Gastrointestinal haemorrhage

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Abstract

Gastrointestinal haemorrhage (GIH) may be subdivided by anatomical site 'that is, upper gastrointestinal haemorrhage (UGIH) or lower gastrointestinal haemorrhage (LGIH), or by mode of presentation' acute or chronic, frank or occult. The mode of presentation may give clues as to the likely cause of the blood loss. Most pathologies leading to GIH can present either acutely, with frank blood loss and signs of circulatory compromise, or with a more insidious presentation. The latter may be with symptoms of either anaemia or related to the ongoing pathological process. This article will consider UGIH and LGIH separately, and the emphasis will be on the acute presentation, investigation and treatment.

Keywords angiodysplasia; diagnostic and therapeutic endoscopy; diverticular disease; gastrointestinal tract; haemorrhage; peptic ulcer

Presentation of gastrointestinal haemorrhage

Presentation of gastrointestinal haemorrhage may be with:

- haematemesis (vomiting of fresh red blood)
- melaena (passage of black, tarry stool caused by the oxidation of haemoglobin in the small intestine; this may persist for several days after upper gastrointestinal haemorrhage without further rebleeding)
- 'coffee ground' vomiting (vomiting of altered black blood as a result of the conversion of haemoglobin to methaemoglobin by acid in the stomach if present for long enough)
- frank rectal bleeding.

Melaena must be distinguished from the sticky grey/black stool caused by oral iron therapy. Haemochezia is the passage of fresh blood through the rectum and, although usually from a cause distal to the ileocaecal valve, may be from upper gastrointestinal haemorrhage, especially if the bleeding is profuse, when it is often accompanied by profound hypovolaemic shock.

Initial management

Initial management of the patient with gastrointestinal haemorrhage from any cause is the same. Resuscitation of the patient should be along ATLS lines, with an orderly approach ensuring that the patient does not become hypoxic (maintaining airway with supplemental oxygen), and supporting the circulation appropriately. At the time of intravenous cannulation, blood samples should be sent for haemoglobin estimation, urea and electrolytes, clotting screen, liver function and cross match. Signs of external blood loss should be noted. An attempt should be made to assess the grade of severity of bleed. Hypovolaemic shock may be graded by the ATLS method (Table 1). During assessment, care should be taken to elucidate the following:

- drug history, including anticoagulant therapy, NSAIDs, COX inhibitors and iron therapy
- past history of abdominal surgery (especially gastric and aortic)
- signs and symptoms of chronic liver disease.

A full history and examination along standard lines is required after starting resuscitation, and this includes a rectal examination. Proctoscopy may be of use in cases of obvious Lower gastrointestinal haemorrhage to exclude ano-rectal causes, however the view is often obscured by blood.

Acute upper gastrointestinal tract haemorrhage

Prevalence and mortality

Acute upper gastrointestinal tract haemorrhage remains a common cause of emergency hospital admission, with 172 admissions per 100,000 of the adult population in the UK in 1997. Approximately 85% of cases present as new emergencies, whereas 15% occur in hospital in-patient populations. However, despite technological advances in endoscopic therapy, the mortality from upper gastrointestinal haemorrhage remains largely unchanged at 6–8%, probably as a result of the ageing population. The management of upper gastrointestinal haemorrhage in the UK has, where geographic considerations allow, been concentrated within larger acute bleed units, and several studies show that improvements in morbidity and mortality are to be gained by this strategy. A multidisciplinary approach to care is required, with full access to appropriate endoscopic facilities, expert endoscopists and specialist endoscopy nursing staff, all of whom need to be available 24 hours a day. Mortality varies between patient groups, with those suffering from upper gastrointestinal haemorrhage whilst an in-patient for other causes having 3 times the mortality as new acute admissions. Mortality for patients aged over 75 is twice that of those aged between 65 and 74 years.

Only 5% of upper gastrointestinal haemorrhage in the UK is due to variceal bleeding. The treatment of this condition tends to be confined to larger sub-specialist units. The commoner causes of upper gastrointestinal haemorrhage in the UK are shown in Table 2.

Diagnosis and initial management

Upper gastrointestinal endoscopy should be performed in all patients presenting with symptoms suggestive of upper gastrointestinal haemorrhage. Generally, presentation with haematemesis and melaena indicates a more severe bleed than melaena alone.

