

## **Title Page**

# **Portal Vein Thrombosis after Laparoscopic Sleeve Gastrectomy: Presentation and Management**

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Short Title: Portal Vein Thrombosis after Laparoscopic Sleeve Gastrectomy

## **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 to other abdominal operations.

**Objective:** We present a series of five 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 between 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.

**Method:** 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 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 nor during the hospital stay, and all patients were discharged by post-operative 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 non-cirrhotic patients with non-occlusive disease, we

65 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 post-operative thrombolysis (Tissue Plasminogen  
Activator) with subsequent therapeutic anticoagulation, followed by oral warfarin or a factor Xa  
inhibitor. There was 1 death from multi-system organ failure in the patient who was referred  
70 from another institution with occlusive disease, initially managed only with an anticoagulation  
infusion.

**Conclusion:** 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  
75 available approaches, we propose that operative portal vein thrombectomy in conjunction with  
fibrinolysis and anticoagulation, offers the best long-term success in patients with occlusive  
PVT.

**Keywords:** PVT; LSG; Portal Vein Thrombosis; Sleeve Gastrectomy; Thrombosis;  
80 Thrombolysis

## **INTRODUCTION**

Venous thrombosis is a common complication after bariatric surgery. The reported incidence is  
90 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 to other/abdominal procedures. Surprisingly, there is a  
higher rate of PVT after LSG even when compared to patients after the duodenal switch  
95 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  
100 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.

## **METHOD**

105 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<sup>2</sup> (range: 42-50). In our series, there were four documented cases of PVT after LSG. The fifth case of PVT after LSG was referred from another center. The patient's demographics are seen in table 1. All four cases had been performed in a standardized manner by two of four  
110 surgeons in our group (BMI). 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 5,000 units subcutaneously every 8 hours and Sequential compression device (SCD) hose. All patient underwent an uncomplicated LSG in reverse Trendelenburg with carbon dioxide insufflation  
115 pressure less than 15 mmHg. Each operation was performed over a 36 French bougie catheter, beginning the sleeve 5 centimeters proximal to the pylorus. The patients received postoperative fluids to maintain a urine output greater than 60 mL/hour. Early ambulation was required. Patients were discharged by the second postoperative day taking adequate oral fluids (greater than 2 liters per day).

120 The patients with PVT presented to the emergency room 10 to 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 CT scans using I.V. 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  
125 thrombus. Further scans were obtained if clinically indicated. All patients were initially treated with systemic therapeutic heparin (aPTT 2 to 3 time's 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

130 the portal venous system (intraclot infusion) (Fig. 1). Intraoperative ultrasound was used to guide  
the thrombectomy. A transabdominal 5 French catheter was placed in a tertiary mesenteric vein  
for uninterrupted postoperative TPA infusion followed by continuous heparin infusion. Two  
patients underwent bowel resection. The third option of catheter-directed TPA into the superior  
mesenteric artery (SMA) was not utilized in any of these patients, although this option could be  
135 considered in high grade non-occlusive 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.  
140 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. Blood work was normal. A CT  
145 scan demonstrated a nonocclusive PVT. The patient was placed on a therapeutic heparin drip  
with a target of aPTT 2 to 2.5 time's baseline (60 to 80 sec). The patient's symptoms resolved  
within 1 day of starting the heparin drip. A subsequent CT scan showed clot regression. The  
patient was discharged on therapeutic rivaroxaban. He has had no symptom recurrence after 10  
months follow-up.

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**CASE 2:** On POD #10, the patient was seen in the emergency room with malaise, severe nausea, mid-abdominal pain and tachycardia. Her WBC was 10,700/mm<sup>3</sup>. Her temperature was normal. A CT scan showed 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  
155 days later, a CTA showed 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 INR on day 6.

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

**CASE 3:** The patient was admitted 32 days post-LSG with malaise, dysphagia, severe nausea,  
165 vomiting, abdominal pain and tachycardia. The WBC was 16,000/mm<sup>3</sup>, and the serum lactate was 4.0 mmol/L. His temperature was normal. A CT scan with I.V. contrast showed partial PV and complete SMV obstruction, ascites, and probable ischemic bowel. The patient was operated emergently. Portal-SMV thrombectomy was accomplished, guided by intraoperative ultrasound (Fig. 2 a & b) 40 cm of necrotic bowel were resected, but not reanastomosed. Marginal bowel  
170 was left, pending a second-look procedure. A 5-French 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  
175 noted and the nonviable bowel resected. Bowel continuity was reestablished. An intraoperative  
ultrasound demonstrated 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 with greater than 1 year of follow up.

