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CASE REPORT

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Splenic artery steal syndrome after liver transplantation: A case series and review of literature

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Key Clinical Message

Splenic steal syndrome (SASS) represents a challenge to interventional radiologists after orthotopic liver transplantation. In this case series, we present three cases of patients who developed SASS after their liver transplants.

KEYWORDS

liver, radiology and imaging, SASS, splenic artery steal syndrome, transplantation

1 | INTRODUCTION

Hypoperfusion of the hepatic artery includes two subtypes, which are splenic steal syndrome (SASS) and gastroduodenal steal syndrome. It is a nonocclusive disease of the hepatic artery that often presents within 60 days of liver transplantation.[1] In addition to hypoperfusion of the hepatic artery, SASS presents with blood flow being diverted to splenic parenchyma through the splenic artery, and this syndrome may be an under-recognized contributor to graft ischemia.[2] Although most cases of SASS are discovered within 60 days of liver transplantation, SASS can present up to 5.5 years posttransplantation.[3] The incidence of SASS ranges from 0.6% to 10.1% in liver transplant recipients.[4] This wide range in incidence is due to no fixed objective diagnostic criteria.[4] It is mainly a diagnosis of exclusion, and two important alternate diagnoses often considered are hepatic artery stenosis and hepatic artery thrombosis. Some studies have placed importance on pretransplant evaluation for risk factors and potential subsequent intervention to reduce the risk of SASS.[5,6] However, studies that have sought out potential risk factors such as spleen-liver ratio, splenomegaly, and pretransplant splenic and hepatic artery size have had mixed results.[7–9] The hepatic artery hypoperfusion leads to a nonspecific hepatic dysfunction which is detected by elevated liver enzymes in the acute stage (<2 months).[7] However, patients often present with other complications such as ascites 2 months after the procedure, which is considered an overt sign of hepatic dysfunction.[2] Cholestasis, biliary ischemia, and hypersplenism are also possible complications of SASS.[1,4,10] Overall, the clinical features of SASS are not consistent, and patients can range from being asymptomatic to having acute liver failure.[3] Histologically, SASS has been noted to demonstrate cholestasis, epithelial ductal regressive changes, and centrilobular zone necrosis.[1,11–13] If transplant biopsy were performed, SASS generally demonstrates mild inflammation, which contrasts with the overt findings seen during acute rejection.[1] Potential consequences of SASS feature early graft dysfunction and biliary ischemia, potentially leading to re-transplantation.[2,4] Some institutions perform either prophylactic or posttransplant treatment procedures in up to 25% of all transplant patients.[4] The outcome of SASS

depends on the time since the hepatic artery suffered ischemia. Therefore, it is necessary to diagnose and treat the complication as soon as possible.[14] The diagnosis of SASS is particularly challenging as it has nonspecific signs such as elevated LFTs, cholestasis and graft dysfunction.[14]

2 | CASE HISTORY/EXAMINATION

2.1 | Case 1

A 70-year-old white female with a history of arthritis was evaluated for preop surgical clearance by her primary care physician prior to scheduling left knee replacement. Blood work at the hospital revealed elevated liver function tests (LFTs). Ultrasound of the abdomen revealed cholelithiasis with intrahepatic and extrahepatic duct dilatation. She underwent (endoscopic retrograde cholangiopancreatography) ERCP with sphincterotomy and biliary and pancreatic stent placement. However, the patient continued to have jaundice and elevated LFTs. She underwent a repeat ERCP with placement of biliary stent and removal of pancreatic stent. The patient eventually developed abdominal pain after stent placement and then presented to the emergency room.

