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Original article

Portal vein thrombosis after laparoscopic sleeve gastrectomy: presentation and management

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Abstract

Background: Portal vein thrombosis (PVT) is a serious problem with a high morbidity and mortality, often exceeding 40% of affected patients. Recently, PVT has been reported in patients after laparoscopic sleeve gastrectomy (LSG). The frequency is surprisingly high compared with other abdominal operations.

Objective: We present a series of 5 patients with PVT after LSG. The treatment was not restricted simply to anticoagulation alone, but was determined by the extent of disease. A distinction is made among nonocclusive, high-grade nonocclusive, and occlusive PVT. We present evidence that systemic anticoagulation is insufficient in occlusive thrombosis and may also be insufficient in high-grade nonocclusive disease.

Setting: Single private institution, United States.

Methods: We present a retrospective analysis of 646 patients who underwent LSG between 2012 and 2015. In all patients, the diagnosis was established with an abdominal computed tomography (CT) scan as well as duplex ultrasound of the portal venous system. All patients received systemic anticoagulation. Depending on the extent of disease, thrombolytic therapy and portal vein thrombectomy were utilized. All patients received long-term anticoagulation.

Results: Four patients with PVT were identified. A fifth patient with PVT after LSG was referred from another center. The mean age of all patients was 49 years. One patient had a history of deep vein thrombosis (DVT). No complications were identified intraoperatively or during the hospital stay, and all patients were discharged by postoperative day 2. The patients presented with PVT at an average of 20 days (range: 10–35) post-LSG. The CT scan was positive for PVT in all patients. In stable noncirrhotic patients with nonocclusive disease, we administered therapeutic anticoagulation. One patient with high-grade, nonocclusive PVT received anticoagulation alone. Patients with occlusive disease were treated with operative thrombectomy including intraoperative and postoperative thrombolysis (tissue plasminogen activator) with subsequent therapeutic anticoagulation, followed by oral warfarin or a factor Xa inhibitor. There was 1 death from multisystem organ failure in the patient who was referred from another institution with occlusive disease, initially managed only with an anticoagulation infusion.

Conclusions: We maintain that portal vein patency is essential to normal gastrointestinal physiology and should be the treatment goal in all patients with PVT. In these patients, the therapeutic option should be guided by the extent of the thrombosis. In view of currently available approaches, we propose that operative portal vein thrombectomy, in conjunction with fibrinolysis and anticoagulation,

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offers the best long-term success in patients with occlusive PVT. (Surg Obes Relat Dis 2016;12:1787–1794.)

Keywords: PVT; LSG; Portal vein thrombosis; Sleeve gastrectomy; Thrombosis; Thrombolysis

Venous thrombosis is a common complication after bariatric surgery. The reported incidence is 3% [1]. Portal vein thrombosis (PVT) occurs less frequently, but has been reported after laparoscopic adjustable gastric band (LAGB), laparoscopic Roux-en-Y gastric bypass (LRYGB), and laparoscopic sleeve gastrectomy (LSG) [2–10]. There appears to be an unexplained statistical increase of PVT after LSG compared with other/abdominal procedures. Surprisingly, there is a higher rate of PVT after LSG even compared with patients after the duodenal switch procedure (DS) [1,6–10]. Most centers use only heparin anticoagulation to treat PVT; however, heparin alone has a significant failure rate, approaching 65% in the best available study [11]. If the PVT is occlusive, an even higher failure rate would be expected. The acute and chronic sequelae of portal vein occlusion are usually disabling and occasionally even fatal.

We present effective alternatives to anticoagulation alone. Particularly in cases of acute occlusive PVT, laparotomy with portal vein thrombectomy, facilitated by intraportal tissue plasminogen activator (TPA) and heparin, is highly effective in restoring portal flow. Portal hypertension is eliminated and normal pancreatic-enteric liver physiology is restored.

