Perioperative Management of Spontaneous Splenorenal Shunts in Orthotopic Liver Transplant Patients

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Abstract

Objectives: **Spontaneous splenorenal shunts cause significant vascular steal from the liver. There is no accepted algorithm for treating spontaneous splenorenal shunts before, during, or after liver transplant, and evidence for efficacy of treatments remains limited.**

Materials and Methods: **We reviewed the literature, and our institution's experience regarding spontaneous splenorenal shunts, including a case series of 6 patients with spontaneous splenorenal shunts undergoing transjugular intrahepatic porto-systemic shunts, a case of intraoperative ligation of a large spontaneous splenorenal shunts during transplant, and 1 patient requiring multiple endovascular interventions to embolize recurrent spontaneous splenorenal shunts after orthotopic liver transplant.**

Results: **Small spontaneous splenorenal shunts may not need intervention, as involution after liver transplant is well known. Transjugular intrahepatic porto-systemic shunts may decrease the porto-systemic gradient in patients with large spontaneous splenorenal shunts, as shown in our review of 6 patients with large spontaneous splenorenal shunts undergoing transjugular intrahepatic porto-systemic shunts. We have demonstrated re-establishment of physiologic flow after ligation of a large spontaneous splenorenal shunt at the time of transplant, supporting operative ligation may be justified if intraoperative compression of the spontaneous splenorenal shunts demonstrates significant improvement of allograft portal venous flow. Ligation of the left renal vein for large spontaneous splenorenal shunts is a safe and effective method of preventing portal venous steal. For concomitant spontaneous splenorenal shunts and portal vein thrombosis, renoportal anastomosis can be performed. We report transient success with endovascular embolization of large spontaneous splenorenal shunts in a patient posttransplant who required multiple interventions.**

Conclusions: **Experience in the approach to and treatment of spontaneous splenorenal shunts in liver transplant recipients is limited. Further investigation into the best approach to treat spontaneous splenorenal shunts is warranted as the presence and persistence of spontaneous splenorenal shunts can lead to allograft dysfunction and possible allograft loss.**

Key words: *Splenorenal shunt, Liver, Transplant*

Introduction

Patients with portal hypertension commonly develop porto-systemic vascular connections as the increased resistance within the liver vascular bed leads to shunting of blood flow. A spontaneous splenorenal shunt (SRS) forms between the splenic vein and the left renal vein (Figures 1A and B) and has been noted in up to one-fifth of patients evaluated for liver transplant.[1-4] As the degree of cirrhosis progresses, higher resistance in the liver

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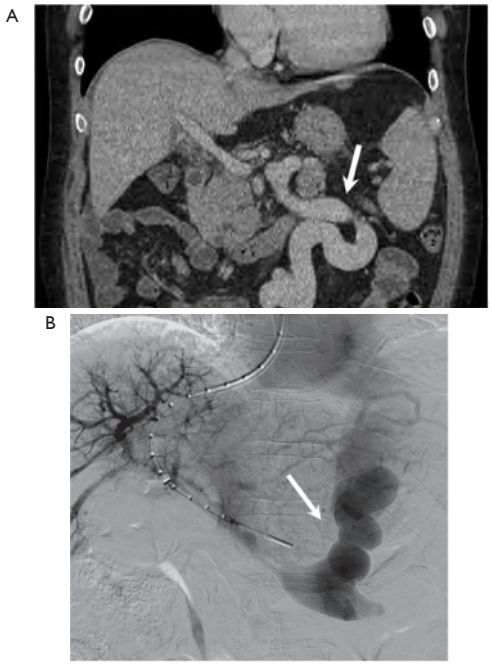
Figure 1. (A, B) Spontaneous Splenorenal Shunt (Arrow)



vascular bed can lead to considerable shunting of portal blood into the left renal vein. This causes the splenorenal shunt to become a high-flow collateral and ultimately can result in significant vascular steal from the transplanted liver.[5, 6]