180 **CASE 4:** The patient presented 2 weeks post-LSG to the emergency room, complaining of  
malaise, nausea and mid abdominal pain. Her WBC was 13,000/mm<sup>3</sup>. Her temperature was  
normal. Other blood work was unremarkable except for an ALT of 180. The CT scan  
demonstrated extensive occlusion of the portal venous system, including intrahepatic left and  
right branches, PV, SMV, SV, as well as ascites and bowel wall thickening.

185 The patient was heparinized, 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 5 French  
190 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  
demonstrated a patent PV with excellent flow. During the night, the heparin drip intended for the  
mesenteric portal catheter was instead given systemically. A CTA demonstrated re-thrombosis.  
The patient was returned to the operating room and, again, underwent thrombectomy with TPA  
195 and heparin infusion. The final ultrasound showed robust hepatopetal flow. The following day, a  
CTA demonstrated 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 with 9 months of follow-up.

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**CASE 5:** This patient underwent LSG at another institution. The surgical technique included sizing with a 38 French bougie and 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 showed 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.

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## **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 less than 1%, with a range of 0.3 to 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 also do patients after LAGBP and after

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duodenal switch, each procedure without a reported increased risk of PVT. One patient in our  
220 series complained of thirst over 4 weeks prior to the diagnosis of PVT (patient #5). 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  
225 from the Netherlands, obesity conferred a two-fold risk of venous thromboembolism (15). A  
prospective cohort study from Vienna showed a linear relationship between increased body  
weight and venous thromboembolism (16). Obesity predisposes to venous thrombosis by  
reduction of fibrinolysis, elevation of clotting factor levels and release of proinflammatory  
mediators (17). All operations, as well as all hospitalizations, are prothrombotic events (18).  
230 However, the patients in this series had laparoscopic procedures requiring less than 35 mins of  
operative time and hospitalizations less than 2 days. Operative conditions included 15 mmHg of  
carbon dioxide insufflation pressure and reverse Trendelenburg position. Hypercapnia may cause  
mesenteric vasospasm (19-21). All of these conditions could reduce portal flow; but these same  
conditions exist for many other laparoscopic procedures not associated with an increased PVT  
235 risk (22).

The patients in our series received DVT prophylaxis including SCD 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 (23, 24). Although patients  
usually had normal vital signs, serum amylase, liver function tests, and lactate levels, the WBC  
240 was increased in 4 of 5 patients. A CT scan with I.V. contrast is indicated in all such patients  
post-LSG (25, 26). 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 categorized as non-occlusive, high-grade nonocclusive, or occlusive thrombosis (Fig 4). In addition, the extent of the thrombus and the condition of the patient should  
245 be considered in making a therapeutic choice. In stable non-cirrhotic patients with non-occlusive 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  
250 alone. Patients with occlusive disease were treated with operative thrombectomy including intraoperative and post-operative mesenteric thrombolysis (TPA) with subsequent therapeutic anticoagulation (heparin). Anticoagulation alone is not sufficient treatment in occlusive disease as demonstrated by case 5 and limited prior literature (11). Occasionally, high-grade partial occlusion does not properly respond, as demonstrated by case 2. Case 2 would have been better  
255 treated, in retrospect, with SMA 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. The futility of clot dissolution in occlusive venous thrombosis, including PVT using heparin anticoagulation is, however, well documented.  
260 Anticoagulation in patients with peripheral DVT was effective in less than 5% of patients treated with this modality alone, whereas thrombolytic therapy resulted in complete clot lysis in 45% and partial clot lysis in 65% (27, 28). Multiple other series demonstrate the high failure rate of anticoagulation alone in occlusive venous thrombosis (29, 30). Recanalization of the portal vein usually does not occur in noncirrhotic patients (11). In partially occluded vessels, heparin does

265 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 (31-35).

270 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 (36-39).