Methods

The charts of 646 LSG patients were reviewed. These patients were operated on between 2012 and 2015. The average age was 49 years (range: 38–58) and average body mass index (BMI) was 42 kg/m^2 (range: 42–50). In our series, there were 4 documented cases of PVT after LSG. The fifth case of PVT after LSG was referred from another center. The patient's demographic characteristics are seen in Table 1. All 4 cases had been performed in a standardized manner by 2 of 4 surgeons in our group. The referred patient had undergone a very similar LSG. All patients received deep vein thrombosis (DVT) prophylaxis according to the consensus recommendation of 2008 [12]. The protocol included perioperative heparin 5000 units subcutaneously every 8 hours and sequential compression device hose. All patients underwent an uncomplicated LSG in reverse Trendelenburg with carbon dioxide insufflation pressure <15 mm Hg. Each operation was performed over a 36F bougie catheter, beginning the sleeve 5 cm proximal to the pylorus. The patients received postoperative fluids to maintain a urine output >60 mL/hr. Early ambulation was required. Patients were discharged by the second postoperative day taking adequate oral fluids (>2 L/d).

The patients with PVT presented to the emergency room 10–32 days after LSG. Initial complaints were nonspecific, including malaise, nausea, and abdominal pain; one patient complained of fever. Patients had screening laboratory testing and abdominal computed tomography (CT) scans using IV contrast. Duplex ultrasound of the PV was performed after the initial CT scan. Subsequent CT angiograms were performed at least once postoperatively to evaluate the status of the thrombus. Further scans were obtained if clinically indicated. All patients were initially treated with systemic therapeutic heparin (activated partial thromboplastin time [aPTT] 2–3 times baseline). Further intervention was determined by the severity of disease, progressing from high-grade nonocclusive to occlusive thrombosis. Patients with occlusive disease were treated with perioperative heparin followed by laparotomy and portal vein thrombectomy facilitated by intraoperative TPA infused directly into the portal venous system (intraclot infusion) (Fig. 1). Intraoperative ultrasound was used to guide the thrombectomy. A transabdominal 5F catheter was placed in a tertiary mesenteric vein for uninterrupted postoperative TPA infusion followed by continuous heparin infusion. Two patients underwent bowel resection. The

Table 1
Patients' demographic characteristics

Patient number	Age	Sex	BMI (kg/m²)	Co-morbidities	Prothrombotic Medications	Operative time (min)	Length of stay (d)
1	45	M	45	OA	No	35	2
2	48	F	47	OA, GERD, OSA	No	30	2
3	56	M	42	MI, CABG, DVT, HTN, OSA,GERD	No	31	2
4	38	F	45	None	No	32	2
5	58	F	50	—	—	—	—

BMI = body mass index; OA = osteoarthritis; GERD = gastroesophageal reflux disease; OSA = obstructive sleep apnea; MI = myocardial infarction; CABG = coronary artery bypass grafting; DVT = deep vein thrombosis; HTN = hypertension.

Patients 1–4 denied smoking and family history of thrombosis. We cannot comment on patient 5, as she was referred from another institution.

