

# Duodenal Perforation With Transplant Hepatic Artery Pseudoaneurysm

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## ABSTRACT

A transplant hepatic artery pseudoaneurysm is a rare postorthotopic liver transplant complication. Bleeding is a common complication of posterior duodenal ulcer secondary to erosion into the gastroduodenal artery. We report the case of a post-transplant patient who presented with massive upper gastrointestinal hemorrhage in the setting of nonsteroidal anti-inflammatory drug use. Endoscopy demonstrated a duodenal ulcer with high-risk stigmata not amenable to hemostasis. Subsequently, an arteriogram revealed a hepatic artery pseudoaneurysm. Transplant professionals should be aware of the possibility of an ulcer eroding into the liver vasculature and in the differential diagnosis for bleeding and pseudoaneurysms in post-transplant patients.

## INTRODUCTION

Bleeding from ulcers were the leading cause of gastrointestinal bleeding in a retrospective analysis of 1,000 consecutive post-liver transplant patients and was associated with decreased patient and graft survival.<sup>1</sup> Bleeding from the gastroduodenal artery (GDA) or intraperitoneal air are well-recognized complications of ulcer perforation, and some distinctive complications have been reported like fistulae into the biliary tree or an ulcer eroding into the liver.<sup>2-4</sup> However, a duodenal ulcer causing hepatic artery (HA) pseudoaneurysm has not been reported in the literature. A transplant HA pseudoaneurysm is a rare post-liver transplantation (orthotopic liver transplant [OLT]) complication.<sup>5</sup> Early diagnosis is critical to allow for expeditious surgical repair and avoid catastrophic sequelae.<sup>6</sup>

## CASE REPORT

A 54-year-old man with a medical history of decompensated nonalcoholic steatohepatitis cirrhosis underwent OLT with a side-to-side cavoplasty and duct-to-duct biliary anastomosis. The vascular reconstruction was an end-to-end portal vein anastomosis and an end-to-end common HA anastomosis. His hospital course was uncomplicated, and he was discharged home with physical therapy on postoperative day 7.

Nineteen months after OLT, the patient was transferred to our institution after presenting to an outside hospital with syncope and resultant fall that led to a trimalleolar left ankle fracture. He had been taking 800 mg of ibuprofen 3 times per day for a few months before presentation for ongoing arthralgias, which he had been advised to discontinue. He was not taking any proton pump inhibitors simultaneously. Upon admission, he reported having dark stools and began vomiting fresh blood with clots. His laboratory results revealed a 5 g drop in hemoglobin, and he was intubated for urgent upper endoscopy. He developed hemorrhagic shock secondary to gastrointestinal hemorrhage and was resuscitated with blood products. An upper endoscopy was performed that revealed a cratered 2–3 cm ulcer in the proximal duodenum with a large vessel visible (Figure 1). Hemostasis was not achieved despite endoscopic therapy with 1.2 mL of epinephrine injection, 2 endoclips, and 1 minute of bipolar electrocautery. Antral biopsies were



**Figure 1.** Endoscopic view of duodenal ulcer with large caliber visible vessel in ulcer base.

not obtained, given the acuity of presentation and unsuccessful hemostasis. Stool *Helicobacter pylori* testing was recommended, but this was not completed during this hospitalization.

Interventional radiology was then consulted for a mesenteric angiogram and possible embolization. Angiography showed no active extravasation; however, it revealed luminal narrowing and irregularity of the proper HA and nonfilling of the GDA (Figure 2). Laboratory results obtained after this procedure showed aspartate aminotransferase of 2,686 U/L and alanine aminotransferase of 3,406 U/L, consistent with ischemic hepatitis. A computed tomography angiogram showed severe



**Figure 2.** Angiogram of celiac axis.

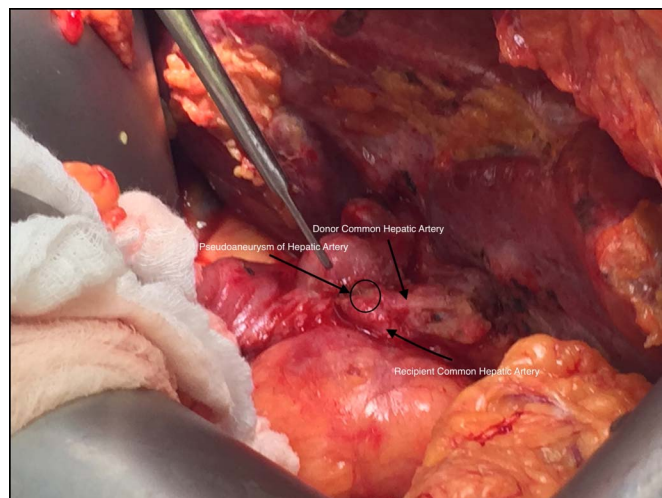
proper HA stenosis and moderate common HA stenosis with GDA occlusion and multifocal hepatic infarcts, in addition to thickening in the first part of the duodenum. It was decided that surgical intervention was needed for repair of the HA with simultaneous assessment of the duodenal ulcer.

On the exploratory laparotomy, the duodenum was adherent to the donor HA. Careful dissection allowed identification of a full-thickness ulceration of the superior aspect of the duodenum (Figures 3 and 4). The HA was seen to be folded at the level of the anastomosis, and at the genu was a pseudoaneurysm. Proximal and distal control was gained, and the aneurysmal segment including previous anastomosis between the recipient and donor HAs was resected. A new end-to-end anastomosis was created. An ultrasound demonstrated excellent flow, and a Graham patch was sutured onto the duodenum over the ulceration. Of note, the GDA could not be labeled in Figure 3 because the recipient artery was trimmed to the take-off of the GDA and turned into a GDA branch patch for anastomosis to the donor artery. A right upper quadrant drain was placed and the patient's abdomen closed uneventfully. He was transferred to the intensive care unit intubated and in a stable condition. He remained on perioperative antibiotics, as abdominal fluid cultures were positive for *Streptococcus mitis* group and *Haemophilus influenzae*.

