Management of massive gastrointestinal haemorrhage

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Abstract

Gastrointestinal (GI) haemorrhage is a common medical emergency, with one patient presenting every 6 minutes in the UK (70–90,000 cases per year). It is associated with a significant mortality rate that has remained relatively static at 10% for more than two decades. Haemorrhage is commonly categorized as bleeding of upper or lower GI origin, but for organization of care, both groups should be regarded as one clinical entity. Rapid assessment, resuscitation and correction of coagulopathy should be undertaken, following local major haemorrhage protocols, with investigation urgently arranged. For upper GI haemorrhage, endoscopy remains the gold standard for simultaneous investigation and treatment. For lower GI haemorrhage, a more nuanced algorithm is available, including CT angiography for actively bleeding or unstable patients, and endoscopic evaluation in select cases. Clinicians may utilize a range of endoscopic and radiological techniques to diagnose and control the source of haemorrhage, which should be tailored to the site of bleeding and pathology. When haemostasis is not achieved, either repeat intervention or a different modality should be selected. Surgery is now infrequently used as a treatment for GI haemorrhage and should be contemplated only once endoscopic and radiological treatments have failed, following discussion between senior clinicians. Postoperative GI bleeding may still be best served with return to theatre, but interventional radiology should also be considered.

Keywords Emergency surgery; endoscopy; gastrointestinal haemorrhage; haematemesis; haematochezia; interventional radiology; lower GI bleed; management; melaena; upper GI bleed

Introduction

Gastrointestinal (GI) haemorrhage is a common medical emergency, with an incidence of 103 per 100,000 population in the UK.[1] This is equal to one patient presenting every 6 minutes, or 85,000 cases per annum.[2–4] It is associated with a significant mortality rate that has remained relatively static at 10% for more than two decades.[4] GI haemorrhage (bleeding) can originate from anywhere along the length of the GI tract, from mouth to anus, or, per stoma in previously operated patients. Acute haemorrhage is traditionally classified as upper or lower GI in origin and refers to overt bleeding rather than more insidious or chronic occult blood loss. The latter is a common cause of iron deficiency anaemia and abnormal stool tests (such as the faecal immunochemical test (FIT)), which can usually be investigated as an outpatient rather than in the emergency setting.[5]

Upper GI haemorrhage

This occurs proximal to the ligament of Treitz (the band of smooth muscle extending from the duodeno-jejunal flexure to the left crus of the diaphragm); it includes bleeding from the oesophagus, stomach, or duodenum, and is accessed with a standard fibre-optic endoscope. Upper GI haemorrhage is subclassified as non-variceal (89%) or variceal (associated with chronic liver disease) (11%). Overall mortality is approximately 10%; unchanged since the 1990s.[2,3]

Lower GI haemorrhage

This occurs distal to the ligament of Treitz; it includes bleeding of the jejunum, ileum, colon, rectum or anal canal. Lower GI haemorrhage is less common, but in-hospital mortality is reportedly as high as 3.4%.[6]

Classifying haemorrhage as upper or lower GI in origin can aid diagnosis and management, but guidelines suggest that for delivery of care, both groups should be regarded as one clinical entity and clinical governance structured as such. Studies have quantified massive (or major) GI haemorrhage as requiring transfusion of at least four units of packed red blood cells.[2] The NHS transfusion service define it as loss of one blood volume in 24 hours (70 ml/kg), 50% of total blood volume within 3 hours, or blood loss more than 150 ml/minute.[7] Clinical indicators of this include systolic blood pressure less than 90 mmHg or heart rate more than 110 beats/minute.[8,9] Landmark UK publications in the last decade include the 2015 National Confidential Enquiry

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into Patient Outcome and Death (NCEPOD) Massive GI haemorrhage report, and the 2018 UK Lower GI Bleeding Collaborative audit.[2,6,10]

In 2015 NCEPOD reported a combined overall mortality of 10.4% for upper and lower GI haemorrhage.[2] Both the NCEPOD report and the Lower GI Bleeding Collaborative found that mortality was associated with three factors:

(i) Requirement for at least four units of red cell transfusion (i.e. massive GI bleeding) doubled overall mortality to 24% in the NCEPOD study (non-variceal upper GI bleeding 21% and lower GI bleeding 20% mortality).[2,6,10] Oakland et al. also reported the same 20% mortality for lower GI bleeding with four or more units of red cell transfusion.[6] Indeed, GI bleeding is the second most common diagnosis after haematological malignancy to result in blood transfusion, accounting for 14%.[8]

(ii) Mortality is also closely associated with degree of shock, and onset of GI bleeding in patients already admitted to hospital was associated with a mortality rate of 18% in the Oakland et al. study (regardless of transfusion) and 37.7% in the NCEPOD study.[2,6,10]

(iii) Comorbidity and appropriateness of intervention appears to contribute significantly to risk of mortality following onset of severe GI haemorrhage; 79% of mortalities reported by NCEPOD were patients on a palliative care pathway at time of death.[2,10]

Presentation

The signs and symptoms of GI haemorrhage are dependent on origin. Upper GI bleeding typically presents with symptoms of melaena (black, malodorous faeces caused by altered haemoglobin), or haematemesis (coffee ground-appearing blood-stained vomitus, caused by blood interacting with gastric acid). Meanwhile lower GI bleeding presents with haematochezia – fresh red bleeding, or passage of clots per rectum. Often the clinical picture can be mixed; brisk upper GI bleeding presenting with haematochezia can lead to the misdiagnosis of lower GI bleeding in as many as 15% of patients, which can lead to a potentially fatal delay in commencing appropriate management. Conversely, patients with bleeding from the caecum or distal small bowel may present with melaena so can be misdiagnosed as an upper GI bleed.[6,11]

It is important to obtain an accurate and thorough history from the patient or carer. Focused questioning of the acutely unwell patient should consider character, frequency and volume of blood loss, associated symptoms such as pain, nausea, vomiting, change in bowel habit and indicators of other pathology such as pyrexia. A complete past medical history must be obtained, with focus on comorbidities that indicate an increased risk of bleeding or help identify the cause. For upper GI haemorrhage, this should include liver disease and presence of varices, alcohol abuse, peptic or duodenal ulcer, reflux disease and any known vascular abnormalities such as angiodysplasia. Symptoms of undiagnosed upper GI malignancy, such as dysphagia and weight loss, must be considered. A history of acute vomiting of any cause must alert the clinician to the risk of a Mallory–Weiss tear. In lower GI haemorrhage, the commonest cause in the UK is diverticular disease, followed by anorectal pathology such as haemorrhoids.[6] Symptoms of colorectal malignancy or inflammatory bowel disease, such as recent change in bowel habit, should be considered. Recent endoscopic instrumentation of the GI tract could suggest an iatrogenic cause of bleeding or may reveal the underlying pathology.

It should be established whether or not the patient is taking medication or undergoing treatment which may damage the GI mucosa causing ulceration and therefore predispose to bleeding; this includes non-steroidal anti-inflammatory drugs (NSAIDs), steroids, or radiotherapy. Up to a third of patients presenting with GI haemorrhage in the UK are prescribed a form of anti-platelet drug, therefore aspirin and other medications which increase the risk of bleeding, such as anticoagulants including warfarin and direct oral anticoagulants (DOACs), should be identified.[9,12] Patients already admitted to hospital may be prescribed low-molecular-weight heparin (LMWH) as prophylaxis or treatment for thromboembolism.

It is essential to perform a full GI examination of the patient. Be aware of signs that help to localize the source of the bleeding, such as an abdominal mass. Examine for the presence of stigmata of chronic liver disease and for the signs of malignancy (e.g. palpable lymphadenopathy). You must perform a digital rectal examination (which allows accurate and objective assessment of melaena versus haematochezia) and, if tolerated by the patient, consider proctoscopy or rigid sigmoidoscopy to identify an anorectal cause. The source of bleeding may be inferred by symptoms and signs, but guidance suggests patients should progress to prompt formal investigation in order to confirm diagnosis.[2,13] As patients already admitted to hospital are at increased risk of mortality, concerns regarding blood loss raised by nursing or allied healthcare staff should be assessed without delay.

