explains the increased mortality in those who develop upper GI bleeding while already an inpatient in hospital. There are a number of possible causes of upper GI haemorrhage and these are summarised in [box 1](#). The management of non-variceal and variceal haemorrhage will now be reviewed.

NON-VARICEAL HAEMORRHAGE

Pre-endoscopic management

Initial assessment and resuscitation

The initial assessment should be a quick but thorough assessment of the patient using the airway, breathing, circulation, disability and exposure (ABCDE) approach as summarised in [box 2](#). A detailed history should be taken, but this should not delay resuscitation in those who are shocked at presentation. In the history, it is important to ask about the quality and quantity of vomited blood (fresh red or coffee grounds), presence of melaena, syncope and alcohol consumption. The past medical history should elicit all significant comorbidities and any past history of upper GI bleeding or relevant surgery. It is important to note any use of non-steroidal anti-inflammatory drugs (NSAIDs), antiplatelet agents, anticoagulants, corticosteroids and selective serotonin reuptake inhibitors (SSRIs) as they have all been associated with an increased risk of GI bleeding.² The examination should include documentation of pulse, blood pressure (including assessment for postural hypotension if appropriate), capillary refill time

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INTRODUCTION

Acute upper gastrointestinal (GI) haemorrhage is a common medical emergency accounting for 50 000–70 000 hospital admissions and 5000 deaths per year. A national UK audit in 2007 estimated the overall mortality of acute upper GI

Box 1 Causes of acute upper gastrointestinal haemorrhage

- ▶ Oesophageal
 - oesophagitis
 - Mallory–Weiss tear
 - oesophageal varices
 - oesophageal ulcers
 - oesophageal cancer
- ▶ Gastric
 - gastric ulcer
 - gastric cancer
 - gastritis
 - gastric varices
 - portal hypertensive gastropathy
 - gastric antral vascular ectasia
 - Dieulafoy lesions
- ▶ Duodenal
 - duodenal ulcer
 - vascular malformations, including aortoenteric fistulae
 - haemobilia (bleeding from the bile duct due to liver biopsy, trauma, arteriovenous malformations, liver tumours).

and early warning score. Close monitoring of all of these parameters, including urine output and blood glucose (especially where chronic liver disease is suspected), is vital. It is also important to note any stigmata of chronic liver disease, which may indicate variceal bleeding. The multidisciplinary team should be informed at an early stage and this might include gastroenterologists, surgeons, anaesthetists and the critical care team. Cardiologists may also need to be involved if the patient has a metallic heart valve or coronary stents (especially drug-eluting stents) requiring antiplatelet therapy.

Resuscitation should promptly and proportionately follow the initial clinical assessment. At least 2 wide-bore peripheral cannulae should be inserted, oxygen provided and consideration be given, if appropriate, to urinary catheterisation, cardiac monitoring, central venous access and invasive arterial pressure monitoring.

In the presence of hypovolaemic shock, while artificial fluids may be given for initial volume support, prompt provision of blood remains paramount to maintain adequate tissue perfusion and oxygenation. If appropriate, local major haemorrhage protocols should be appropriately activated to ensure efficient and timely blood product acquisition. The threshold for transfusion should be carefully weighed up in each patient and based on haemodynamic status, predicted blood loss and coexistent comorbidities that might increase the risk of complications from anaemia.³ International consensus guidelines, however, recommend that blood be

Box 2 Initial approach to the management of acute upper gastrointestinal bleeding

Airway

- ▶ Assess patency.
 - If reduced consciousness, consider definitive airway to protect against aspiration and facilitate safe endoscopy.
- ▶ Breathing
 - Maintain oxygen saturations at 94%–98% with oxygen as required.
 - Check for aspiration (consider chest X-ray).
- ▶ Circulation
 - Check and monitor pulse, blood pressure, capillary refill time and urine output.
 - 2× wide-bore cannulae and baseline blood tests.
 - Intravenous fluids±blood/blood products.
 - Correct clotting abnormalities.
 - In brisk bleeds, consider activating major haemorrhage protocol.
 - If vascular access is difficult, consider placement of intraosseous device.
 - Consider need for central venous line/arterial line and involvement of critical care disability.
 - Monitor conscious level using Glasgow Coma Scale.
 - Check blood glucose.
- ▶ Exposure
 - Examine for stigmata of chronic liver disease (which may suggest varices).
 - Note any abdominal tenderness/guarding (may indicate perforated viscus).

