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CASE REPORT | LIVER

Splenic Artery Embolization for Treatment of Refractory Ascites After Liver Transplantation

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Abstract

Post-transplantation refractory ascites is uncommon; however, it can be a serious problem, increasing both morbidity and mortality in patients. Despite scant literature available, splenic artery embolization (SAE) has been shown to be an effective treatment for refractory ascites after cadaveric orthotopic liver transplantation (OLT). We report a successful use of therapeutic SAE for refractory ascites post-OLT.

Introduction

Post-transplantation refractory ascites is uncommon; however, it can be a serious problem, increasing both morbidity and mortality in patients. One study reported the incidence of post-transplantation ascites to be 7%, with the major mechanism being post-sinusoidal portal hypertension.[1] Another retrospective study showed persistent ascites, for more than 4 weeks after liver transplantation, was present in 5.6% of patients.[2] Procedures such as TIPS or porto-caval shunts have been used to treat refractory ascites; however, these were challenged with high morbidity and mortality. Splenic artery embolization (SAE) is one method to achieve decreased portal vein (PV) perfusion.

Case Report

We report of a 49-year-old white man with a medical history significant for diabetes mellitus and end-stage liver disease due to chronic hepatitis C and alcohol-related cirrhosis. He had a MELD score of 20 and underwent an orthotopic liver transplant (OLT) from a deceased donor. For 18 months prior to his transplant, he had required large-volume paracentesis with removal of more than 8 L of ascitic fluid every 2 weeks. He was also taking furosemide 40 mg daily; however, due to a rise in creatinine to 2.5 mg/dL, diuretics were discontinued 3 months prior to his transplant.

The immediate post-operative course was uncomplicated, and he was discharged home on standard post-transplant immunosuppression and prophylactic medications. His liver enzymes, AST, ALT, and bilirubin declined after OLT, though he continued to have ascites, and his renal function did not fully recover. He was hospitalized 2 weeks post-OLT with ascites, and more than 9 L of ascitic fluid was drained during paracentesis. He was again hospitalized at 9 weeks post-OLT with tense ascites, and required removal of 12.1 L of fluid.

Cytology on the ascitic fluid was negative for malignancy. A transjugular liver biopsy was suggestive of outflow obstruction with mild sinusoidal dilatation of unclear significance (Figure 1). Histopathology of the

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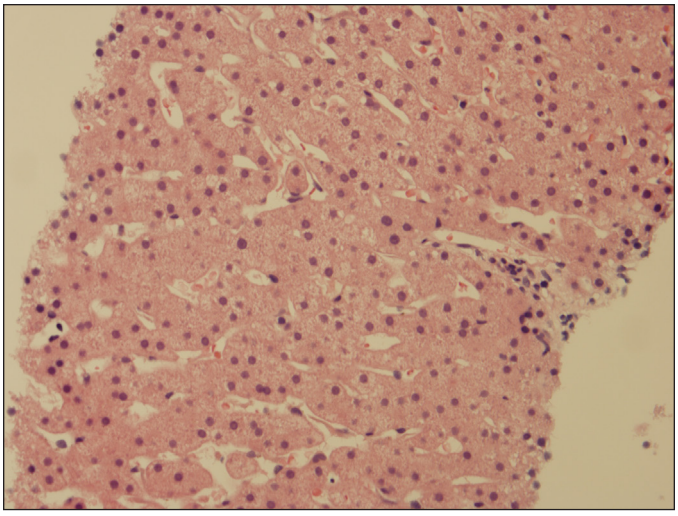

Figure 1. Liver biopsy showing mild sinusoidal dilatation.

portal triad did not show any abnormalities (Figure 2). He was also noted to have persistent splenomegaly on imaging (Figure 3). A magnetic resonance venography showed patent hepatic vasculature. An echocardiogram was unremarkable. Due to the liver biopsy findings suggesting outflow obstruction, persistent portal hypertension on imaging, and refractory ascites, there was an increased suspicion for vascular obstruction. A CO_2 venogram of the inferior vena cava (IVC) showed no evidence of an anastomotic stenosis. There was absence of a trans-anastomotic caval pressure gradient (suprahepatic IVC 11 mm Hg, suprarenal IVC 11 mm Hg, and infrarenal IVC 11 mm Hg).