Initial management

Initial management of haemorrhage involves standard resuscitation measures. Assessment of the patient's airway and respiratory system is performed, with attention made to ensure adequacy of ventilation. Reduced consciousness and subsequent aspiration of either blood or gastric contents can result in airway obstruction and complications such as respiratory tract infection. Oxygen saturation and respiratory rate are recorded and, in the event of inadequate ventilation, simple airway manoeuvres (head tilt, chin lift and jaw thrust) and adjuncts (oropharyngeal or nasopharyngeal airway) utilized. The patient's heart rate and blood pressure are recorded and wide bore peripheral venous access obtained (at least two 16-to-18-gauge intravenous cannula). In the event of inadequate ventilation and perfusion or inability to secure intravenous access, seek immediate support via a medical emergency or arrest team in order to progress resuscitation. All hospitals in the UK have a major haemorrhage protocol which care-providers should be familiar with and able to activate when necessary. Failure to respond to initial resuscitation, as judged by prompt and ongoing observation using a system such as the National Early Warning Score (NEWS), should mandate referral to critical care outreach services.[13]

Blood tests including haemoglobin (Hb), haematocrit, urea, creatinine, electrolytes, liver function, coagulation profile (international normalized ratio (INR), prothrombin time (PT), activated partial thromboplastin time (APTT) and fibrinogen) are

obtained and cross-matched blood obtained after discussion with the transfusion laboratory. When available, thromboelastometry (TEM, previously known as rotational thromboelastometry (ROTEM)) can provide rapid assessment of coagulation status. Elevated serum urea, often used to differentiate upper and lower GI blood loss, may be observed in the case of upper GI haemorrhage as blood bound protein is metabolized to blood urea nitrogen (BUN) and then reabsorbed. Measurement of lactate from either arterial or venous blood gas allows for a prompt assessment of tissue perfusion and the patient's blood volume status, and has been demonstrated to be a sensitive predictor of mortality.[14]

Previously healthy and young patients have robust compensatory mechanisms to ensure adequate circulatory pressure. Increase in myocardial contractility, heart rate and peripheral vasoconstriction maintain circulatory pressure at near normal level for as long as possible and therefore a falling blood pressure is considered a late sign and should not be relied upon in the initial stages to guide assessment of severity of shock. The Advanced Trauma Life Support (ATLS) guidelines on haemorrhage severity and class of hypovolaemic (haemorrhagic) shock is a useful tool in the estimation of blood loss in patients with significant GI bleeding (Table 1).

A prompt intravenous fluid bolus of 500 ml crystalloid is recommended for initial volume replacement.[15] In the event of major haemorrhage, judicious use of blood products will be required, but over-transfusion should be avoided due to the risk of circulatory overload and transfusion reaction. After consultation with appropriate expertise (gastroenterology, surgery or critical care) it may be prudent to adopt a policy of 'permissive hypotension' (maintaining blood pressure at a level required to maintain tissue perfusion and cognition) until definitive control of the source of bleeding can be established. This should only be undertaken with senior clinical oversight, due to the risk of under-resuscitation.

Hospitals will have a transfusion policy where, for stable patients, packed red blood cell transfusion is recommended below a threshold (typically Hb <70 g/litre).[15] In the UK, the National Institute for Health and Care Excellence (NICE) guidelines recommend a transfusion target of Hb 80 g/litre in patients with cardiovascular disease and 70 g/litre in those without.[7] However, in the event of major haemorrhage, haemoglobin may not reduce immediately as the patient loses both red cells and plasma volume. Meta-analysis shows restrictive transfusion is associated with improved outcomes in patients without cardiovascular disease but results are less clear and may be harmful in those with coronary artery disease, stroke or peripheral vascular disease.[16]

Activation of a major haemorrhage protocol triggers the rapid and continuous issue of red cells and other blood products from the blood bank in pre-agreed ratios, usually 1:1 packed red cells: fresh frozen plasma (FFP), with platelets and cryoprecipitate directed by laboratory results. Staff members in haematology, theatres and critical care are usually alerted when the protocol is activated. There is little published evidence to suggest specific platelet or FFP target levels, but several guidelines recommend patients who are actively bleeding and have thrombocytopaenia (platelet count $<50 \times 10^9$/litre) receive platelets.[7,10] Use of FFP and cryoprecipitate (a more concentrated source of fibrinogen) will depend on coagulation tests, and both products require liaison with a haematologist before administration.

In summary, for patients presenting with GI bleeding, it must be remembered that a normal haemoglobin and systolic blood pressure does not exclude a life-threatening haemorrhage that, if left unrecognized, may manifest with sudden circulatory collapse. Resuscitation must be prompt and effective, with early assistance from relevant expertise in critical care and gastroenterology/surgery. Inexperienced team members must have a low threshold for activating major haemorrhage protocols.

Coagulopathy and clotting

Use of anticoagulant and antiplatelet medication is widespread as either primary or secondary thromboembolic prevention. This inevitably results in increased risk of bleeding and presents an extra challenge in the management of iatrogenic coagulopathy in the setting of acute haemorrhage. During acute GI bleeding, anticoagulants and antiplatelets are often withheld whilst the primary haemorrhage is addressed, although there is some evidence of long-term cardiovascular harm resulting from this practice.[10] The following summary applies to both upper and lower GI

Classification of shock

	Class of haemorrhagic shock			
	I	II	III	IV
Blood loss (ml)	Up to 750	750–1500	1500–2000	>2000
Blood loss (% blood volume)	Up to 15	15–30	30–40	>40
Pulse rate (per minute)	<100	100–120	120–140	>140
Blood pressure	Normal	Normal	Decreased	Decreased
Pulse pressure (mmHg)	Normal or increased	Decreased	Decreased	Decreased
Respiratory rate (per minute)	14–20	20–30	30–40	>35
Urine output (ml/hour)	>30	20–30	5–15	Negligible
Central nervous system/mental status	Slightly anxious	Mildly anxious	Anxious, confused	Confused, lethargic

From 'ATLS – Advanced trauma life support. (2012). Chicago, Ill.: American College of Surgeons, Committee on Trauma'.

Table 1

bleeding, but exact management will vary between patients and even institutions.

In those taking warfarin and other vitamin K antagonists who experience life-threatening haemorrhage, warfarin is reversed with intravenous vitamin K and prothrombin complex concentrate (PCC) (Beriplex®). This contains human coagulation factors II, VII, IX, X and endogenous inhibitor proteins S and C and it is considered preferable to fresh frozen plasma (FFP) due to ease of administration, rapid normalization of INR and low risk of volume overload.[17] Guidance suggests that restarting warfarin seven days after bleeding minimizes both re-bleeding risk and thromboembolic events.[10]

In patients with high thrombotic risk (such as prosthetic mitral valve, atrial fibrillation with prosthetic heart valve or mitral stenosis, or venous thromboembolism within the last 3 months) LMWH can be considered at 48 hours if the patient is stable with normalized coagulation. If a patient has massive haemorrhage whilst prescribed LMWH or unfractionated heparin (UFH), protamine is used as a selective antidote.