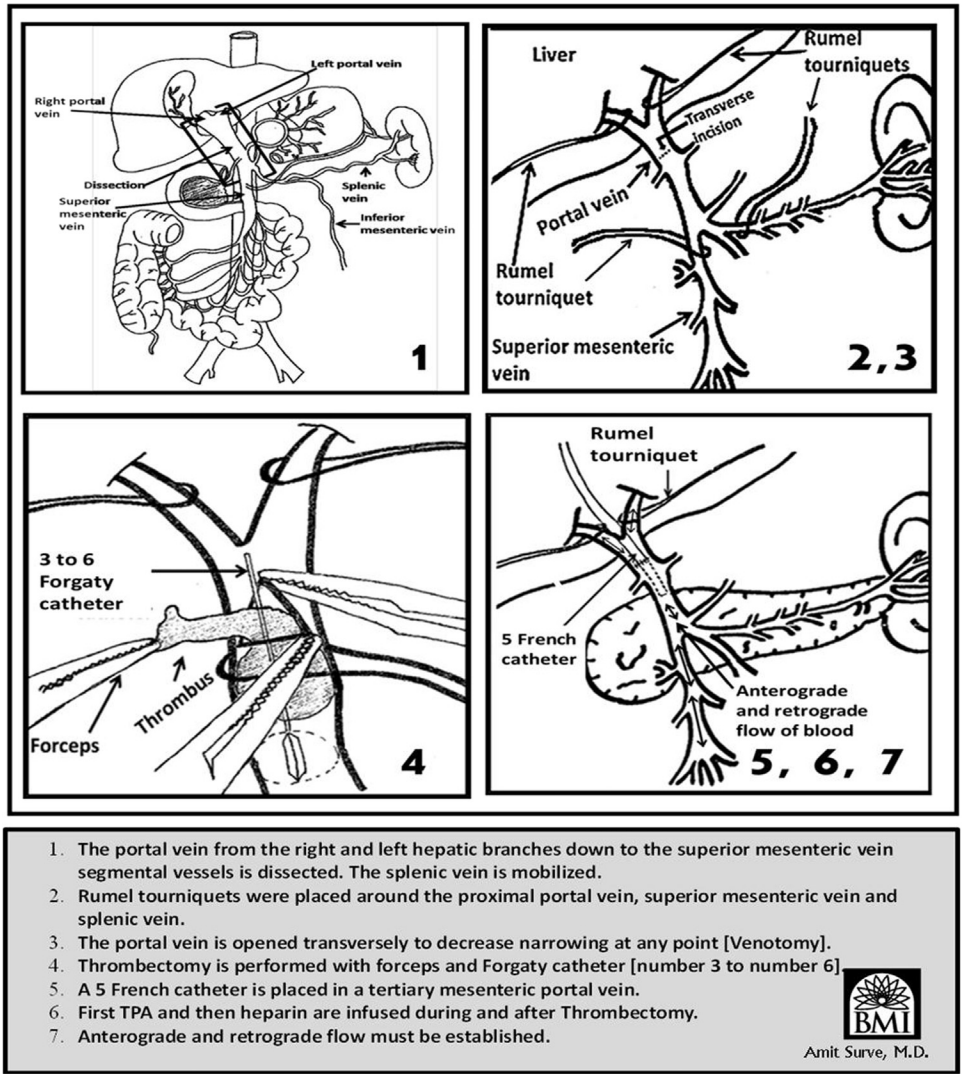

Fig. 1. Hand-drawn sketch showing step-by-step technique of portal vein thrombectomy.

third option of catheter-directed TPA into the superior mesenteric artery was not utilized in any of these patients, although this option could be considered in high-grade nonocclusive PVT. Patients underwent a postoperative Computed tomography angiography (CTA) to evaluate portal vein (PV) patency. Further studies were ordered only as clinically indicated. All patients received long-term anticoagulation with warfarin or, preferably, a factor Xa inhibitor (rivaroxaban, apixaban). We achieved at least 6 months of subsequent anticoagulation therapy as recommended by our consulting hematologist. All patients were evaluated for thrombophilia.

Case reports

Case 1

Two weeks post-LSG, this patient presented to the emergency room with malaise, nausea, mild central abdominal pain, and temperature of 101°F. Bloodwork was normal. A CT scan found a nonocclusive PVT. The patient was placed on a therapeutic heparin drip with a target of aPTT 2–2.5 times baseline (60–80 s). The patient's symptoms resolved within 1 day of starting the heparin drip. A subsequent CT scan found clot regression. The patient was discharged on therapeutic rivaroxaban. He has had no symptom recurrence after 10 months follow-up.

Case 2

On postoperative day (POD) 10, the patient was seen in the emergency room with malaise, severe nausea, mid-abdominal pain, and tachycardia. Her white blood cell count (WBC) was 10,700/mm^3. Her temperature was normal. A CT scan found nonocclusive PVT extending into superior mesenteric vein (SMV) and splenic vein (SV). The patient was started on heparin. Her symptoms resolved within 36 hours. Three days later, a CTA found small-bowel thickening but stable nonocclusive thrombus. Therapeutic heparin

was continued for 5 days. The patient had been started on warfarin and was discharged with a therapeutic international normalized ratio on day 6.