At 4 months post-OLT, our patient had required 5 large-volume paracenteses for refractory ascites. His creatinine continued to slowly rise to a peak of 2.8 mg/dL. SAE was then performed, after which he required an additional

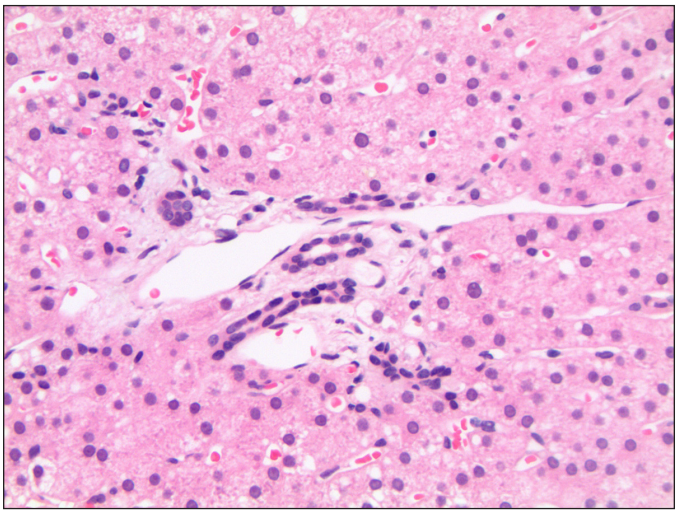

Figure 2. Liver biopsy showing an unremarkable portal tract.

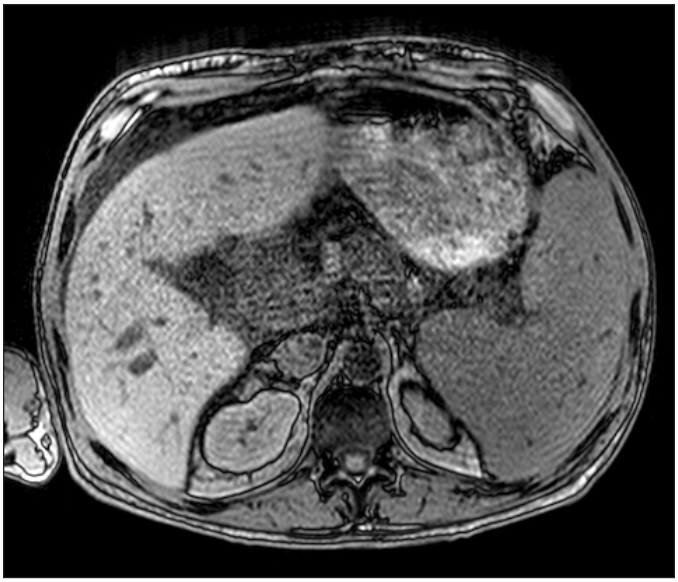

Figure 3. Abdominal MRI showing splenomegaly.

paracentesis to remove 3 L of fluid. He was then discharged home on furosemide 40 mg daily. One month after the embolization the ascites had resolved, so furosemide was discontinued and he has not required further paracentesis. His renal function returned to baseline levels, but never fully recovered.

Discussion

Ascites in patients after liver transplantation is caused mainly by portal hyperperfusion and high flow through the collaterals and portal vasculature. SAE decreases this by diminishing portal inflow pressures and decreasing graft congestion.[3] The use of SAE for both the prophylaxis and treatment of refractory ascites after liver transplantation has been previously reported in the literature. One study reported that prophylactic SAE shortened operating time, reduced blood loss, and reduced the volume of post-transplant ascites. Post-operative ascites on day 7 in prophylactic SAE was 400 mL/day, whereas ascites in non-prophylactic SAE was 1,500 mL/day (p=0.02).[4] Another study reported 6 cases with post-transplant refractory ascites who underwent therapeutic SAE, 5 of which experienced a complete clinical resolution of ascites after a median time of 49.5 days (mean 83 days; range 12-295 days).[5] Potential complications of SAE include splenic abscess, sepsis, splenic infarction, and portal vein thrombosis, especially if performed in the proximal aspect of the splenic artery.[5-7] Further data is needed to prospectively identify patients who may benefit from early SAE to reduce patient morbidity and improve quality of life. Additional studies are needed to understand the long-term outcomes for patients undergoing SAE in the setting of refractory ascites post-OLT.

Disclosures

Author contributions: A. Meighani and SMR Jafri wrote the manuscript. M. Raoufi was the pathologist and helped prepare the manuscript. R. Salgia edited the manuscript and was the project mentor. A. Meighani is the article guarantor.

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