transfused if the haemoglobin (Hb) level is ≤ 70 g/L.³ Recent evidence has shown that adopting a restrictive transfusion strategy, and not transfusing until the Hb falls below <70 g/L, is associated with improved survival, a reduced risk of rebleeding and complications.⁴ This strategy was associated with improved 45-day survival outcomes compared with patients transfused at an Hb of 90 g/L, and the survival advantage was greater in patients with cirrhosis than those with peptic ulcer disease.⁴ The harmful effects of transfusion may relate to impaired haemostasis, adverse effects on coagulation and increased splanchnic blood flow and pressure that might impair clot formation and stability.^{5 6}

The National Institute for Health and Care Excellence (NICE) has recently issued guidance on blood product management in upper GI bleeding.⁷ Specifically:

- ▶ Platelets should only be given if the patient is actively bleeding or haemodynamically unstable and has a platelet count of $<50 \times 10^9$ /L.
- ▶ Fresh frozen plasma should be given if the fibrinogen level is <1 g/L or the prothrombin time (PT) or activated partial thromboplastin time is >1.5 times normal.

- ▶ Prothrombin complex should be provided to those on warfarin and actively bleeding.
- ▶ Recombinant factor VIIa should only be used when all of the above measures have failed.

Risk stratification

It is important that patients with upper GI bleeding are stratified into low and high risk on admission according to validated prognostic scales. High-risk patients can be identified for early intervention, thereby reducing morbidity and mortality, and certain low-risk patients can even be discharged safely. The Blatchford score (table 1) should be used in every patient on initial presentation.⁸ The Rockall score (table 2) can also be used but requires knowledge of endoscopic findings to be fully completed.⁹ In a multicentre UK study, it was found that patients with a Blatchford score of zero can be safely discharged with a view to early outpatient endoscopy.¹⁰ In this study, there was no requirement for blood transfusion or endoscopic intervention, nor any deaths, in patients with a Blatchford score of 0. This study also showed that these low-risk patients are suitable for discharge and outpatient management and that formally adopting the Blatchford score into clinical practice significantly reduced the number of low-risk admissions.

Pre-endoscopic pharmacotherapy

- ▶ *Proton pump inhibitor (PPI) therapy*: Gastric acidity inhibits platelet aggregation, clotting and clot stability through increased fibrinolysis.¹¹ PPI therapy facilitates

clot formation, stabilisation and eventually mucosal healing of peptic ulceration.¹¹ PPI therapy is commonly given to patients prior to endoscopy; however, neither intravenous nor oral PPI therapy in this setting has been shown to alter clinically important outcomes such as mortality, rebleeding rate and need for surgery.^{11a} Patients kept nil by mouth often receive intermittent intravenous PPI therapy; however, this is less cost-effective than oral therapy.^{11a} There is some evidence that pre-endoscopy PPI therapy can reduce the presence of high-risk stigmata at endoscopy and need for subsequent endoscopic therapy; however, one such randomised controlled trial (RCT) was in Asian patients who are more sensitive to PPI therapy and therefore derive greater treatment effects.^{12 13} Both British and Scottish Gastroenterology societies do not advocate the use of PPIs prior to endoscopy, and their use should certainly not delay early endoscopy.¹⁴

- ▶ *Tranexamic acid*: The CRASH-2 trial showed that a short course of tranexamic acid given to adult patients with traumatic bleeding within 3 h of injury significantly reduced all-cause mortality with no apparent increase in the risk of adverse thrombotic events.¹⁵ Due to this, tranexamic acid is advocated in many trauma protocols. In GI bleeding, however, the results so far have not been as conclusive. A recent meta-analysis of seven RCTs showed that tranexamic acid did not reduce bleeding, bleeding-related mortality, surgery or transfusion requirements but may reduce all-cause mortality.¹⁶ Due to the small numbers of patients studied and trial heterogeneity, it is currently not recommended for routine use in GI bleeding. The HALT-IT trial is a recently commenced international randomised, double-blind placebo-controlled study determining the effect of early administration of tranexamic acid in patients with GI bleeding. It is due to report its findings in 2017.

