The inferior mesoiliacal shunt: A novel shunt for refractory rectal variceal bleeding due to splanchnic thrombosis

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ABSTRACT

Surgical shunt therapy may be required when pharmacologic, endoscopic, and radiologic treatment of chronic splanchnic vein thrombosis have failed. In this case report, we present a new interposition shunt for the treatment of refractory rectal variceal bleeding: the inferior mesoiliacal shunt between the inferior mesenteric vein and the left common iliac vein using a cryopreserved iliac venous graft. The postoperative course was complicated by shunt thrombosis at day 2, probably owing to inadvertent interruption of anticoagulation and a decrease in the shunt flow rate. Surgical thrombectomy was performed successfully. The patient presented no relapse of rectal bleeding and was asymptomatic and well at the 12-month follow-up. (J Vasc Surg Cases and Innovative Techniques 2020;6:562-5.)

Keywords: Splanchnic vein thrombosis; Polycythemia vera; Inferior mesoiliacal shunt

Splanchnic vein thrombosis (SVT) is an uncommon manifestation of venous thromboembolism involving one or more splanchnic vein(s).[1,2] The clinical presentation is heterogenous, but the most frequent complication remains upper and/or lower gastrointestinal bleeding owing to portal hypertension (PH).[2] Nevertheless, bleeding episodes may be refractory to endoscopic and radiologic management and may necessitate surgical therapy, mainly gastric devascularization procedures and venous shunts. The type of surgical shunt to be constructed depends on the nature of the remaining patent veins. Therefore, in case of extensive diffuse SVT, construction of classical shunts including portocaval, mesocaval, and splenorenal may anatomically not always be possible.

We describe a new interposition shunt; the inferior mesoiliacal (IMI) shunt between the inferior mesenteric vein (IMV) and the left common iliac vein (LCIV).

CASE REPORT

A 56-year-old woman with refractory rectal variceal bleeding, hepatic hydrothorax, and mild ascites was referred to our center. She has a history of JAK2-V617F-positive polycythemia vera that was discovered when she presented with a SVT 14 years earlier. She developed a progressive chronic thrombosis, partly owing to poor adherence to anticoagulant therapy. Physical examination revealed mild ascites, splenomegaly, and no signs of encephalopathy. Laboratory findings showed chronic anemia (hemoglobin 10 g/dL), iron deficiency (14 μg/dL), and platelet count was 189 $\times$ 10^9/L. Liver tests were normal. Endoscopy revealed no esophagogastric varices, but large rectal varices. Computed tomography (CT) angiography showed a portal cavernoma with only a partially patent splenic vein (Fig 1, *A-C*), a patent collateral venous network between the spleen and the rectum draining into the left internal iliac vein (Fig 1, *D*), and a partially open prominent IMV (Fig 1, *E*). Cavography showed a narrowed lumen of the left renal vein. We opted to construct a shunt between the IMV and the LCIV to selectively decompressing the IMV territory and the rectal varices.

Explorative laparotomy confirmed complete obstruction of the extrahepatic portomesenteric venous axis, including the superior mesenteric vein (SMV). The IMV was thrombosed in its proximal portion until the confluence with the splenic vein, but open and dilated distally. The IMV was dissected free after mobilizing the descending colon. A transit time flow measurement showed an hepatofugal flow of 50 mL/min and an elevated portal pressure of 30 mm Hg. The LCIV was then mobilized by transecting the mesocolon and ligating the inferior mesenteric artery (Fig 2, *A*). An IMI shunt between the IMV and the LCIV was then created in an end-to-side fashion with running Prolene 7.0 interposing a cryopreserved iliac venous allograft of diameter 8 mm with length of 10 cm, provided by the European Homograft Bank in Brussels (Figs 2, *B* and 3). Intraoperative measurement indicated an immediate drop of the

