Approach to upper gastrointestinal bleeding

Upper GI bleeding is the most common complication of peptic ulceration and portal hypertension.

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Upper gastrointestinal haemorrhage has a variety of causes (Table 1) and is the commonest complication of peptic ulceration and portal hypertension. Peptic ulceration in the duodenum or stomach and oesophageal varices are the conditions most often responsible for patients who have the potential to present with life-threatening haemorrhage.[1-6] The key elements of an approach to this medical and surgical emergency are outlined in Fig. 1.

Resuscitation and assessment

Whatever the actual cause, the most important initial management is to assess the haemodynamic parameters and institute appropriate resuscitation measures early. In those who are haemodynamically unstable aggressive resuscitation has been shown to improve outcome. Ninety five per cent will stabilise and it is important that they are fully assessed by history and physical examination.[1,3,4] This allows management to be strategised based on whether the individual has variceal or non-variceal bleeding. The former is most likely if they have stigmata of chronic liver disease and portal hypertension and the latter assumed in the absence of these findings.

Risk stratification

A variety of clinical factors and laboratory tests can be incorporated into risk stratification. The Rockall system[5,6] (Table 2) is the most commonly used and is applicable for both variceal and non-variceal bleeding. These systems predict the likelihood of continuing to bleed or of rebleeding, and the risk of death. These clinical factors, age over 60, and shock on admission are highly predictive.[7] Concurrent medical therapy is particularly important as NSAIDs and anticoagulants, which are commonly prescribed in the elderly, have a direct deleterious effect on coagulation. The

Table 1. Causes of upper gastrointestinal bleeding-related degree of bleeding severity

	Site		
Degree of bleeding	**Oesophageal**	**Gastric**	**Duodenal**
Major common	Oesophageal varices	Gastric varices	Benign ulcer
	Mallory-Weiss tear	Portal hypertensive gastropathy	
		Benign ulcer	
Major uncommon		Dieulafoy's lesion	Haemobilia
			Haemosuccus pancreas
			Aorto-enteric fistula
		Gastric cancer	
Usually minor	Oesophagitis	Gastritis	
	Oesophageal cancer	Gastric antral vascular ectasia	

Table 2. Modified Rockall risk stratification scoring system

	Score			
Variable	**0**	**1**	**2**	**3**
Age	Under 60	60 - 79	Over 80	
Shock	No shock	Pulse over 100	BP under 100	
Co-morbidity	None		Cardiac failure Ischaemic heart disease	Renal failure Liver failure Disseminated malignancy
Diagnosis	Mallory-Weiss tear	All other diagnoses	Upper GI malignancy	
Major signs of recent haemorrhage	None /dark spot		Blood in the upper GI tract Adherent clot Visible or spurting vessel	

Maximum score prior to endoscopy: 7. Risk of death; score 0 - 2 ≈<5%; score 3 - 5 ≈10 - 40%; score 6 - 8 ≈>50%.
Maximum score following endoscopy: 11. Risk of death; score 0 - 3 ≈<5%; score 4 - 7 ≈10 - 40%; score 8 - 11 ≈>50%.