There is also lack of evidence for stopping antiplatelets to reduce risk of re-bleeding. When used for secondary prevention, Oakland et al. suggest continuing aspirin, but when used for primary prevention, they suggest that aspirin can be stopped indefinitely. Liaison with cardiology will be required for patients taking dual antiplatelet therapy for coronary stents (commonly a P2Y12 antagonist such as clopidogrel, in addition to aspirin). If considered high risk for thromboembolism (within 12 months of drug eluting coronary stents, or within one month of bare metal stents), dual antiplatelet therapy − or aspirin alone − may have to be continued. Restarting drug therapy following the bleeding period should be prompt, but there is little evidence on timing. It must be remembered that discontinuing dual antiplatelet therapy in the at-risk period confers up to 40% risk of acute myocardial infarction or death.[18]

DOACs have a short half-life unless there is concomitant renal failure. Dabigatran inhibits thrombin, whereas most others inhibit factor Xa. Life-threatening bleeding will require liaison with haematology as reversal is complex and drug dependent. Idarucizumab (Praxbind®) is available for the reversal of dabigatran, and andexanet alfa (Ondexxya®) has been licensed for the reversal of apixaban and rivaroxaban in patients with life-threatening bleeding. If the specific reversal agents are not available, PCC can also be used, but always after liaison with a haematologist. Resuming anticoagulation or substituting for warfarin at seven days is recommended.[6] Tranexamic acid is commonly administered to patients presenting with GI haemorrhage. However, the landmark trial "Effects of high-dose 24 hour infusion of tranexamic acid on death and thromboembolic events in patients with acute GI bleeding" (HALT-IT) found no difference between tranexamic acid infusion and placebo for mortality, blood transfusion and re-bleeding.[19] With only a very small increase in venous thromboembolism, most guidelines no longer recommend tranexamic acid for GI bleeding.

In summary, administration of reversal agents and prothrombin complexes is determined by liaising with haematology, and for patients who are treated with anticoagulant or antiplatelet agents for a medical comorbidity, specialist cardiac or stroke advice is essential, both when considering the stopping of medication or upon restarting.

Upper GI haemorrhage

Upper GI bleeding is the more common GI haemorrhage, with an incidence greater than 100 per 100,000 population.[1,3] It is a medical emergency, with an overall mortality rate of 10%.[1,3] This has persisted since the first large UK study in 1995 (by the senior author of this article).[1,3] In the first, audit mortality was 14%, and has reduced slightly since then, remaining static at 10% since 2003.[4] Patients are typically admitted under the medical team that provide emergency oesophago-gastro-duodenoscopy (OGD, or simply 'endoscopy'), as this is the diagnostic and treatment modality of choice. It is not recommended that overall leadership and oversight of care for patients with GI bleeding is rigidly divided, however, upper GI haemorrhage differs to lower GI in that prompt upper GI endoscopy establishes control of bleeding in more than 95% of cases.[2]

Pathology

Peptic ulcers

Peptic ulcer disease is the most common cause for upper GI bleeding and accounts for approximately 31−67% of presentations.[12] Ulceration beyond the mucosa into the submucosa results in inflammation which causes weakening and necrosis of the arterial wall, and leads to pseudoaneurysm formation, rupture and haemorrhage. Up to 90% of duodenal ulcers and 70% of gastric ulcers are associated with infection of *Helicobacter pylori*. This Gram-negative bacterium causes disruption of the mucosal barrier resulting in inflammation and ulceration of the gastric and duodenal mucosa. Incidence of peptic ulcer disease has declined significantly since the identification of *H. pylori* and the widespread use of proton pump inhibitors (PPIs).[12,20]

NSAIDs are also associated with peptic ulcer disease, by inhibiting cyclooxygenase and decreasing mucosal prostaglandin synthesis. This results in an impaired mucosal barrier, and a 40-fold increased risk of gastric ulceration and eightfold increased risk of duodenal ulceration (up to 20% of long-term NSAID users will have mucosal ulceration). For this reason, NICE recommends that all NSAIDs are stopped during acute haemorrhage. The decision to restart after haemorrhage should be considered carefully on a case-by-case basis. If the benefit of treatment appears to outweigh the potential risk of further bleeding, then a prophylactic PPI should be prescribed concurrently, which reduces the risk of new peptic ulcer formation by 50%−80%.[7,12] Benign peptic ulcers are assessed endoscopically, where they are typically described as having smooth, rounded edges. The Forrest classification (Table 2) categorizes ulcers into three classes, which helps guide management and risk-stratifies those patients at high risk of re-bleeding and mortality. Any ulcer other than a 2c or 3 is considered high risk.[13]

Gastritis, duodenitis and oesophagitis

Stress gastritis is commonly seen in critically unwell inpatients and typically results from disruption to mucosal defences (ordinarily maintained by mucus, bicarbonate and prostaglandins that protect the gastric mucosa from the intra-luminal acidic content). NICE recommends routine use of PPI for prophylaxis in critically ill patients admitted to intensive care.[7,20] Patients at risk of oesophagitis tend to have a history of gastrooesophageal reflux disease and a hiatus hernia may be present. The stratified

Forrest classification

Class	Description	Endoscopic Intervention	Rebleeding rate
1A	Active spurting	Yes	55%
1B	Active oozing	Yes	55%
2A	Nonbleeding visible vessel	Yes	43%
2B	Adherent clot	Consider	22%
2C	Flat pigmented spot	No	10%
3	Clean ulcer base	No	5%

Adapted from: Nelms, D. W. & Pelaez, C. The Acute Upper Gastrointestinal Bleed. Surg Clin North Am 98, 1047–1057 (2018).

Table 2

squamous epithelium that lines the oesophagus lacks many of the mucosal defences that protect against the caustic effects of gastric acid. Increased acid exposure and reflux of gastric contents into the distal oesophagus results in the same inflammation and erosion that results in haemorrhage associated with peptic ulceration.

Mallory–Weiss syndrome

Longitudinal lacerations in the gastric cardia or at the gastro-oesophageal junction account for 4%–8% of upper GI bleeding and occur as a result of a sudden increase in intra-abdominal pressure (usually during vomiting, e.g., during hyperemesis of pregnancy, or following alcohol intake or food poisoning). Spontaneous resolution of bleeding is common, and intervention is only required in 10% of cases. Lesions not actively bleeding can be managed with PPI and anti-emetic medication alone; rebleeding is rare (7%).[11,20] This should be differentiated from Boerhaave syndrome (oesophageal rupture), where there is a full thickness tear of the oesophageal wall following increased intra-oesophageal pressure (also caused by vomiting); bleeding is less common.

Malignancy

Tumours of the upper GI tract rarely present with acute haemorrhage and only form approximately 4–8% of acute upper GI bleeds. Tumours are often asymptomatic until a late stage, therefore at presentation disease is often advanced. Nevertheless, if ulcers and lesions appear suspicious (elevated, irregular borders with associated abnormal mucosal folds), biopsy should be undertaken, as approximately 6% of gastric ulcers contain underlying malignancy. It is also necessary to repeat the endoscopy after 6–8 weeks as a non-healing ulcer is suspicious for an underlying malignancy. Duodenal ulcers are less commonly malignant.

Dieulafoy lesions/vascular ectasia

These are a rare cause of upper GI haemorrhage. They are large, but histologically normal, arterioles which protrude through the submucosa and mucosa and can occur anywhere in the GI tract – however typically they are found on the lesser curve of the stomach and within 6 cm of the gastro-oesophageal junction. Exposure to the acidic intra-luminal environment can result in necrosis and rupture of the affected arteriole, resulting in sudden, brisk bleeding in a patient with no other significant symptoms or risk factors.[13,20]

Post-surgical intervention

Specialist hepatobiliary, upper GI and vascular centres will have preferred treatment strategies based on expertise in specific radiological, endoscopic or surgical techniques, and clinicians caring for such patients should familiarize themselves with local treatment options. Haemobilia can result from any hepatopancreaticobiliary tract procedure (e.g. endoscopic retrograde cholangiopancreatography (ERCP)), following trauma, or as a complication of pancreatitis (hemosuccus pancreaticus). Endoscopic control of biliary bleeding is often difficult to establish, therefore computed tomography angiography (CTA) followed by angiographic embolization or surgery, might be appropriate.[20] GI bleeding post-hepatobiliary surgery is not uncommon, for example bleeding from the gastroduodenal artery (GDA) stump after pancreaticoduodenectomy (a Whipple's procedure). This is often managed by radiological embolization of the GDA.