After orthotopic liver transplant, porto-systemic collaterals typically involute as the low-resistance vascular bed of the allograft leads to decreased flow through the aberrant channels.[7-9] However, large shunts, those measuring larger than 10 mm, are less likely to collapse and may continue to steal flow from the new organ. Additionally, shunts may reconstitute, and the steal may worsen if the intrinsic allograft vascular resistance becomes elevated, as may occur with rejection, fluid overload, and other post-transplant phenomena.[10-12] Splenorenal shunts are of particular concern as they tend to be large and may go undetected at the time of transplant. Nonetheless, it remains controversial as to whether the persistence of porto-systemic collaterals and vascular steal is uniformly detrimental to long-term allograft function. There is also no consensus on what intervention is best for treatment of large, persistent shunts.

Materials and Methods

A review of the literature and our institution's experience regarding SRS in liver transplant candidates and recipients was performed. This included a review of preoperative patients undergoing transjugular intrahepatic porto-systemic shunts (TIPS) who also had an SRS, intraoperative evaluation and management of transplant recipients with known SRS, and postoperative management of patients after liver transplant with a large residual SRS. Our institution's experience regarding SRS, including a case series of 6 patients with SRS undergoing TIPS, 1 case of intraoperative ligation of a large SRS at the time of transplant, and 1 patient requiring multiple endovascular interventions to embolize a recurrent SRS after orthotopic liver transplant, also was reviewed.

Results

Diagnosis

The presence and size of an SRS in a liver transplant candidate should be identified preoperatively if possible. Aucejo and associates reported a strategy of preoperative triple-phase computed tomography with angiographic reconstructions as well as sonography and subsequent intraoperative graft flow measurements after completion of the hepatic artery anastomosis to improve management of portosystemic shunts during liver transplant.[13] Unfortunately, in some instances, the splenorenal shunt is either not identified preoperatively, cannot be accessed during surgery, or does not become clinically apparent until after transplant. The treatment of such shunts becomes difficult, as there are limited therapeutic options.

Intervention

There is no accepted algorithm for treating splenorenal shunts before, during, or after liver transplant, and evidence for efficacy of different treatment plans is largely limited to case reports.[1, 2, 5, 8, 10, 14-27] Possible treatment options include preoperative further selective shunting of flow such as transjugular intrahepatic portosystemic shunts, intraoperative assessment of portal flow with a decision to intervene if there is evidence of inadequate flow to the allograft, automatic intraoperative ligation of the shunt, close monitoring of the shunt and the functional status of the transplant liver without intervention, creation of portorenal anastomosis in the case of portal vein thrombosis, or postoperative percutaneous interventions to embolize a symptomatic shunt.

Preoperative intervention

Transjugular intrahepatic porto-systemic shunts were developed as a way to create a direct shunt between the portal and hepatic venous systems as a nonoperative attempt both to relieve portal hypertension and decrease intrahepatic venous pressure as blood is shunted into the systemic circulation. Given that both TIPS and an SRS essentially serve the same purpose of shunting blood to the systemic circulation, we hypothesized that TIPS creation may decompress a coexisting SRS.

We retrospectively reviewed our records of patients undergoing TIPS who also had an SRS (Table 1). Six patients were identified, and the report from TIPS was reviewed for the porto-systemic gradient before and after TIPS, the size of the TIPS stent, diameter of the balloon used, and procedural complications. The only cases in which patients did not have concomitant intervention on the SRS directly were included. Spontaneous splenorenal shunt size decreased in 3 patients (Figures 2 A-D), remained unchanged in 2 patients, and was unable to be evaluated in the last secondary to significant streak artifact from metallic coils from prior gastric variceal embolization. Spleen size as well as inferior vena cava diameter decreased in 3 patients but increased in the other 3 patients. In the 4 patients with ascites before TIPS, 3 showed resolution after the procedure. Overall, the porto-systemic gradient decreased in 5 of the 6 patients after TIPS (Table 2). While this analysis of data is small and retrospective, it does demonstrate that a decrease in SRS size can be achieved preoperatively.

