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Resuscitation Varices are ubiquitous in patients with portal hypertension irrespective of the aetiology and usually present with an acute, and significant mortality. The lower oesophagus is the most common site and the diagnosis should be suspected in a patient known to have cirrhosis, but confirmation of the source is required following initial resuscitation. Variceal haemorrhage is a medical emergency and failure to control variceal bleeding with current medical management occurs in 10–20% of cases. Patients with massive haemorrhage should be admitted to an intensive therapy unit, venous access obtained through two - Summary box 69.9 Causes of portal hypertension

Upper GI Overt blood bleeding from varices If severe and associated with variceal Hypersplenism Development of Portal varices hypertension CT/endoscopy Refractory ascites transplant candidate Figure 69.12 Management of complications of portal hypertension. CT, computed tomography; GI, gastrointestinal; TIPSS, transjugular intrahepatic portosystemic stent shunt. Pre-sinusoidal Extrahepatic: portal vein thrombosis, splenic vein thrombosis (pancreatitis, pancreatic tumour), myelofibrosis, arterioportal shunt, tropical splenomegaly Intrahepatic: schistosomiasis, congenital hepatic fibrosis and portal infiltration (sarcoidosis), drugs and toxins, veno-occlusive disease Sinusoidal Cirrhosis Post-sinusoidal Hepatic vein occlusion (Budd–Chiari syndrome), veno-occlusive disease, congestive cardiac failure Endoscopic Anaemia surveillance Endoscopic treatment loss Tamponade +/- endoscopic TIPSS treatment Massive bleeding Shunt or Recurrent devascularisation Liver transplant bleeding may need splenectomy (portal vein thrombosis may result) Treat only if signs Endoscopic of bleeding surveillance Peritoneovenous shunt or TIPSS if

large-bore peripheral cannulae and resuscitation commenced, ideally with blood. Liver function tests will reveal underlying liver disease and a coagulation profile will identify any coagulopathy. Hypervolaemia may increase portal pressure and exacerbate bleeding. Ten milligrams of vitamin K are administered intravenously but a coagulopathy requires FFP and activation of a major transfusion protocol. Thrombocytopenia secondary to hypersplenism is treated if the platelet count is $<50 \times 10^9$ Treatment protocols include the use of splanchnic vasoconstrictors, such as terlipressin, octreotide and somatostatin, and prophylactic antibiotics. When bleeding continues treatment options are sclerotherapy, banding, balloon tamponade and TIPSS. The use of oesophageal balloons should be avoided, which is usually possible when experienced endoscopists are available. As soon as the patient is haemodynamically stable the diagnosis should be confirmed endoscopically as 30% will have a non-variceal source of bleeding. Variceal bleeding is often associated with hepatic encephalopathy and endotracheal intubation may be required prior to

endoscopy to protect the airway and prevent aspiration (Figure 69.13). Robert William Sengstaken Sr , 1923–1978, neurosurgeon, Garden City , New York, NY , USA. Arthur Blakemore , 1897–1970, surgeon, the Columbia College of Physicians and Surgeons, New York, NY , USA

Balloon tamponade and self-expanding stents Balloon tamponade is effective for massive or refractory variceal bleeding but is only recommended as a 'bridge' to definitive treatment. If the rate of blood loss prohibits endoscopic evaluation, a Sengstaken–Blakemore tube (originally described in 1950) or a Minnesota tube (addition of an oesophageal aspiration port) can be inserted to provide temporary haemostasis (Figure 69.14). Once inserted, the gastric balloon is inflated with 300 mL of air and retracted to the gastric fundus and the oesophago-gastric varices tamponaded by inflation of the oesophageal balloon to 60 mmHg. The two remaining channels allow gastric and oesophageal aspiration, and the position of the tube is confirmed radiologically . A strict protocol for the management of balloon tamponade is important to avoid complications particularly oesophageal pressure necrosis. Recently , self-expanding covered metal oesophageal stents have also been employed for the emergency treatment of oesophageal varices and results are equivalent to balloon tamponade unless the bleeding site is intragastric.