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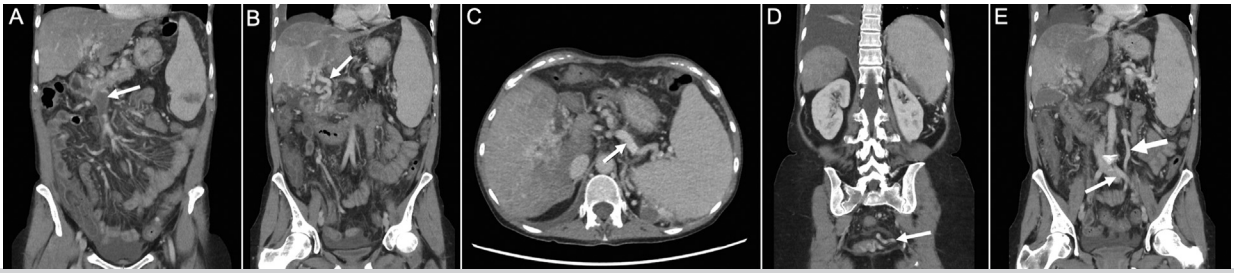

Fig 1. Preoperative computed tomography (CT) angiogram demonstrating extensive splanchnic vein thrombosis (SVT); superior mesenteric vein (SMV) thrombosis **(A)**, cavernous transformation of the portal vein **(B)**, partial patent splenic vein **(C)**, patent collateral venous network between the spleen and the rectum draining into the left internal iliac vein **(D)**, and proximally occluded, but distally dilated open inferior mesenteric vein (IMV) (*thick arrow*), and patent left common iliac vein (LCIV) (*thin arrow*) **(E)**.

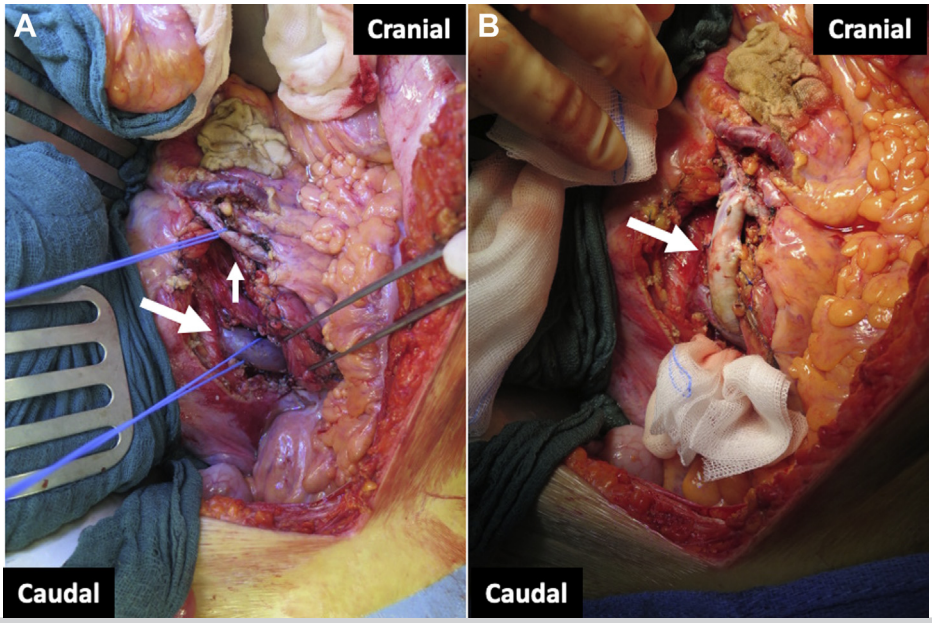

Fig 2. Intraoperative images. **A,** Inferior mesenteric vein (IMV) encircled (*thin arrow*) and left common iliac vein (LCIV) encircled (*thick arrow*). **B,** Side-to-side anastomosis of IMV to LCIV using a cryopreserved iliac venous graft (*arrow*).

venous pressure from 30 mm Hg to 8 mm Hg and a high flow of 500 mL/min through the newly created IMI shunt.

Additionally, the splenic artery was ligated and a partial gastric devascularization was performed. Low-molecular-weight heparin therapy 40 mg twice daily was ordered 6 hours after surgery.