Intraoperative intervention

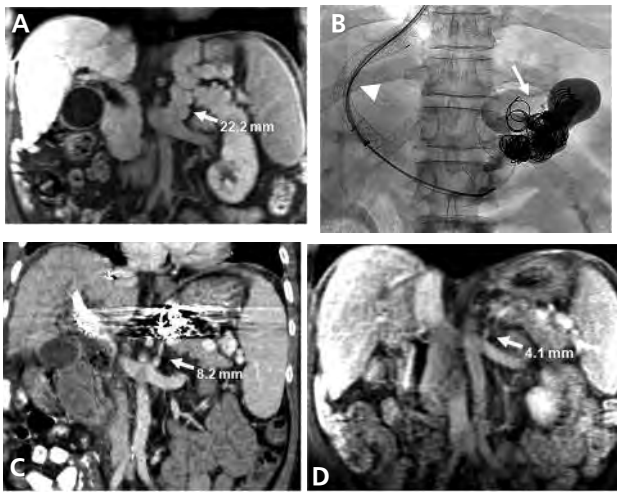
Presence of an SRS does not necessitate intervention, particularly if adequate vascular flow is achieved in the allograft at the time of implantation. Intraoperative measurements of portal flow in the transplanted liver can guide intervention.[14] The degree of steal from the SRS can be measured quantitatively by occluding the shunt and determining the increase in flow through the portal system of the allograft.

Table 1. Treatment Modality and Effect on SRS

Patient	Intervertion Performed	Effect on SRS Size
1	Preoperative TIPS	*
2	Preoperative TIPS	Decreased
3	Preoperative TIPS	Decreased
4	Preoperative TIPS	Decreased
5	Preoperative TIPS	No change
6	Preoperative TIPS	No change
7	Intraoperative ligation of SRS	Decreased
8	Preoperative percutaneous embolization of SRS	Decreased+

*unable to measure, +required further intervention

Figure 2. (A-D) SRS After TIPS



A. SRS 22.2 mm in diameter before intervention (arrow)
B. TIPS creation (arrowhead), concomitant coiling of SRS (arrow)
C. One month post-TIPS, SRS 8.4 mm in diameter (arrow)
D. Four months post-TIPS, SRS 4.1 mm in diameter (arrow)

Table 2. Effect of TIPS on SRS Diameter and Porto-Systemic Gradient

Patient	SRS Diameter Before TIPS	SRS Diameter After TIPS	Portosystemic Gradient Before TIPS	Portosystemic Gradient After TIPS
1	13 mm	*	12 mm Hg	4 mm Hg
2	9 mm	8 mm	6 mm Hg	5 mm Hg
3	22 mm	5 mm	*	11 mm Hg
4	10 mm	3 mm	13 mm Hg	2 mm Hg
5	14 mm	14 mm	21 mm Hg	7 mm Hg
6	14 mm	14 mm	18 mm Hg	7 mm Hg

*unable to measure

Margarit and associates presented case reports of intraoperative measurements of portal venous flow through the transplanted liver with and without compression of the splenorenal shunt.[28] In these cases, they could demonstrate significant improvement of portal venous flow after compression of the SRS up to 8 times that of their initial flow measurements, thereby justifying the need for ligation of an SRS in these patients.

We have reported a case of an SRS steal severe enough to cause rapid postoperative deterioration of the recipient necessitating reoperation (Table 1). Despite intraoperative identification and ligation of the SRS with subsequent improvement of portal flow, the patient was unable to be closed primarily and died later that day.[14] Ligation of the left renal vein (LRV) via a retroperitoneal approach is an appealing alternative for such patients as it allows

access without having to reopen the abdomen posttransplant.