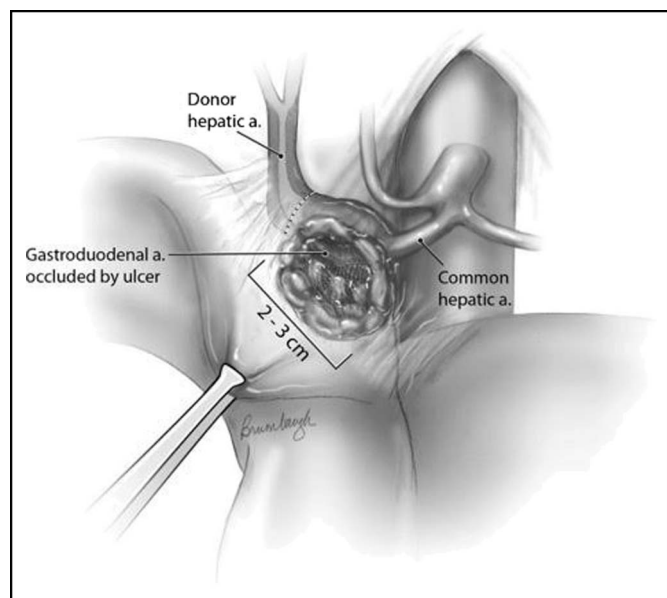
On postoperative day 2, the patient was extubated. On postoperative day 4, the patient continued to do well and had an open reduction and internal fixation of the left trimalleolar ankle fracture. During this time, his liver functions normalized and repeat liver ultrasound demonstrated normal flow through the transplant HA. The patient was discharged on postoperative day 13 to a subacute rehab facility and was medically stable.

## DISCUSSION

We describe a duodenal ulceration in a patient with previous OLT, resulting in HA pseudoaneurysm. The majority of



**Figure 3.** Intraoperative images.



**Figure 4.** Illustration of hepatic artery pseudoaneurysm from duodenal ulcer. This illustration was drawn by Jennifer Brumbaugh, MA, and published with her permission.

post-OLT patients with HA pseudoaneurysm present early and they are related to technical factors, infections, or bile leaks.<sup>5</sup> This case is unusual because the pseudoaneurysm was related to a duodenal ulcer that developed late after transplant. A delayed presentation has been seen as a complication of HA angioplasty in post-OLT patients.<sup>4</sup>

The use of nonsteroidal anti-inflammatory drugs (NSAIDs) is well known to cause gastrointestinal mucosal injury by a variety of mechanisms: inhibition of mucosal prostaglandin synthesis, impaired mucosal barrier properties, topical irritation to the epithelium, and the reduction of blood flow to the mucosa for healing.<sup>7,8</sup> The damage due to NSAIDs can occur throughout the digestive tract, and the analgesic properties of the drug often masks the symptoms until the patient is in a dire condition, as seen with this patient.<sup>9</sup> To our knowledge, the patient had not reported any gastrointestinal symptoms, such as abdominal pain, before this presentation. Although he did not have any testing for *H. pylori*, his ulcer was felt to be because of his chronic NSAID use, which is a known risk factor. Generally, anterior ulcers are at higher risk of perforation, whereas posterior ulcers are at a higher risk of bleeding via erosion into the GDA.<sup>10</sup> In our patient, angiography demonstrated nonfilling of GDA, and computed tomography angiogram showed GDA occlusion. The GDA would have been ligated at the time of liver transplant, and it is typically done to reduce competing flow away from the proper HA. Although there are some reports of ulcers eroding into the liver or forming fistulas with the biliary tree, there do not appear to be any reports of ulcers causing erosions into a transplanted HA.<sup>11,12</sup> In addition, we could find no reports of pseudoaneurysms being caused by an eroding ulcer. Rather the major risk of pseudoaneurysms after OLT comes from

biliary-enteric anastomosis and postoperative biliary leaks.<sup>13</sup> There are also case reports documenting post-OLT pseudoaneurysms from acute pancreatitis and septic emboli. The pseudoaneurysms that form usually occur at the arterial anastomosis, as it did with the patient in this report.<sup>14</sup>

Besides successfully stopping the patient's use of NSAIDs earlier, there seems to be little the healthcare team could have done to predict or prevent the outcome of this patient. The team significantly improved the outcome by operating early before the pseudoaneurysm could rupture, as HA pseudoaneurysm rupture in post-OLT patients is associated with a 53% mortality rate.<sup>13</sup> Jeng et al have demonstrated in their analysis of 50 patients with HA pseudoaneurysm that early detection is a key factor affecting the clinical outcome of these patients.<sup>6</sup> Although clearly this is a rare occurrence, transplant hepatologists and surgeons should be aware of the possibility of an ulcer causing damage to the liver vasculature and include it in their differential diagnosis for bleeding and pseudoaneurysms after OLT.

## DISCLOSURES

**Author contributions:** All authors contributed equally to the manuscript. AP Shah is the article guarantor.

**Acknowledgement:** Illustration of the hepatic artery pseudoaneurysm and the posterior duodenal ulcer was hand drawn by Jennifer Brumbaugh, MA, and published with her permission.

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**Informed consent** was obtained for this case report.

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