CT of the abdomen and pelvis revealed severe dilatation of intrahepatic bile ducts of both right and left hepatic lobes with abrupt cutoff of the hepatic ducts just above the confluence suggesting high-grade obstruction from tumor. Patient was referred to our institution where she was found to have intrahepatic biliary ductal dilation with suggestion of obstruction from the mass at the hilum. Bile duct biopsy revealed atypical epithelial cells suspicious for carcinoma. Pathology was reviewed at the hospital and the diagnosis of adenocarcinoma was confirmed. Over the next 3 months, patients complained of a loss of appetite and had significant weight loss. She also started complaining of upper abdominal pain after stent placement. She complained of nausea with dry heaves. She complained of weakness, lightheadedness, itching, and insomnia. Since the jaundice began, a severe rash developed on her abdomen and legs. Patient then underwent right hepatic lobectomy, periportal lymph node dissection, bile duct resection, roux-en-y hepatico-jejunostomy for the diagnosed Klatskin's tumor. She was eventually transferred to the SNICU, as planned, post operatively. She tolerated the procedure well, but her postoperative course has been complicated by liver failure, likely SFSS (small for size syndrome) complicated by SBP (spontaneous bacterial peritonitis) and further decompensation including encephalopathy and hepatorenal syndrome.

She eventually underwent proximal splenic artery embolization to decrease portal hypertension. She has been on broad spectrum antibiotics and Caspofugin for bacterial and fungal infections. The patient was transferred to the hospital floor where she gradually improved, but her liver function remained poor. However, the patient had worsening confusion and lactulose was restarted with subsequent improvement. She eventually experienced diarrhea, so the lactulose was discontinued. The patient subsequently developed hypernatremia and had ascites requiring multiple paracenteses. She continued taking antibiotics and antifungals for her infections. Despite the medications, her labs continued to show liver dysfunction with evidence of hepatorenal syndrome. She was readmitted to the SNICU (surgical and neurosciences intensive care unit) due to difficulty with hypotension, and where she was restarted on CRRT (continuous renal replacement therapy).

She continued to have difficulty with sepsis/ongoing infections with some isolates growing resistant organisms, sepsis, liver failure, fluid collections requiring IR drainage, hypotension with pressor requirement, renal failure with persistently low urine output, and lactic acidosis.

2.2 | Case 2

A 74-year-old white female with a history of end stage renal disease (ESRD) from alcoholic cirrhosis complicated by portal hypertension, ascites, esophageal varices and hepatic encephalopathy. The patient underwent orthoptic liver transplantation and was able to cope with the procedure well. Post transplantation, the patient was transferred to SNICU, and LFTs were monitored. The LFTs were elevated, particularly the AST and ALT.

There was a slight elevation in the creatinine post transplantation, which was indicative of acute kidney injury (AKI). Pretransplantation US showed diminished diastolic flow. Posttransplantation liver US demonstrated reversal of diastolic flow in the hepatic artery with high resistivity index and high portal vein velocity concerning for hepatic arterial hypoperfusion.

The interventional radiology (IR) team was consulted to evaluate SASS. A Celiac angiogram (Figure 1A,B) was performed which demonstrated findings consistent with SASS. The patient underwent successful embolization of splenic artery with Amplatzer plug. Pre-embolization US showed reversal of hepatic artery flow with diminished diastolic flow (Figure 2). Post embolization US demonstrated improved flow in the hepatic artery with improved peak velocities and forward diastolic flow (Figure 2B). After the procedure, there were no complications, and the patient was transferred to the hospital floor. LFTs improved post