There are only two case reports of portal vein thrombectomy in the literature. One of these surgeons also placed a portal catheter for direct mesenteric anticoagulation (40). 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. Also, the amount of TPA and heparin infusion can 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 prior to systemic endothelial heparin binding and clearance of the administered heparin (41).

280 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<sup>1</sup>.

## **CONCLUSION**

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. 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 (SMA) 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**

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<sup>1</sup>In addition to the cases presented in the series, one of us (Dr. L. Belnap) has had an 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.

I certify that the manuscript did not involve the use of animal or human subjects.

Since this is a retrospective study: the formal consent is not required for this type of study.

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## Figure Legends

Figure 1: Hand drawn sketch showing step by step technique of portal vein thrombectomy

Figure 2: a) Duplex US: Thrombus in the Superior Mesenteric Vein

410 b) Duplex US: Thrombus extending into right portal vein

Figure 3: a) Occlusive thrombus of proximal portal vein extending into the R & L portal bifurcation

b) Proximal portal vein thrombus- note R & L portal vein branch extension. The thinner clot  
415 tracked down from the superior mesenteric vein

c) Fogarty catheter passing into the intrahepatic R & L portal branches

d) Repair of portal venotomy

Figure 4: Proposed algorithm for the treatment of portal vein thrombosis

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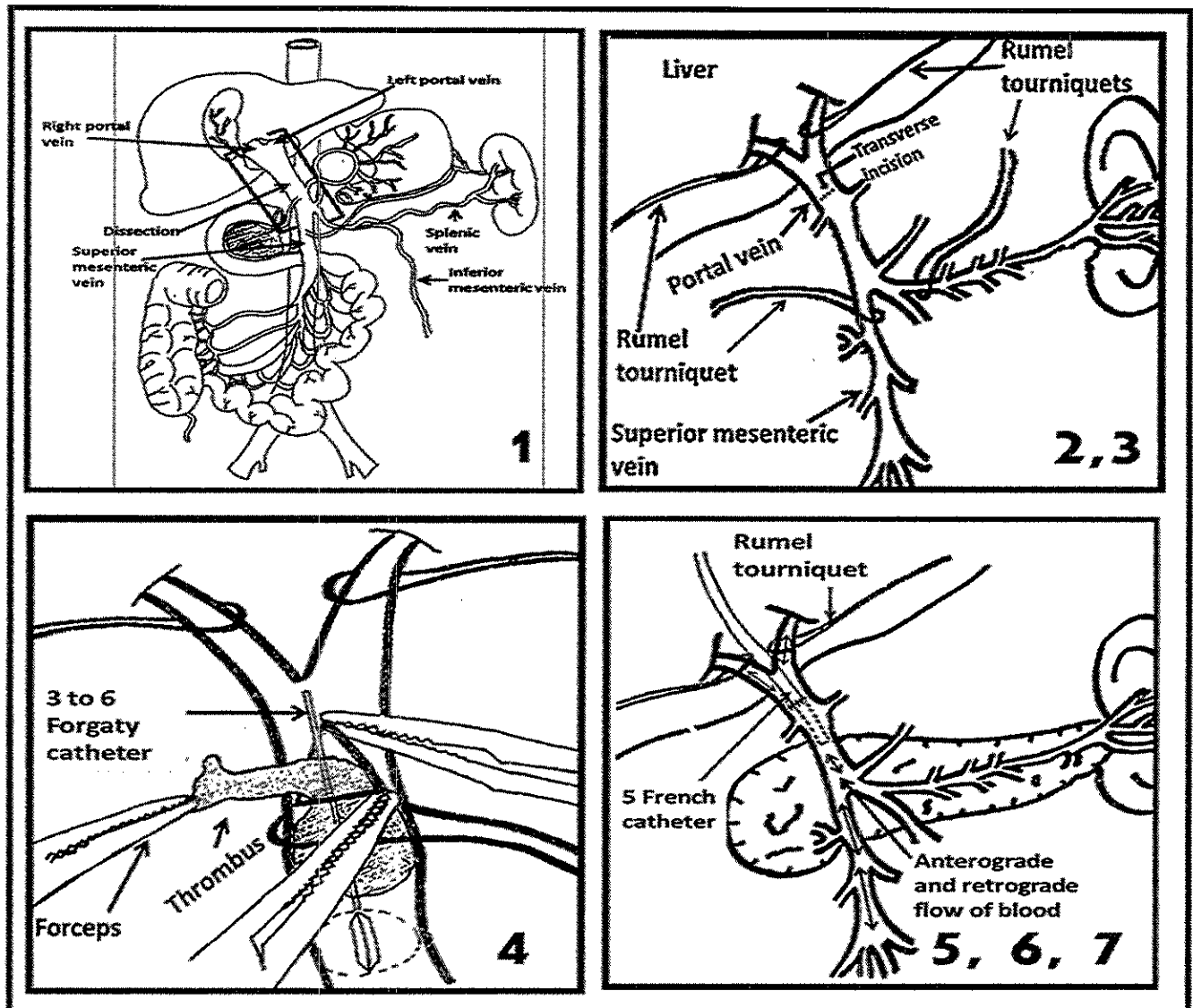
**Table 1: Patients demographics**