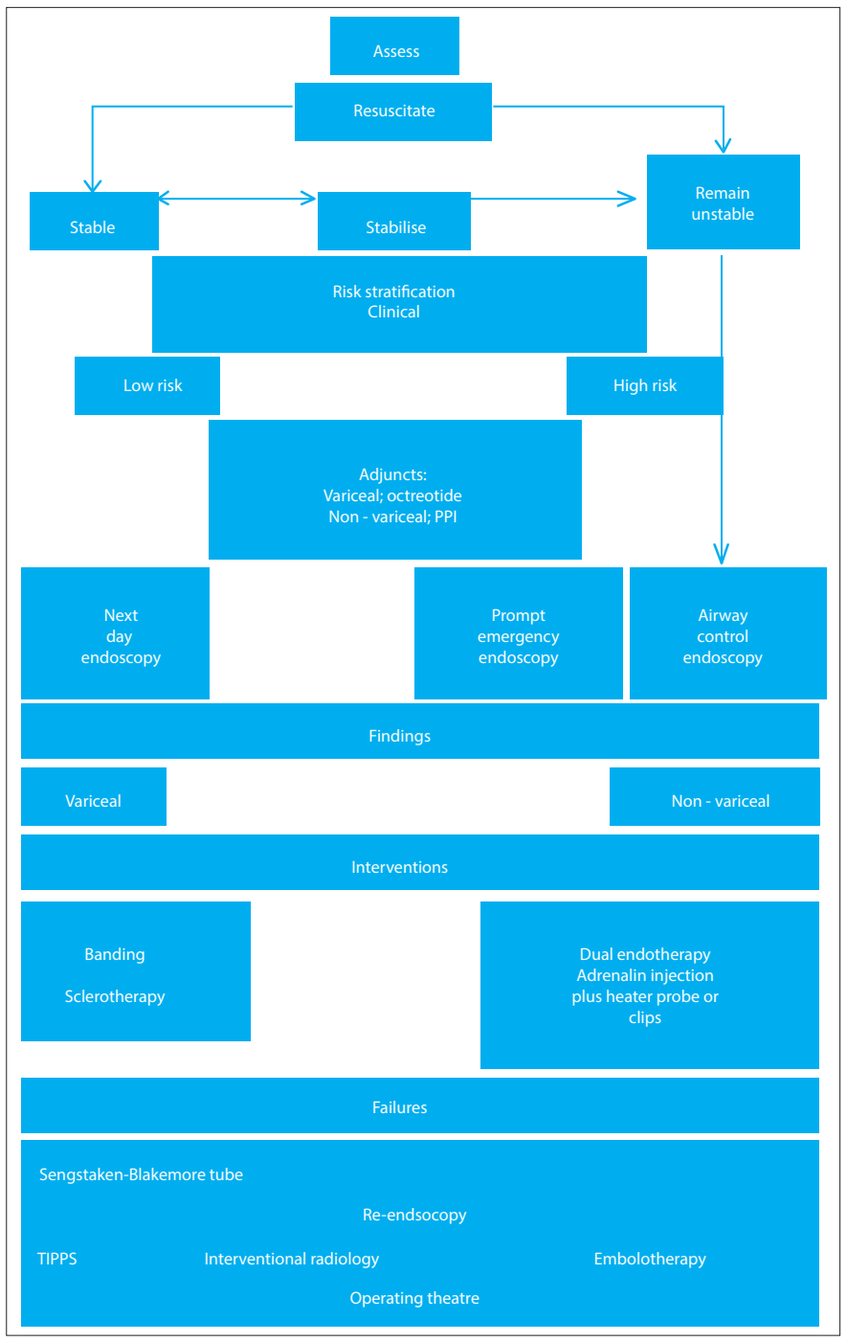

Fig. 1. Key elements of an approach to gastrointestinal haemorrhage.

former, which affects platelet function, increases rebleeding risk but there is no active therapy to negate this effect in the acute situation. Aberrant warfarin anticoagulation can and should be corrected by blood product replacement.[2,3] In addition, medical comorbidities also contribute to the risk and require active management.

Monitoring and adjunctive measures

These stratification systems can also direct appropriate monitoring of high-risk patients prior to endoscopy, which is the next step. During resuscitation and stabilisation appropriate adjunctive measures are of proven benefit. In clinically high-risk patients with suspected non-variceal bleeding, intravenous PPI therapy will elevate the pH to above 6, which allows a stable clot to be formed and once formed to be less likely to lyse.[8-10] Similarly, in variceal haemorrhage the use of vasopressors or somatostatin analogues, which reduce splanchnic flow, have been shown to be of benefit.[2,3,11] The risk of infection, or of infection being precipitated, in patients with chronic liver disease and variceal haemorrhage, has a deleterious effect on outcome. Hence antimicrobial therapy is both rational and of confirmed benefit.[2,3]

Endoscopy

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The patient generally should be haemodynamically stable prior to endoscopy and for the vast majority this is possible. It is important that they are transfused to at least an Hb of 8 g/dl, particularly if they were shocked on admission. It is essential that appropriate monitoring is utilised. Pulse oximetry and supplemental oxygen through nasal prongs should be used routinely, as should ECG monitoring. Those patients who are difficult to stabilise should be endoscoped in the operating theatre with the surgeon present and airway protection, otherwise adverse events are likely and the outcome of endoscopic therapy will be compromised.[1-4] Conscious sedation is often helpful, but in the elderly and the potentially unstable patient it is important that it is titrated gradually to get the patient co-operative and a non-moving target without deep sedation in which the airway may be compromised.[1-4]