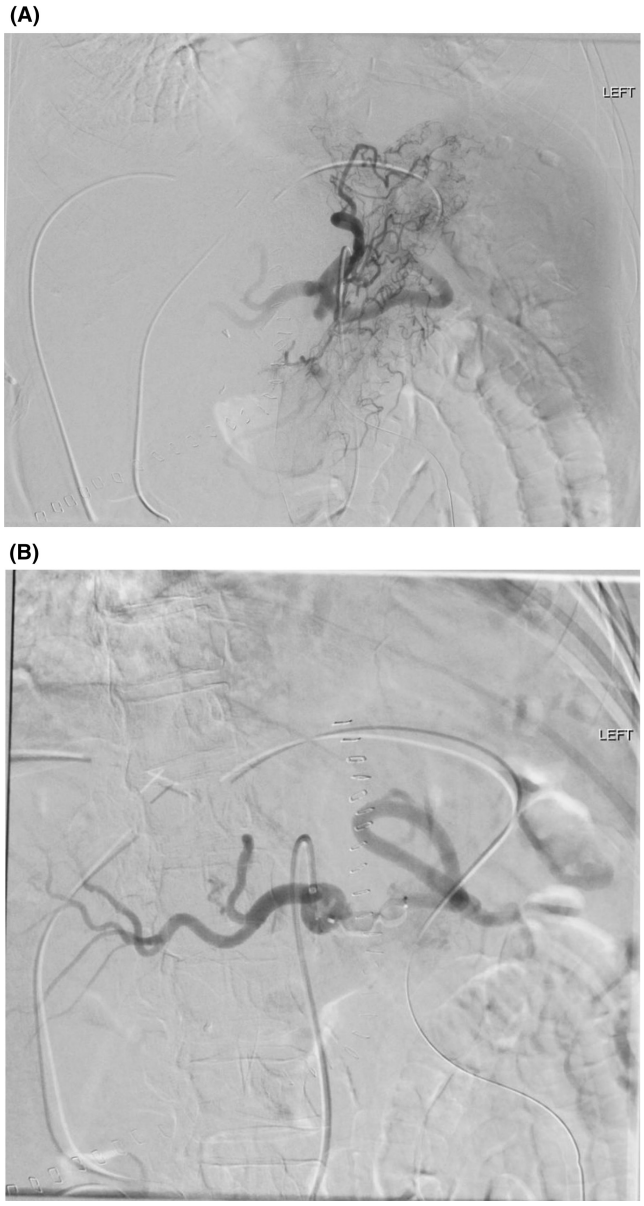

FIGURE 1 (A) Contrast injection of celiac axis shows preferential flow into the splenic artery and little contrast into the hepatic artery. (B) Restoration of hepatic artery flow post splenic artery embolization.

