

Case report



Two cases of variceal haemorrhage during living-donor liver transplantation

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Editor's key points

- Two cases of massive haemorrhage from varices during liver transplantation are described.
- Raised venous pressure after portal cross-clamping is a potential mechanism.
- Observation of the nasogastric tube during cross-clamping is recommended.

Some patients with cirrhosis experience rupture of venous varices before operation, and liver transplantation is a therapy of last resort for these patients. However, we have experienced two cases of intraoperative rupture in whom no abnormalities of the venous varices were seen on endoscopy before operation. One patient with ruptured gastrointestinal varices was treated by direct surgical ligation and the other with ruptured oesophageal gastric varices, spontaneously recovered with a Sengstaken–Blakemore tube. These cases suggest that acute variceal haemorrhage should always be considered as a possibility during living-donor liver transplantation in patients with a history of upper gastrointestinal bleeding. Careful observation of the nasogastric tube is important during clamping of the hepatic portal vein.

Keywords: living-donor liver transplantation; portal hypertension; variceal haemorrhage

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Recurrent life-threatening variceal haemorrhage secondary to portal hypertension is an indication for liver transplantation. However, clamping of collateral veins and portal hypertension can cause massive intraoperative haemorrhage. There have been reports of the use of decompression of portal hypertension using a transjugular intrahepatic portosystemic shunt.¹ However, in our institution, we usually use splenic artery ligation and a portosystemic shunt.^{2,3} Despite these measures, we experienced two cases of massive haemorrhage due to oesophagogastric varices and gastrointestinal varices after clamping of the hepatic portal vein or associated collateral vein. The cases were treated by surgical intervention and by compression with a Sengstaken–Blakemore (SB) tube.

Case report

Case 1

A 53-yr-old man with uncompensated cirrhosis due to hepatitis B and hepatocellular carcinoma received a living-donor liver transplantation (LDLT) from his healthy son. His BMI was 25.9 kg m⁻² (height, 172 cm; weight, 66.2 kg). He had previously experienced episodes of massive oesophageal variceal bleeding which were treated by endoscopic injection sclerotherapy (EIS). Preoperative oesophageal endoscopy

showed no signs or symptoms of rupture. Laboratory evaluations included: haemoglobin, 9.9 g dl⁻¹; white blood cell count, 4400 µl⁻¹; platelet count, 37 000 µl⁻¹; aspartate aminotransferase (AST), 78 IU litre⁻¹; alanine aminotransferase (ALT), 52 IU litre⁻¹; albumin (Alb), 2.5 g dl⁻¹; prothrombin time–international normalized ratio (PT–INR), 1.99; total bilirubin (T-Bil), 6.3 mg dl⁻¹; creatinine, 0.61 mg dl⁻¹; and NH₃, 45 µg dl⁻¹. His Child–Turcotte–Pugh score was 13 and the model of end-stage liver disease (MELD) score was 21. A large number of collateral veins were observed on contrast abdominal computed tomography (CT) and prophylactic splenic artery modulation was used to decompress the portal hypertension. Anaesthesia was induced with midazolam, remifentanyl, and isoflurane with rocuronium for tracheal intubation. Anaesthesia was maintained with isoflurane and remifentanyl. The pre-anhepatic phase proceeded uneventfully, with stable systemic arterial pressure and central venous pressure (CVP) remaining at 5–10 mm Hg. However, after the ligation of the ligamentum teres hepatis, arterial pressure gradually reduced. A definite bleeding source was not identified in the surgical field, but sudden gastric distension was observed. The CVP also gradually decreased and rapid transfusion was initiated. The circulation was supported pharmacologically with phenylephrine and a norepinephrine infusion at 0.05–0.1 µg kg⁻¹ min⁻¹ to

maintain a mean arterial pressure >55 mm Hg. The nasogastric tube was blocked by clots, but sudden haemorrhage from the mouth and nose helped to identify the bleeding as coming from an upper gastrointestinal varix. The stomach was opened in an attempt to control the bleeding under direct vision. The varix was oversewn in stages by intermittently releasing the applied pressure and allowing time for blood volume to be restored. All respective collateral veins were ligated after liver implantation, and the haemodynamic status was gradually stabilized. In total, 90 units of packed red cells, 150 units of fresh-frozen plasma (FFP), and 60 units of platelets were required. After transplantation, further transfusions were required, but the haemodynamic status and laboratory data remained stable (haemoglobin: 9.1, 8.9, and 9.1 mg dl⁻¹ and INR: 1.61, 1.44, and 1.54 on postoperative days 1, 2, and 3). Endoscopic examination confirmed the absence of bleeding and the trachea was extubated in the intensive care unit on postoperative day 1. No bleeding from the varix occurred after operation.