- ▶ *Management of concomitant medications*: In the acute bleeding phase, all medications that might aggravate GI bleeding should be discontinued where possible. These include NSAIDs, aspirin, clopidogrel and anticoagulants. SSRIs have also been linked with an increased risk of GI bleeding.¹⁷ Patients with complicated cardiovascular disease, such as those with prosthetic valves or drug-eluting coronary stents, requiring anticoagulant or antiplatelet therapies represent a particular management challenge. Early involvement of the cardiologists is vital to minimise adverse cardiac events from the cessation of these therapies. Haematologists may also need to be consulted if the patient is on standard or novel oral anticoagulants.

Prokinetic agents: Good endoscopic views are vital to maximise the chance of finding the bleeding point and delivering therapy. Erythromycin has prokinetic properties, and a recent meta-analysis of 4 RCTs has reported that it significantly improves endoscopic visibility when given around 30 min before endoscopy.¹⁸ It may also reduce the need for second-look endoscopy and length of stay.

Table 1 Blatchford score

Admission risk marker	Score component value
Blood urea (mmol/L)	
≥6.5 <8.0	2
≥8.0 <10.0	3
≥10.0 <25.0	4
≥25	6
Haemoglobin (g/L) for men	
≥12.0 <13.0	1
≥10.0 <12.0	3
<10.0	6
Haemoglobin (g/L) for women	
≥10.0 <12.0	1
<10.0	6
Systolic blood pressure (mm Hg)	
100–109	1
90–99	2
<90	3
Other markers	
Pulse ≥100 (per min)	1
Presentation with melaena	1
Presentation with syncope	2
Hepatic disease	2
Cardiac failure	2

Table 2 Rockall score

Variable	Score			
	0	1	2	3
Age	<60	60–79	>80	
Shock	No shock	Pulse >100 Blood pressure >100 systolic	Systolic blood pressure <100 mm Hg	
Comorbidity	Nil major		Heart failure, ischaemic heart disease, major comorbidity	Renal failure, liver failure, metastatic cancer
Postendoscopy				
Diagnosis	Mallory–Weiss	All other diagnoses	Gastrointestinal malignancy	
Evidence of bleeding	None		Blood, adherent clot, spurting vessel	

Endoscopic management

Early endoscopy allows for further risk stratification of patients with upper GI bleeding. Those with low-risk lesions such as a non-bleeding Mallory–Weiss tear, clean-based ulcer or ulcer with a pigmented spot in the base can be identified for early discharge. Alternatively, those with high-risk lesions (active arterial bleeding, non-bleeding visible vessels and adherent clots) can be identified for prompt endoscopic intervention to reduce the chance of further bleeding. In these individuals, early endoscopic therapy (within 24 h) reduces transfusion requirements, rebleeding and need for surgery.¹⁹ Despite these beneficial effects, early endoscopy has surprisingly not been shown to reduce mortality significantly (number needed to treat (NNT) 35–500).²⁰ It has been speculated that this may be because the major determinant of survival is the presence and severity of concurrent medical comorbidities rather than the achievement of haemostasis.⁹ Providing daily endoscopy access throughout the year, and therefore access to endoscopy within 24 h, remains a significant challenge for many hospital trusts.