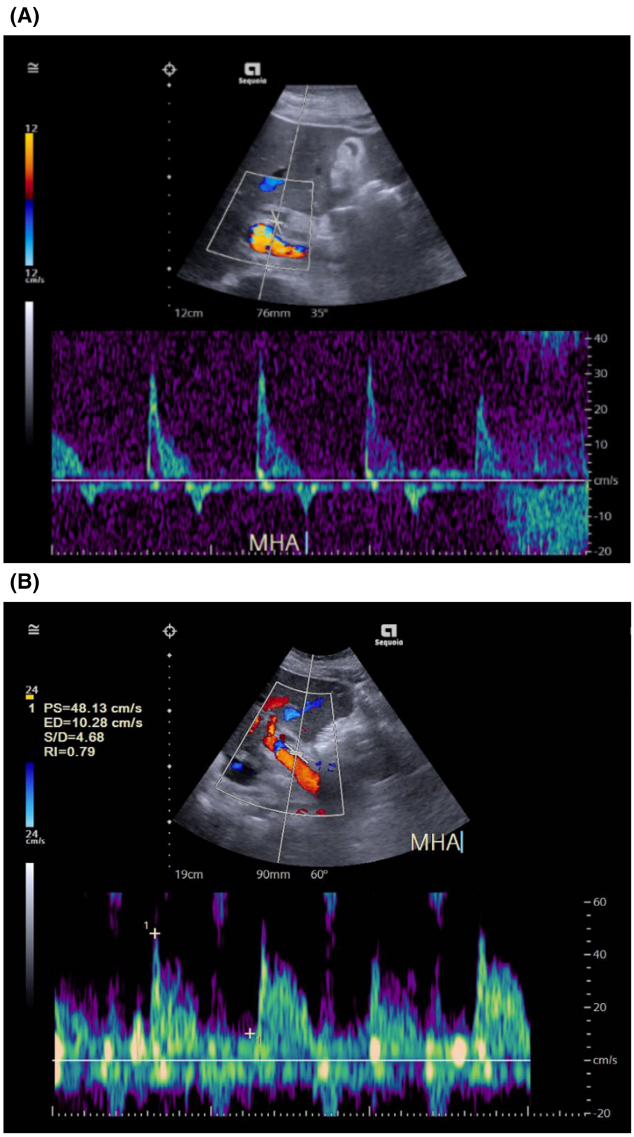

FIGURE 2 (A) Pre-embolization hepatic artery flow reversal with diminished diastolic flow. (B) Restoration of hepatic flow post splenic artery embolization.

embolization and were within normal range 2 days following the procedure. There was a spike in the LFTs that raised concern for acute rejection. The patient underwent liver biopsy showing mild to moderate acute rejection, which was treated with pulse dose steroids. Post treatment, LFTs decreased gradually.

2.3 | Case 3

A 68-year-old white male complaining of fatigue and had a history of ESRD, decompensated cirrhosis secondary to alpha-1 antitrypsin deficiency and nonalcoholic steatohepatitis, which was complicated by a single episode of variceal hemorrhage. The patient subsequently underwent orthoptic liver transplantation. He also had a history of nonocclusive portal and mesenteric vein thrombosis secondary to antithrombin III deficiency. Intraoperatively, a large pre-existing portal vein thrombus was removed. He was transferred to the surgical intensive care unit (SICU) posttransplantation. Posttransplantation US of the liver demonstrated no presence of thrombus in the hepatic or portal veins. His LFTs were high posttransplantation but decreased gradually.

His postoperative course was complicated with an AKI and was managed with n-acetylcysteine. The patient's LFTs returned to normal 3 months following the transplantation. However, creatinine remained slightly elevated and

was presumed to be due to Calcineurin inhibitor nephrotoxicity. LFTs continued to fluctuate throughout his follow-up course. On a follow-up liver US, he was found to have persistent dilated portal vein and splenomegaly, and spectral broadening of the hepatic vein waveforms. Portal hypertension without portal vein thrombosis was suspected. He was then referred to IR service for a transjugular biopsy with pressure measurement. The biopsy demonstrated no liver fibrosis and no rejection, but the portal vein branch morphology was found to be abnormal.

The patient underwent a hepatic arteriogram (Figure 3) and a hepatic venogram with pressure measurement. He was found to have hypoperfusion to the common hepatic artery secondary to stealing from the splenic artery. The IR team was consulted for a possible splenic artery embolization. He underwent 70% embolization of spleen using 500–700-micron PVA particles. Patient tolerated the procedure well and the post procedural course was unremarkable. He was discharged a couple of days later.

There was a sudden spike in the ALP and GGT levels after the procedure, and the ultrasound demonstrated an enlarged spleen, which was presumed to be due to necrosis from the recent embolization. LFTs returned to normal levels a few weeks later.