Aorto-enteric fistulae can occur following vascular intervention to the aorta or GI tract, and often there is a 'herald bleed' followed by massive exsanguinous haemorrhage. Urgent radiological investigation may be helpful in such vascular complications, but immediate reconstructive surgery is often indicated, with extra-anatomic bypass reconstruction, removal of any infected synthetic material (e.g. aortic graft) and closure of any enterotomy. Perioperative mortality is inevitably extremely high.[11] This diagnosis must also be considered for patients with negative endoscopic findings but prior history of vascular surgery.

Staple line bleeding

Bleeding is a recognized complication of procedures such as laparoscopic sleeve gastrectomy. This may present with haematemesis or signs of intra-abdominal haemorrhage. Similarly, intragastric balloon placement may cause Mallory-Weiss like bleeding or ulceration requiring urgent endoscopic removal of the balloon. In such cases, resuscitation followed by discussion with a bariatric service is essential. Once stable, the patient will usually require transfer to allow definitive treatment in a centre that is able to provide appropriate anaesthetic and high-dependency care. Similarly, bleeding following oesophagogastric surgery, whether open, laparoscopic or robotic should be managed as any surgical complication, in tandem with haemorrhage resuscitation, and urgent surgical expertise sought from a regional oesophagogastric service as to the suitability of radiological, endoscopic or surgical investigation.

Variceal bleeding

Accounting for between 4% and 20% of upper GI bleeding, varices are abnormally dilated veins which occur as a result of portal hypertension and development of portosystemic shunts, commonly found in the distal oesophagus and proximal stomach. Most cases are secondary to cirrhosis, but rarely varices may be caused by non-cirrhotic portal hypertension (veno-occlusive disease) or portal vein thrombosis. Gastric varices are further subdivided into gastro-oesophageal varices and isolated gastric varices depending upon their anatomical location.[7,21] The management of patients with varices differs from those with non-variceal bleeding, as variceal bleeding is associated with a 20% risk of mortality within 6 weeks following the first episode of variceal bleeding. On presentation, patients with suspected varices should be treated with a splanchnic vasoconstrictor, continued until definitive haemostasis is achieved or until after 5 days following presentation, with caution taken for those with ischaemic heart disease or peripheral vascular disease.[15] NICE and British Society of Gastroenterology (BSG) guidelines currently recommend terlipressin, however some authors recommend a somatostatin analogue (octreotide), which is licensed in North America.[12,15] Patients with variceal bleeds are at high risk of bacterial infection and antibiotics have been shown to reduce the risk of re-bleeding, infection and overall mortality, therefore prophylactic broad-spectrum antibiotics are appropriate.[15]

Pre-endoscopic care

Timing of endoscopy

The haemodynamically unstable patient with evidence of active bleeding should undergo endoscopy after initial resuscitation, and all patients requiring admission should receive endoscopy within 24 hours. UK hospitals should have access to 24-hour endoscopy services and an on-call endoscopy team for GI bleeding.[2] Critical care opinion must be sought if the patient does not respond to initial resuscitation, and endoscopic investigation under general anaesthetic in theatre may be required, with inotropic and other critical care support provided. There is an association between endoscopy performed more than 24 hours after admission and increased risk of mortality, evidencing the need for early intervention, ideally within 6–24 hours.[22] Even after a period of stabilization, if the patient further deteriorates, immediate repeat intervention is necessary. Correction of abnormal coagulation should not delay endoscopy if bleeding is life threatening.[9,12,13] While most endoscopy is performed in a dedicated endoscopy suite, emergency or out-of-hours endoscopy is commonly performed either in the emergency operating theatre or even at the bedside in intensive care. The management plan formulated by the endoscopist needs communicating to the clinical team responsible for continuity of care and written or verbal communication must be promptly reviewed upon the patient's return to the bed space. Pre-endoscopic nasogastric drainage is not certain to offer benefit.[13]

Risk stratification

Given the associated morbidity and mortality associated with acute upper GI haemorrhage and re-bleed rate of approximately 5–20%, even after successful endoscopic intervention, it is essential to risk-stratify patients to identify those at high risk of serious adverse events. This allows prediction of further endoscopic intervention and aides triage of patients. Current NICE guidelines advocate a two-step risk assessment for the assessment of acute upper GI haemorrhage.[7] Prior to endoscopy and within 24 hours of admission, the Glasgow–Blatchford score (Table 3) is used to risk-stratify patients. Low-risk patients (scoring 0) may be appropriately discharged with suitable further outpatient investigation. The higher the Blatchford score, the greater the risk of adverse clinical outcome and more urgent endoscopy should be considered. After endoscopy, NICE recommends use of the full Rockall scoring system (Table 4) to predict risk of re-bleeding or mortality following endoscopic intervention; a score of more than 2 indicates increased risk. All patients with GI haemorrhage should have an agreed re-bleed plan, which should be based on individual risk and pathology. This second stage risk stratification helps clinical teams anticipate further treatment and intervention and will usually involve repeat endoscopy.[7]

Proton pump inhibitors (PPIs)

These medications act by irreversibly blocking the H^+/K^+ gastric proton pump in gastric parietal cells, preventing the luminal secretion of H^+ ions, and reducing up to 99% of gastric acid production. The use of PPIs in acute upper GI haemorrhage prior to endoscopy remains a controversial topic. Most guidelines agree that following an endoscopic diagnosis of ulcerative disease with at risk features, high-dose PPI is recommended. The European Society of GI Endoscopy (ESGE) recommends

Glasgow–Blatchford criteria

Criteria (on admission)		Score
Hb – Male (g/litre)	**Hb – Female (g/litre)**	
120–130	100–120	1
100–120		3
<100	<100	6
Urea (mmol/litre)		
6.5–8		2
8–10		3
10–25		4
≥25		6
Systolic blood pressure (mmHg)		
100–109		1
90–99		2
<90		3
Others		
Pulse ≥100		1
Melaena		1
Syncope		2
Hepatic disease		2
Cardiac failure		2

From: Blatchford O, Murray W, Blatchford M. A risk score to predict need for treatment for upper gastrointestinal haemorrhage. Lancet 2000; 356: 1318–21. Hb, haemoglobin.

Table 3

Post-endoscopy Rockall score

Criteria (on admission)	Score
Age*	
<60	0
60–79	1
≥80	2
Shock*	
Pulse >100	1
Systolic blood pressure <100 mmHg	2
Comorbidity*	
Cardiac, other major	2
Renal/liver failure, cancer	3
Endoscopic diagnosis	
Normal, Mallory–Weiss	0
Ulcer, erosion, oesophagitis	1
Cancer	2
Endoscopic SRH	
Clean base ulcer, flat pigmented spot	0
Active bleeding, clot, vessel, blood	2

*Denotes components of pre-endoscopy Rockall Score. From: Rockall T, Logan R, Devlin H et al. Risk assessment after acute upper gastrointestinal haemorrhage. Gut 1996; 38: 316–21. SRH, stigmata of recent haemorrhage.

