Upper gastrointestinal bleeding

Introduction

Acute gastrointestinal haemorrhage is classified according to whether it originates from the upper or lower gastrointestinal tract, the demarcation of which is the ligament of Treitz. This article will review the epidemiology, aetiology, assessment and treatment of acute upper gastrointestinal bleeding.

Epidemiology

Acute upper gastrointestinal haemorrhage (AUGIH) remains a common reason for admission to hospital and 14% of cases occur in those who are already inpatients (Rockall et al, 1995). The overall incidence of AUGIH is approximately 100/100 000 adults/year in the UK with a significant increase in incidence with age (from 23/100 000 in those aged under 30 years to 485/100 000 in those aged over 75 years). The incidence of AUGIH in men is more than double that of women at all ages apart from the elderly where the difference is less marked.

Aetiology

The most common gastrointestinal lesion to cause haemorrhage is peptic ulceration (35–50% of AUGIH) which may be oesophageal, gastric, duodenal or mixed (British Society of Gastroenterology Endoscopy Committee, 2002). Others include malignancy (most commonly adenocarcinoma of the stomach), varices, Mallory–Weiss syndrome, oesophagitis and erosive disease. Each diagnostic group has its own aetiological factors, such as *Helicobacter pylori* infection and non-steroidal anti-inflammatory drugs (NSAID) usage in peptic ulceration.

Ulceration at specific sites will increase risk of a major haemorrhage because of the proximity of major arteries. For instance peptic ulcers at the lesser curve of the stomach may result in bleeding from the left gastric artery, in the first part of the duodenum posteriorly from the gastroduodenal artery, or in the posterior wall of the stomach from the splenic artery. Varices are a consequence of portal hypertension, the most common causes of which are alcohol and hepatitis. Varices are most frequently oesophageal although they may rarely affect the stomach.

Other causes include gastrointestinal stromal tumours which may ulcerate and bleed, vascular lesions such as angiodysplasia, haemobilia (which may be iatrogenic, e.g. post-endoscopic retrograde cholangiopancreatography), trauma, and in patients with a history of abdominal aortic aneurysm repair, an aorto-enteric fistula should be considered.

Clinical features

The diagnosis of AUGIH is a clinical one with a classic presentation of haematemesis or 'coffee-ground' vomiting in a shocked patient. Malaena (a black, tarry, offensive stool) will always follow a significant haemorrhage. In patients with a rapid haemorrhage, fresh blood may be passed per rectum (haemochezia) and thus may be difficult to distinguish from a lower gastrointestinal haemorrhage. A history must be taken for NSAID use, alcohol intake, previous AUGIH, and the patient examined for signs of chronic liver disease which may be suggestive of varices. The degree of shock must be quickly assessed by observing pulse rate, blood pressure, capillary refill time and respiratory rate, and these parameters should be regularly monitored once fluid resuscitation has begun.

Haemoglobin levels do not give a useful estimate of the volume of haemorrhage in the acute setting as sufficient time for haemodilution to occur will not have elapsed and so the haemoglobin concentration may be normal in the setting of a large bleed. Patients with pre-existing iron-deficiency anaemia who have had a small bleed will have a pre-existing low haemoglobin concentration which may result in overestimation of the volume of haemorrhage.

Management

Resuscitation and drug therapy

The patient must be adequately fluid-resuscitated before endoscopy and haemostasis is attempted. Intravenous access comprising two wide bore cannulae should be gained and resuscitation started, initially with crystalloids and subsequently with cross-matched blood. A response to resuscitation is indicated by a normalizing pulse rate, blood pressure, capillary refill time, central venous pressure and urine output, so central venous access and urinary catheterization are essential for monitoring severe cases.

There is some evidence that high-dose proton pump inhibitors can reduce rebleeding rates, especially in the sub-

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Figure 1. Incidence (per 100 000 adults per year) of acute upper gastrointestinal haemorrhage with age in men and women in the UK (Rockall et al, 1995).