Endoscopy: early endoscopy has three roles in upper gastrointestinal haemorrhage

- to establish the cause of the bleed
- to allow prognostic information to be gained
- for therapeutic purposes (haemostasis).

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Classification of hypovolaemic shock

	Class 1	Class 2	Class 3	Class 4
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	<100	>100	>120	>140
Blood pressure (mmHg)	Normal	Normal	Decreased	Decreased
Pulse pressure (mmHg)	Normal or increased	Decreased	Decreased	Decreased
Respiratory rate	14–20	20–30	30–40	> 35
Urine output (ml/hr)	> 30	20–30	5–15	Negligible
CNS/mental state	Slightly anxious	Mildly anxious	Anxious, confused	Confused, lethargic
Fluid replacement (3:1 rule)	Crystalloid	Crystalloid	Crystalloid and blood	Crystalloid and blood

Table 1

Scoring systems for upper gastrointestinal haemorrhage have been devised dependent on endoscopic findings. The Rockall score has been used to predict death from upper gastrointestinal haemorrhage using a composite score of independent risk factors (Table 3). The score for each variable is summated. A Rockall score of 0 equates to a < 5% risk of rebleed, whereas the risk is 40% if the score is over 8. Fluid resuscitation should be initiated immediately and normally with boluses of colloid solution. If shock is profound and refractive to a 2 litre fluid bolus, then O negative blood may be required, although full cross-matching is usually possible. An arbitrary level of 10g/dl Hb is often taken as a level at which to transfuse. Ideally, the patient should be stabilized prior to endoscopic assessment and this should take place within 24 hours of hospital admission. Occasionally, a patient with profuse and continuing upper gastrointestinal haemorrhage will need endoscopy despite never being able to achieve normalized haemodynamic parameters. Attempts should be made to keep the patient warm during resuscitation, with warmed fluids and by covering with warming blankets.

A variety of wide-channelled endoscopes with facility for high-flow lavage and suction should be available for use in the bleed unit, along with a variety of lavage tubes and over tubes to facilitate evacuation of clot and repeated passage of the endoscope. Endoscopy is usually performed under sedation with a benzodiazepine and supplemental oxygen with monitoring of blood pressure and pulse oxymetry. However there are occasions when a full general anaesthetic is required, either to protect the airway in a severely compromised patient, or because the patient is already being ventilated.

Endoscopic attempts at haemostasis should be made if the diagnosis is that of bleeding varices or peptic ulcer disease with stigmata of recent haemorrhage (SRH, Table 4). Active bleeding from an ulcer (spurt or ooze from a visible vessel) and a non-bleeding visible vessel or adherent clot are indications for endoscopic therapy due to the high risk of rebleeding. Adherent clot should always be washed off to identify ulcer stigmata below, prior to therapy. In contrast, a clean ulcer base with or without black or red spots should be treated conservatively.

Causes of acute upper gastrointestinal haemorrhage

Diagnosis	Approximate prevalence (%)*
Peptic ulcer	35–50
Gastroduodenal erosions	8–15
Oesophagitis	5–15
Varices	5–10
Mallory Weiss Tear	10–15
Malignancy of the upper gastrointestinal tract	5–10
Vascular malformations	5
Miscellaneous	5

*Data from published series.

Table 2

Therapy

Treatment of a bleeding peptic ulcer

Injections of solutions into the ulcer base – in a major meta-analysis of trials, no one solution has been shown to be superior to 1 in 10,000 adrenaline solution. This is injected in aliquots of 0.5–1.0 ml around the ulcer base, up to a maximum of 10 ml. Other agents, such as sclerosants, have been shown to cause unacceptable rates of bowel wall necrosis. Fibrin glue and thrombin injections have also been used with varying results, but are not yet widely accepted.

Thermal treatments are all coaptive, that is, they involve the apposition of the vessel walls followed by thermal injury to obtain haemostasis. The most commonly used are the heater probe and bipolar coagulation probes. Both should be used with the widest possible diameter probe for the best haemostatic effect, and both involve the transfer of bursts of thermal energy in 20–30 J fractions until the bleeding stops. The Gold probe has a combined coagulation tip and injection needle.

Recent evidence suggests that in cases with major stigmata of recent haemorrhage, a combination of coaptive therapy and injection therapy can produce lower rates of rebleeding than monotherapy alone.