Table 4

intravenous high-dose PPI (omeprazole 80 mg/hour) be given as a bolus on presentation, followed by continuous infusion (omeprazole 8 mg/hour) for all patients requiring admission.[23] This recommendation is refuted by NICE who advise against offering PPI prior to emergency endoscopy, citing lack of evidence that it reduces re-bleeding rate or mortality, and that it may downgrade underlying pathology and delay definitive endoscopic management.[7] NCEPOD found that 73% of patients with acute bleeding received PPI, indicating a clear disparity between national standards and clinical practice.[2] Post-endoscopy, NICE does recommend commencing a PPI in those with stigmata of recent haemorrhage.[7]

Prokinetics

ESGE recommends intravenous erythromycin (250 mg) 30–120 minutes prior to gastroscopy to improve mucosal visualization, by accelerating gastric emptying. There is no evidence to support the use of metoclopramide.[12,13,23]

Endoscopic management

Non-variceal upper GI bleeding

NICE recommends one of the following methods to achieve haemostasis:

- Mechanical treatment: direct compression of a bleeding vessel using a designed haemostatic device such as an endoscopic clip (endoclip)
- Thermal coagulation (with or without adrenaline injection): using either contact thermal haemostasis (monopolar diathermy) or non-contact (such as argon plasma coagulation; especially useful in the management of angiodysplasia)
- Fibrin or thrombin treatment (with or without adrenaline injection): compounds which may be applied over a large area. These substances mechanically adhere to bleeding points and activate coagulation factors. They are useful when managing large areas of oozing such as in gastritis, malignancy, or portal hypertensive gastropathy.[9,11,12,15,20]

Injection of adrenaline alone has been demonstrated to be inferior to the above methods; however, it can be utilized as an adjunct. Several novel modalities have been proposed as either an adjunct or a therapeutic alternative to treat non-variceal GI bleeding. Products such as Hemospray® (a haemostatic powder spray) (Cook Medical, USA) have been advocated, particularly in areas difficult to access endoscopically using traditional techniques (such as the lesser curve of the stomach, posterior bulb of the duodenum and gastric cardia). However, there are few randomized, prospective studies.[15,24]

Variceal upper GI bleeding

NICE and the British Society of Gastroenterology (BSG) recommend variceal band ligation (VBL) for oesophageal varices. This involves deployment of a small rubber band around the varices to induce strangulation and thrombosis of the vessel. Following the procedure some patients may develop ulceration at the site of deployment, but this may be reduced with a PPI.[21,24]

Gastric varices should be offered N-butyl-2 cyanoacrylate injection as first-line therapy. Commonly referred to as glue, this strongly adhesive substance is injected into bleeding varices, and has been found to be superior to VBL for achieving haemostasis and reducing re-bleed rates in this sub-cohort of patients. Thrombin injection may also be used for this purpose and has a reported haemostasis rate of 94% with a re-bleed rate of 18%.[7,13]

When these methods fail to adequately achieve haemostasis, it may be necessary to consider a second-line endoscopic technique to control haemorrhage. Balloon tamponade (e.g. Sengstaken–Blakemore tube) can be used in most cases of oesophageal and junctional variceal haemorrhage. These are successful in controlling haemorrhage in 91% of cases, but re-bleed rates are high (approximately 50%), which means they are often utilized in a temporizing manner and should be removed after 2 days. The tubes are poorly tolerated and may result in pressure necrosis, aspiration pneumonia and, rarely, oesophageal perforation. Patients should be intubated and monitored in an intensive care setting. Self-expanding metal stents have been used for the same purpose and can remain in place for 14 days.[7,23]

Use of Hemospray® and other novel haemostatic techniques have been trialled in the management of variceal bleeding, but more studies are required before such techniques are implemented routinely.[7,13]

Interventional radiology

According to NICE, interventional radiology should be considered for an unstable patient who has re-bled after endoscopic treatment. However, if there is high risk of re-bleeding or doubt about haemostasis at initial endoscopy, or evidence of re-bleeding after initial control, further endoscopy should be planned in the first instance.[7] North American guidelines suggest that CTA should be considered in specific circumstances – arterial bleeding that cannot be controlled endoscopically, unclear

source of bleeding, or negative endoscopic findings.[25] Similarly, in unique circumstances such as the early postoperative period following upper GI or bariatric surgery, or after trauma, it may be preferred to proceed to radiological management over endoscopy. Such cases will require discussion with senior endoscopy and interventional radiology clinicians before progressing to radiological investigation and onward treatment.

It must be remembered that interventional radiology services are not available out of hours in every hospital, whereas following the NCEPOD and upper GI bleed audit, every hospital should be able to provide emergency endoscopy.[2] CTA with triple phase contrast (arterial, portal venous, and delayed phase) is available in all radiology departments with CT, but onward radiological intervention with angiography may require transferring a patient between centres and is not without risk. An unstable patient may progress to repeat endoscopic or even surgical management if interventional radiology is not readily available.

Angiography has a sensitivity of 86% and specificity of 95% for obscure GI blood loss and may be used to help identify vascular malformations, neoplasms, and can exclude small and large bowel sources of bleeding. However, in order to accurately identify the source of blood loss, patients must be bleeding at a rate of 0.5 ml/minute.[9,10,25] If a bleeding source is identified, then the interventional radiologist may attempt selective angiography of the mesenteric vessels and radiological embolization where appropriate. The most common source of bleeding and target for embolization in upper GI bleeding is the gastroduodenal artery. Haemostasis is achieved using coils, however several other products including polyvinyl alcohol particles and Gelfoam® are available.

Angiographic embolization may result in abdominal pain, ischaemia, arterial injury, and contrast induced nephropathy, but is considerably less morbid than traditional surgical salvage. Some authors describe superselective catheterization of the bleeding vessel, embolization of arteries less than 1 mm, or avoidance of larger bore products such as Gelfoam® as reducing the risk of bowel ischaemia.[26] In the case of bleeding varices, when endoscopic control has failed, a trans-jugular intrahepatic portosystemic shunt (TIPSS) procedure can be undertaken. This procedure involves radiologically guided deployment of a stent bridging the portal and hepatic veins, creating a portosystemic shunt across the liver parenchyma, resulting in rapid reduction in portal pressure. These procedures are performed in specialist centres and there are several considerations prior to treatment. Early liaison with a specialist hepatobiliary centre and interventional radiology service is essential when standard endoscopic management has failed.[7,13]

Surgery

Historically, surgery was considered to be a reasonable treatment option for the patient with upper GI haemorrhage, particularly in the pre-endoscopic era. However, surgery is now the least frequently utilized treatment modality, when all other means to control haemorrhage have failed. Some authors have specified failed or unavailable endoscopic or radiological intervention, co-existing pathology such as perforation, or massive uncontrolled haemorrhage as indications for surgical management.[27] Improvements in endoscopic management and increased availability of interventional radiology have seen a fall in surgery for all GI bleeding of 50% over 10 years.[27] The 2007 BSG audit reported that only 2.3% of patients underwent surgical management of uncontrolled haemorrhage.[4] The surgical approach depends upon the origin and underlying pathology causing haemorrhage (Table 5). The most common surgical procedure is under-running or over-sewing of a bleeding duodenal or gastric ulcer, and once haemostasis is obtained, definitive ulcer treatment is usually medical. If there is associated perforation, gastric or duodenal resection with reconstruction may be required, which may require temporizing surgery before upper GI expertise or transfer is obtained.[27] Mortality following surgery is high (29%) and has remained static.[13,20]

Follow-up/ongoing care

There is evidence to suggest that early nutrition is safe and may reduce length of stay compared to delayed reintroduction of nutrition post-upper GI bleeding.[28] Intravenous PPI is recommended for 72 hours after successful haemostasis or where there are stigmata of recent haemorrhage with no active bleeding observed.[7] Long-term primary and secondary PPI prophylaxis can be used; however, side effects include increased risk of hip fracture, *Clostridium difficile* infection, pneumonia, and other symptoms such as diarrhoea. It is essential during endoscopy

Historically described surgery for upper gastrointestinal bleeding

Disease process	Surgical options
Peptic ulcer	Oversew
	Three-point ligation of gastroduodenal artery
	Vagotomy and pyloroplasty
	Vagotomy and antrectomy
	Highly selective vagotomy
Mallory–Weiss tear	Oversew
Dieulafoy lesion	Oversew
	Wedge resection
Varices	Portacaval shunt
	Mesocaval shunt
	Distal splenorenal shunt
Gastric cancer	Distal gastrectomy
	Total gastrectomy
	D2 lymphadenectomy
Haemobilia	Selective ligation
	Resection of aneurysm
	Non-selective ligation
	Liver resection
Aortoduodenal fistula	Angiography and stent (if haemodynamically stable)
	Open repair
	Extra-anatomic bypass

From Feinman, M. & Haut, E. R. Upper gastrointestinal Bleeding. Surg Clin North Am 94, 43–53 (2014).