Case 2

A 52-yr-old man with uncompensated cirrhosis due to hepatitis C received an LDLT from his healthy daughter. His BMI was 30 kg m⁻² (height, 158 cm; weight, 62 kg). He had previously experienced episodes of massive oesophageal variceal bleeding treated by EIS and endoscopic variceal ligation (EVL). However, preoperative oesophageal endoscopy showed no signs or symptoms of rupture. Laboratory evaluations included: haemoglobin, 10.1 g dl⁻¹; white blood cell count, 6140 µl⁻¹; platelet count, 69 000 µl⁻¹; AST, 44 IU litre⁻¹; ALT, 26 IU litre⁻¹; Alb, 2.4 g dl⁻¹; PT-INR, 1.32; T-Bil, 3.42 mg dl⁻¹; creatinine, 3.48 mg dl⁻¹; and NH₃, 133 µg dl⁻¹. His Child-Turcotte-Pugh score was 14, and MELD score was 26. Collateral veins including splenorenal and periumbilical shunts were observed on preoperative abdominal contrast CT. Prophylactic splenic artery modulation for decompression of portal hypertension was not conducted due to his poor condition, particularly renal dysfunction. Anaesthesia was induced with midazolam, remifentanyl, and isoflurane, with rocuronium for tracheal intubation. Intraoperative anaesthesia was maintained with isoflurane and remifentanyl. The patient was haemodynamically fairly stable during the pre-anhepatic phase but, when the portal vein was clamped, there was immediate bleeding from the mouth and nasogastric tube. Massive transfusion and inotropic support with norepinephrine was needed to maintain arterial pressure. Bleeding from an oesophageal gastric varix rupture was suspected and confirmed by endoscopy. Bleeding could not be controlled endoscopically, so an SB tube was introduced. The position of the tube was confirmed by chest X-ray. Bleeding was controlled gradually as surgery progressed. Collateral veins were ligated directly after implantation. The SB tube was removed immediately after transplantation, and bleeding from the nasal tube did not reoccur. In total, 86 units of packed red cells, 110 units of FFP, and 40 units of platelets

were required. The haemodynamic status gradually stabilized without inotropic support. No further transfusion was required and on postoperative days 1, 2, and 3, haemoglobin was 9, 9.2, and 11.2 mg dl⁻¹ and INR was 1.17, 1.12, and 1.13. The trachea was extubated on the intensive care unit on postoperative day 1 after endoscopic examination. The postoperative course was unremarkable regarding general condition, including the lung, liver, and renal function.

Discussion

We describe two cases of massive intraoperative haemorrhage due to rupture of oesophageal or gastrointestinal varices after clamping of collateral hepatic portal veins. One case was treated by supported therapy and direct ligation while the other was treated by compression with an SB tube.

Both cases occurred during the anhepatic phase when the hepatic portal vein was clamped. There have been a few similar previous case reports of massive bleeding from varices due to portal hypertension during liver transplant.^{4, 5} In a case of massive upper gastrointestinal haemorrhage immediately after cross-clamping of the inferior vena cava and hepatic portal vein, arterial pressure was maintained by inotropes and repeated transfusion, and the varices were oversewn successfully without relapse.⁴ Bladder tamponade due to vesical varices has been reported during the anhepatic phase of liver transplantation and was treated by direct ligation.⁵ In our report, haemorrhage also occurred due to a temporary increase in portal vein pressure. In this scenario, veno-venous bypass, which reduces systemic venous congestion and may protect the recipient from the effects of reduced cardiac output and altered systemic vascular resistance, is an option. However, it is controversial.⁶ At our centre, veno-venous bypass is instituted only as a last resort in the presence of marked haemodynamic instability. Both the piggyback technique and the establishment of a portocaval shunt require temporary restriction of flow through the portal system, which in turn creates an increase in portal pressure. This pressure alteration could induce variceal rupture at the time of flow restriction, and this may have been the mechanism in our cases.

In both cases, an NG tube had already been placed blindly by the surgeon in ward just before they came to the operating theatre. In Case 1, although a gastrointestinal varix ruptured, the tube was obstructed and the stomach provided a temporary blood reservoir resulting in delayed diagnosis. However, in Case 2, rupture of the oesophageal varix was diagnosed immediately because of the nasogastric tube. Careful attention to the nasal tube is recommended in patients with a previously treated varix.

Most patients with cirrhosis and varices have haemorrhage within a year of diagnosis, and about two-thirds of these patients will have recurrent variceal bleeding, most within 6 months of the index episode.⁷ In both of our patients, at least 1 yr had passed since the last bleeding episode, and endoscopy before operation showed no signs

of bleeding. Intraoperative drugs for portal hypertension, such as β -adrenergic blockers or vasopressin, may prevent bleeding by reducing the pressure.⁸ The SB tube is used less commonly as endoscopic intervention is now available, but has been used successfully for gastrointestinal haemorrhage related to liver transplantation,⁹ and should be considered as a potential intervention in an emergency situation.

In conclusion, acute variceal haemorrhage should always be considered during LDLT in patients with a history of upper gastrointestinal bleeding. When clamping the hepatic portal vein, the nasogastric tube should be carefully observed.

Conflict of interest

None declared.

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