Argon plasma coagulation has also been used to good effect and involves the transfer of electrical energy by the inert gas argon. Its role may be limited to superficial lesions.

Rockall Scoring system for upper gastrointestinal haemorrhage

Variable	0	1	2	3
Age (years)	<60	60–80	>80	
Shock	Systolic BP >100mmHg Heart rate <100	Systolic BP > 100mmHg Heart Rate >100	Systolic BP < 100mmHg	
Comorbidity	None		Cardiac failure Ischaemic heart disease Any major comorbidity	Renal failure Liver disease Disseminated malignancy
Diagnosis	Mallory Weiss tear No identified lesion No SRH	All other diagnoses	Malignancy of upper gastrointestinal tract	
Major SRH	None Dark spot		Blood in UGI tract Adherent clot Visible or spurting vessel	

Table 3

Endoscopic clips are also gaining a role for the treatment of visible vessels and are usually used in conjunction with injection therapy.

Treatment of other lesions depends on the pathology.

Mallory Weiss tears usually stop bleeding spontaneously without the need for treatment, but injection therapy may be needed occasionally. Telangiectasia may be treated with either argon plasma coagulation or heater probe. The Dieulafoy lesion is difficult to diagnose but may respond best to either band ligation treatment as for varices or argon plasma coagulation.

The management of variceal bleeds is shown in Figure 1.

Drug treatment for upper gastrointestinal haemorrhage

There is now good evidence for the use of acid suppressant drugs in peptic ulcer disease (PUD). The stability of a clot is reduced in acid pH because a pH > 6 is needed for platelet aggregation. Several trials have shown reduced hospital stay, transfusion requirements and rebleed rates with the use of proton pump inhibition. If major stigmata of recent haemorrhage are present, then intravenous proton pump inhibitor (PPI) treatment is now routine.

There is no clear evidence for the use of splanchnic vasoconstrictors such as somatostatin, and neither is there compelling evidence for the use of antifibrinolytic drugs such as tranexamic acid.

Stigmata of haemorrhage and associated rebleed rates (modified Forrest classification)

Class	Endoscopic findings	Rebleed rate (%)
Ia	Spurting arterial vessel	80–90
Ib	Oozing haemorrhage	10–30
IIa	Non-bleeding visible vessel	50–60
IIb	Adherent clot	25–35
IIc	Ulcer base with black spot	0–8
III	Clean base	0–12

Table 4

Post-endoscopy management

Standard monitoring of blood pressure, pulse and urine output should be routine for all patients with upper gastrointestinal haemorrhage.

If the endoscopy has shown that the patient is stable and at low risk of rebleeding, then early feeding can be instituted. There is no evidence that this increases rebleed rates.

Repeat endoscopy and further endotherapy may be needed if there is evidence of continued haemorrhage or rebleeding, and some would advocate a planned endoscopy to reassess in high-risk patients, in order to establish the regression of MSRH.

Uncontrolled upper gastrointestinal haemorrhage

If active, non-variceal upper gastrointestinal haemorrhage cannot be arrested, there are two treatment options.

Surgery is the mainstay of treatment. However, recent evidence suggests that in experienced units, repeat endoscopy and endotherapy may give equivalent results to surgery. There may of course be occasions when the endoscopic evidence suggests that this would be unwise.

More recent reports of endovascular treatment in high-risk patients would suggest that there is a role for this therapy in units with access to skilled personnel and appropriate facilities. Particular vessels (gastroduodenal artery) may be embolized with a combination of endovascular sponges and coils and the angiogram may delineate abnormal anatomy prior to surgery (angiodysplasia). However, such treatment may take several hours, and in a sick patient who has rebled, prompt surgical control of the haemorrhage is more often the safer option.