Twenty-five days later, the patient was admitted with a bowel perforation caused by segmental SMV thrombosis. She was taken to surgery, where a 55-cm segment of infarcted small bowel was resected with a primary anastomosis. The portal vein was patent. She received postoperative heparin and was discharged 1 week later on rivaroxaban. She has had no recurrent symptoms with follow-up exceeding 1 year.

Case 3

The patient was admitted 32 days post-LSG with malaise, dysphagia, severe nausea, vomiting, abdominal pain, and tachycardia. His WBC was 16,000/mm^3 and serum lactate was 4.0 mmol/L. His temperature was normal. A CT scan with intravenous (IV) contrast found partial PV and complete SMV obstruction, ascites, and probable ischemic bowel. The patient was operated on emergently. Portal-SMV thrombectomy was accomplished, guided by intraoperative ultrasound (Fig. 2 A, B); 40 cm of necrotic bowel was resected but not reanastomosed. Marginal bowel was left, pending a second-look procedure. A 5F feeding tube was placed in a tertiary mesenteric venule. The portal system was perfused during and after the operation with TPA, followed by continuous heparin. The abdomen was partially closed, leaving the mesenteric catheter in place.

A second-look procedure was undertaken the following day. Further bowel demarcation was noted and the nonviable bowel resected. Bowel continuity was reestablished. An intraoperative ultrasound found an open portal system. A later CTA confirmed a patent portal system. The patient was transitioned from heparin to warfarin and discharged on POD 11. He remains symptom-free at >1 year follow-up.

Case 4

The patient presented 2 weeks post-LSG to the emergency room complaining of malaise, nausea, and midabdominal pain. Her WBC was 13,000/mm^3. Her temperature was normal. Other bloodwork was unremarkable except for alanine aminotransferase of 180. The CT scan found extensive occlusion of the portal venous system, including intrahepatic left and right branches, PV, SMV, and SV, as well as ascites and bowel wall thickening.

The patient was administered heparin and then taken to the operating room. An intraoperative duplex ultrasound confirmed the CT scan results. Through a portal venotomy, extensive thrombosis was removed from the PV, SMV, and associated branches (Fig. 3 A, B). The left portal vein could not be opened satisfactorily, but the right portal vein had excellent retrograde flow. Vigorous hepatopetal flow was established. A mesenteric catheter was placed through a 5F mesenteric catheter (Fig. 3 C, D). The portal system was infused with TPA during and after the operation, followed by a heparin drip. At the completion of the operation, the ultrasound again found a patent PV with excellent flow. During the night, the heparin drip intended for the mesenteric portal catheter was instead given systemically. A CTA found rethrombosis. The patient was returned to the operating room and again underwent thrombectomy with TPA and heparin infusion. The final ultrasound found robust hepatopetal flow. The following day, a CTA found excellent portal flow. The SV and left hepatic vein remain thrombosed but there was no evidence of portal hypertension, not even left-sided portal hypertension. The patient was symptom-free with a patent portal system. She remains on rivaroxaban and had no return of symptoms at 9 months of follow-up.

Case 5

This patient underwent LSG at another institution. The surgical technique included sizing with a 38F bougie and

