CASE REPORT

Upper gastrointestinal bleeding in cirrhosis: varix or no varix?

Alexandre Oliveira Ferreira,¹ Helena Tavares Sousa,¹ Jorge Brito,² Luis Rosa³¹Department of Gastroenterology and Hepatology, Centro Hospitalar Barlavento Algarvio, Portimão, Portugal²Department of Radiology, Centro Hospitalar Barlavento Algarvio, Portimão, Portugal³Department of Radiology, Fundação Champalimaud, Lisbon, Portugal

Correspondence to

Dr Alexandre Oliveira Ferreira, alex.fsof@gmail.com

SUMMARY

Upper gastrointestinal bleeding from variceal origin is a frequent complication in the cirrhotic population. Duodenal variceal haemorrhage, however, is infrequent and the endoscopic management of such lesions is not straightforward. Non-endoscopic options include vasoactive drugs, transjugular intrahepatic portosystemic shunt (TIPS), transvenous obliteration and surgery as rescue therapy. We present a patient with Child-Pugh A hepatitis C virus-cirrhosis with acute bleeding from a duodenal varix. It was managed with elastic band ligation but late rebleeding occurred after 6 weeks. Gastroduodenoscopy revealed active bleeding from the ligation eschar. Band ligation and sclerosis were attempted but unsuccessful. Terlipressin was started and the patient referred for TIPS. Surprisingly, angiography showed a normal hepatic vein pressure gradient; therefore, TIPS was not performed. Haemorrhage ceased with medical treatment alone. The patient remained stable and was discharged after 10 days, being currently under evaluation for hepatitis C therapy.

BACKGROUND

Ectopic varices are accountable for 1–5% of all gastrointestinal (GI) variceal bleeding.^{1–2} They are usually related to portal hypertension, intra-abdominal surgery, vascular thrombosis or vascular malformations.³

Because of its low incidence, the evidence supporting the currently adopted therapeutic methods is scant and there are no randomised trials on this subject. Case reports and case series include medical treatment with vasoactive drugs, elastic band ligation, cyanoacrylate⁴ and human thrombin injection^{5–6} as endoscopic modalities, transjugular intrahepatic portosystemic shunt (TIPS) and balloon-occluded retrograde transvenous obliteration (BRTO) by means of interventional radiology and, finally, performing a surgical shunt.³

Terlipressin is a synthetic analogue of vasopressin available in Europe but not in the USA. It can be administered in boluses and is considered the first-line pharmacological therapy in variceal bleeding after being shown to reduce treatment failure and mortality.⁷ Terlipressin has an important systemic vasoconstrictor effect more noticeable on the splanchnic arteries, causing an increase in systemic vascular resistance and arterial pressure as well as a significant (roughly 20%) and sustained (up to 4 h) decrease in portal vein pressure and flux.^{8–10}

This case illustrates the difficulty in endoscopic management of ectopic varices but more importantly it emphasises the need to integrate all information

available and interpret it cautiously. In our case it is important to note the significant portal pressure lowering effect of terlipressin and active bleeding.

CASE PRESENTATION

A 45-year-old white man with a history of active alcoholism and chronic hepatitis C presented to the emergency department for haematemesis and haematochezia beginning 5 days before. The patient was alert and eupneic. He had a pale tone and was sudoretic, tachycardic (140 bpm) and hypotensive (87/36 mm Hg). Apart from an enlarged palpable liver, physical examination was unremarkable for other stigmata of chronic liver disease.

Blood tests were as follows: haemoglobin 5.8 g/dL (13.5–18); mean cell volume (MCV) 112 fL (78–100); white cell count (WCC) 14 000/ μ L (4000–10 000); platelet count 92 000/ μ L (150 000–400 000); international normalised ratio (INR) 1.80; bilirubin 0.5 mg/dL (0.2–1.0); albumin 3.2 g/dL (3.2–4.8); aspartate aminotransferase (AST) 47 U/L (10–34), alanine transaminase (ALT) 13 U/L (10–44) and γ -glutamyl transpeptidase (GGT) 156 U/L (11–50). His hepatitis C virus (HCV) viral load was 894 IU/mL and model for end-stage liver disease (MELD) score 13.

After adequate haemodynamic resuscitation, with fluids and red blood cell (RBC) packs, the patient underwent urgent upper GI endoscopy, which showed innocent small oesophageal varices, mild portal hypertensive gastropathy and no gastric varices. Finally, an oozing bleeding from a varix located at the second portion of the duodenum (figure 1) was noticed and successfully controlled by means of two-rubber-band ligation (figure 2).