Surgery for upper gastrointestinal haemorrhage is less commonly performed than in previous decades because of the decrease in PUD as a result of acid suppressant drugs and because of improvements in endoscopic therapy. Patients currently presenting to the surgical team are older, with worse disease and more medical comorbidity. They may also have a larger transfusion requirement as a result of continued bleeding in spite of repeat endoscopic treatment. As a result of the decreased incidence,

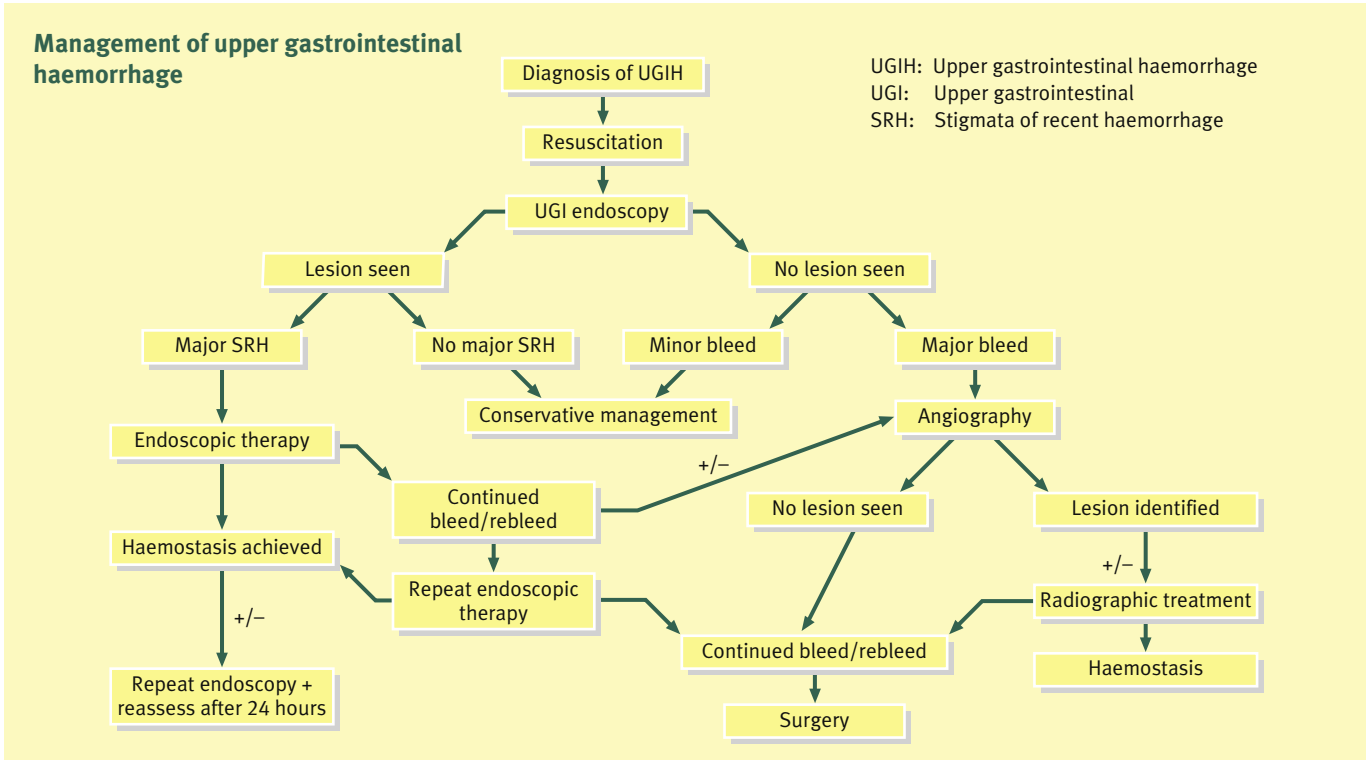


Figure 1

current trainee surgeons therefore have much less experience in operative treatment for upper gastrointestinal haemorrhage.

Bleeding duodenal ulcer

These are often posterior wall lesions involving a branch or the main trunk of the gastroduodenal artery. A small upper midline laparotomy is made and the duodenum identified. Kocherization of the duodenum by releasing its lateral peritoneal attachments may be needed to improve exposure. A longitudinal duodenotomy is made between stay sutures, avoiding the pylorus. Finger pressure can control any bleeding point, whilst irrigation and suction clear the operative field. A number 1 or 0 PDS or vicryl stitch is used to under-run the vessel, using a small needle in order to avoid taking too deep a bite, as the bile duct lies behind.

If there is only a little scarring, then the enterotomy may be closed. If not, then it is closed in the manner of a pyloroplasty. If there is a large ulcer defect, then the duodenum may be closed with a Finney pyloroplasty.