Table 5

that a *Campylobacter*-like organism (CLO) test is performed and, if positive, *H. pylori* eradication treatment commenced. *H. pylori* eradication is linked to improved outcomes, reduced rates of re-bleed and if commenced immediately, improved compliance is observed. Patients should be re-tested for *H. pylori* at least 6 weeks after initial positive test, and 2 weeks after completing the course of PPI. Testing for the presence of *H. pylori* whilst taking PPI treatment may result in a false-negative result. Retesting can be performed via urease breath test or increasingly via a stool antigen test.[7]

Following variceal bleeding, repeat endoscopy is recommended at frequent intervals as recurrent varices may warrant further band ligation. A non-selective β-blocker such as propranolol or carvedilol may be used to reduce portal pressure by means of splanchnic vasoconstriction and a reduction in cardiac output. These have no role in preventing the development of varices, but may be useful for prevention of bleeding in patients with established cirrhosis and varices.[21] All patients with portal hypertension should be referred to a hepatology service for ongoing management; screening for hepatocellular carcinoma and surveillance will also need to be established alongside these treatment options.[7]

Lower GI haemorrhage

Lower GI haemorrhage is less common, with an incidence of 33–77 per 100,000 population. This group forms approximately 3% of all acute surgical admissions.[6,10] Small volume lower GI bleeding is much more common in the population and likely to be assessed by general practitioners. Unlike upper GI haemorrhage, 80% of lower GI bleeding stops spontaneously after initial resuscitation and correction of coagulopathy with only a small proportion of patients requiring intervention.[6] In most UK hospitals, lower GI haemorrhage is referred to emergency general surgery; however, when the endoscopy suite is required, liaison with gastroenterology will be needed.

Overall mortality from lower GI bleeding is 3.4%, and mortality is generally related to comorbidity rather than gross exsanguination.[6,10] It is essential to exclude an upper GI source of blood loss, with rapid transit of blood through the GI tract. Where there is any doubt regarding origin of haemorrhage, particularly in the haemodynamically compromised patient with blood per rectum, rapid assessment for upper GI bleeding and consideration of upper GI endoscopy with push enteroscopy (allowing examination 50–100 cm beyond the ligament of Treitz into distal duodenum and proximal jejunum) can take place if expertise and equipment allows. The consequence of missing a true upper GI haemorrhage can be catastrophic, and occurs in 15% of patients with assumed lower GI bleeding.[29] For approximately 10% of patients with a lower GI bleed, the source of the bleeding is never identified. Where patients remain haemodynamically stable, there is the option to repeat investigations or to progress to more specialized tests including ^{99m}Technetium-labelled red blood cell scintigraphy, CT or magnetic resonance enterography, video capsule endoscopy, push enteroscopy or double balloon enteroscopy.[5] These investigations are of value when obscure small intestinal bleeding is suspected, but availability is limited and may require specialist referral, therefore are beyond the scope of most guidelines.[5]

Pathology

Diverticular bleeding

This is the most common cause for lower GI bleeding, accounting for approximately 30–65% of cases in Western populations.[6] Diverticular disease is increasingly common with advancing age, but patients in their early 30s may present with it. The cause is uncertain, but traditionally was thought to be related to lack of fibre and lifestyle factors, such as smoking and obesity in western countries. Increasingly, it is thought of as part of the inflammatory bowel disease spectrum and may have some heritability. Bleeding occurs as a complication when small vessels in the wall of a diverticulum are eroded, most prevalent in the sigmoid and descending colon. Incidence of re-bleeding after a single diverticular bleed is low (15%) but is much higher after subsequent bleeding (approximately 50% of patients with two episodes will have a further bleed).[26,30] At endoscopy, North American guidelines suggest stigmata of haemorrhage from colonic diverticulum is treated with clips, band ligation, or coagulation.[31] Diverticula of the small bowel, commonly jejunum, may occasionally manifest with rectal haemorrhage. Diagnosis usually requires radiological imaging; both upper and lower GI endoscopy can be negative. Such cases, although rare, support the need for progressing quickly to urgent radiological imaging in patients who have not had a diagnosis made at endoscopy.

Vascular abnormalities

Angiodysplasia is responsible for approximately 5–10% of acute lower GI bleeding. Angiodysplastic lesions mainly affect the caecum and ascending colon but can be small bowel in 15% of patients. These result from abnormally dilated mucosal capillaries communicating with tortuous and dilated submucosal veins, and have a typical flat, red, regular bordered appearance at endoscopy. Incidence increases with age. Although spontaneous resolution is high (90%), there is a relatively high re-bleed rate with 26% of patients having re-bled after 1 year and 45% after 3 years. Other vascular abnormalities in the lower GI tract include varices (commonly rectal), and Dieulafoy lesions, however the incidence is rare (<3%).[26,27,30]

Neoplasms and polyps

Colorectal cancer is the fourth most common cancer in the UK with 42,000 new diagnoses per year. Rectal bleeding is a high-risk symptom that warrants urgent investigation. Colorectal tumours can present with bleeding, usually slow in nature, although it is important to note that benign polyps can also present with haemorrhage. Fresh red rectal bleeding is mostly associated with left-sided tumours (accounting for >60% of cancers). Overall, neoplasms and polyps are responsible for 2–15% of acute lower GI bleeding.[6]

Colitis

Ischaemic colitis accounts for approximately 5–20% of presentations. It may affect any part of the colon but typically affects the vascular watershed area of the splenic flexure. This area of colon is supplied by the marginal artery of Drummond, which bridges the middle colic (from the superior mesenteric) and left colic (from the inferior mesenteric) arteries. The condition is thought to be caused by inadequate blood supply to the affected colonic wall, leading to erosive lesions and secondary bleeding.

There are several risk factors for the development of ischaemic colitis (which may result from either arterial or venous hypoxia), however the condition is broadly divided into occlusive and non-occlusive disease, and may be thrombotic or embolic in origin. Management is dependent on the degree of ischaemia, with full-thickness necrosis and gangrene indicating the need for surgical resection; however, in most cases the condition will be transient, and resolution of symptoms observed with appropriate non-operative management. Less commonly, inflammatory colitis (encompassing inflammatory bowel disease, mainly ulcerative colitis) and infective colitis may also result in catastrophic GI haemorrhage. In a patient with lower GI haemorrhage who had previously undergone pelvic irradiation (e.g. for prostate cancer), radiation proctitis or colitis can occur months or years after completing treatment.

Meckel's diverticulum

Often referred to by the rule of two's (under 2 inches in length, within 2 feet from the ileocaecal valve, affecting 2% of the population, typically presenting under the age of 2 and containing two types of heterotrophic mucosa), a Meckel's diverticulum is the most common congenital malformation in the GI tract. They may present with lower GI haemorrhage originating from the distal small bowel, as a result of acidic secretions from ectopic gastric mucosa causing ulceration.[26] The anatomical location means upper and lower GI endoscopy may not reveal the diagnosis, and CT angiography or even surgery may be required in such cases.

Anal lesions and post-procedure bleeding

The management of anal lesions (most commonly haemorrhoids; 5−20%), post-polypectomy (2−7%) or postoperative bleeding can be identified from adequate history and examination.[6] As with other causes of lower GI bleeding, lesions within the anal canal will often spontaneously stop. Where active bleeding is observed during examination (including with proctoscope or rigid sigmoidoscope), attempts can be made at haemostasis through direct application of pressure, cautery or through sutures. This may need to be done under general anaesthesia − blind suturing of the anal canal should be avoided, and instead appropriate input from a coloproctologist should be sought.

