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Extensive Portomesenteric Venous Thrombosis after Laparoscopic Sleeve Gastrectomy, Treated with Catheter-Directed Lysis

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Background	A 46-year-old morbidly obese African American female presented 13 days postlaparoscopic sleeve gastrectomy with extensive portomesenteric venous thrombosis.
Summary	The patient presented with abdominal pain that she described as central, sharp, and intense. Computed tomography (CT) of the abdomen and pelvis were suggestive of extensive portal vein thrombosis with involvement of the superior mesenteric vein that was confirmed with a follow-up ultrasound doppler study. Treatment was coordinated between general surgery and interventional radiology. The patient received catheter-directed thrombolysis via the superior mesenteric artery for 18 hours, after which she was followed closely in the intermediate care unit for four days. Following lysis therapy, the patient was discharged on enoxaparin. Later hematological workup revealed no underlying coagulopathy. A rare complication of bariatric surgery, portomesenteric venous thrombosis has been shown to occur following laparoscopic sleeve gastrectomy more so than other bariatric procedures.
Conclusion	The risk of deep venous thrombosis continues well into the postoperative period for some bariatric patients. There is minimal level I evidence to support screening risk factors for those without a personal or family history of previous thrombotic event or to recommend a dose or duration of therapy for continuing prophylactic anticoagulation post-discharge. Future prospective controlled trials may help identify those patients who would benefit most from extended thromboprophylaxis.
Keywords	Porto-mesenteric venous thrombosis; laparoscopic sleeve gastrectomy

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Case Description

Laparoscopic sleeve gastrectomy (LSG) is one of the most common bariatric procedures performed. With concurrent nutrition and exercise education programs, patients in the majority of cases experience drastic weight loss and health benefits. However, morbid obesity is an important risk factor for postoperative venous thromboembolism (VTE). It is generally agreed upon that perioperative prophylaxis against thrombotic complications is imperative for these patients. The current American Society for Metabolic and Bariatric Surgery (ASMBS) guidelines regarding VTE prophylaxis recommend that all bariatric patients receive mechanical prophylaxis and are ordered for early ambulation.

Although all bariatric patients are at risk for developing VTE, this is a relatively uncommon complication. Frequently reported complications of LSG include staple line leak and hemoperitoneum. During the procedure, dye is injected into the stomach to assess for staple line leakage. At the end of the procedure, the viscera are examined for sites of active bleeding. The authors present a rare case of postoperative portomesenteric venous thrombosis (PMVT) that occurred after LSG in a patient with a negative coagulopathy panel.

A 46-year-old morbidly obese African American woman presented with abdominal pain 13 days postlaparoscopic sleeve gastrectomy. Her pain was central and sharp in quality. She denied nausea, vomiting, or change in bowel habits. She also denied fever, chills, or dysuria. She was afebrile, hemodynamically stable, and her body mass index was 42.92. She had diffuse tenderness to deep palpation but did not exhibit peritoneal signs on exam. Labs were pertinent for mild leukocytosis, but electrolytes, blood urea nitrogen, and creatinine were within normal limits. Computed tomography (CT) of the abdomen and pelvis (Figure 1 and Figure 2) were suggestive of extensive portal vein thrombosis (PVT) that was confirmed with a follow-up ultrasound doppler study showing no flow in both branches as well as in the main portal vein (Figure 3 and Figure 4).

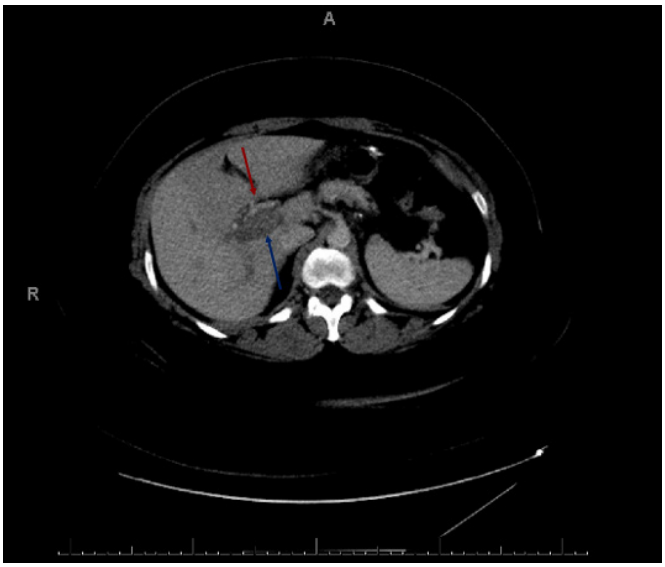

Figure 1. Axial CT abdomen/pelvis with oral and intravenous contrast showing opacification of the hepatic artery (red arrow) and unopacification and distension of the main portal vein (blue arrow), suggestive of an acute thrombus.

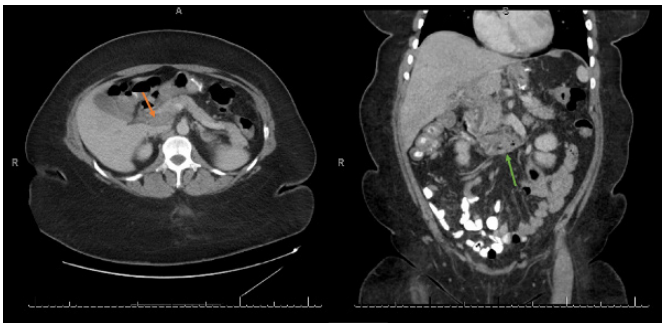

Figure 2. Axial and Coronal CT abdomen/pelvis with oral & intravenous contrast showing hypoattenuation of the main portal vein (orange arrow), intrahepatic portal veins, and involvement of the superior mesenteric vein (green arrow).