The aim is to provide swift haemostasis with a safe operation, doing the minimum possible. Occasionally, severe and chronic ulceration render the duodenum unsafe to close and, in this circumstance, a partial gastrectomy is performed, with closure of the duodenal stump by Nissen's method (with or without T-tube drainage).

There is no indication for vagotomy in the era of PPIs, except for severe and recurrent ulceration that has occurred whilst on medication.

Bleeding gastric ulcer

A bleeding gastric ulcer may be under-run after biopsy of its edge to exclude malignancy (accessed via an anterior gastrotomy). Alternatively, the lesser curve may be excised (Pauchets manoeuvre). For a large antral ulcer, antrectomy may be necessary. High lesser curve ulcers may be excised, although proximity to the left gastric vessels may require mobilization of the full lesser curve.

The Dieulafoy lesion is treated by suture under-running. Mallory Weiss tears rarely need operative intervention.

Follow-up

Gastric ulcers should always be reassessed by endoscopy to ensure healing and a benign aetiology. All patients with bleeding peptic ulcer should be treated with PPIs, and follow-up endoscopy should assess the *H. pylori* status to allow eradication.

Varices

Variceal bleeding is a relatively uncommon cause of upper gastrointestinal haemorrhage. Most cases are in known patients with cirrhosis and therefore tend to be managed in specialized liver units.

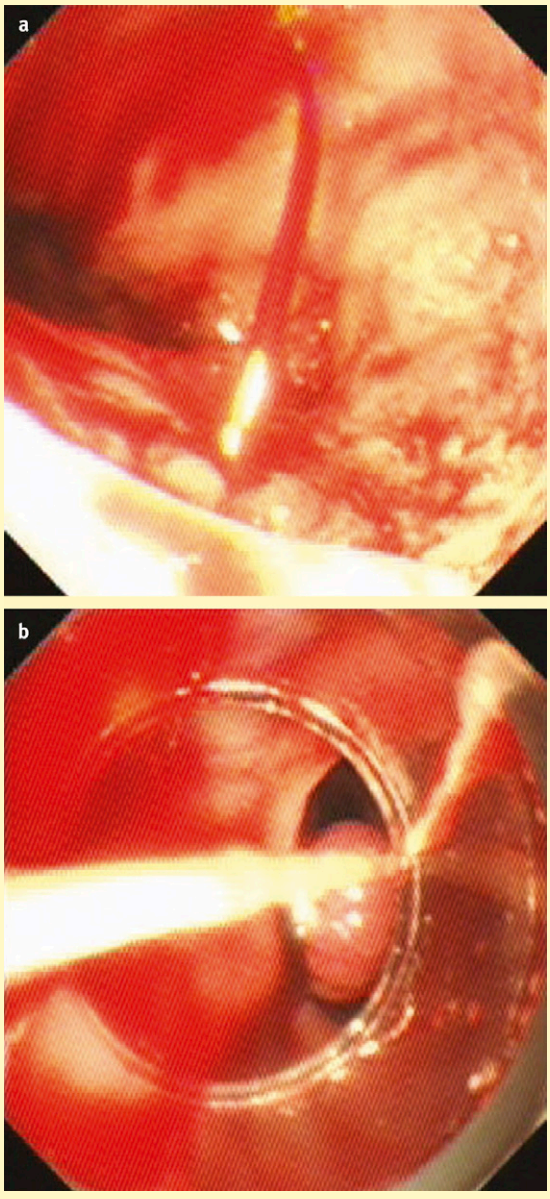
It is vital that specialist experience is sought early in the management of these patients. In contrast to non-variceal bleeding, there is a definite role for splanchnic vasoconstrictor drugs. Both terlipressin (a precursor to vasopressin) and octreotide reduce the portal blood flow and have been shown to reduce transfusion requirements, although not mortality, in these patients. Beta blockade has been shown to have similar results.

In cases with known or suspected variceal haemorrhage, drug treatment may be instituted prior to endoscopy. Initial control with balloon tamponade from a Minnesota or Sengstaken tube may allow stabilization and facilitate transfer to a unit with expert endoscopic experience.

Endoscopic treatment consists of injection sclerotherapy, cyanoacrylate (glue) injection and band ligation, and their use depends on the experience of the operator. More definitive procedures such as transjugular intrahepatic portosystemic shunting (TIPPS) may be appropriate, but surgery, including devascularization procedures and surgically created shunts, are less commonly used. Figure 2 shows a bleeding varix which has been banded.

