Oesophageal Varices

Variceal haemorrhage occurs from dilated veins (varices) at the junction between the portal and systemic venous systems. Varices tend to be in the distal oesophagus and/or the proximal stomach but isolated varices may be found in the distal stomach, large and small intestine. The majority of patients with variceal bleeding have chronic liver disease. Bleeding is characteristically severe and may be life-threatening. The size of the varices and their tendency to bleed are directly related to the portal pressure, which is usually directly related to the severity of underlying liver disease. Large varices with red spots are at highest risk of rupture.\[1]\n
Epidemiology\[2, 3\]

- Bleeding from oesophageal varices is responsible for 5-11% of all cases of upper gastrointestinal bleeding (UGIB).
- In Western countries, alcoholic and viral cirrhosis are the leading causes of portal hypertension and oesophageal varices.
- 30% of patients with compensated cirrhosis and 60-70% of patients with decompensated cirrhosis have gastro-oesophageal varices at the time of presentation.
- Oesophageal varices develop in approximately 8% of patients with chronic liver diseases per year for the first two years and in 30% of patients by the sixth year.
- The risk of bleeding from oesophageal varices in the first year after identification is 30%.

Aetiology

The causes of oesophageal varices are anything that can cause portal hypertension. Some examples are in the table that follows.

<table>
<thead>
<tr>
<th>Causes of oesophageal varices</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Prehepatic</strong></td>
</tr>
<tr>
<td>- Portal vein thrombosis.</td>
</tr>
<tr>
<td>- Portal vein obstruction - congenital atresia/stenosis.</td>
</tr>
<tr>
<td>- Increased portal blood flow - fistula.</td>
</tr>
<tr>
<td>- Increased splenic flow.</td>
</tr>
<tr>
<td><strong>Intrahepatic</strong></td>
</tr>
<tr>
<td>- Cirrhosis due to various causes, including alcoholic hepatitis and chronic hepatitis (eg. viral or autoimmune).</td>
</tr>
<tr>
<td>- Idiopathic portal hypertension (hepatoportal sclerosis).</td>
</tr>
<tr>
<td>- Acute hepatitis (especially alcoholic).</td>
</tr>
<tr>
<td>- Schistosomiasis.</td>
</tr>
<tr>
<td>- Congenital hepatic fibrosis.</td>
</tr>
<tr>
<td>- Myelosclerosis.</td>
</tr>
<tr>
<td><strong>Posthepatic</strong></td>
</tr>
<tr>
<td>- Compression (eg, from tumour).</td>
</tr>
<tr>
<td>- Budd-Chiari syndrome.</td>
</tr>
<tr>
<td>- Constrictive pericarditis (and rarely right-sided heart failure).</td>
</tr>
</tbody>
</table>
Factors that increase the risk of variceal bleeding[4]
These are also the same factors that increase the risk of portal hypertension:

- Decompensation of liver disease.
- Malnourishment.
- Alcohol intake.
- Physical exercise.
- Circadian rhythms.
- Increased intra-abdominal pressure.
- Aspirin.
- Non-steroidal anti-inflammatory drugs (NSAIDs).
- Bacterial infection (cause of initial, and recurrence of, bleeding).

Presentation

Symptoms

- Haematemesis (most commonly), melaena.
- Abdominal pain.
- Features of liver disease and specific underlying condition.
- Dysphagia/odynophagia (pain on swallowing; uncommon).
- Confusion secondary to encephalopathy (even coma).

Signs

- Peripherally shut down.
- Pallor.
- Hypotension and tachycardia (ie shock).
- Reduced urine output.
- Melaena.
- Signs of chronic liver disease.
- Reduced Glasgow Coma Scale.
- Signs of sepsis may also commonly be present.

Investigations

Endoscopy is required at an early stage.[5, 6]

- FBC - haemoglobin may be low; MCV may be high, normal or low; platelets may also be low; WCC may be raised.
- Clotting including INR.
- Renal function.
- LFTs.
- Group and cross-match.
- CXR - patients may have aspirated or have chest infection.
- Asitic tap may be needed if bacterial peritonitis is suspected.
- Investigations as indication for the underlying cause of portal hypertension (see separate Portal Hypertension article).

Differential diagnosis

Other causes of UGIB, as listed in the separate Upper Gastrointestinal Bleeding (includes Rockall Score) article.
Management[7]

See also separate Upper Gastrointestinal Bleeding (includes Rockall Score) article.