Urgent Suspected Endoscopy resuscitation variceal Airway bleeding protection Antibiotics Terlipressin Shunt surgery or devascularisation Consider transplantation Figure 69.13 The management of variceal bleeding. TIPSS, transjugular intrahepatic portosystemic stent shunt. Oesophageal Sclerotherapy Bleeding Eradication varices or banding control programme Fundal Cyanoacrylate Bleeding varices (thrombin) continues Balloon tamponade Repeat Bleeding sclerotherapy controlled Significant Continued TIPSS bleeding bleeding continues

Endoscopic treatment The two most commonly used endoscopic techniques are endoscopic band ligation to the base of the varix and injection of a sclerosant into or around the varix. Following resuscitation, endoscopy is performed in a head-down position with good suction available. A double-channel endoscope with a bridge is essential to facilitate suction during injection and provide manoeuvrability of the needle, and power washers dramatically improve visualisation. Some time should be spent assessing the bleeding, confirming it is variceal and obtaining a stable position. When the bleeding varix or varices are identified only the source should be treated. Sclerotherapy or banding both achieve effective control with banding reducing rebleeding; a single treatment is usually sufficient. Transjugular intrahepatic portosystemic stent shunts The emergency management of variceal haemorrhage is extremely difficult when pharmacological and endoscopic therapies have failed. Treatment of these patients now relies on TIPSS, a radiological procedure first described in 1969 but not widely available until the development of endovascular stents in 1985. TIPSS has replaced surgical portocaval shunt and is now accepted as the preferred method for treating refractory portal hypertension. A TIPSS is inserted under local anaesthetic, analgesia and sedation using fluoroscopic guidance and ultrasonography . Via the internal jugular vein, superior vena cava and hepatic vein, a guidewire is inserted through the hepatic parenchyma into a branch of the portal vein. The tract is dilated; a metallic stent is then inserted and expanded, forming a portovenous channel (Figure 69.15). A satisfactory drop in portal venous pressure is usually associated with good control of the variceal haemorrhage. The main early complication is perforation of the liver capsule, with potentially fatal intraperitoneal haemorrhage. TIPSS occlusion may produce further variceal haemorrhage and occurs more

commonly in patients with well-compensated liver disease and good synthetic function. The incidence of post-TIPSS encephalopathy is comparable to that following surgical shunts (40%) and due to portal blood avoiding hepatic detoxification, if severe, flow is reduced by inserting a smaller stent. The main contraindication to TIPSS is portal vein occlusion, and long-term stenosis occurs in 50% of patients at 1 year. Surgical shunts The increasing availability of liver transplantation and TIPSS has greatly reduced the indications for surgical portosystemic shunts, which, because of their high morbidity and mortality, are now rarely considered for variceal haemorrhage. The current indication is the failure of medical management in non-cirrhotic patients with extrahepatic portal vein occlusion. Surgical shunts effectively prevent rebleeding from oesophageal

Oesophageal aspiration channel

Oesophageal balloon: 40 mmHg

Gastric balloon: at least 300 mL of air
Gastric aspiration channel

Figure 69.14 Oesophageal and gastric balloon tamponade with a Sengstaken-Blakemore or

Minnesota tube. The tube must be carefully managed. Figure 69.15

An angiogram following insertion of a transjugular intra-hepatic portosystemic stent shunt (TIPSS)

(open arrow). Contrast in

the portal vein flows through the metallic stent and outlines the right hepatic vein. Pressure measurements are taken from within the portal vein before and after insertion. Solid arrows indicate coils placed at the site of previous embolisation.

(c) or gastric varices by reducing portal pressure and are divided into selective, splenorenal and non-selective portocaval. Selective shunts attempt to preserve hepatoportal blood flow while decompressing the left side of the portal circulation, which is responsible for oesophageal and gastric varices (Figure 69.16 Selective shunts have a lower incidence of encephalopathy but there is no evidence that prophylactic shunting is beneficial. Recurrent or refractory variceal bleeding Sugiura procedure The Sugiura procedure for oesophageal varices combines splenectomy with oesophagogastric devascularisation, permanently interrupting the intraoesophageal portacaval shunt while preserving perioesophageal varices. The surgery is performed on the stomach wall and all venous tributaries are divided as for highly selective vagotomy except on both the lesser and greater curves. The upper half of the stomach and 8–10 cm of oesophagus are cleared (less than originally described but avoiding entering the chest). After devascularisation with careful preservation of the collateral channels and the vagus, a large oesophageal stapler is introduced into the lower oesophagus, which is transected just above the cardia.

Figure 69.16 Surgical shunts for portal hypertension involve shunting portal blood into the systemic veins. This commonly involves a side-to-side portocaval anastomosis (a) or end-to-side portocaval (b) , mesocaval 'H graft'

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