Figure 1 Oozing varix on the second portion of the duodenum.

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Figure 2 Endoscopic variceal band ligation.

Somatostatin was administered for 5 days. A total of seven RBC packs were needed.

A triphasic CT of the abdomen (figure 3) revealed an enlarged liver with slightly lobulated contour, a normal spleen and portal vein but with signs of portosystemic collateral vessels with para-oesophageal varices and enlargement of the paraumbilical vein.

The evolution was uneventful and the patient was discharged after 14 days.

After 4 weeks he was again admitted to the emergency department for haematemesis with hypotension (84/43 mm Hg) and tachycardia (102 bpm), with a haemoglobin of 7.7 g/dL.

Urgent endoscopy revealed an oozing bleeding from the previous ligation ulcer on the second portion of the duodenum (figure 4). Elastic band ligation and sclerosis was attempted with marginal effect (figure 5). Terlipressin was started and the patient referred for TIPS placement.

On angiography the hepatic vein (HV) was catheterised and measurement of suprahepatic free and wedge pressure was remarkable for the absence of a gradient. Selective arteriography of the coeliac trunk and superior mesenteric artery was also performed, being negative for active bleeding. TIPS placement was not attempted based on the absence of an HV gradient on the haemodynamic study.

Figure 3 CT scan of the abdomen (venous phase) showing an enlarged liver with slightly lobulated contour and repermeabilisation of the paraumbilical vein.

Figure 4 Oozing from the ligation eschar.

Liver biopsy was undertaken and showed evidence of cirrhosis.

Terlipressin was continued for 5 days (2 mg every 4 h for the first 3 days, followed by 1 mg every 4 h on the last 2 days) and a total of three RBC packs were administered.

OUTCOME AND FOLLOW-UP

The patient remained stable for a week and was discharged afterwards.

He had no rebleeding and is currently abstemic for 6 months.

He is currently under evaluation for HCV treatment.

DISCUSSION

Duodenal variceal bleeding is an infrequent condition that poses several management issues, especially due to the lack of high-quality evidence. Currently it is advocated to start vasoactive medication and to try endoscopic measures if technically feasible, with band ligation for small varices only (due to the risk of a large mucosal defect) and cyanoacrylate injection being the most consensual.^{4 11–13} There are reports on alternative endoscopic techniques like human thrombin injection^{5 6} that may be less likely to cause thromboembolic complications.¹⁴ In case of treatment failure, options include interventional radiology either

Figure 5 Bleeding after failure of endoscopic haemostasis.

with TIPS placement or with other less studied modalities like BRTO, percutaneous transhepatic variceal embolisation (PTVE) or partial splenic embolisation.^{15 16} For TIPS, careful evaluation of liver function using Child Pugh and MELD is mandatory. With BRTO and PTVE there seems to be an increased risk for recurrence of ectopic varices elsewhere.¹⁷ Portal decompressive surgery is nowadays seen as the last savage option, as it has fallen in disfavour for the promising and less invasive radiology procedures.

In our case the main event was the surprisingly normal pressure gradient assessment by the transjugular haemodynamic study, which precluded TIPS placement, due to uncertainty on the liver cirrhosis diagnosis.

All previous diagnostic examinations were reviewed and a liver biopsy was performed, confirming the diagnosis of liver cirrhosis. Indeed, putting all into perspective, we concede that the personal history was compelling but not enough and that the lab tests were supportive but with limited predictive value. However, the portosystemic collaterals were undoubtful due to portal hypertension, related to liver cirrhosis, which was later confirmed by histology. Thus, this patient did have Child-Pugh A liver cirrhosis with portal hypertension which was significantly decreased, to normal values, presumably because of recent bleeding and terlipressin at the time of the catheterism.

At the end he was fortunate that the bleeding stopped and the indication for TIPS waived.

Learning points

There are several key points to note on the present case:

- ▶ Difficulty in diagnostic and management of ectopic varices.
- ▶ The effect of terlipressin as an effective portal pressure lowering drug as measured by angiography.
- ▶ The management of liver cirrhosis and portal hypertension requires a careful interpretation of all available information and active multidisciplinary discussion.
- ▶ Transjugular intrahepatic portosystemic shunt is an irreversible procedure with a high complication rate and should be reserved for very selected cases.

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Contributors All the authors were involved in patient care and in the writing and correction of the manuscript.

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