A range of techniques can be used to stop and prevent further bleeding including injection (adrenaline), thermo-ablative (coagulation probes) and mechanical (clipping) therapies. Dual therapy is now advocated, for example, adrenaline injection around and into the bleeding point followed by coagulation therapy or clip application to the visible vessel. One meta-analysis of 16 RCTs reported that a combination approach reduces rebleeding rate, need for surgery and mortality.²¹ Adrenaline injection promotes haemostasis by a combination of local vasoconstriction and tamponade effect; thermo-ablative or mechanical therapies then directly deal with the offending bleeding vessel. A repeat endoscopy should be considered when initial endoscopic treatment was considered to be suboptimal or if the risk of rebleeding is deemed high or potentially life-threatening as this reduces rebleeding rate.²²

Topical haemostatic powders have been a recent addition to the therapeutic arsenal in GI bleeding and can be considered in selected cases, for instance, in those

where active bleeding is present and dual therapy is not possible or has failed. Such therapies include Hemospray (TC-325, Cook Medical), Endoclot (EndoClot Plus) and Blood Stopper (Ankaferd İlaç Kozmetik AŞ). Hemospray is already in use in many UK centres and is an inorganic powder, which can be sprayed onto a bleeding vessel via a catheter through the endoscope. Once in contact with blood it absorbs water and forms a cohesive and adhesive gel, which stops bleeding through a combination of mechanical effects (tamponade) and possible pro-coagulatory effects on platelets and clotting factors.²³ In a prospective, single-arm, pilot study of Hemospray involving 20 adult patients with active bleeding (Forrest score 1a or 1b ulcers), 95% (19/20) achieved acute haemostasis with no signs of ongoing bleeding at 72 h, and no adverse events were reported at 30 days.²⁴ In a small case series, Hemospray has also shown efficacy in the management of non-ulcer bleeding including portal hypertensive gastropathy, vascular ectasia, haemorrhagic gastritis and cancer bleeding.^{25 26} There are no randomised or placebo-controlled data on the effectiveness of topical haemostatic powders.

In those where endoscopic therapy fails to control bleeding, the options include interventional radiology for angiographic embolisation or surgery. These options also may be considered for those that rebleed despite dual endoscopic therapy.

Postendoscopic pharmacotherapy

1. *PPI therapy*: A meta-analysis of 24 RCTs has shown that PPIs reduce the rate of rebleeding (NNT=13), need for surgery (NNT=34) and need for further endoscopic therapy (NNT=10) but did not affect overall mortality.²⁷ Subgroup analysis of these trials subsequently showed that the reduction in mortality was significant when confined to seven trials in high-risk patients (active bleeding or non-bleeding visible vessel) who received endoscopic therapy and remained significant when the analysis was confined to four trials that used high-dose intravenous therapy (omeprazole 80 mg bolus followed by 8 mg/h infusion for 72 h) following endoscopic therapy.²⁷ There was no effect on mortality in the other three trials that

used lower-dose intravenous or oral PPI therapy. It is, therefore, recommended that this high-dose intravenous bolus then continuous infusion regimen be used in patients with high-risk endoscopic lesions treated endoscopically.

2. *Helicobacter pylori*: A urease test should be routinely performed at the time of endoscopy in patients with peptic ulceration. In the setting of a bleeding ulcer, there is evidence that the urease test is less accurate; a subsequent urease breath test may be considered due to its greater sensitivity.²⁸ *H. pylori* eradication is effective in reducing recurrent peptic ulcer bleeding and is more effective than PPI therapy alone (NNT=6 if no maintenance PPI therapy given and NNT=20 if maintenance PPI therapy given).²⁹ If *H. pylori* is present, then it is recommended that PPI therapy be given for at least 3 weeks after eradication therapy.³⁰
3. *Management of antiplatelet and anticoagulant agents postendoscopy*: After resolution of GI bleeding, some patients may require ongoing treatment with potential gastric irritants or anticoagulants; there is some evidence that they may benefit from concomitant maintenance PPI therapy.³¹ In those with peptic ulcer bleeding on aspirin, resuming aspirin therapy with PPI cover immediately after endoscopic therapy has been shown to significantly increase the 30-day bleeding rate but reduce all-cause mortality at 8 weeks.³² In patients requiring dual antiplatelet therapy (aspirin and clopidogrel), concomitant treatment with omeprazole significantly reduced the rate of ulcer bleeding with no significant increase in cardiovascular events.³³ The decision to recommence antiplatelet or anticoagulant medications after upper GI bleeding can be a difficult one. This must be done on an individual case-by-case basis after careful consideration of the risks and benefits and discussion with the patient and any relevant specialties, such as cardiology.