A control duplex ultrasound examination and CT scan on postoperative day 2 could not identify the shunt. Thrombosis of the homologous graft was confirmed upon reexploration. Fresh clots were extracted and a good flow (250 mL/min) was restored. Continuous intravenously heparin drip, not catheter directed, was administered and switched to therapeutic low-molecular-weight heparin after 72 hours, later converted to maintenance Phenprocoumon. Shunt patency was confirmed by postoperative CT (Fig 4). The patient developed a chyle leak that resolved with medium chain triglyceride diet, but delayed her discharge until postoperative day 36. Maintenance therapy consisted of diuretics, anticoagulation (target international normalized ratio of >2.5), and nonselective beta-blockers.

At the 1-year follow-up, the patient presented no recurrent episodes of significant rectal bleeding. Duplex ultrasound examination confirmed the patency of the IMI shunt. Control endoscopy showed no gastric and only small rectal varices. Written informed consent was obtained from the patient for publication.

DISCUSSION

The clinical presentation of SVT is heterogenous, depending on the extent of thrombosis and the nature of the veins involved.[1,2] In our patient, diffuse SVT resulted in recurrent episodes of rectal variceal bleeding despite adequate pharmacologic and endoscopic treatment.

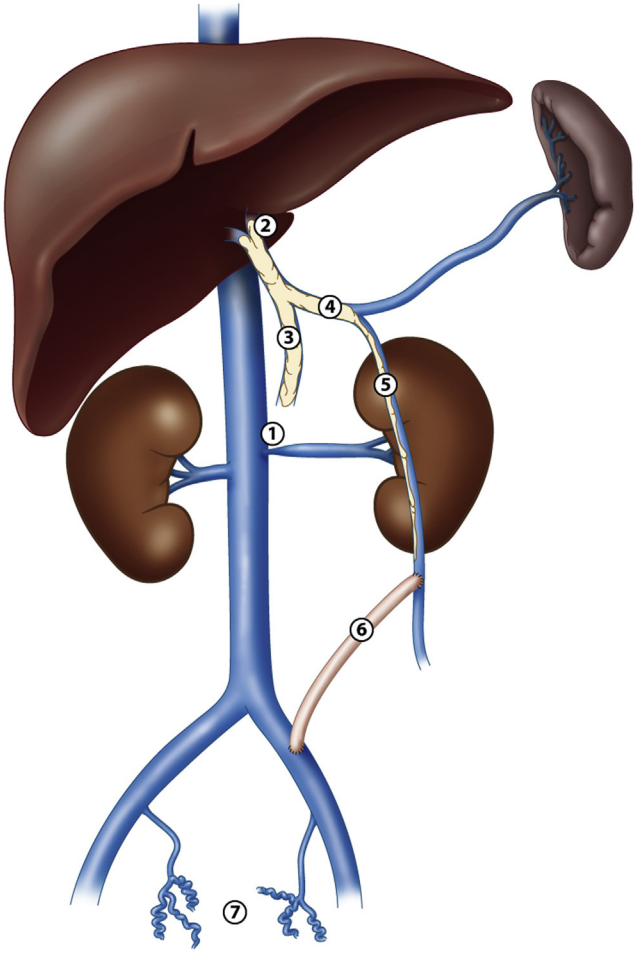

Fig 3. Graphic representation of the extensive splanchnic vein thrombosis (SVT) and orientation of inferior mesoiliacal (IMI) shunt. (1) Narrow caval ostium of the left renal vein. (2) Portal cavernoma. (3) Superior mesenteric vein (SMV) thrombosis. (4) Partial left splenic vein thrombosis. (5) Partial inferior mesenteric vein (IMV) thrombosis. (6) IMI shunt. (7) Rectal varices.