Management for acute variceal haemorrhage consists of vasoactive drugs, endoscopic band ligation and antibiotics prophylaxis. Transjugular intrahepatic portosystemic shunt (TIPS) is reserved for those who do not respond or are unlikely to respond to initial standard management.[8]

National Institute for Health and Care Excellence (NICE) guidance[9]

- Terlipressin should be offered to patients with suspected variceal bleeding at presentation. Treatment should be stopped after definitive haemostasis has been achieved, or after five days, unless there is another indication for its use.
- Prophylactic antibiotic therapy should be offered at presentation to patients with suspected or confirmed variceal bleeding. Antibiotic use significantly reduces the mortality of patients who develop acute UGIB in association with chronic liver disease.[1]
- Oesophageal varices:
  - Band ligation should be used for patients with UGIB from oesophageal varices.
  - TIPS should be considered if bleeding from oesophageal varices is not controlled by band ligation.
  - NICE recommends that there is sufficient evidence to show that stent insertion is effective for selected patients with oesophageal varices in whom other methods of treatment have failed to control bleeding.[10]
- Gastric varices:
  - Endoscopic injection of N-butyl-2-cyanoacrylate should be offered to patients with UGIB from gastric varices.
  - TIPS should be offered if bleeding from gastric varices is not controlled by endoscopic injection of N-butyl-2-cyanoacrylate.

Resuscitation

- Assess airway - can easily be compromised, especially if reduced Glasgow Coma Scale.
- In the community, call an ambulance urgently - assess level of shock, lay the patient down and raise their legs (but, if actively vomiting then there is risk of aspiration, so the recovery position is more appropriate); provide oxygen and gain intravenous access if possible.
- Wide-bore access is needed, with a minimum of two cannulae (central access may also be required).
- Provide 40% oxygen if tachypnoeic or confused.
- Begin fluid resuscitation with rapid infusion of crystalloid and colloid solution.[4]
- Give blood (ideally cross-matched) as soon as possible - if a delay is likely then group O rhesus negative blood may need to be given. The initial aim is to correct hypovolaemia - this may require several litres.[1]
- Patients should be monitored with a cardiac monitor, blood pressure, pulse rate and urine output (catheterise until the patient is stabilised).
- Insert a nasogastric tube to assess the severity of the bleeding and to lavage gastric contents before performing endoscopy.[11]
- Correct anaemia and coagulopathy; blood transfusion will be necessary.[12] Many of the patients will have a coagulopathy and so give intravenous vitamin K immediately. If INR is prolonged, also give fresh frozen plasma. Platelet transfusion may be required.[13]
- There is some concern that saline infusions may worsen ascites but the nature of the emergency warrants their use.

Consider inserting a central venous line/pulmonary artery catheter in haemodynamically unstable or elderly patients, or those with cardiac or pulmonary disease, to monitor fluid balance (overuse can produce oedema, ascites and hyponatraemia).

Endoscopy

- Urgent endoscopy is necessary for variceal haemorrhage. Emergency fibre-optic endoscopy confirms the diagnosis and source of bleeding.
- Urgent endoscopy in unwell patients is not without its risks; adequate staffing and the presence of resuscitation facilities are required.
- Endoscopy allows the bleeding point to be visualised and treated:
  - Band ligation is the first choice of treatment.
  - Emergency sclerotherapy is still widely used as a first-line therapy for variceal bleeding in patients with cirrhosis, especially when band ligation is not available. However, drug treatment may stop bleeding in the majority of these patients. There is no convincing evidence to support the use of emergency sclerotherapy for variceal bleeding in cirrhosis as the initial single treatment when compared with vasoactive drugs.[14]

Vasoactive drugs

- The use of vasoactive agents (terlipressin, octreotide or somatostatin) is associated with a significantly lower risk of acute all-cause mortality and transfusion requirements and improved control of bleeding and shorter hospital stay.[15]
- Terlipressin is an analogue of vasopressin and studies have shown it to be superior to placebo in variceal haemorrhage.[4] Terlipressin is considered the vasoactive agent of choice in acute variceal bleeding.[16] It should be given to all patients presenting with suspected variceal bleeding prior to endoscopy and following confirmation.[1]
- Vasopressin and terlipressin should not be used in severe hypovolaemic shock and patients with severe cardiac disease.