VARICEAL HAEMORRHAGE

Varices are the cause of bleeding in around 11% of patients presenting with upper GI haemorrhage, and in

the majority the varices are oesophageal.¹ In cirrhosis, oesophageal varices tend to develop when the hepatic venous pressure gradient (HVPG) is above 10 mm Hg (normal <5 mm Hg).³⁴ The mortality of variceal bleeding remains high at between 15% and 20%, and outcome closely correlates with the severity of underlying liver disease. The latter can be assessed by calculating the Child–Pugh score (table 3). Patients presenting with possible variceal haemorrhage should be assessed, resuscitated and risk-stratified as previously described for non-variceal haemorrhage. Additional attention should be given to the correction of coagulopathy and thrombocytopenia as this is frequently present in those with advanced liver disease. Prompt involvement of the gastroenterology team is important to adequately optimise patients for early endoscopy.

Endoscopic therapy

Oesophageal varices: Variceal band ligation is superior to injection sclerotherapy in terms of rebleeding rate, mortality rate and rate of death due to rebleeding.³⁵ Band ligation requires fewer endoscopic sessions to achieve variceal obliteration and is also associated with fewer adverse events such as sepsis, oesophageal ulceration and stricture formation.³⁵ Due to these reasons, band ligation is the therapy of choice for the management of oesophageal variceal bleeding.

Gastric varices: Gastric varices can be classified according to their location and relationship to oesophageal varices. Those that continue from oesophageal varices and extend for <5 cm along the lesser curvature of the stomach are termed gastroesophageal varices (GOV) type 1. Those that continue from oesophageal varices but extend towards the fundus along the greater curvature are GOV type 2. Isolated gastric varices (IGV) are not in continuation with oesophageal varices and can be in the fundus (IGV type 1) or anywhere distally (IGV type 2). Two RCTs have shown that, irrespective of classification, cyanoacrylate superglue injection is more effective

Table 3 Child–Pugh classification of chronic liver disease

Clinical/laboratory findings	Score		
	1	2	3
Encephalopathy	None	Mild (grade 1–2)	Severe (grade 3–4)
Ascites	None	Mild/slight	Moderate/large
Bilirubin (μM/L)	<34	34–51	>51
Albumin (g/L)	>35	28–35	<28
Prothrombin time prolongation (s)	<4	4–6	>6
International normalised ratio (INR)	<1.3	1.3–1.5	>1.5
Chronic liver disease is classified into Child–Pugh class A to C using the total score from the above:			
Total score	Child–Pugh class		
5–6	A		
7–9	B		
10–15	C		

than band ligation in terms of initial haemostatic control, rebleeding rate, need for blood transfusion and treatment-induced ulcer bleeding.^{36 37} Gastric variceal glue injection is not without risk as it can be associated with glue embolisation to the lungs with haemodynamic compromise and hypoxia. Glue injection requires both endoscopic skill and excellent coordination with nursing staff to minimise the risk of damaging the endoscope permanently with glue or gluing the endoscope or injector needle onto the varix itself. Thrombin injection of gastric varices has also been used similarly with promising results; however, to date no RCT data exist.³⁸

Pharmacotherapy for variceal bleeding

Vasoactive therapy: Vasoactive drugs reduce portal hypertension by decreasing portal blood flow. Terlipressin is preferred over somatostatin or its analogues (such as octreotide) as it is the only one to have shown a reduction in mortality, with an NNT of 8.3 to prevent one death.³⁹ A typical terlipressin dosing regimen is 2 mg intravenous stat followed by 1–2 mg every 4–6 hours. Terlipressin should be started promptly if variceal bleeding is suspected and continued after endoscopy for at least 48 h as beneficial effects have been demonstrated both before and after endoscopic diagnosis or therapy.³⁹