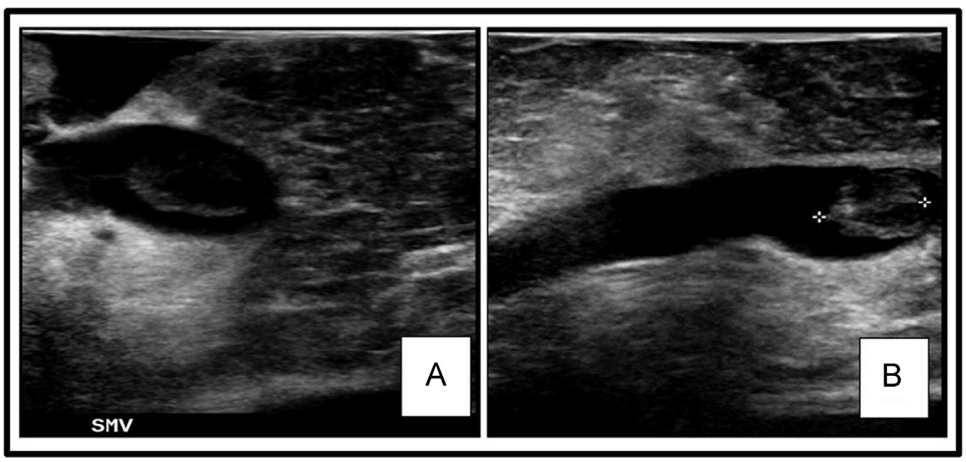

Fig. 2. (A) Duplex ultrasound: thrombus in the superior mesenteric vein. (B) Duplex ultrasound: thrombus extending into right portal vein.

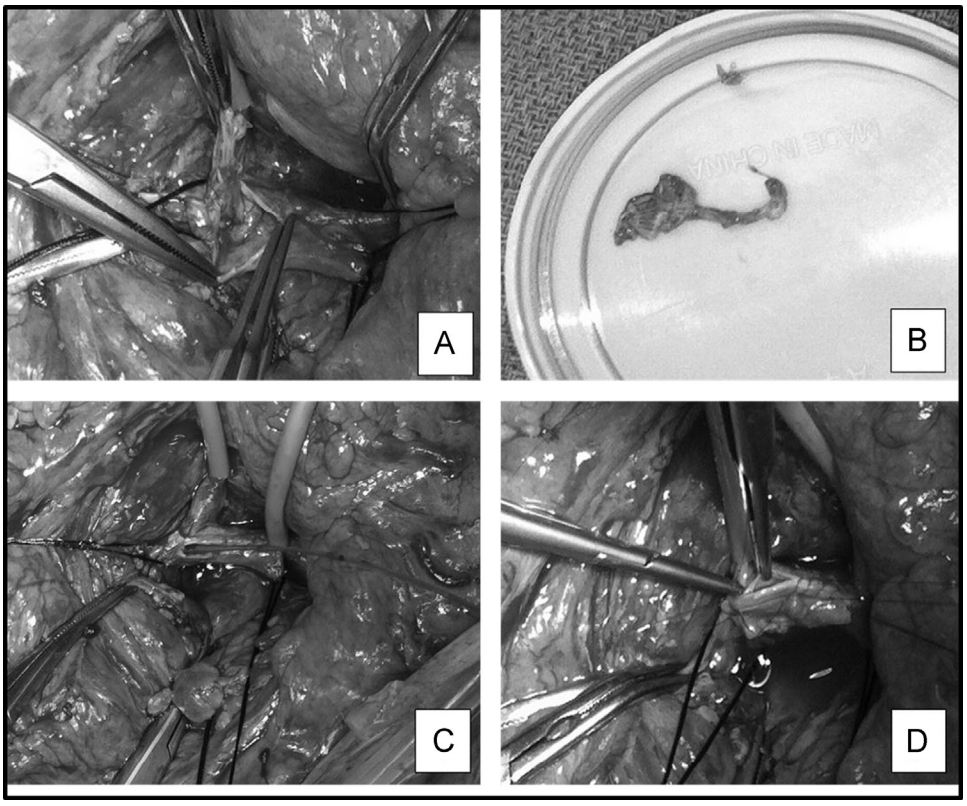

Fig. 3. (A) Occlusive thrombus of proximal portal vein extending into the right and left portal bifurcation. (B) Proximal portal vein thrombus; note right and left portal vein branch extension. The thinner clot tracked down from the superior mesenteric vein. (C) Fogarty catheter passing into the intrahepatic right and left portal branches. (D) Repair of portal venotomy.

perioperative DVT prophylaxis. Postoperatively the patient complained of difficulty consuming adequate fluid.

She presented to the emergency room at that institution with malaise, nausea, and mid-abdominal pain. A contrast CT scan found PVT with complete occlusion. The patient was immediately placed on a heparin drip. However, in the course of the ensuing 30 hours, her condition deteriorated. She developed multiple system failure. At that point, she was transferred to our facility and taken to the operating room emergently. The patient underwent portal vein thrombectomy. In spite of successfully reestablishing portal flow, her condition deteriorated with progressive multiple organ failure. Ultimately, support was withdrawn and the patient expired approximately 3 days after the onset of her symptoms.

