Acute Gastric Variceal Bleeding During Orthotopic Liver Transplant

Stephen Aniskevich, Timothy S. Shine, David P. Shapiro

Abstract

We present a case of intraoperative gastric variceal bleeding during liver transplant. After an uneventful induction and surgical dissection, our patient developed hemodynamic instability during the anhepatic phase. We believe that an increase in portal pressures, owing to clamping of the portal system, led to spontaneous variceal rupture; however, placement of an oral gastric tube or transesophageal echocardiography probe may have contributed to this also. After intraoperative banding, the patient was stabilized and surgery proceeded uneventfully. The patient had no long-term sequelae. Anesthesiologists involved in the care of patients with end-stage liver disease should be aware of this infrequent intraoperative complication and be prepared to treat it appropriately.

Key words: *Transesophageal echocardiography, Intraoperative variceal banding, Liver transplantation, Portal hypertension*

Introduction

Gastrointestinal bleeding is a well-established complication of end-stage liver disease; however, intraoperative, upper gastrointestinal bleeding during liver transplant is rare (1). Clamping the portal vein during the anhepatic phase of transplant leads to an increase in portal pressure, which can result in variceal rupture. Instrumentation of the esophagus with an oral gastric tube and transesophageal echocardiography probe also could result in variceal injury. Because liver transplant is often associated with hemodynamic instability, vigilance on the part of the anesthesiologist can detect this complication early and prevent long-term sequelae. We present a case of intraoperative gastric variceal bleeding during an orthotopic liver transplant.

Case Report

A 55-year-old white woman (87 kg, 168 cm) presented for an orthotopic liver transplant for end-stage liver disease secondary to nonalcoholic steatohepatitis. Complications included mild encephalopathy, diuretic refractory ascites, partial portal vein thrombosis, and portal hypertension with a history of esophageal variceal bleeding treated with variceal banding.

While waiting for the transplant, the patient received a total of 4 upper endoscopies. Two endoscopies, performed at 6 and at 12 months before transplant, showed esophageal varices (grade 2) and were successfully banded each time. The endoscopy performed 6 months preoperatively also showed some evidence of bleeding and grade 1 gastric varices. Additionally, the patient received upper endoscopies at 5 months and again at 2 weeks preoperatively, which showed no gastric varices and trace esophageal varices.

On the day of transplant, the patient was brought to the operating suite where she underwent uneventful, rapid-sequence induction using propofol, succinylcholine, and fentanyl. After intubation and invasive line placement, an orogastric tube was placed to decompress the stomach and then removed. A transesophageal echocardiogram probe then was placed to obtain a mid-esophageal 4-chamber view. Initial evaluation from a mid-

From the Mayo Clinic Florida, Jacksonville, FL, USA
Address reprint requests to: *Stephen Aniskevich MD, Mayo Clinic Florida, Dept of Anesthesiology, 4500 San Pablo Rd. Jacksonville, Fl 32224*
Phone: *+1 904 953 2000* ***Fax:*** *+1 904 956 3332* ***E-mail:*** *aniskevich.stephen2@mayo.edu*

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esophageal depth showed a hyperdynamic heart with no regional wall motion abnormalities.

Surgery was uneventful through the dissection phase; however, 5 minutes after entering the anhepatic phase of surgery, the patient developed hemodynamic instability evidenced by systolic blood pressure decreasing from a baseline of 100-120 to 70- 80 mm Hg. Central venous pressure remained at 10 to 12 mm Hg, and there was no evidence of inferior vena cava compression by the surgeon or clamps. No arrhythmias were noted, and no obvious bleeding source could be identified within the surgical field. Re-evaluation of the heart by transesophageal echocardiogram showed no evidence of ischemia. Transgastric views were attempted at this time, but the heart could not be visualized well. Laboratory results showed a decrease in the hemoglobin concentration from 111 g/L (11.1 g/dL) to 103 g/L (10.3 g/dL). The patient responded to frequent small boluses of fluids and phenylephrine.

After recirculation, the patient continued to have hemodynamic instability, requiring administration of vasopressin and norepinephrine to maintain systolic blood pressures in the low 90s. Hemoglobin levels also started a slow downward trend to a nadir of 71 g/L (7.1 g/dL), despite aggressive administration of packed red blood cells. The patient also showed evidence of fibrinolysis on thromboelastography, which was corrected with platelets, fresh frozen plasma, cryoprecipitate, and a small dose of aminocaproic acid.

Owing to continued hemodynamic instability, a full transesophageal echocardiogram examination was reattempted; however, transgastric views were still unobtainable. At this time, blood was noted in the oropharynx and coming from the mouth. The transesophageal echocardiogram probe was removed with no evidence of tissue debris. An orogastric tube was reinserted, and approximately 700 cc of bloody fluid was removed from the stomach. The orogastric tube then was placed to suction, and an additional 200 cc of blood was removed during the course of the next 30 minutes. Gastric lavage was performed with 1 liter of saline without clearing; therefore, an emergent intraoperative upper endoscopy was performed.

The upper endoscopy revealed 1 actively bleeding gastric varices at the level of the gastroesophageal junction. The gastroenterologist successfully placed 4 bands to control the bleeding. After endoscopy, the patient's hemoglobin stabilized at 100g/L (10 g/dL), and norepinephrine was titrated downward. Vasopressin was discontinued before the end of surgery.

Her postoperative course in the intensive care unit was complicated by delayed graft function, but she had no evidence of rebleeding, and was eventually discharged home on postoperative day 9.

Discussion

Gastrointestinal bleeding is a well-established complication of end-stage liver disease; however, intraoperative upper gastrointestinal bleeding during liver transplant is rare (1). More commonly, gastrointestinal bleeding is a preoperative or postoperative complication of liver transplant. A variety of causes has been ascribed (2-5).

Our patient presented at the operating suite with several risk factors for upper gastrointestinal bleeding, including portal hypertension, prior upper gastrointestinal bleeding from esophageal varices, ascites, and poor coagulation due to her cirrhosis. Because of these risks, our practice is to limit the initial transesophageal echocardiogram examination to a mid-esophageal level to try to minimize variceal rupture. We hypothesize that clamping of the portal system at the start of the anhepatic phase caused portal pressure to increase, leading to spontaneous rupture of the gastric varices. The resulting hemodynamic instability prompted a more-detailed transesophageal echocardiogram examination with entrance into the stomach for transgastric views. Whether this contributed or exacerbated to the bleeding is unknown, but we were unable to obtain good views of the cardiac structures. This could suggest a large amount of blood already in the stomach, which prevented good contact of the ultrasound probe with the stomach lining. An alternative hypothesis is that insertion of the oral-gastric tube for stomach decompression resulted in variceal rupture, and the bleeding worsened after clamping owing to increases in the portal pressure.

The diagnosis of intraoperative variceal rupture can be difficult owing to the hemodynamic instability commonplace during liver transplant. Vigilance may be the anesthesiologist's best ally in detecting and treating this complication. It is important to rule out the usual causes of hypotension, such as obvious

bleeding in the surgical field, cardiac disturbances, or electrolyte abnormalities. When variceal bleeding is suspected, early endoscopic evaluation and intervention should be performed.

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