Antibiotic therapy: Bacterial infections are common in cirrhotic patients with variceal bleeding and manifest in around 66% within 2 weeks; in 30%–40% an infection is present on admission.⁴⁰ It has been hypothesised that sepsis-induced systemic endotoxin release promotes an increase in portal pressure due to local vasoconstriction and a rise in intrahepatic vascular resistance⁴¹; sepsis has thus been implicated in precipitating variceal bleeding. Cirrhotic patients with variceal bleeding who either have sepsis on admission or who go on to develop it have high rates of failure to control bleeding, and increased rebleeding and mortality.^{41a} Prophylactic antibiotic use has been shown to significantly reduce mortality in cirrhotic patients with variceal bleeding, and thus antibiotics should be promptly commenced on admission.⁴² Options include ceftriaxone, norfloxacin, ciprofloxacin or other broad spectrum antibiotics such as tazocin; however, local infection control policies should be followed.

PPI therapy: Shallow ulcers normally occur at variceal banding sites in the oesophagus and occasionally these can bleed. In a small randomised placebo-controlled trial after variceal banding, the number of ulcers at day 10 was the same in each group; however, those receiving pantoprazole (40 mg daily for 10 days after banding) as opposed to placebo had smaller ulcers. Although not statistically significant, all 3 post-banding ulcer bleeds occurred in the placebo group.⁴³ In another RCT, the risk of bleeding was significantly lower in patients given 10 mg of rabeprazole daily

following band ligation compared with a similar group receiving no PPI. Due to significant benefits, the trial was stopped early.⁴⁴ PPIs are thus commonly employed after variceal band ligation.

Rescue therapy for variceal bleeding

Balloon tamponade: In around 10%–20% of patients, variceal bleeding continues despite combined pharmacological and initial endoscopic therapy. Although repeat endoscopy can be considered if the patient is clinically stable, if there is cardiovascular compromise then balloon tamponade with a Sengstaken–Blakemore tube can be life-saving. Balloon tamponade can also be useful in torrential variceal bleeding where endoscopy fails to identify or adequately treat bleeding varices, but in this setting it is usually used as a bridge to either a further attempt to treat the varices endoscopically or radiological placement of an intrahepatic portosystemic shunt. Control of bleeding is achieved in around 80% of patients; however, complications occur in as many as 20% including aspiration, tube migration and oesophageal necrosis or perforation.⁴⁵ RCTs have not shown any benefit of balloon tamponade over standard vasoactive therapies or endoscopic sclerotherapy.^{46 47} As a result, balloon tamponade is usually used as a rescue measure.

Transjugular intrahepatic portosystemic shunt (TIPSS): Portal hypertension is present when the HVPG—the difference between the wedged and free hepatic venous pressures—is >5 mm Hg. It is considered clinically significant when it exceeds 10 mm Hg as this usually results in the development of varices.⁴⁸ In patients with variceal bleeding, an HVPG of >20 mm Hg is associated with failure to control bleeding, a higher rate of rebleeding and higher 1-year mortality.⁴⁹ A TIPSS is the percutaneous placement of a stent between the hepatic vein and intrahepatic segment of the portal vein in order to reduce portal pressure. Although a very useful technique to control recurrent variceal bleeding, it can also be placed to reduce other complications of liver failure such as refractory ascites or hepatic hydrothorax. A reduction in HVPG to <12 mm Hg or by 20% from the baseline value reduces the risk of variceal haemorrhage and improves survival.⁵⁰ The two most important complications of the TIPSS are hepatic encephalopathy due to systemic exposure to toxin-containing blood and heart failure due to the sudden increase in cardiac preload.