Patient number	Age	Gender	BMI (kg/m <sup>2</sup> )	Co-morbidities	Prothrombotic Medications	Operative time (mins)	Length of stay (days)
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	-	-	-	-

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

Abbreviation: OA= Osteoarthritis, GERD= gastroesophageal reflux disease, OSA= obstructive sleep apnea, MI= myocardial infarction, CABG= coronary artery bypass grafting, DVT= deep vein thrombosis, HTN= hypertension

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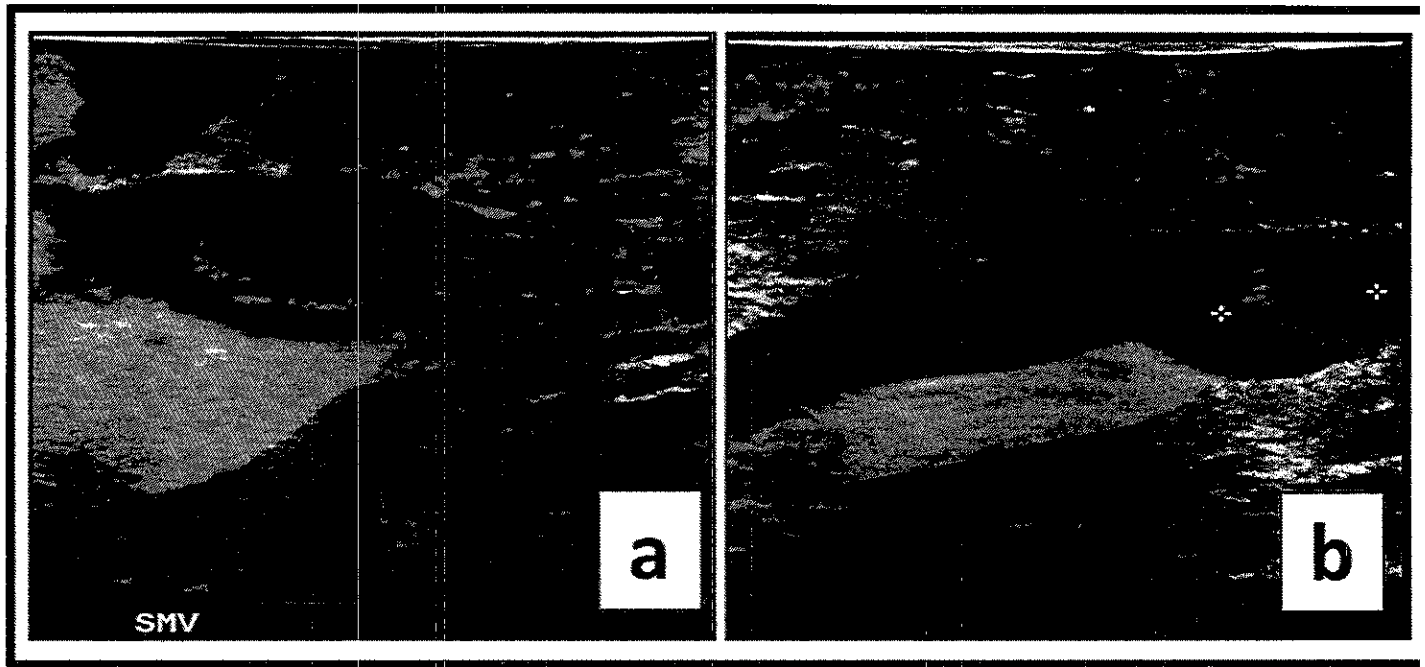


