Successful use of a transjugular intrahepatic portosystemic stent shunt to control severe refractory oesophageal variceal haemorrhage in a poor risk patient

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Summary: This report describes a 44 year old man with a severe gastrointestinal haemorrhage from oesophageal varices. Bleeding could not be controlled with conservative therapy and sclerotherapy. He was successfully treated with a radiologically guided transjugular intrahepatic stent shunt at a time when his condition was too poor to attempt an open surgical procedure.

Introduction

Haemorrhage from oesophageal varices in patients with portal hypertension remains a common and serious medical emergency associated with significant mortality and morbidity. Injection sclerotherapy is the most effective primary therapy for acute variceal bleeding. Pharmacotherapy and balloon tamponade have useful supporting roles.¹² Despite therapy, some 10–20% of patients may continue bleeding³–⁵ and in these cases the prognosis is poor.³ We report a case of severe refractory bleeding in a patient with poor outlook, eventually controlled with a transjugular intrahepatic portosystemic stent shunt (TIPSS). This should lead to wider consideration and evaluation of this technique as an alternative to surgery in critically ill patients.


References

Case report

A 44 year old man was admitted to hospital with haematemesis and melaena. He had consumed excess alcohol (100 units/week) for the last 10 years. Examination revealed postural hypotension, irregular hepatomegaly, grade one encephalopathy, no oedema or ascites. Investigations showed: bilirubin 44 μmol/l; albumin 24 g/l; aspartate transaminase 50 IU/l; gamma glutamyl transferase 296 IU/l; alpha fetoprotein 2 IU/l; prothrombin time 23 seconds (control 16 seconds); haemoglobin 5.9 g/dl; platelets $5 \times 10^9$/l.

He was resuscitated with blood and blood products and the bleeding stopped spontaneously within 24 hours. Subsequently he had a further bleeding episode on day 3 when endoscopy showed three large actively bleeding variceal columns. Sclerotherapy was performed but bleeding continued, and balloon tamponade, intravenous vasopressin and isosorbide dinitrate were used to stabilize the bleeding. Sclerotherapy performed the next day was successful in arresting the haemorrhage.

Further haemodynamically significant bleeding on day 12 (when routine sclerotherapy had been planned) was controlled by intravenous vasopressin/isosorbide dinitrate followed by sclerotherapy. On the 18th day following admission he had a fourth bleeding episode and, at endoscopy, two large bleeding variceal columns were seen. Injection sclerotherapy was performed but brisk bleeding continued despite the additional use of balloon tamponade and vasopressin/nitrate. All endoscopic procedures were carried out by a consultant gastroenterologist with considerable experience of emergency sclerotherapy. Olympus GIF 1TV10 endoscopes were used, sclerotherapy was performed with a disposable 25 gauge needle with intended intravariceal injection of 5% ethanolamine oleate up to 5 ml per column.

As he had continued to bleed, by this stage his prognosis was extremely poor; the Childs–Pugh score was 15. He has a large amount of tense ascites and grade four encephalopathy with plasma albumin 25 g/l, prothrombin time 29 seconds and bilirubin 162 μmol/l. Endotracheal intubation and intermittent positive pressure ventilation was necessary because of pneumonia and persistent hypoxia.

A transjugular intrahepatic stent shunt was inserted as an emergency manoeuvre rather than undertake an open surgical procedure. Following right internal jugular vein puncture, the right hepatic vein was catheterized and the right main portal vein punctured through a 10 Fr. sheath with a coaxial catheter needle system (William Cook Europe, Bjaeverskov, Denmark). The track was dilated to 8 mm and a 68 mm long, 10 mm diameter Wallstent (Schneider, Minneapolis, MN, USA) was inserted. Following insertion of the shunt, the mean portosystemic pressure gradient decreased from 20 to 13 mmHg. Fluoroscopy and ultrasound were used to guide placement. The patient was mechanically ventilated throughout the procedure which resulted in cessation of active bleeding. Subsequently there were no further bleeding episodes and great improvement in all clinical and laboratory features was seen. Repeat endoscopy on day 32 showed no residual varices. The patient was discharged well (Childs–Pugh class A) on day 37 taking propranolol and lactulose. At follow-up at 12 months he remains well, abstinent from alcohol, with no evidence of decompensation or complications. Duplex colour Doppler scanning shows the shunt to be patent and functioning.

Discussion

Injection sclerotherapy is currently the most effective treatment for acute variceal bleeding. Compared to balloon tamponade and pharmacotherapy, it shows improved bleeding control, reduced rate of rebleeding and improved survival. Endoscopic variceal ligation seems to be an effective alternative to sclerotherapy but is not as well proven or widely available. Despite maximal conservative therapy a significant proportion may continue to bleed or rebleed in hospital. The additional success rate for third and subsequent sclerotherapy sessions is low. When two sclerotherapy sessions have failed to arrest the bleeding, surgical shunting or oesophageal transection is often advised. However, by this stage the general condition of many patients is so poor it precludes surgery. The mortality of emergency surgical procedures in patients with severe disease, such as the one described, is high (50–80%). New approaches to the management of refractory bleeding are needed.

Percutaneous intrahepatic stent shunts were first described in an animal model in 1969 and transjugular intrahepatic shunting was first reported in humans in 1982. Since then the technique has been used successfully in a small number of patients, in the USA and mainland Europe, to reduce portal pressure and stop variceal bleeding in patients with cirrhotic portal hypertension refractory to sclerotherapy with and without variceal embolization. Associated morbidity and mortality was relatively low. The patient described here had the most severe disease state at the time of shunting of those yet described. Percutaneous decompression of the portal venous system minimizes the risks associated with bleeding or general anaesthesia associated with open surgery. In this
patient it allowed control of variceal bleeding refractory to sclerotherapy, tamponade and pharmacotherapy without recourse to surgery.

Beta-blockers prevent variceal haemorrhage and reduce mortality when used in secondary prophylaxis. Partial decompression of the portal hypertension had been achieved by mechanical means but a significant portosystemic gradient remained. Propranolol was continued in an attempt to further reduce his risk of recurrent variceal haemorrhage. Although there are insufficient data reported on combining pharmacotherapy and TIPSS, it has been shown that combinations of different pharmacological therapies have useful additive portal hypotensive effects. Long-term follow-up is unavailable and the exact place of TIPSS in the management of lower risk patients and its relationship to non-operative and operative modalities needs to be defined. It should be considered as a potentially life-saving procedure in critically ill patients with variceal bleeding.

References


Myositis due to cholesterol emboli

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Summary: Myositis due to spontaneous cholesterol embolization is uncommon and usually associated with cutaneous abnormalities at presentation. A case of myositis due to cholesterol emboli is reported. The patient presented with painful weak legs, and the diagnosis was confirmed by muscle biopsy.

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