The common sites of bleeding must be visualised – that is the distal oesophagus, the incisura of the stomach, the pre-pyloric region and the first part of the duodenum. The view may not be optimal due to old or fresh blood and clots when they are present. These may prove difficult for the suction channel to cope with. In such situations it is important to have an experienced endoscopist and to carefully move past the blood rather than try to suction it away and

block the endoscope. Automatic irrigation devices may be helpful to displace clots and get a better view of the bleeding site.[1,12]

The ulcers can be classified into different categories according to the Forrest classification (Fig. 2). This defines the risk of rebleeding and is the determinant of which ulcers should have endotherapy to either control the bleeding or reduce the risk of it recurring.[1,4,8,9]

Endotherapy

Non-variceal haemorrhage

There are various endotherapies that have been employed to control the bleeding endoscopically. For non-variceal haemorrhage the tried and tested modality is injection with 1/10 000 solution of adrenaline.[1,4,9] Use at least 10 ml. This will cause intense vasoconstriction and narrowing of the vessel lumen by a sheer volume effect. This will allow localisation of the bleeding point.

It has been shown that more lasting control can be achieved by adding one of the following two techniques – thermal coaption with a heater probe or gold probe.[1,4,9,12] This is placed directly on the visible vessel to obliterate the bleeding vessel's lumen by pressure and direct thermal coagulation. Alternatively, use endoscopic clipping devices which pass through the accessory channel of the endoscope and deploy a detachable clip to the vessel.[1,4,8,9,12]

Aberrant warfarin anticoagulation can and should be corrected by blood product replacement.

For the majority of patients these methods should control even active bleeding 80 - 90% of the time. Duodenal ulcers are often more difficult technically than gastric ulcers as the endoscope manoeuvrability is limited in the confined space of a scarred duodenum. If there is failure to control active bleeding, if facilities and logistics allow and the patient is stable then interventional angiography may control the situation by embolotherapy with a variety of different agents. Coils are used to control the major inflow and outflow followed by chemical obliteration of the microcirculation around the area to ensure that more permanent control is obtained.[13]

These techniques are usually employed in the high-risk elderly patient with co-morbidities in whom surgery carries a prohibitive risk. However, there remains a place for prompt surgery as the only hope for salvage, particularly in those patients with persistent shock.[7]

Once controlled, the IV PPI should be continued for 48 hours[9,10] and the ulcer then effectively managed by curative oral eradication therapy, of which the current first-line therapy is a 2-week course of amoxycillin and clarithromycin with a bd double-dose PPI.[14]

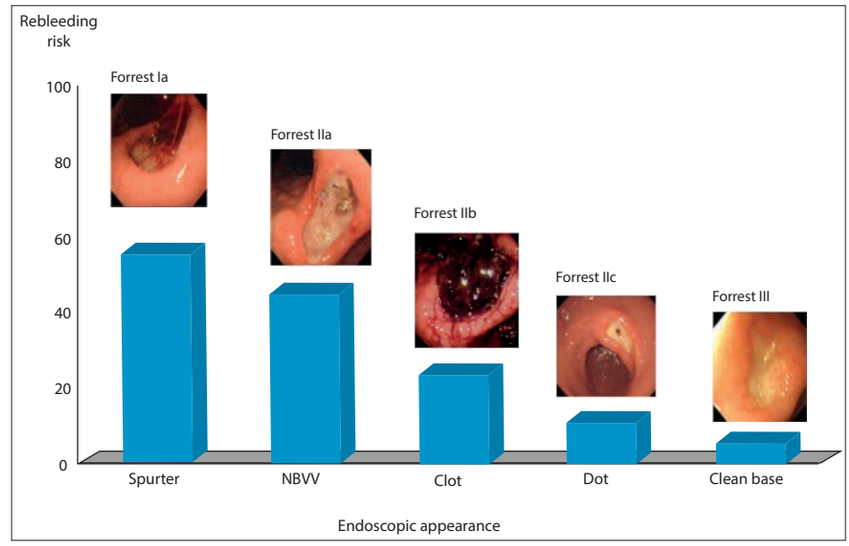

Fig. 2. Forrest classification of peptic ulcer bleeding related to risks of rebleeding. (NBVV = non-bleeding visible vessel.)