Variceal obturation with glue

- This involves embolisation of varices with a glue-like substance (N-butyl-2-cyanoacrylate).
- It is particularly good for gastric or gastro-oesophageal variceal bleeding.
However, there is a risk of embolisation to the lung, spleen or brain.

**Balloon tube tamponade (Sengstaken-Blakemore tube)**
- Balloon tamponade should be considered as a temporary salvage treatment for uncontrolled variceal haemorrhage.\(^1\)
- The Sengstaken-Blakemore tube (preferably kept in the fridge to stiffen rubber and make passage easier) is inserted through the mouth and into the stomach.
- The gastric balloon is inflated with air and the gastric balloon is then pulled up against the oesophagogastric junction, compressing submucosal varices.
- The Sengstaken-Blakemore tube also contains an oesophageal balloon which is only rarely required when the gastric balloon does not work.
- If bleeding continues, it may be that the tube is wrongly positioned or bleeding is from another source.

**Transjugular intrahepatic portosystemic shunt**
- Transjugular intrahepatic portosystemic stent shunting is recommended as the treatment of choice for uncontrolled variceal haemorrhage.\(^1\)
- The hepatic vein is cannulated percutaneously via the internal jugular vein, using a needle under ultrasound or fluoroscopic guidance.
- A tract is created through the liver from the hepatic to the portal vein - thus reducing portal pressure. This is dilated and an expandable metal stent inserted to create a shunt.
- This has a high success rate but encephalopathy is found in 25% of cases (as portal blood diverted from the liver) and shunt occludes within one year in up to 50% of cases.\(^4\)

**Surgery**\(^7\)
- Oesophageal transection and gastric devascularisation are rare procedures but may have a role for patients with portal and splenic vein thrombosis who are not suitable candidates for shunt procedures and who continue to have variceal bleeding despite endoscopic and pharmacological treatment.\(^17\)
- Liver transplantation is the treatment of choice for patients with advanced liver disease.

**Other aspects of management of variceal haemorrhage**
- Avoid sedatives if possible.
- Patients are best managed on ITU/HDU.
- Antibiotic prophylaxis - infection occurs in up to 50% of patients and is associated with a worse outcome. It is thought that bacteria release endotoxins that enhance portal pressure and impair coagulation.\(^18\) Antibiotics are therefore given early - eg, ciprofloxacin.
- Treat hepatic encephalopathy if this is also present.

**Prevention**\(^8\)
- No specific treatment has been shown to prevent the formation of varices.
- Prevention of first variceal haemorrhage depends on the size/characteristics of varices. In patients with small varices and high risk of bleeding, non-selective beta-blockers are recommended, while patients with medium/large varices can be treated with either beta-blockers or oesophageal band ligation.
- Prevention of recurrent variceal haemorrhage consists of the combination of beta-blockers and endoscopic band ligation.

**Endoscopic screening**
- All patients with newly diagnosed cirrhosis should have screening endoscopy, looking for oesophageal varices.
- Presence of moderate or large varices requires beta-blockers in the first instance (indefinite treatment).
- If there are contra-indications to beta-blockers, the varices should be banded or sclerosed.\(^19\)
- In the long term, repeated endoscopic screening is usually required - eg, 2- to 3-yearly in cases of small varices.
- Patients who have survived an oesophageal variceal bleed should receive beta-blockers, ± nitrates and endoscopic screening and treatment.

The use of measures of hepatic vein pressure gradient with an aim to reduce it under 12 mm Hg is associated with a significant reduction in mortality.\(^20, 21\)

**Prognosis**\(^22\)
- Patients who have bled once from oesophageal varices have a 70% chance of rebleeding. Approximately one third of further bleeding episodes are fatal.
- The risk of death is maximal during the first few days after the bleeding episode and decreases slowly over the first six weeks.
- Associated renal, respiratory, cardiovascular and immune disorders account for 20-65% of mortality in patients with oesophageal varices.

**Further reading & references**

1. Management of acute upper and lower gastrointestinal bleeding; Scottish Intercollegiate Guidelines Network - SIGN (September 2008)


9. Acute upper GI bleeding; NICE Clinical Guideline (June 2012)

10. Stent insertion for bleeding oesophageal varices; NICE Intervventional Procedure Guidance, April 2011


Disclaimer: This article is for information only and should not be used for the diagnosis or treatment of medical conditions. Patient Platform Limited has used all reasonable care in compiling the information but makes no warranty as to its accuracy. Consult a doctor or other healthcare professional for diagnosis and treatment of medical conditions. For details see our conditions.