Self-expanding metal stents (SEMS): Small case series have reported on the use of removable or biodegradable SEMS for refractory variceal bleeding.^{51 52} Preliminary data suggest that they are effective in achieving haemostasis; however, SEMS removal followed by conservative treatment is associated with a high rebleeding rate (60% at 9 days), suggesting that this approach should be used as a bridging strategy to more definitive therapy such as TIPSS or liver

transplantation.⁵¹ NICE has produced a detailed appraisal of existing studies that can be accessed freely.⁵³

Secondary prevention of variceal haemorrhage

Without additional therapy, patients with variceal bleeding that survive an index bleed have a 60% chance of rebleeding within 1–2 years with a 33% mortality.⁵⁴ Propranolol has been shown to significantly reduce rebleeding and mortality in these patients and should be commenced soon after stabilisation.⁵⁵ The combination of β -blockers and nitrates is superior to β -blocker monotherapy but is associated with more side effects and is poorly tolerated.⁵⁶

Carvedilol is a non-selective β -blocker with α (1)-adrenergic blocking activity, which has been shown to have a greater portal hypotensive effect than propranolol alone in patients with cirrhosis.⁵⁷ Carvedilol is effective in the primary prophylaxis of variceal bleeding and also effective in patients who fail to reduce HVPG on propranolol.^{58–59} Based on these data, some centres use carvedilol in preference to propranolol.

Periodic surveillance gastroscopy with or without further variceal band ligation has been shown to significantly reduce the median rebleeding rate to around 32%.⁶⁰ It is currently best practice for patients to be placed on life-long propranolol or carvedilol and a 'banding programme' of surveillance gastroscopy until variceal obliteration has been achieved, followed by surveillance gastroscopies every 3–12 months depending on the findings. The interval between surveillance endoscopies is still debated and practice varies across units in the UK; however, many perform this at 1–2-weekly intervals given previously published guidelines.⁶¹ More recent data report similar effectiveness (rebleeding rate, variceal recurrence and mortality) whether banding was performed at two weekly intervals or monthly, although those in the monthly endoscopy group had fewer banding ulcers.⁶² In those unsuitable for surveillance gastroscopy, a combination of non-selective β -blocker and nitrate is recommended.¹⁴ Patients who rebleed despite optimal pharmacological and endoscopic therapy or those who are intolerant to that approach, can be considered for TIPSS.

SUMMARY

Acute upper GI bleeding is a common gastroenterological emergency with high mortality, particularly if variceal or in the elderly patient with comorbidities. Prompt clinical assessment, resuscitation and risk stratification using validated scoring systems are vital to ensure a positive outcome for the patient. Gastroenterologists should be informed early as should the surgical team and, if necessary, critical care physicians. Cardiologists should also be involved early if the patient has a metallic heart valve or a drug-eluting stent to facilitate prompt decisions over

Box 3 Key messages in upper gastrointestinal bleeding

- ▶ All patients should be adequately resuscitated prior to endoscopy and risk-stratified with the Blatchford score.
- ▶ Early involvement of the multidisciplinary team is important (gastroenterologists and surgeons, and, if necessary, critical care, cardiology and haematology).
- ▶ There is no firm evidence supporting the pre-endoscopic administration of proton pump inhibitors (PPIs).
- ▶ After endoscopic ulcer therapy, patients should receive a high-dose bolus of a PPI followed by a continuous infusion for 72 h.
- ▶ Prophylactic antibiotics and terlipressin should be administered promptly in suspected variceal haemorrhage.

antithrombotic agents, although this could be done after endoscopy if performed within 24 h. Where possible, blood should only be transfused if the Hb is <70 g/L; however, each case should be carefully risk-assessed. Endoscopy within 24 h reduces rebleeding rates and need for surgery, and is likely to reduce length of stay. PPI therapy before endoscopy is controversial and not recommended but is still widely practised. After endoscopy, PPI therapy has significant benefits especially in those who have had appropriate endoscopic therapy. Variceal haemorrhage requires early specialist gastroenterological input and management due to the complexity of decompensated liver disease and high mortality. Any patient with suspected variceal haemorrhage should receive broad-spectrum antibiotics and terlipressin (unless contraindicated). The provision of daily endoscopy lists and prompt access to specialist gastroenterological reviews throughout the year remains a very significant challenge facing many hospital trusts.

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