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Upper GI Bleeding in a Post-Liver Transplant Patient

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Question

A 45-year-old man presented to the emergency room with large-volume hematochezia and subsequent hematemesis. His medical history is notable for alpha-1 antitrypsin deficiency with eventual liver failure. He underwent orthotopic liver transplantation 9 months prior, which was complicated by ischemic cholangiopathy with recurrent cholangitis and need for serial percutaneous cholangiogram tube exchanges. He underwent retransplantation 2 months before presentation, this time with a relatively uncomplicated postoperative course. The patient was not on aspirin or any nonsteroidal anti-inflammatory drugs. On this presentation, he was afebrile but hemodynamically unstable with a blood pressure of 82/60 mmHg and a heart rate of 110 bpm. Nasogastric lavage returned bright red blood with clot. Laboratory values were as follows: White blood cell count, 8300/ μ L (neutrophils, 82%); hemoglobin, 8.2 g/dL; platelet count, 192,000/ mm^3 ; blood urea nitrogen, 24 mg/dL; creatinine, 1.0 mg/dL; prothrombin time, 12.4 seconds; and International Normalized Ratio, 1.2. Between admission and intervention, the patient required a total transfusion of 12 U of packed red blood cells for hemodynamic support.

Upper gastrointestinal endoscopy revealed a normal esophagus. The stomach contained a large amount of blood clot, but no site of active bleeding. Examination of the duodenum showed an adherent blood clot emanating from the ampulla of Vater (Figure A). The patient was referred to interventional radiology for visceral angiography (Figure B).

What is the diagnosis?

Visceral angiography showed a bilobed pseudoaneurysm of the extrahepatic portion of the hepatic artery at the site of the anastomosis (Figure C, *arrow*) with signs of active bleeding. Three overlapping 5-mm coronary graft stents were deployed across the defect. A postprocedural arteriogram demonstrated total exclusion of the pseudoaneurysm without extravasation (Figures D and E, pre- and postinjection, respectively). Computed tomography with angiography was performed 24 hours later and showed continued absence of extravasation (Figure F, *arrow* on stent). Given his hemodynamic instability and suspicion of infection, the patient was started on broad-spectrum antibiotics. The patient was discharged home 4 days later in stable condition. Throughout the hospital course, the patient's transaminases remained at baseline without evidence of ischemic hepatopathy.

The differential diagnosis of upper gastrointestinal bleeding in the early posttransplant period includes anastomotic bleeding, variceal bleeding owing to progressive liver failure from primary graft nonfunction, peptic ulcer bleeding owing to cytomegalovirus and herpes

simplex virus infection, stress ulcers from common medications such as corticosteroids or mycophenolate, and hemobilia owing to hepatic artery fistula.

The reported incidence of hepatic artery pseudoaneurysm (HAP) after transplantation is 1%–2%, with mortality approaching 70%.¹ Patients usually present within 3 weeks posttransplantation with acute blood loss anemia and low-grade fevers. Luminal bleeding occurs through a fistulous tract between the aneurysm and either directly with the duodenum or indirectly via the biliary tree. An extrahepatic HAP typically occurs as a result of localized infection associated with a subhepatic collection frequently related to a hepaticojejunostomy, biliary leak, small bowel perforation, or intra-abdominal sepsis. The inflammatory milieu limits the resilience of vascular tissue resulting in a mycotic pseudoaneurysm. Other risk factors for HAP are technical difficulty in creation of the arterial anastomosis, re-exploration, or repeat liver transplantation. In contrast, an intrahepatic HAP is more likely to occur with instrumentation of the liver and trauma to the hepatic artery from capsular puncture, as with percutaneous liver biopsy or percutaneous cholangiogram tube placement. The clinical trajectory in these types of injuries can sometimes follow a more protracted course.

Treatment options for HAP are either through interventional radiology or surgery. Endovascular covered stenting can exclude the aneurysmal portion of the hepatic artery while maintaining perfusion to the liver.² Coil embolization can be used emergently to achieve hemostasis but can be complicated by ischemia to the allograft. Surgical ligation similarly can cause ischemic injury, although resection of the affected segment with saphenous bypass graft interposition has been described.³ Antibiotics are used either prophylactically or to treat ongoing infection.

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Figure A.

Figure B.

Figure C.

Figure D.

Figure E.

Figure F.