1. The portal vein from the right and left hepatic branches down to the superior mesenteric vein segmental vessels is dissected. The splenic vein is mobilized.
2. Rumel tourniquets were placed around the proximal portal vein, superior mesenteric vein and splenic vein.
3. The portal vein is opened transversely to decrease narrowing at any point [Venotomy].
4. Thrombectomy is performed with forceps and Forgaty catheter [number 3 to number 6].
5. A 5 French catheter is placed in a tertiary mesenteric portal vein.
6. First TPA and then heparin are infused during and after Thrombectomy.
7. Anterograde and retrograde flow must be established.



Amit Surve, M.D.

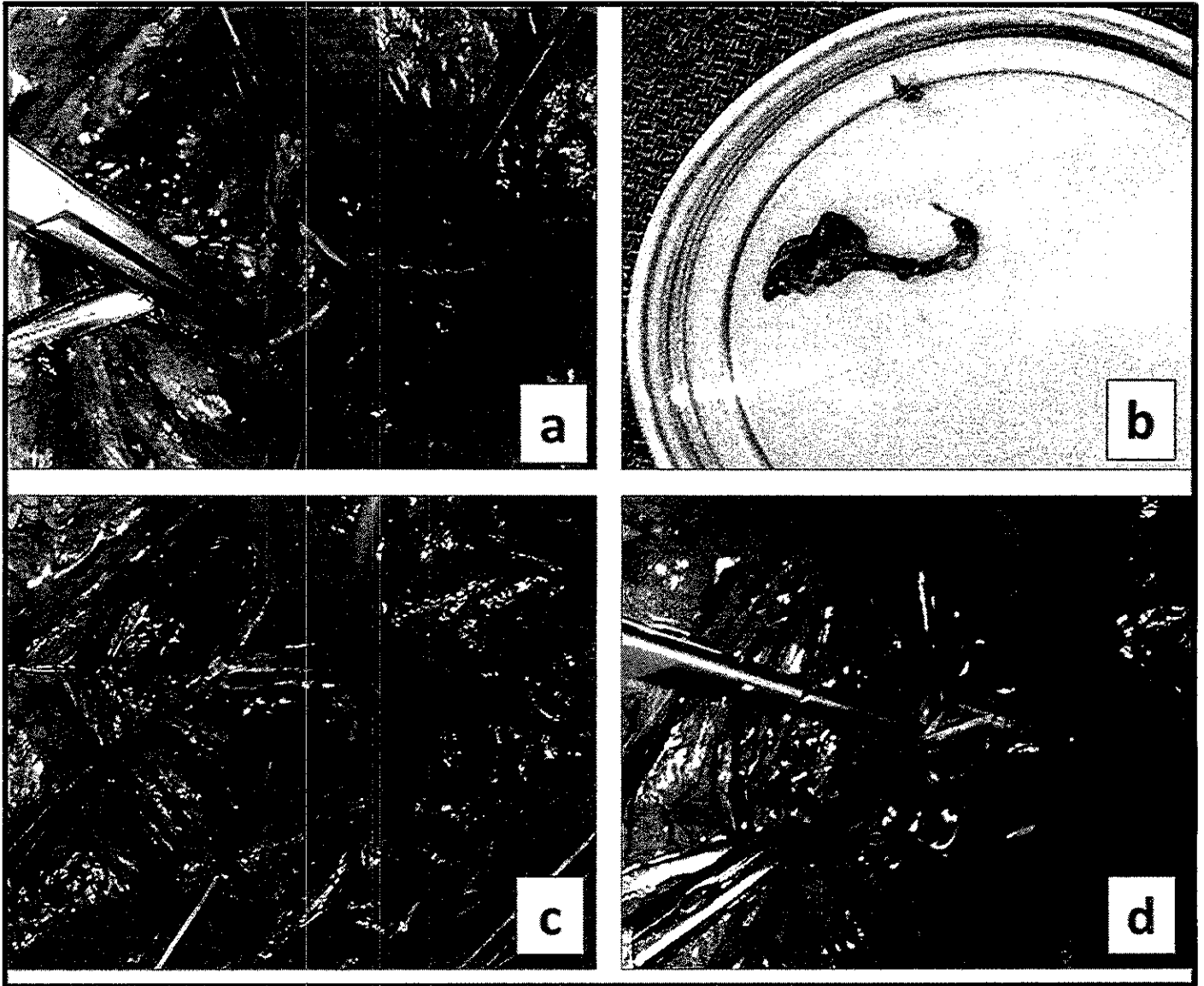
**Figure 1: Hand drawn sketch demonstrating step by step technique of portal vein thrombectomy**