3 | DISCUSSION

The most frequently relied upon modality for the diagnosis of SASS is angiography.[8] It is defined as a subjective slow flow of blood in the hepatic artery relative to that in the splenic artery in the absence of significant artery abnormalities such as hepatic artery stenosis, thrombosis, or kinks.[15] Uflacker and coworkers diagnosed SASS when there is a visualization of hepatic artery during the portal-venous phase of angiogram.[3] However, others have not relied on this definition or threshold.[5] Other modalities for detection of SASS include regular Doppler ultrasonography (US).[14] US is often performed daily within the first week after OLT. Emergency angiography is performed when a weakened or decreased signal is detected on US.[14] US findings are nonspecific and are not often used to describe SASS cases since findings overlap with other conditions such as infection, transient graft edema, or rejection.[16–18] The most described US findings include a high level of arterial resistive index (above 0.8).[19] Reversal of diastolic flow or low diastolic flow is also often observed.[3,4] Furthermore, arterial velocities are rarely reported.[17] Nevertheless, US findings are not often used for diagnosis of SASS.[14] In fact, only 30% percent (34 out of 113) of the SASS cases reported US findings. Other methods for the diagnosis of SASS include a splenic volume greater than 830 cm^3, which has an almost 75% accuracy in diagnosing SASS.[7,8] Splenic artery diameter of >4.0 mm or splenic artery to hepatic artery ratio of >1.5 are considered indicators of SASS.[7,8] Some studies have also indicated a difference of at least 6 mm between hepatic and splenic artery diameters as being predictive of SASS.[6] However, no imaging modality truly solidifies the diagnosis[20], and the diagnosis is only confirmed upon seeing improved graft function and increased hepatic arterial perfusion after correcting splenic artery perfusion[6].

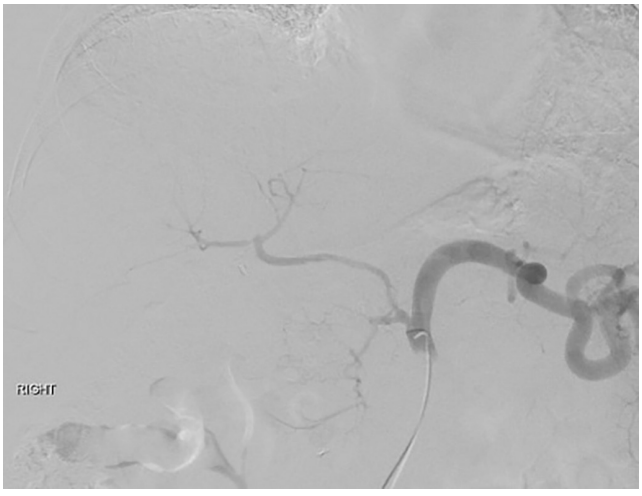

FIGURE 3 Contrast injection of celiac axis shows preferential flow into the splenic artery and little contrast into the hepatic artery.

All our patients were managed with splenic artery embolization. Embolization in the first case was with coils. However, embolization in the second and third cases were done with Amplatzer plug and 500–700-micron PVA particles respectively. Our treatment methods were consistent with the modalities at other institutions. Many institutions have considered splenic artery coil embolization as the primary treatment for SASS.[15,17] Patients with SASS often have a thick splenic artery with a diameter that is often thicker than 5.0 mm or 1.5 times that of the hepatic artery.[14] Therefore, fast arterial flow can push the coil into the branch of splenic artery leading to ischemic necrosis of the spleen and possibly septicemia.[10] Furthermore, the incidence of infection after coil embolization was reported to be as high as 50%.[15]

Currently, the treatments for SASS range from interventional radiological ones to surgical ones such as splenic artery ligation, proximal and distal embolization, banding and splenectomy.[13,14,21–23] Despite the wide range of possibilities, proximal splenic artery embolization remains the most popular and preferred intervention because it is less invasive than surgical options with less risk of both intra and postoperative bleeding.[11] It is also known that proximal embolization

TABLE 1 Treatment modalities for SASS patients at other institutions and their clinical outcomes.

Author (year published)	Treatment of SASS (2010-present)	Number of patients with SASS treated	Number of patients with clinical improvement	Percentage clinical improvement
Garcia-Criado et al. (2014)[26]	Splenic artery ligation and Amplatzer splenic artery plug	7	7	100
Zhu et al. (2012)[27]	Splenic artery coil embolization	8	8	100
Uslu et al. (2012)[28]	Splenic artery coil embolization or vascular plug	20	20	100
Mogl et al. (2010)[5]	Splenic artery and gastroduodenal artery coil embolization	24	23	95.8
Grieser et al. (2010)[8]	Splenic artery coil embolization	12	NA	NA
Li et al.(2016)[29]	Splenic artery embolization	50	45	90
Liu et al. (2015)[30]	Splenic artery embolization	3	3	100
Pérez et al. (2011)[11]	GDA embolization and partial splenic artery embolization	1	1	100