Where control in this manner fails, or where another source of bleeding is suspected, patients should be managed in the same manner as a more proximal cause. In the patient who has had a recent polypectomy, there is no need for radiological investigation before proceeding to colonoscopy, which is the diagnostic and therapeutic modality of choice according to BSG guidelines.[10] Endoclips with or without adrenaline are recommended to control bleeding. Heater probe and bipolar diathermy anywhere other than in the rectum (below the peritoneal reflection) should be used with caution as the bowel wall is thinner and at increased risk of perforation following polypectomy.[10,15]

Bleeding following right hemicolectomy with a stapled ileocolonic anastomosis is common and can manifest with significant amounts of brisk lower GI blood loss originating from the staple line. This is usually in the immediate postoperative period, but occasionally patients may be readmitted after discharge. Supportive treatment via resuscitation with blood products, and correction of coagulopathy is recommended as first-line treatment, but if haemorrhage is not controlled, re-operation may be necessary.

In the patient presenting with bleeding per-stoma, digital examination, and thorough assessment of the stomal orifice should be undertaken. Similarly, patients with a defunctioning stoma can present with rectal bleeding originating from the distal rectum or anal canal, especially if radiotherapy has been used. If a local cause is not found, then the patient should be managed the same as all others, with note made of the type of previous surgery and presence or not of rectum and anal canal. Such background should be communicated clearly to the endoscopist or radiologist when undertaking further investigations, and specific equipment requested for stomal endoscopic examination.

Risk stratification

BSG guidelines recommend a two-stage approach to the assessment of the patient with lower GI bleeding.[10,15] Initially the patient should be assessed for signs of haemodynamic instability and shock, which indicate emergent investigation for potential definitive management. Haemodynamically stable patients or those with a Shock Index (SI) of 1, should be assessed using the Oakland score (Table 6). This system allows identification of patients who are unlikely to suffer a serious adverse event (score 8).[10] In the absence of any other indications for admission, this score also calculates safe discharge for expedited outpatient investigation. The Oakland score is validated and specific to lower GI bleeding in the UK and is superior to other risk assessment tools in predicting safe discharge, transfusion requirements and re-bleed, but is inferior to other scoring systems in predicting mortality.

Management

CT angiography and interventional radiology

When there is haemodynamic instability or where active bleeding is suspected, BSG and North American guidelines suggest that CTA should be performed.[10,31] When a bleeding source is identified through extravasation (blush) of contrast, if local expertise is available, patients may then undergo targeted intervention through angiographic embolization. Alternatively if this is not available on-site, once CTA has identified active bleeding, onward transfer to a centre that can offer angiographic embolization may be prudent if the patient can be stabilized. If a patient is unstable or the intervention would be delayed (e.g. due to proximity of the nearest interventional centre), such findings will give an indication to the endoscopist of the origin of bleeding and can help guide endoscopic intervention locally. Again, it must be reiterated that if CT angiography is negative, an upper GI bleeding source must be considered, and such patients should proceed to endoscopy. There are no high-quality studies comparing embolization and endoscopic intervention in the management of lower GI bleeding. Empirical arterial embolization may also be beneficial, even where no active bleeding is seen, and this may be particularly useful when dealing with bleeding from a tumour. There are risks associated with embolization including bowel ischaemia (7−24%) and rebleeding (10−50%). Empirical embolization is associated with a higher 30-day mortality (31%) compared to targeted treatment (9%).[32] BSG guidelines therefore conclude that the decision to proceed to

Oakland score

Predictor	Score component value
Age	
<40	0
40–69	1
≥70	2
Gender	
Female	0
Male	1
Previous LGIB admission	
No	0
Yes	1
ORE findings	
No blood	0
Blood	1
Heart rate	
<70	0
70–69	1
90–109	2
≥110	3
Systolic blood pressure	
<90	5
90–119	4
120–129	3
130–159	2
≥160	0
Haemoglobin (g/litre)	
<70	22
70–89	17
90–109	13
110–129	8
130–159	4
≥160	0

Patients scoring ≤8, with no other indications for hospital admission are suitable for immediate discharge from accident and emergency and outpatient investigation. ORE, digital rectal examination; LGIB, lower gastrointestinal bleeding.
From Oakland, K. et al. Diagnosis and management of acute lower gastrointestinal bleeding: guidelines from the British Society of Gastroenterology. Gut; 68: 776–89 (2019).

Table 6

embolization or primary therapeutic colonoscopy following CTA should be based on local expertise and patient factors.[10]

A systematic review of embolization for lower GI haemorrhage reported 97% overall success, 21% re-bleed and 7% ischaemia rates (most were minor).[32,33] A patient who has undergone embolization of any section of the GI tract should be monitored for signs of significant end-organ ischaemia, which could require operative management. However, routine colonoscopy to assess this is not necessarily required in cases of lower GI bleed treated with selective embolization.[33]

Endoscopy (flexible sigmoidoscopy and colonoscopy)

Identification of a bleeding point and ability to achieve haemostasis is often more challenging due to limited views of the unprepared bowel (by the presence of faecal matter and blood). It may be possible to give the patient bowel preparation or an enema to improve visualization, but views may still be unsatisfactory, with widely ranging reports of diagnostic yield. The timing of endoscopic intervention remains controversial with many conflicting studies; however, BSG guidelines suggest that for patients with evidence of major bleeding, this should take place on the next available list and within 24 hours after admission to hospital.[10] Conversely, North American guidelines suggest non-emergent inpatient colonoscopy is sufficient due to lack of improved clinical outcomes (re-bleeding and mortality).[31]

The endoscopic management of lower GI bleeding includes using the same haemostatic techniques as used in acute upper GI haemorrhage, namely mechanical treatment, thermal coagulation and fibrin or thrombin products. No one technique appears superior to another, however the BSG recommends the use of mechanical clips as first-line modality in diverticular bleeding due to low risk, widespread availability, and ease of use.[10] Angiodysplastic lesions may be more amenable to thermal coagulation.[30] CO_2 and gas exchange should be used, and diathermy and argon plasma coagulation limited in this setting due to the risk of gas explosion. Sub-mucosal infiltration of adrenaline may be useful in obtaining initial haemostasis, but its use should be limited in the rectum and anal canal due to the risk of migration into the systemic circulation.[30]

Surgery

In limited circumstances, such as catastrophic post-surgical bleeding, return to the operating theatre is the treatment of choice; otherwise, it should be considered a salvage option, only to be used when other means to control haemorrhage have failed. Where there is an identified colonic bleeding source, but haemorrhage control has failed, a segmental colectomy may be indicated. This also allows treatment of any colonic inflammatory disease process, such as ischaemic colitis or diverticulitis. Where no identifiable bleeding source is identified, blind subtotal colectomy has been reported, but with poor outcomes.[34] On-table entero-endoscopy through a small bowel enterotomy can be technically challenging; radiological localization preoperatively with CTA is more likely to yield successful surgical intervention.[27,35] Occasionally, laparoscopy or diagnostic laparotomy may be indicated from radiological and endoscopic investigations and after discussion between relevant specialists; but this is no longer first line management of lower GI haemorrhage.

Summary

Massive GI haemorrhage is a common emergency presentation and is associated with significant risk of mortality. Outcomes can be improved by rapid assessment and resuscitation, correction of coagulopathy, and early diagnosis and intervention using endoscopy and radiology expertise.

Endoscopy and interventional radiology (CTA followed by embolization) are now the investigative and therapeutic modalities of choice, with surgery only considered as a last resort when other treatment strategies have failed. ◆

REFERENCES

1 Rockall TA, Logan RF, Devlin HB, et al. Incidence of and mortality from acute upper GI haemorrhage in the United Kingdom. Steering committee and members of the national audit of acute upper GI haemorrhage. *BMJ* 1995; **311:** 222–6.