Ligation of the LRV has been suggested as a method to resolve the negative hemodynamic sequelae of an SRS on the allograft. Castillo and associates recently reported intraoperative ligation of the LRV based on intraoperative portal flow and portal pressure measurements. In that series, portal vein flow less than 1200 mL/min in the allograft after reperfusion raised concern for portal hypoperfusion, necessitating intervention.[4] Ligation of the LRV may be more appealing than ligation of the SRS itself, as the varix is very thin-walled and inadvertent damage could precipitate dramatic blood loss. Additionally, it has been well established that LRV ligation is a good surgical option and does not negatively affect renal function.[29]

An SRS smaller than 10 mm in diameter measured at the level of transition into the LRV will typically collapse and involute after liver transplant as portal pressures normalize and flow decreases through aberrant collaterals. Therefore, in patients with an SRS less than 10 mm in diameter, intervention may be unnecessary.[6-8] However, the posttransplant patient with a persistent splenorenal shunt should be monitored carefully for evidence of compromised allograft function, as there is increased risk for liver dysfunction, portal vein thrombosis, and hepatic encephalopathy in these patients. Regular evaluation of liver function tests as well as surveillance sonographic duplex imaging should be performed. Anecdotally, we have found reversal of the direction of flow in the intrahepatic left portal vein to be an early indicator of SRS shunting, well before partial or complete portal vein thrombosis.

The sequelae of continued vascular steal by SRS can be devastating to the transplanted liver and ultimately can lead to loss of the allograft. Thus, for SRSs larger than 10 mm, automatic ligation of the aberrant shunt may be justified.[15-18] Lee and associates described ligation of the LRV for splenorenal shunts larger than 10 mm as a safe and effective method of preventing vascular steal from the portal system of the allograft.[16] In their review of 44 patients who had undergone ligation of a large splenorenal shunt during living donor liver transplant, no procedural-related renal dysfunction was observed and there were no cases of steal-related phenomena. Ligation of an SRS may be more important in the setting of live-donor transplant because of the relatively small size of the donor allograft relative to a preexisting SRS. Posttransplant shunting may be more pronounced than in the deceased donor scenario.[18]

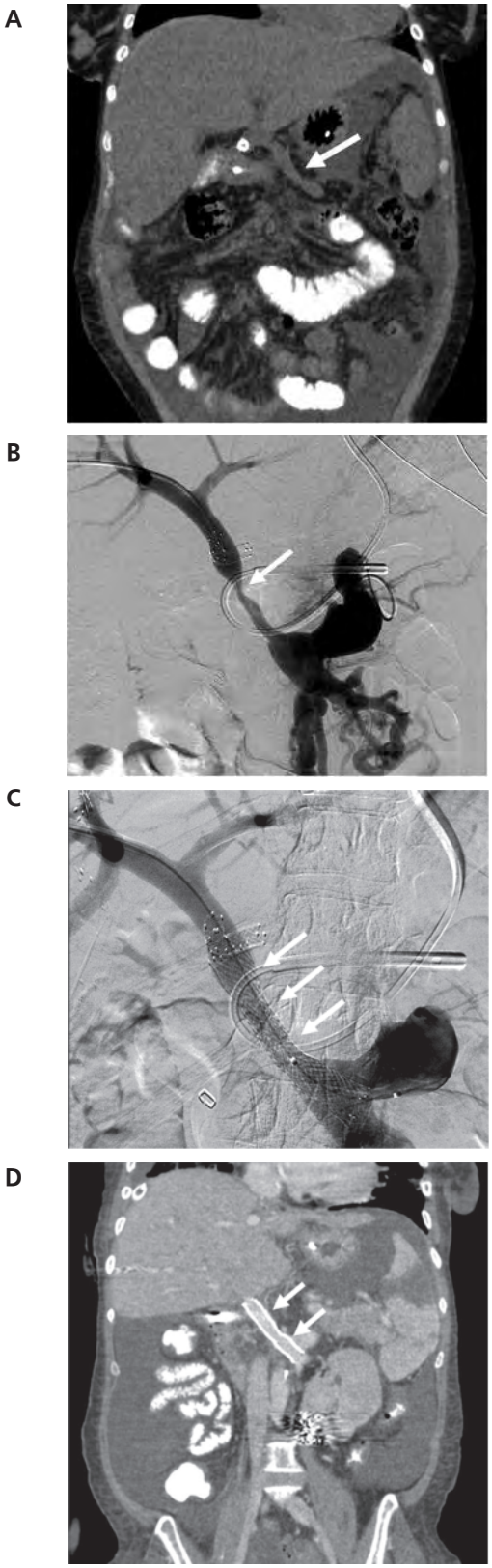
Orthotopic liver transplant candidates with portal vein thrombosis present additional operative challenges.[19] The location and extent of the portal vein thrombosis are important factors in the operative technique used during transplant. Eversion thromboendovenectomy is an option for more-localized portal vein thrombosis, but if there is extensive involvement of the porto-mesenteric circulation, more complex interventions such as venous jump graft reconstructions, arterialization of the portal vein, or combined liver and small bowel transplant are required. Recently, focus has shifted to cavoportal hemitransposition and renoportal anastomosis (Figure 3A).[20-23] In patients with portal vein thrombosis and SRS, the retroperitoneal approach presents an elegant surgical solution as it ensures adequate portal perfusion and simplifies the overall procedure by redirecting shunted blood to the liver and obviating the need for splenectomy. Kato and associates described this technique in 5 patients and demonstrated prompt reperfusion of the allograft with adequate decompression of the bowel as well.[20] Four of 5 patients were alive with well-functioning grafts 16 months after transplant. Anecdotally, we have encountered stenosis of a renoportal conduit (Figure 3B) that resulted in canalization of the collapsed SRS as well as additional varices requiring additional percutaneous intervention to re-establish hepatopetal flow (Figures 3C and D).