Discussion

Current literature reports that LSG confers an increased risk of PVT compared to other abdominal procedures in general and, specifically, to other bariatric procedures. The incidence of PVT in our series is $<1\%$, with a range of .3%–1% reported in literature [1,6–10]. A precise explanation for this increased risk is unclear. These patients do have a reduced fluid intake with the reduction of gastric capacity, but so do patients after laparoscopic gastric banded plication and after duodenal switch—each procedure without a reported increased risk of PVT. One patient (patient 5) in our series complained of thirst >4 weeks before the diagnosis of PVT. However, dehydration alone does not appear to adequately explain why patients after LSG are at increased risk for PVT.

Obesity itself is a hypercoagulable state associated with increased thrombotic events [13]. All patients undergoing bariatric surgery carry this risk [14]. In a population-based control study from the Netherlands, obesity conferred a 2-fold risk of venous thromboembolism [15]. A prospective cohort study from Vienna reported a linear relationship between increased weight and venous thromboembolism [14]. Obesity predisposes to venous thrombosis by reduction of fibrinolysis, elevation of clotting factor levels, and release of proinflammatory mediators [16]. All operations, as well as all hospitalizations, are prothrombotic events [17]. However, the patients in this series had laparoscopic procedures requiring <35 minutes of operative time and hospitalizations <2 days. Operative conditions included 15 mm Hg of carbon dioxide insufflation pressure and reverse Trendelenburg position. Hypercapnia may cause mesenteric vasospasm [18–20]. All of these conditions could reduce portal flow; but these same conditions exist for many other laparoscopic procedures not associated with an increased PVT risk [21].

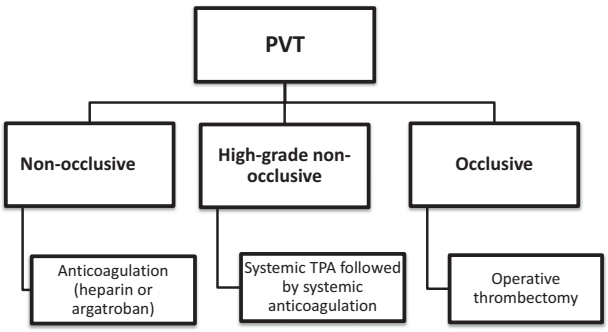

Fig. 4. Proposed algorithm for the treatment of portal vein thrombosis.

The patients in our series received DVT prophylaxis including sequential compression device hose and perioperative heparin. The clinical presentation was nonspecific in these patients, but all did complain of malaise, nausea, and abdominal pain, consistent with other series [22,23]. Although patients usually had normal vital signs, serum amylase, liver function tests, and lactate levels, the WBC was increased in 4 of 5 patients. A CT scan with IV contrast is indicated in all such patients post-LSG [24,25]. After the diagnosis of PVT is established, immediate anticoagulation with heparin is indicated even if surgery is contemplated. Ultimate treatment of PVT is determined by the severity of disease, which is categorized as nonocclusive, high-grade nonocclusive, or occlusive thrombosis (Fig. 4). In addition, the extent of the thrombus and the condition of the patient should be considered in making a therapeutic choice. In stable noncirrhotic patients with nonocclusive disease, we recommend therapeutic anticoagulation (heparin or argatroban) with a follow-up CT scan. Clot regression, or at least stabilization, should be documented or additional therapy should be added. In our 2 patients treated with heparin, symptoms improved within 36 hours of instituting therapy. Most cases of PVT are nonocclusive and usually respond to anticoagulation alone. Patients with occlusive disease were treated with operative thrombectomy including intraoperative and postoperative mesenteric thrombolysis (TPA) with subsequent therapeutic anticoagulation (heparin). Anticoagulation alone is not sufficient treatment in occlusive disease as reported by case 5 and limited prior literature [11]. High-grade partial occlusion does not properly respond on occasion, as reported by case 2. Case 2 would have been better treated, in retrospect, with superior mesenteric artery catheter-directed TPA infusion followed by systemic anticoagulation.