2 NCEPOD. Time to Get Control? A review of the care received by patients who had a severe GI haemorrhage. 2015. London, https://www.ncepod.org.uk/2015gih.html

3 Rockall TA, Logan RF, Devlin HB, et al. Variation in outcome after acute upper GI haemorrhage. The national audit of acute upper gastrointestinal haemorrhage. *Lancet* 1995; **346:** 346–50.

4 Hearnshaw SA, Logan RFA, Lowe D, et al. Acute upper gastrointestinal bleeding in the UK: patient characteristics, diagnoses and outcomes in the 2007 UK audit. *Gut* 2011; **60:** 1327–35.

5 Murphy B, Winter DC, Kavanagh DO. Small bowel gastrointestinal bleeding diagnosis and management-A narrative review. *Front Surg* 2019; **6:** 25.

6 Oakland K, Guy R, Uberoi R, et al. Acute lower GI bleeding in the UK: patient characteristics, interventions and outcomes in the first nationwide audit. *Gut* 2018; **67:** 654–62.

7 Dworzynski K, Pollit V, Kelsey A, et al. Management of acute upper gastrointestinal bleeding: summary of NICE guidance. *BMJ* 2012; **344**: e3412.

8 Norfolk D. Handbook of transfusion medicine. 5th ed. Norwich: TSO, 2013.

9 Adler DG. Assessment of upper gastrointestinal bleeding. 2021. London, https://bestpractice.bmj.com/topics/en-gb/456

10 Oakland K, Chadwick G, East JE, et al. Diagnosis and management of acute lower gastrointestinal bleeding: guidelines from the British Society of Gastroenterology. *Gut* 2019; **68:** 776–89.

11 Nelms DW, Pelaez CA. The acute upper gastrointestinal bleed. *Surg Clin North Am* 2018; **98:** 1047–57.

12 Sverdén E, Markar SR, Agreus L, et al. Acute upper gastrointestinal bleeding. *BMJ* 2018; **363**: k4023.

13 Siau K, Chapman W, Sharma N, et al. Management of acute upper gastrointestinal bleeding: an update for the general physician. *J R Coll Physicians Edinb* 2017; **47:** 218–30.

14 El-Kersh K, Chaddha U, Sinha RS, et al. Predictive role of admission lactate level in critically ill patients with acute upper gastrointestinal bleeding. *J Emerg Med* 2015; **49:** 318–25.

15 Siau K, Hearnshaw S, Stanley AJ, et al. British Society of Gastroenterology (BSG)-led multisociety consensus care bundle for the early clinical management of acute upper gastrointestinal bleeding. *Frontline Gastroenterol* 2020; **11:** 311–23.

16 Docherty AB, O'Donnell R, Brunskill S, et al. Effect of restrictive versus liberal transfusion strategies on outcomes in patients with cardiovascular disease in a non-cardiac surgery setting: systematic review and meta-analysis. *BMJ* 2016; **352**: i1351.

17 Witt DM, Nieuwlaat R, Clark NP, et al. American Society of Hematology 2018 guidelines for management of venous thromboembolism: optimal management of anticoagulation therapy. *Blood Adv* 2018; **2:** 3257–91.

18 Iakovou I, Schmidt T, Bonizzoni E, et al. Incidence, predictors, and outcome of thrombosis after successful implantation of drug-eluting stents. *JAMA* 2005; **293:** 2126–30.

19 HALT-IT Trial Collaborators. Effects of a high-dose 24-h infusion of tranexamic acid on death and thromboembolic events in patients with acute gastrointestinal bleeding (HALT-IT): an international randomised, double-blind, placebo-controlled trial. *Lancet* 2020; **395:** 1927–36.

20 Feinman M, Haut ER. Upper gastrointestinal bleeding. *Surg Clin North Am* 2014; **94:** 43–53.

21 Garcia-Tsao G, Sanyal AJ, Grace ND, et al. Prevention and management of gastroesophageal varices and variceal hemorrhage in cirrhosis. *Hepatology* 2007; **46:** 922–38.

22 Lau JYW, Yu Y, Tang RSY, et al. Timing of endoscopy for acute upper gastrointestinal bleeding. *N Engl J Med* 2020; **382:** 1299–308.

23 Gralnek IM, Stanley AJ, Morris AJ, et al. Endoscopic diagnosis and management of nonvariceal upper gastrointestinal hemorrhage (NVUGIH): European Society of Gastrointestinal Endoscopy (ESGE) Guideline - update 2021. *Endoscopy* 2021; **53:** 300–32.

24 Ibrahim M, El-Mikkawy A, Abdel Hamid M, et al. Early application of haemostatic powder added to standard management for oesophagogastric variceal bleeding: a randomised trial. *Gut* 2019; **68:** 844–53.

25 Expert Panels on Vascular Imaging and Gastrointestinal Imaging, Singh-Bhinder N, Kim DH, et al. ACR appropriateness Criteria® nonvariceal upper gastrointestinal bleeding. *J Am Coll Radiol* 2017; **14:** S177–88.

26 Aoki T, Hirata Y, Yamada A, et al. Initial management for acute lower gastrointestinal bleeding. *World J Gastroenterol* 2019; **25:** 69–84.

27 Donohoe CL, Rockall TA. Is there still a role for the surgeon in the management of gastrointestinal bleeding. *Best Pract Res Clin Gastroenterol* 2019; **42–43**: 101622.

28 Obeidat M, Teutsch B, Floria D-E, et al. Early nutrition is safe and does not increase complications after upper gastrointestinal bleeding-a systematic review and meta-analysis of randomized controlled trials. *Sci Rep* 2024; **14**: 10725.

29 Laine L, Shah A. Randomized trial of urgent vs. elective colonoscopy in patients hospitalized with lower GI bleeding. *Am J Gastroenterol* 2010; **105:** 2636–41. quiz 2642.

30 Marion Y, Lebreton G, Le Pennec V, et al. The management of lower gastrointestinal bleeding. *J Visc Surg* 2014; **151:** 191–201.

31 Sengupta N, Feuerstein JD, Jairath V, et al. Management of patients with acute lower gastrointestinal bleeding: an updated ACG guideline. *Am J Gastroenterol* 2023; **118:** 208–31.

32 Yap FY, Omene BO, Patel MN, et al. Transcatheter embolotherapy for gastrointestinal bleeding: a single center review of safety, efficacy, and clinical outcomes. *Dig Dis Sci* 2013; **58:** 1976–84.

33 Yu Q, Funaki B, Ahmed O. Twenty years of embolization for acute lower gastrointestinal bleeding: a meta-analysis of rebleeding and ischaemia rates. *Br J Radiol* 2024; **97:** 920–32.

34 Gianfrancisco JA, Abcarian H. Pitfalls in the treatment of massive lower gastrointestinal bleeding with 'blind' subtotal colectomy. *Dis Colon Rectum* 1982; **25:** 441–5.

35 Jakobs R, Hartmann D, Benz C, et al. Diagnosis of obscure gastrointestinal bleeding by intra-operative enteroscopy in 81 consecutive patients. *World J Gastroenterol* 2006; **12:** 313–6.

Practice points

- Upper and lower gastrointestinal (GI) haemorrhage are common emergency presentations, associated with significant risk of mortality
- Validated risk scores for both upper and lower GI haemorrhage are available to assist with selection and timing of investigation
- Prompt endoscopy is the investigation and treatment modality of choice for upper GI haemorrhage
- For actively bleeding or unstable patients with lower GI haemorrhage, management via CT angiography is recommended with progression to selective embolization. For stable patients, colonoscopy or flexible sigmoidoscopy may be recommended
- Surgery remains the last treatment option once endoscopic and radiological treatments have failed, or there has been rebleeding even after repeat attempts to control the source of haemorrhage. Post-operative bleeding may still be best served with return to theatre, after discussion with senior clinicians, but interventional radiology is often considered