Acute lower gastrointestinal haemorrhage

This may be defined as bleeding from a point below the duodeno-jejunal flexure. Excluding causes in the anal canal, lower gastrointestinal haemorrhage is significantly less common than upper gastrointestinal haemorrhage (approximately 20% of all cases of gastrointestinal haemorrhage). Most cases of lower gastrointestinal haemorrhage will settle spontaneously and a significant proportion will remain without definite diagnosis.



a Jet of blood from a bleeding oesophageal varix. **b** Varix after endoscopic banding.

Figure 2

Presentation

Presentation of lower gastrointestinal haemorrhage may be associated with:

- bloody diarrhoea
- abdominal pain
- rectal and anal symptoms.

Fresh rectal bleeding may also originate from a brisk upper gastrointestinal cause, in which case the patient will be profoundly shocked.

Common cases of lower gastrointestinal haemorrhage are shown in Table 5.

Management

As stated above, the majority of cases of acute lower gastrointestinal haemorrhage will settle spontaneously and investigations can be performed at leisure. Occasionally this is not the case and initial resuscitation is as described for upper gastrointestinal haemorrhage.

Acute colonic bleeding

This makes up 20% of all acute gastrointestinal haemorrhage; 80% of these cases stop spontaneously. The majority of cases occur in the elderly, with a mean age of > 65 years. The cause of bleeding may remain unconfirmed, even after investigation, including pathological examination of the resected colon. The two most common causes of acute colonic bleeding are angiodysplasia and diverticular disease. Carcinoma of the colon presents with acute haemorrhage only rarely. Inflammatory bowel disease presents uncommonly as a fulminant colitis, which may lead to exanguinating haemorrhage.

Angiodysplasia

Since the introduction of angiography, angiodysplasia has become a more common diagnosis. Eighty percent of lesions affect the right colon up to the hepatic flexure, while 20% affect the left colon and sigmoid. Bleeding from angiodysplasia may present either with occult bleeding and anaemia or profuse rectal bleeding. Endoscopy

Causes of acute haemorrhage of the lower gastrointestinal tract

- Diverticular disease
- Angiodysplasia
- Inflammatory conditions (e.g. Crohn's disease/ulcerative colitis)
- Drugs (e.g. NSAIDs, anticoagulants)
- Neoplastic (benign, malignant)
- Rare (e.g. endometriosis, bleeding Meckel's diverticulum)
- Iatrogenic (e.g. following endoscopic biopsy)

Table 5

may show a characteristic cherry red spot (Figure 3). It is not easy to see subtle mucosal detail in a patient with a brisk colonic bleeding and no bowel prep, so most cases of angiodysplasia are diagnosed by angiography. Asymptomatic pick-up rates for angiodysplasia in colonoscopies performed for other reasons show that approximately 25% of the elderly will have visible lesions. If a lesion is found but there is no active bleeding, then a dilemma exists for the surgeon. A high proportion of this group will also have diverticular disease, which is the most common cause of colonic haemorrhage and the commonest cause to require operation.

Diverticular disease

This is the most common cause of acute lower gastrointestinal haemorrhage. Since the advent of visceral angiography, it is clear that many cases of lower gastrointestinal haemorrhage that were attributed to diverticular disease had alternative causes. Diverticular disease probably accounts for 50–60% of lower gastrointestinal haemorrhage. Bleeding results from rupture of the nutrient vessels to the colonic mucosa as they pass through the neck of the diverticula. Trauma or, more likely, inflammation may cause this. Diverticulitis *per se* is not a cause of diverticular bleeding.

Most diverticular disease affects the left colon. However, in proportion to its incidence, diverticular disease of the right side is more likely to be a cause of bleeding. Lower gastrointestinal haemorrhage from diverticular disease can result in painless profuse fresh rectal bleeding. Less than 10% of acute diverticular bleeds need treatment, although one-third will rebleed at a later time. Drugs such as anticoagulants and NSAIDs have been shown to be exacerbating factors in lower gastrointestinal haemorrhage, and NSAIDs increase the risk of diverticular bleeding.

An algorithm for the management of lower gastrointestinal haemorrhage is shown in Figure 4.