Table 3. Child-Pugh classification for risk stratification in portal hypertension

	Point allocation		
Parameter	**One**	**Two**	**Three**
Bilirubin (µmol/l)	Less than 34	34 - 50	More than 50
Albumin (g/l)	More than 35	28 - 35	Less than 28
INR	Less then 1.7	1.71 - 2.3	More than 2.3
Ascites	None	Mild	Moderate to severe
Encephalopathy	None	Grade I - II	Grade III - IV (refractory)
Prognostic significance	**A**	**B**	**C**
Points score	5 - 6	7 - 9	10 - 15
One-year survival (%)	100	81	45
Two-year survival (%)	85	57	35

Variceal bleeding

In patients with variceal bleeding, the bleeding often stops in those with good liver reserve. Once again the risk stratification allows prognostication and the risk of rebleeding in hospital. Though the Rockall score has been validated for variceal haemorrhage the Child-Pugh score[15] (Table 3) is most commonly used to stratify risk.

At endoscopy a full inspection of the foregut must be undertaken. This will allow identification of non-variceal causes of haemorrhage, which occur in 10% of cases, and those who may be bleeding from gastric varices or portal hypertensive gastropathy.[3,11,16] The method of choice is banding, as shown in

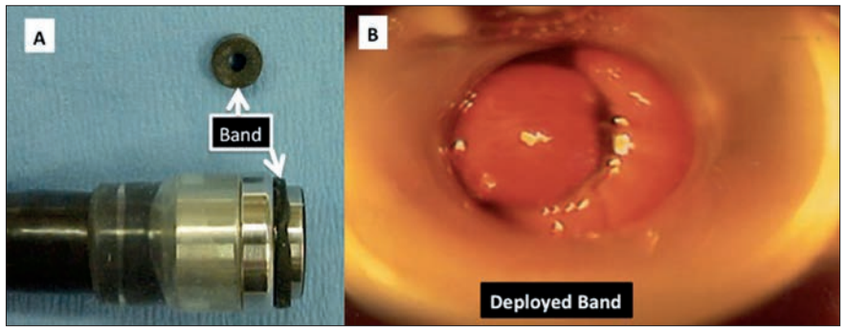

Fig. 3(A). Superiorly band, showing deployed width of 2 mm. Inferiorly band loaded on banding device width 9 mm. (B). Endoscopic view of deployed band.

Fig. 3. This has largely superseded injection sclerotherapy with ethanolamine oleate, as it has a higher complication rate than banding.[16] Once again, those who continue to bleed are best managed with airway control prior to endoscopy in the operating theatre complex or the ICU. The Sengstaken-Blakemore tube can be used when visualisation of the oesophagus is not possible due to active bleeding. In this situation the gastric balloon should be blown up and traction applied.[17] This is usually effective but should be followed by immediate endoscopy once deflated and removed. Alternatively, a covered 30 mm oesophageal stent has been used as an alternative form of tamponade[18] but the interventional procedure of choice is a TIPS shunt[19] which, when placed successfully, will reduce the portal pressure below 12 mmHg and control the bleeding. Once bleeding has been controlled, appropriate supportive medical management should be continued. Those patients with Child-Pugh category C disease often have been precipitated into hepatic failure by the bleed and require additional management for this complication. These individuals are also likely to have a prolonged INR and require repeated transfusion of fresh frozen or freeze dried plasma.[3]

Subsequent management is two-fold. Firstly, to reduce the risk of further bleeding by establishing non-selective B blockade and continued banding of the varices until they are eradicated.[3,11] The second consideration is to establish the cause of the portal hypertension and manage it appropriately. Management at this stage is best handled by a multidisciplinary team comprising a gastroenterologist/hepatologist and a surgeon. These patients need an adaptable long-term strategy, which needs to be individualised and includes the option of liver transplantation where appropriate.[3,11]

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Summary

- Acute upper GI bleeding requires an accurate assessment at presentation with a focus on resuscitation and appropriate monitoring.
- Thereafter it is important to determine if portal hypertension and varices are likely from the history and physical examination.
- This will dictate the supportive and adjunctive therapy for each disease process.
- Risk stratification should be done early and allows prognostication, which allows prioritisation of the degree of monitoring required and the urgency of endoscopy.
- Endoscopy will determine the cause in the vast majority of patients and allow control of those suitable for endotherapy.
- Once the index bleed is under control, the underlying disease can be determined and co-morbidities optimised so an effective long-term management plan can be formulated.