Postoperative intervention

In some instances, ligation of the SRS is either not possible or deemed unwarranted at the time of transplant. However, if postoperative imaging shows changes in flow dynamics that lead to a clinically significant vascular steal from the allograft, intervention may be necessary. Interruption of the SRS can be achieved either by reoperation with ligation of the SRS or by percutaneous techniques leading to occlusion of the shunt.

Reoperation allows for direct ligation of the shunt with or without concomitant splenectomy. Splenectomy is a controversial technique in these patients as it can lead to an increased incidence of portal vein thrombosis and sepsis in already

Figure 3. (A-D). Renoportal Anastomosis, Stenosis, Stenting



A. Renoportal anastomosis (arrow)
B. Stenosis of renoportal anastomosis (arrow)
C. Stenting of renoportal stenosis (arrows)
D. One month poststenting of renoportal anastomosis (arrows)

immunocompromised patients. Nonetheless, case reports have shown improved portal flow when an SRS ligation is combined with splenectomy.[15] However, reoperation after liver transplant can be technically difficult, making minimally invasive percutaneous approaches more appealing.

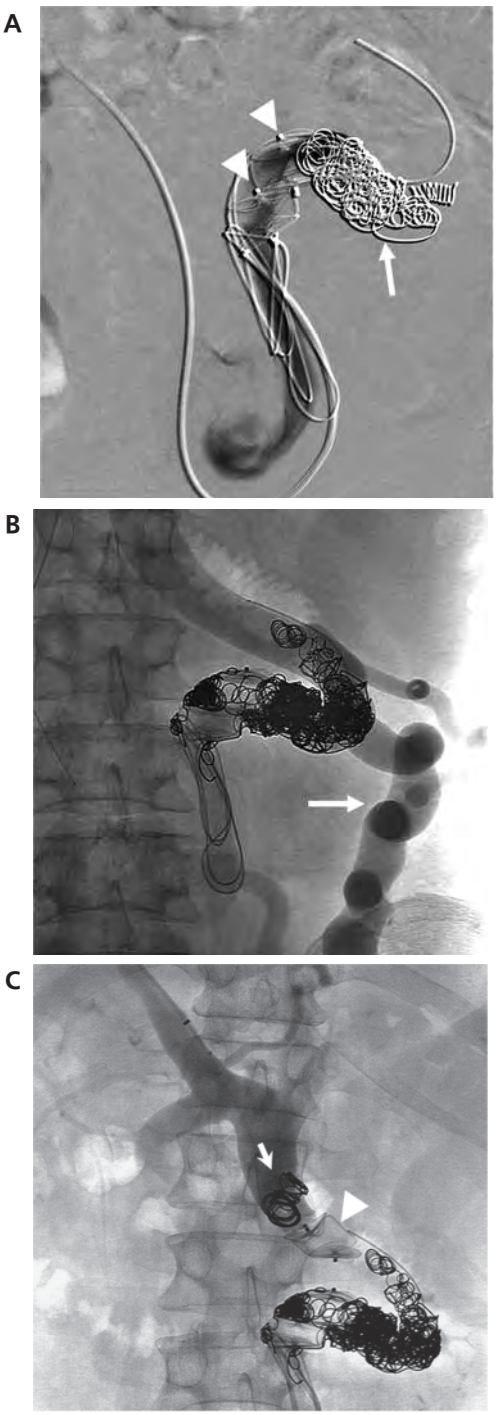
Case reports have shown successful occlusion of splenorenal shunts using percutaneous approaches employing stent grafts, plugs or coils, and thrombotic agents. Amplatzer plugs can be used in the splenic vein to occlude the SRS directly. In cases where the plugs cannot safely be deployed because of the size of the SRS or fear of migration, covered stent grafts can be used from the superior mesenteric vein to the portal vein to preferentially redirect flow from the portal system and the splenic vein.[24-27]