Unfortunately, the distinction between nonocclusive, high-grade nonocclusive, and occlusive disease has not been defined in the PVT literature. However, the futility of clot dissolution in occlusive venous thrombosis, including PVT using heparin anticoagulation, is well documented. Anticoagulation in patients with peripheral DVT was effective in $<5\%$ of patients treated with this modality alone, whereas thrombolytic therapy resulted in complete clot lysis in 45% and partial clot lysis in 65% [26,27]. Multiple other series report a high failure rate of anticoagulation alone in occlusive venous thrombosis [28,29]. Recanalization of the portal vein usually does not occur in noncirrhotic patients [11]. In partially occluded vessels, heparin does maintain the exposure of the thrombus to endogenous fibrinolytic agents, especially plasmin. This mechanism of thrombolysis is not possible in the setting of complete vessel occlusion. Plasmin cannot reach the clot. Indeed, systemic TPA has a high failure rate. The loss of portal flow has many acute and chronic consequences including bowel infarction, ascites, variceal bleeding, encephalopathy with neuropsychiatric dysfunction, and even fatal outcome [30–34]. Attempts to establish portal vein patency have been undertaken by percutaneous transhepatic and percutaneous jugular portal vein thrombolytic therapy with AngioJet suction of the clot. These approaches have met with limited success and are now rarely attempted [35–38].

There are only 2 case reports of portal vein thrombectomy in the literature. One of these surgeons also placed a portal catheter for direct mesenteric anticoagulation [39]. The use of a mesenteric catheter placed at the time of open thrombectomy has many advantages. Direct mesenteric clot perfusion with TPA can be continued postoperatively without the higher bleeding risk of inducing a systemic thrombolytic state. The amount of TPA and heparin infusion can also be substantially reduced. Less TPA is required because first-pass liver degradation is avoided. Less heparin is necessary because the mesenteric venous blood is immediately anticoagulated before systemic endothelial heparin binding and clearance of the administered heparin [40]. Excellent mesenteric venous anticoagulation is achieved with a much lower systemic aPTT (goal of 1.5–2.0 above baseline). Furthermore, the mesenteric catheter easily allows a direct mesenteric venogram without arterial injection, reducing the amount of contrast and potential renal toxicity. This venogram can be used to determine the duration of thrombolytic therapy. The benefits of operative portal vein thrombectomy far outweigh the risk of the procedure. In our series, the only adverse outcomes of the procedure itself were the requirement for blood transfusion and postoperative ileus.[1]

Conclusions

This paper emphasizes the following points: (1) The symptoms of PVT are often subtle and nonspecific, requiring a high index of suspicion for accurate diagnosis. (2) Early diagnosis is very important for best patient outcomes.

[1] In addition to the cases presented in the series, Dr. L. Belnap has had extensive experience in liver transplantation and portal vein thrombectomy over 25 years, involving many other underlying disease entities. Postoperative patency results have been excellent and complications are largely restricted to transfusion and postoperative ileus.

(3) The distinction between nonocclusive, high-grade nonocclusive, and occlusive thrombosis should determine appropriate treatment. (4) CT angiography is indicated for diagnosis, staging, and follow-up of treatment efficacy; duplex ultrasound is less accurate. (5) Therapeutic options include simple anticoagulation, catheter-directed thrombolytic therapy, and portal vein thrombectomy. (6) Anticoagulation and even catheter-directed superior mesenteric artery thrombolysis usually will not reestablish portal flow in patients with occlusive PVT. (7) Transabdominal mesenteric catheter-directed thrombolysis followed by heparin anticoagulation likely reduces the potential systemic risk of bleeding and is highly effective. (8) Portal vein patency should be the goal of therapy, not just stabilization of the thrombotic process.

Statement of Human and Animal Rights

This study did not involve the use of animal or human patients. Because this is a retrospective study, formal consent is not required.

Disclosures

The authors have no commercial associations that might be a conflict of interest in relation to this article.

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