**Figure 2**

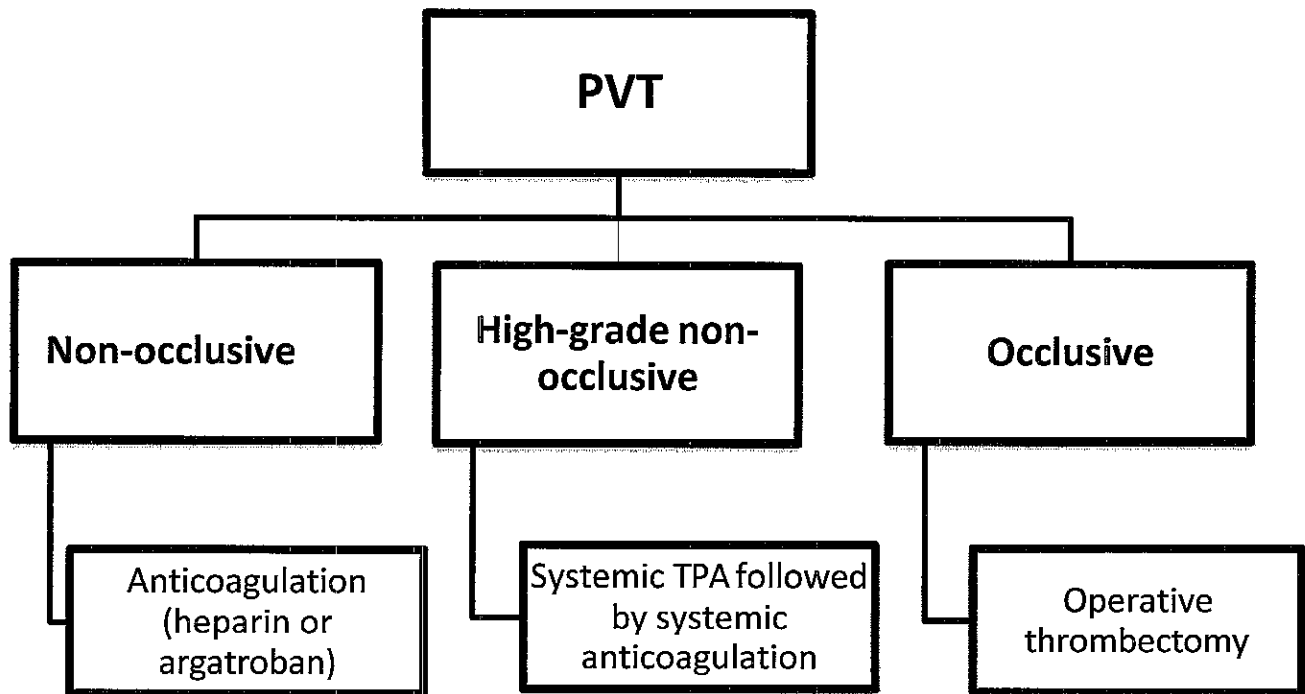
**a) Duplex US: Thrombus in the Superior Mesenteric Vein**

**b) Duplex US: Thrombus extending into right portal vein**



**Figure 3:**

- a) Occlusive thrombus of proximal portal vein extending into the R & L portal bifurcation
- b) Proximal portal vein thrombus- note R & L portal vein branch extension. The thinner clot tracked down from the superior mesenteric vein
- c) Fogarty catheter passing into the intrahepatic R & L portal branches
- d) Repair of portal venotomy



**Figure 4: Proposed algorithm for the treatment of portal vein thrombosis**

Abbreviations: PVT=Portal vein thrombosis, TPA=tissue plasminogen activator