Investigation and treatment

In cases of lower gastrointestinal haemorrhage when proctoscopy reveals no source of bleeding, an upper gastrointestinal endoscopy should be performed. The rationale behind this is that acute upper gastrointestinal haemorrhage can present both with fresh rectal bleeding and melaena, is more common than lower gastrointestinal haemorrhage and is less likely to stop spontaneously, and is therefore more life-threatening.

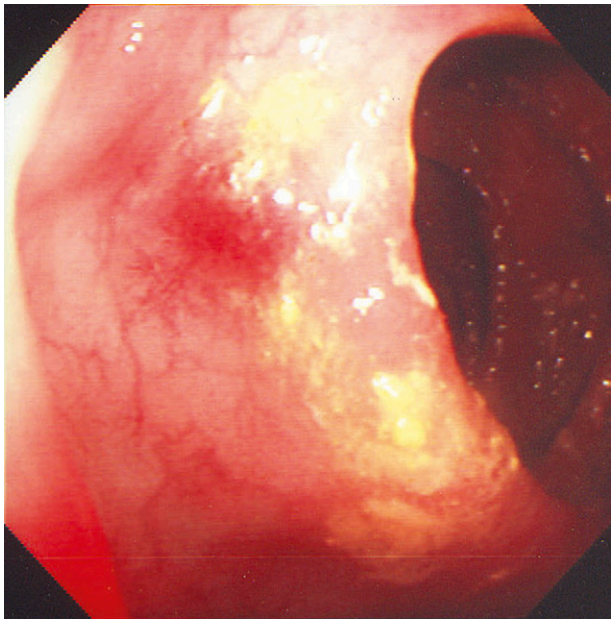


Figure 3 Cherry red spot of colonic angiodysplasia.

Arteriography: the choice of investigation thereafter depends on the availability of resource and operator skill available. If there is a profuse, ongoing bleed then it is likely that colonoscopy would be unhelpful as there would be difficulty in visualizing the mucosa due to clot and faeces. In this instance, arteriography may demonstrate extravasation of contrast into the bowel lumen. This can be demonstrated at rates of 1–1.5 ml/min. Selective catheterization of the superior mesenteric artery is performed initially, as the source of bleeding more commonly lies in this distribution. Abnormalities include venous abnormalities such as early venous filling, venous lakes and tufts, and therefore venous phase imaging is crucial. If no abnormality is found, the IMA can then be catheterized. Non-actively bleeding angiodysplasia should not be assumed to be the cause of haemorrhage, as discussed above. Bleeding from small intestinal origin may not be detected by endoscopy. If more than 0.5 ml/min of blood is being lost in an active bleed, then mesenteric angiography may detect the site. Other lesions such as angiodysplasia and polyps may be detected by an abnormal vascular pattern. Therapeutic techniques such as coil or sponge embolization may arrest bleeding points or reduce the rate of blood loss prior to surgery, but there is a small incidence of ischaemic colitis when these techniques are used. Vasopressin infusion is less often used due to its high failure rate, side effects (especially in an aged patient group) and the improvements in angioraphic embolisation techniques.

Endoscopy: the success of colonoscopy during acute episodes of lower gastrointestinal haemorrhage depends on the experience of the operator, with some units reporting a diagnostic yield of over 75%. There is, however, wide variation in the literature. Colonoscopy can be the first line of treatment as well as the first line of investigation. Diathermy, both monopolar and bipolar, argon plasma coagulation, laser and heater probe have all been reported as successful treatments in angiodysplasia. Heater probe and injection therapies have been used to control diverticular bleeds. A significant risk of colonic perforation is present in this situation, especially if endotherapeutic techniques are used near the neck of diverticula.

Isotope scanning following intravenous technetium radiolabelled autologous cells (red cell scan) may be helpful in cases of occult bleeding, but are generally unhelpful in acute lower gastrointestinal haemorrhage. Red cell scans can detect rates of bleeding from 0.05–0.1 ml/min, but a positive result is rarely very specific in terms of anatomical localisation.

Contrast studies play no role in the investigation of acute lower gastrointestinal haemorrhage.

Surgery is required for ongoing haemorrhage when either arteriographic or endoscopic treatment has been unsuccessful or the patient is unstable with continuing haemorrhage.