is much more likely to maintain collateral flow to the spleen than distal embolization.[3] Table 1 summarizes the most common methods for managing SASS. To explore hepatic artery perfusion after embolization, an angiogram should be performed, and in successful cases, this angiogram demonstrates increased, prompt flow in the hepatic artery. In addition, it demonstrates increased enhancement of the liver parenchyma during the late arterial phase.[3]

Other treatments such as temporary splenic artery blockage have also been reported in the literature.[24,25] This method can lead to decreased splenic artery flow without irreversible local ischemic necrosis of the spleen. More studies are needed to investigate this intervention. Nevertheless, it appears to be a promising therapeutic intervention to SASS. Our case series highlights the fact that proximal splenic artery embolization is the most common treatment for SASS after orthotopic liver transplantation. It also sheds light on relatively new and effective treatments such as temporary blockade of splenic artery. Furthermore, it shows how imaging modalities such as hepatic arteriogram and venograms with pressure measurement can facilitate the diagnosis of this syndrome. Table 1 shows most common treatments and their clinical outcome for SASS patients.

There are several possible mechanisms leading to SASS. One possible cause of SASS is portal venous hyperperfusion, which is observed by Doppler US as altered arterial blood supply to the liver and associated with increased portal venous flow.[4] There are two possible mechanisms by which the hyperperfusion could lead to sinusoidal injury in the graft: (1) The direct effect via elevated portal venous pressures (2) Hepatic artery buffer response secondary to rapid adenosine washout, which can lead to hepatic artery hypoperfusion.[3,4] The adenosine promotes vasodilation in arteries, so its washout can lead to hepatic artery vasoconstriction.[32]

As research moves forward and more risk factors are found to be associated with SASS, a greater importance may be placed upon prophylactic treatment, such as preoperative splenic artery embolization. Mogl et al. demonstrated reduced risk of SASS complications after SASS prophylaxis compared to postoperative treatment.[5] A randomized control trial by Umeda et al. also demonstrated that preoperative embolization in patients who had severe portal hypertension resulted in reduced hepatic hypoperfusion along with lower operative time and blood loss.[17] Other interventions such as splenic artery ligation or banding are often used as prophylactic measures and can be used if the diagnosis of SASS is made posttransplantion.[31]

One major limitation of this case series is the limited number of patients included in the report. It is also important to mention that the case series is possibly limited by confounders that might have affected their clinical outcome. All the patients in this case series were older than 65. Therefore, treatment methods may not be necessarily generalized to younger and healthier populations.

4 | CONCLUSION AND RESULTS

Splenic artery embolization was the most common treatment modality for our SASS patients. Our patients had significant clinical improvement after their treatment. However, more novel treatments need to be investigated in the future. Imaging modalities such as ultrasound,

arteriogram and venograms with pressure measurements were used to diagnose this syndrome. An angiogram can be used after the procedure to ensure prompt flow in successful cases.

AUTHOR CONTRIBUTIONS

Assim Saad Eddin: Supervision; writing – original draft; writing – review and editing. **Abhiram Kamaraju:** Writing – original draft; writing – review and editing. **Umar Ramzan:** Writing – review and editing. **Jay Yu:** Supervision; writing – review and editing. **Surbhi Dadwal:** Writing – review and editing. **Sandeep Laroia:** Conceptualization; supervision; writing – review and editing.

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CONFLICT OF INTEREST STATEMENT

The authors hereby agree that there are no conflicts of interest to disclose.

DATA AVAILABILITY STATEMENT

All data underlying the results are available as part of the article and no additional source data are required.

CONSENT

Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

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