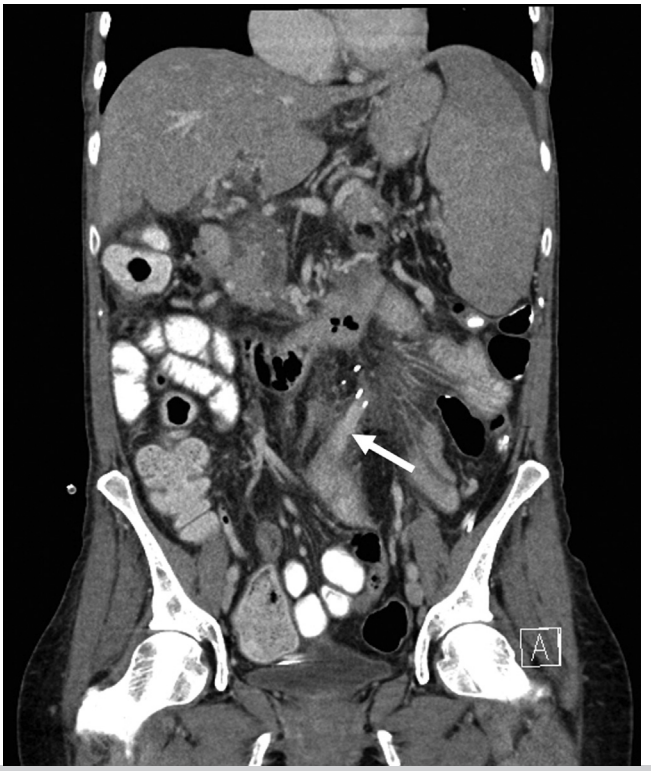

Fig 4. Postoperative computed tomography (CT) angiogram showing a patent inferior mesoiliacal (IMI) shunt (*arrow*) between the inferior mesenteric vein (IMV) and the left common iliac vein (LCIV).

We report a novel type of shunt: the IMI shunt selectively decompressing the IMV into the LCIV.

The traditional mesocaval shunt is a H-shunt between the SMV and the inferior vena cava and uses either a prosthetic or an autologous or homologous jugular or iliac graft.[3,4] At surgical exploration, occlusion of the SMV was confirmed precluding construction of this type of shunt. A splenorenal shunt was considered, but was contraindicated because of a stricture at the caval ostium of the left renal vein. Anatomically, the only option left was to selectively decompress the distally dilated IMV. The latter was easily identified, and encircled. Shunts between the IMV and the vena cava have been described previously.[5,6] We decided to use the LCIV, not described before in literature, as the landing zone for our shunt because the distance between the IMV and the LCIV was shorter compared with the inferior vena cava. An 8-mm interposition homograft was used to bridge the two veins. This diameter was opted for in accordance to the vessels diameter; the IMV was smaller in diameter but larger than a normal IMV. Very likely there is a correlation between size of the vein and patency of the shunt in which half of the size of the shunt is a good estimate. We did not opt for a direct transposition onto the LCIV in order to decompress the IMV proximally and distally from the shunt. Shunts between the IMV and the gonadal vein or the left renal vein have also been described, but could not be considered because of the stenosis of the left renal vein orifice that would compromise outflow.[7,8]

In addition to the IMI shunt, the stomach was partially devascularized to prevent upper gastrointestinal bleeding. A transjugular intrahepatic portosystemic shunt procedure was technically impossible considering the chronic obstructed portal vein. To reduce PH with its associated hypersplenism and hepatic hydrothorax, the splenic artery was additionally ligated.[9] Because the short gastric vessels were preserved, splenic infarction did not occur.

Despite obtaining high flows (500 mL/minute) through the shunt intraoperatively, corresponding with previously published flow values through portosystemic shunts,[10]

early thrombosis occurred. At immediate reexploration, no signs of technical failure like kinking or torsion were observed, but a high tendency for clotting was noticed. Thrombectomy was performed and this resulted in rapid restoration of adequate flow. Of note however, the flow went from 500 to 250 mL/min. This could possibly be explained by the decrease in PH owing to the patency of the IMI shunt itself, until a steady state is reached, a phenomenon that can take a few hours. In additions, we cannot exclude that ligating the splenic artery contributed to a decrease of PH and, second, the flow through the newly created shunt.

Retrospectively, our patient had accidentally not received the prescribed postoperative anticoagulation and this, in the absence of technical factors and a decrease in the shunt flow rate, probably triggered the thrombosis. A continuous heparin drip was intravenously administered postoperatively, and thrombosis did not reoccur.

In conclusion, this case illustrates the efficacy of a new IMI shunt in the treatment of refractory rectal variceal bleeding in patients with diffuse SVT and proximally occluded but still distally open IMV. Strict postoperative surveillance and continued anticoagulation are essential. Longer term patency and efficacy of this new decompressive shunt needs to be determined.

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