However, in our experience, the percutaneous approach to SRS occlusion may require multiple interventions from different approaches to achieve permanent success (Table 1). In 1 of our patients undergoing retransplant with Mayo End-Stage Liver Disease score of 54 at the time of surgery, we decided not to intervene on a known 3-cm SRS as his portal flow was greater than 2 L/min during portal bypass, and our focus was on timely conclusion of the procedure. Sonographic monitoring showed adequate hepatopetal portal flow for the first 2 months postoperatively. However, when the transaminase levels increased to more than 1.67 μkat/L and hepatofugal flow appeared on ultrasound, percutaneous endovascular intervention was performed. Initial therapy with Amplatzer plugs was ineffective but subsequently, the SRS was occluded with an array of platinum embolization coils and injection of Gelfoam slurry into the interstices of the coils (Figure 4A). Unfortunately, the SRS reopened 1 year after the percutaneous intervention (Figure 4B). Recently, we reattempted percutaneous closure of the SRS and are hopeful for a more-durable repair (Figure 4C).

Discussion

The pathophysiology and consequences of portal hypertension often lead to formation of porto-systemic venous collaterals that preferentially shunt blood flow away from the high resistance hepatic vascular bed to the lower resistance systemic circulation. When the cirrhotic liver is replaced, these collaterals become harmful to the new, low-

Figure 4. (A-C). Endovascular Repair of Splenorenal Shunt Postorthotopic Liver Transplant



A. Endovascular occlusion of SRS by 2 Amplatzer plugs (arrowheads) and multiple coils (arrow)
B. Spontaneous recanalization of SRS (arrow) after endovascular intervention
C. Endovascular reocclusion of SRS with additional coils (arrow) and plugs (arrowhead)

resistance allograft, shunting portal blood away from the liver and ultimately causing portal vein thrombosis and graft loss. Preoperative imaging should assess size and location of any shunts, and a collaborative decision can be made about if and when to intervene. Treatment options include preoperative TIPS to decompress the SRS, automatic intraoperative ligation of the shunt, intraoperative assessment of portal flow with decision to intervene if there is evidence of inadequate flow to the allograft, close monitoring of the shunt and the functional status of the transplant liver without intervention for a small SRS, creation of portorenal anastomosis in the case of portal vein thrombosis, or postoperative percutaneous interventions such as embolization of the shunt. Because published literature on this topic consists primarily of case reports, and the experience of most transplant teams is limited to scant numbers of cases, further investigation into the best approach to an SRS is warranted, as the presence and persistence of collaterals such as an SRS can lead to allograft dysfunction and possible graft loss if continued vascular steal from the portal system occurs after transplant.

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