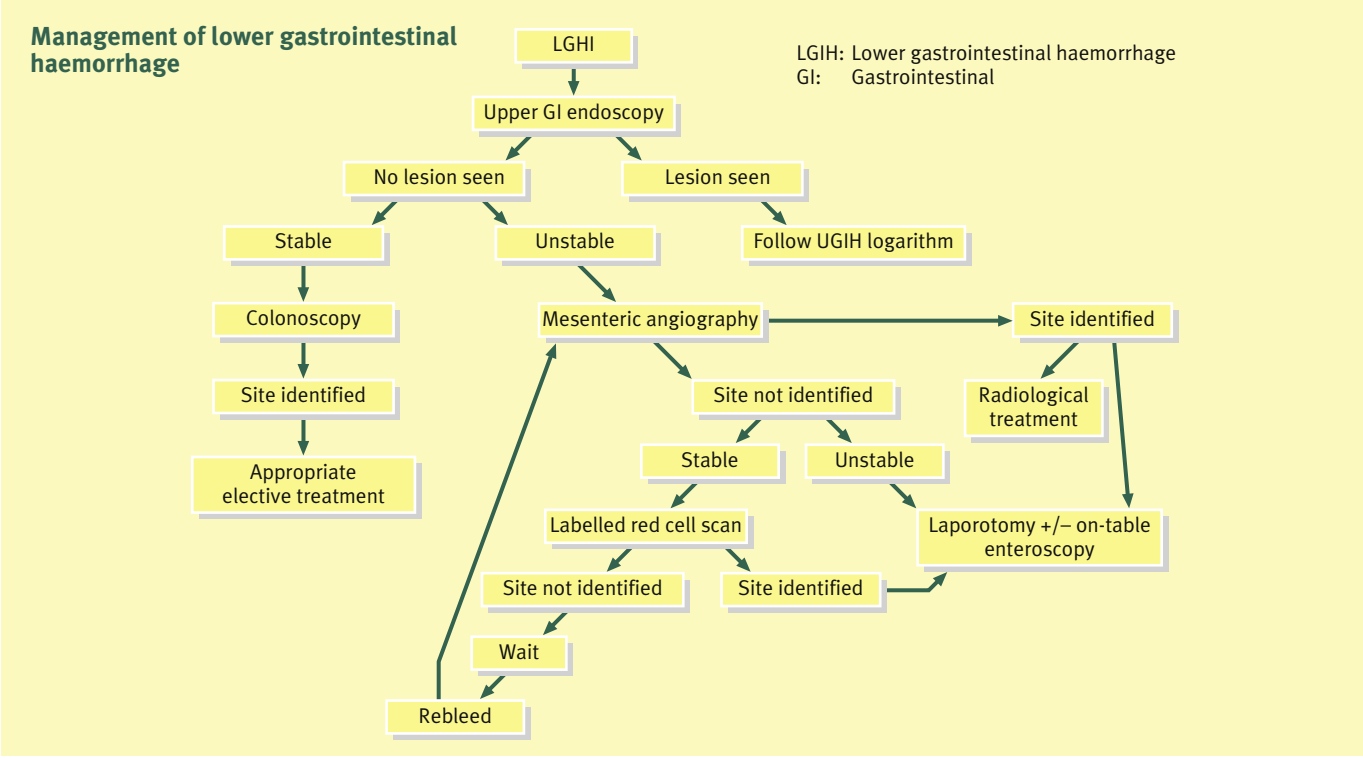


Figure 4

Often the source of blood loss will have been identified prior to laparotomy by one of the above techniques. However, there will be a subgroup of patients where either dual pathologies have been noted (diverticular disease and angiodysplasia being the most common) or the cause has not been established. In these circumstances, on-table colonoscopy and a second operator are invaluable. Antegrade saline lavage during colonoscopy may increase the rate of diagnostic yield and also cleanses the colon prior to any anastomosis. If, despite these efforts, a cause can not be established, then sub-total colectomy should be performed. It is imperative in this situation that a cause of haemorrhage proximal to the ileocaecal valve be excluded by on-table enteroscopy. If enteroscopy is not possible or if the operator has doubt as to the site of bleed, then subtotal colectomy with end ileostomy should be performed. Bleeding from a cause in the small intestine will soon become apparent in the postoperative period. The number of patients requiring this procedure should be negligible, with appropriate facilities for intraoperative endoscopy. ◆

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