

Aneurysms of the Splenic Artery in Liver Transplant Recipients

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Abstract

Splenic artery aneurysms occur infrequently in the normal population, but presents with increased incidence among cirrhotic patients with portal hypertension. The optimal management of splenic artery aneurysms and the prevention of spontaneous rupture in these patients present a therapeutic challenge for transplant physicians and surgeons. We discuss a case of SAA rupture in a liver transplant recipient, and evaluate the available treatment options described in the literature.

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Case Report

Aneurysms of the splenic artery (SA) account for 60% of all visceral artery aneurysms, but are rarely encountered in the general population. In patients with chronic liver disease and portal hypertension, however, the reported incidence of splenic artery aneurysms (SAA) is as high as 10% in certain series [1]. The management of SAAs in liver transplantation (LT) candidates presents a unique challenge due to the presence of portal hypertension and coagulopathy among patients with decompensated cirrhosis, the potential use of the SA for arterial anastomosis during LT, and the propensity for aneurysm rupture in the early postoperative period. We herein describe a case of SAA rupture in an LT recipient, and discuss optimal prevention and treatment strategies.

Case Presentation

A woman of South Asian descent was referred to our center at the age of 44 with cirrhosis of unclear etiology. She had undergone an extensive work-up which failed to identify a recognizable cause for her liver disease. The major manifestations of her liver disease consisted of jaundice, fatigue, portal hypertension, and one episode of hematemesis secondary to esophageal varices, requiring endoscopy and variceal band ligation. Her medical history was otherwise only significant for typhoid fever and ascites secondary to peritoneal tuberculosis in the distant past while she was in her native country of India.

After a thorough medical and psychosocial evaluation, she was deemed a suitable candidate for LT and was placed on the national transplant waiting list. She remained on the waiting list for over a decade with a Model for End-stage Liver Disease (MELD) score of approximately 11, with preserved renal function, an international normalized ratio (INR) of 1.3, a total bilirubin of 1.5 mg/dL, and

a platelet count of 75 K/ μ L. She did not experience any further variceal bleeding, but developed symptomatic splenomegaly, as evidenced by abdominal discomfort, anorexia, early satiety, and malnutrition. On computed tomography imaging she was found to have massive splenomegaly with the tip of the spleen extending into the mid pelvis (**Figure 1**), as well as a 2.5 cm SAA (**Figure 2**). It was determined at this point that the preferred course of action would consist of operative splenic artery ligation and splenectomy at the time of LT. Due to concern for the life-threatening risk of spontaneous SAA rupture and debilitating symptoms from massive splenomegaly, we submitted an application to the United Network for Organ Sharing (UNOS) Regional Review Board and obtained MELD exception points in order to expedite transplantation.

At the age of 59, the patient underwent orthotopic LT with a whole liver graft from a donation after cardiac death donor. Following revascularization of the liver graft, a splenectomy was performed. Additionally, the proximal SA was ligated near its origin from the celiac trunk to interrupt arterial blood inflow into the aneurysm. On postoperative day 4, the patient experienced an acute episode of hypotension which quickly progressed to pulseless electrical activity (PEA) arrest. Spontaneous circulation was restored after 3 minutes of cardiopulmonary resuscitation. Following the arrest, the patient's abdomen was noted to be firm and distended, and the abdominal drains had increased sanguinous output. Concurrently, the hematocrit dropped acutely from 35% to 21%, and the patient was returned emergently to the Operating Room.

Upon reopening the abdomen, we encountered a large-volume hemoperitoneum, and the SAA was found to have ruptured and



Figure 1 Coronal post-contrast venous phase computed tomography image demonstrating massive splenomegaly, with the tip of the spleen extending into the pelvis.



Figure 2 Axial post-contrast arterial phase computed tomography image at the level of the pancreas showing a 2.5 cm aneurysm in the distal splenic artery (arrow).

bleeding rapidly. Despite having achieved proximal and distal ligation at the time of initial LT, there was continued arterial filling of the aneurysmal sac suggesting the presence of collateral inflow. We proceeded to ligate the SA at multiple locations along the length of the vessel, and the aneurysm itself was oversewn. The subsequent postoperative recovery was uneventful, and the patient was discharged home on post-transplant day 16. At 3 years post-LT, the patient continues to do well with normal liver graft function and no further complications related to the SAA.

Discussion

Several putative mechanisms have been proposed for the higher incidence and increased size of SAAs in the setting of portal hypertension. These include increased visceral blood flow secondary to collateral formation, dilatation and elongation

of the splenic artery, elevated cardiac output and splanchnic vasodilation induced by hyperglucagonemia, and hormonal changes in cirrhotic patients leading to weakening of the arterial wall [2]. Despite the increased frequency of occurrence, SAAs are almost always asymptomatic in pretransplant patients. The diameter of SAA in cirrhotic patients is usually limited to <3 cm, and spontaneous rupture of the aneurysm is seldom seen [3]. Furthermore, repair in the pretransplant period is discouraged due to the elevated risks of bleeding and hepatic decompensation in cirrhotic patients, the creation of adhesions which can increase the difficulty of the subsequent transplant operation, and the potential need for using the SA at the time of LT. Therefore, we recommend deferring treatment for SAAs until the time of transplantation if possible.

Conversely, several instances of SAA rupture in the early post-transplant period have been reported in the literature [1, 3-5], with most cases occurring within the first 10 days after LT. Spontaneous rupture of SAAs shortly after transplantation is thought to be precipitated by abrupt alterations in celiac blood flow associated with liver graft implantation, the use of high-dose corticosteroids to prevent graft rejection [2], and the activation of collagen lysis within the wall of the aneurysm immediately following laparotomy [6]. Treatment of SAA should be instituted at the time of LT in order to reduce the risk of aneurysm rupture in the early postoperative period.

Therapeutic options for SAAs include operative ligation, or alternatively, endovascular repair with embolization or stent graft implantation. Surgical management of SAA at the time of transplantation involves achieving proximal and distal ligation of the SA, with resection of the aneurysm sac if feasible [7]. Transcatheter embolization of SAAs can be accomplished using coils or N-butyl cyanoacrylate glue, with a reported technical success rate of 88% [8]. Treatment failure is often a consequence of incomplete sealing or recanalization via short gastric vessels or other collaterals, resulting in persistent filling of the aneurysm sac. The use of transcatheter embolization has also been associated with the development of a post-embolization syndrome, characterized by splenic infarcts and abscess formation [9]. In cases where no surgical treatment can be performed during LT, the use of percutaneous embolization within 24 hours post-transplant have been described with minimal morbidity [3-5]. Endovascular stent graft placement is another viable therapeutic option, but its use for visceral aneurysms is based only upon anecdotal experience at this time [10].

Conclusions

Patients with chronic liver disease and portal hypertension demonstrate a higher incidence of SAA, which presents a therapeutic challenge for transplant physicians and surgeons. The treatment of SAA in the pretransplant period is discouraged in most cases due to the low rate of spontaneous rupture and high operative risks in this patient population. On the contrary, the likelihood of SAA rupture is highest within the first few days after LT, and preventive strategies should be implemented during or immediately after the transplant procedure. Surgical management of SAA should include proximal and distal ligation of the SA with resection of the aneurysm if possible. Endovascular repair options include embolization or stent graft placement, and can be considered in cases where adequate operative treatment has not been achieved.

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