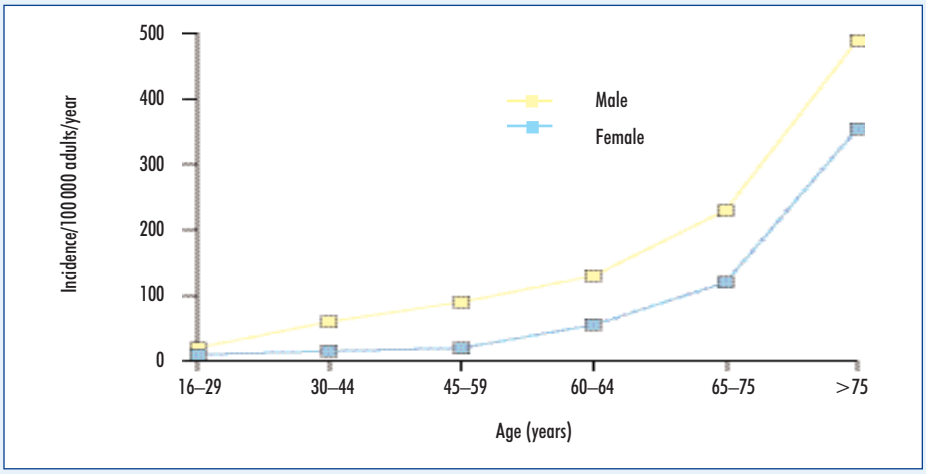

Table 1. Causes of acute upper gastrointestinal haemorrhage

Diagnosis	Approximate %
None	23
Peptic ulcer	29
Malignancy	1
Varices	8
Mallory–Weiss tear	13
Erosive disease	13
Oesophagitis	10
Other	4

From Rockall et al (1995)

group of patients with non-bleeding visible vessels. Clot extruding from a breach in a vessel wall is liable to lyse at low pH and proton pump inhibitors administered as a bolus and subsequently by continuous infusion can reliably raise the gastric pH. A recent meta-analysis has, however, shown that proton pump inhibitor therapy reduces the risk of re-bleeding and the requirement for surgery, but has no benefit on overall mortality (Leontiadis et al, 2005).

Upper gastrointestinal endoscopy

The aim of endoscopic therapy is to make a diagnosis, arrest haemorrhage and to prevent re-bleeding with the ultimate purpose of reducing transfusion requirements, surgical intervention rates, morbidity and mortality. It also provides the surgeon with information as to the site of the lesion in cases requiring urgent surgery. Peptic ulcers with stigmata of recent haemorrhage should all be treated endoscopically and where adherent clot is present this should be washed off and the underlying lesion treated appropriately (Bleau et al, 2002).

Endoscopic therapies for bleeding peptic ulcers take the form of injecting substances, applying thermal energy and mechanical haemostatic devices.

Various injectable substances have been assessed in the treatment of AUGIH including adrenaline and many sclerosing agents such as ethanolamine and absolute alcohol. However, the British Society of Gastroenterology guidelines (British Society of Gastroenterology Endoscopy Committee, 2002) recommend the injection of adrenaline solution in quadrants around the bleeding point and then into the bleeding vessel, which achieves haemostasis in 95% of cases. There is little evidence that the use of sclerosants confers any advantage in terms of re-bleeding rates and they may cause the potentially life-threatening complication of tissue necrosis and perforation. Adrenaline causes an initial tamponade as well as arterial constriction leading to thrombosis of the bleeding vessel. Procoagulants such as fibrin glue (Rutgeerts et al, 1997) and thrombin have been used with some success, but they are expensive and difficult to use.

The aim of applying thermal energy to a bleeding point is to coagulate the vessel. A Teflon-coated heater probe may be placed in contact with the bleeding point after the ulcer base has been cleaned with a water jet, resulting in coagulation. Bipolar diathermy, the Nd:YAG (neodynium:yttrium argon garnate) laser and argon plasma coagulators are other means of delivering thermal energy, and all these methods appear to be equally efficacious in reducing re-bleeding.

Hemoclips (Olympus, Tokyo, Japan) are endoscopically-placed titanium clips that are directly applied to bleeding vessels and are then shed into the lumen a few days later. They have been shown to play an important role in the management of larger vessels, beyond the ability of injection and thermal methods. There are, however, technical difficulties associated with their use (Chung et al, 1999).

Failure of endoscopic therapy

Surgery (including ulcer excision, underrunning of bleeding vessels, partial gastrectomy) is indicated where endoscopic therapy is not possible or where it has failed to control massive haemorrhage. Angiography may allow radiological embolization of an actively bleeding vessel (e.g. the gastroduodenal artery in a duodenal ulcer). It is especially useful when the source of haemorrhage is unclear.

Management of oesophageal varices

Varices are a common problem with studies showing that 90% (Grace, 1997) of patients with cirrhosis will develop varices and that 30% (North Italian Endoscopic Group for the Study and Treatment in Esophageal Varices, 1988) of these will go on to haemorrhage. Of paramount importance is fluid resuscitation and airway protection. Normal saline should be avoided in patients with chronic liver disease owing to their impaired renal sodium excretion and the possible development of ascites (Williams and Westaby, 1994).

Pharmacological measures to reduce variceal bleeding aim to reduce portal pressures, by reducing portal collateral blood flow either with splanchnic vasoconstrictors (e.g. vasopressin, somatostatin and their analogues and propanolol) or with vasodilators (e.g. nitroderivatives and calcium-channel blockers) (Dagher et al, 2000).

In the endoscopic management of varices, banding has superseded injection sclerotherapy as the treatment of choice. Uncontrolled variceal haemorrhage may be controlled with a Sengstaken–Blakemore tube; however, this is a temporary measure until definitive treatment. A transjugular intrahepatic porto-systemic shunt (TIPSS) is a minimally invasive method of reducing portal pressure. If all attempts at haemostasis have failed oesophageal transection may be life-saving in some cases.

Risk of further bleeding

Further bleeding from peptic ulcers is associated with poor outcome and is predicted on the basis of findings at endoscopy such as the stigmata of recent haemorrhage, as described by Forrest et al in the 1970s (*Table 2*).

Table 2. Forrest endoscopic classification of stigmata of recent haemorrhage

Type		Risk of re-bleeding
Ia	Spurting haemorrhage	80–90%
Ib	Oozing haemorrhage	10–30%
IIa	Visible haemorrhage	50–60%
IIb	Adherent clot	25–35%
IIc	Black spot	0–8%
III	Clean ulcer base	0–12%

From Forrest et al (1974)

Risk assessment in AUGIH

Age is a major risk factor for death in AUGIH. Data show that under the age of 60 years and with no significant co-morbidities, mortality is of the order of 0.1% whereas in the 80 years and over age group mortality is 13% (*Figure 2*) (Rockall et al, 1995).

Other risk factors for death include major co-morbidities (such as malignancy, organ failure and respiratory disease), shock, re-bleeding, stigmata of recent haemorrhage, and the diagnosis (with highest mortality rates in AUGIH secondary to malignancy and varices).

Using these parameters a mortality risk assessment score has been devised (Rockall et al, 1996) and externally validated (Vreeburg et al, 1999) which gives a simple numerical score which can reproducibly predict mortality. Patients are given an initial score at presentation on the basis of age, the presence of shock and co-morbidities and then a final score according to the diagnosis and any stigmata of recent haemorrhage (*Table 3*).

The pre-endoscopy score of 0–7 has a step-wise increase in mortality, as does the post-endoscopy score with a mortality of 0% in those scoring 2 or less. Re-bleeding confers a 2–5-fold increase in mortality.

This scoring system has a variety of uses including the identification of patients at very low risk in whom admission for only a short time or even outpatient management might be safe. **BJHM**

Conflict of interest: none.

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Figure 2. Mortality for emergency admissions by age group with 95% confidence intervals.

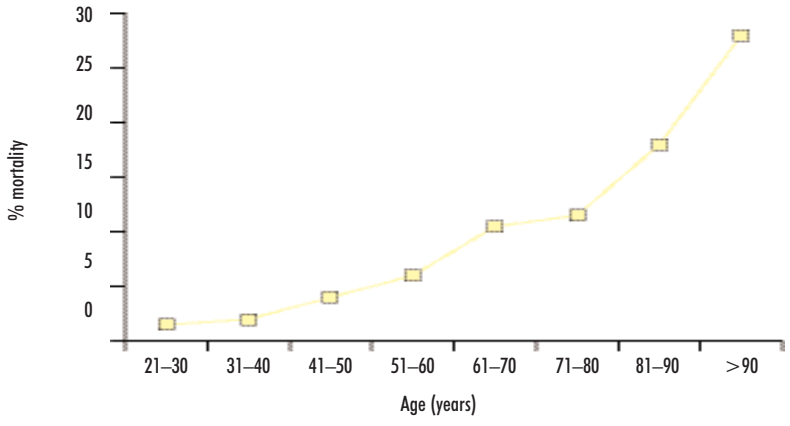

Table 3. Numerical risk score. Maximum additive score before diagnosis = 7, maximum additive score following diagnosis = 11

Score	0	1	2	3
Age (years)	<60	60–79	>80	
Shock	None	Pulse >100 bpm SBP <100mmHg	SBP>100mmHg	
Co-morbidity	None		Cardiac failure, ischaemic heart disease, major co-morbidity	Renal failure, liver failure, disseminated malignancy
Diagnosis	Mallory–Weiss tear, no lesion identified	All other diagnoses	Malignancy of upper GI tract	
Major SRH	None or dark spot only		Blood in upper GI tract, adherent clot, visible or spurting vessel	

bpm = beats per minute; GI = gastrointestinal; SBP = systolic blood pressure; SRH = stigmata of recent haemorrhage

KEY POINTS

- Acute upper gastrointestinal haemorrhage is a common reason for admission to hospital and cause of morbidity in inpatients.
- The diagnosis is a clinical one.
- Various therapeutic modalities are available.
- A numerical risk score can predict mortality and identify those who are at low risk.