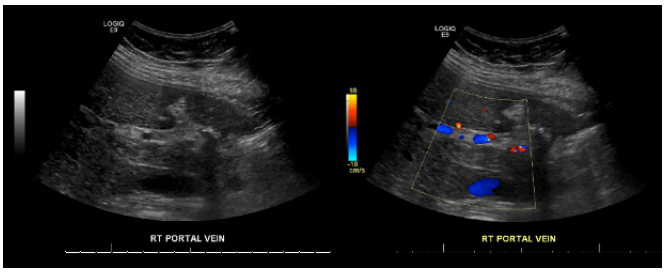

Figure 3. Echogenic intraluminal material in the right portal vein with no flow on color-flow doppler.

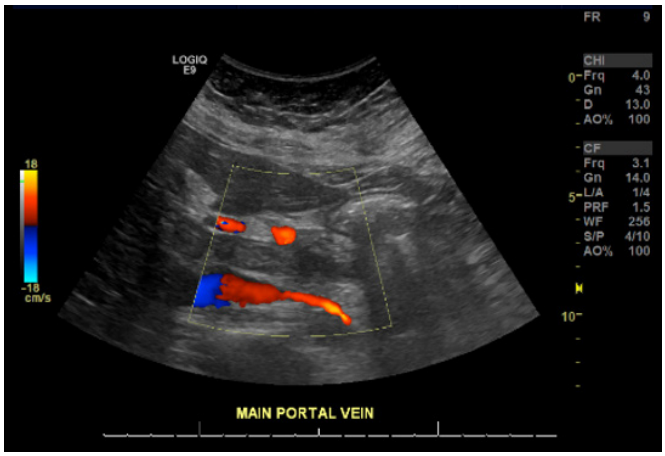

Figure 4. Color-flow doppler of main portal vein showing no flow.

Heparin was promptly initiated at 80 units/kg intravenously, and the decision to proceed with catheter-directed thrombolytic therapy was made with interventional radiology. Delayed mesenteric angiogram failed to opacify the portal venous system, consistent with extensive PVT, and a catheter was then placed percutaneously into the superior mesenteric artery via the femoral artery for tissue plasminogen activator infusion. The patient underwent 18 hours of thrombolysis, during which she was followed closely in the intermediate care unit. Follow-up CT scan four days later showed stable thrombus without signs of ischemia. The patient remained hemodynamically stable and showed no clinical signs of intestinal necrosis or embolism throughout admission. The patient was subsequently discharged on enoxaparin 1 mg/kg subcutaneously twice a day and scheduled for outpatient follow-up in six months to repeat imaging studies to determine resolution of thrombus and therefore determine necessity of continuing anticoagulation indefinitely and/or transitioning to novel oral anticoagulants. Later hematological workup (Table 1) revealed no underlying coagulopathy and only showed a mildly elevated apolipoprotein A.

Twenty days following thrombolysis, the patient underwent magnetic resonance cholangiopancreatography (MRCP) for reasons unrelated to this case, and the results showed persistent thrombus in the main portal vein (Figure 5).

Thrombophilia workup results	
Lipoprotein A	elevated (198 nmol) (increases prothrombotic fibrin clot phenotype)
Hb electrophoresis	Normal/Undetected
Lupus anticoagulant	
Protein C	
Protein S	
G20210A mutation	
Factor V Leiden	
BCR/ABL mutation	
JAK2 mutation	
ANA	
Rheumatoid Factor	

Table 1. Results of a thrombophilia workup to rule out a likely underlying coagulopathy were only positive for a mildly elevated lipoprotein A.

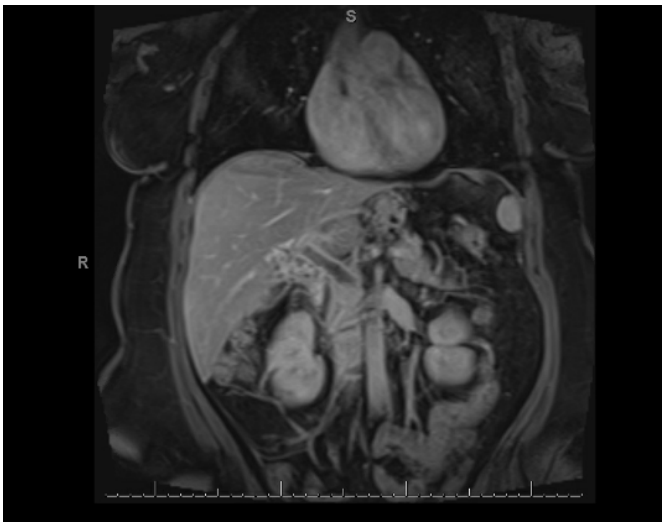

Figure 5. MRCP showing stable thrombus in the main portal vein (arrow).

At the patient's scheduled six-month follow-up for repeat imaging, she was unfortunately found to have portal cavernous transformation on CT (Figure 6). This is a consequence of portal hypertension, suggesting that partial recanalization seems to have been achieved, enough to allow venous outflow from the mesentery; however, a chronic thrombus had nonetheless developed and created a state of chronic portal hypertension. The patient will now be monitored for the clinical sequelae of portal hypertension and carefully screened for masked malignancies.

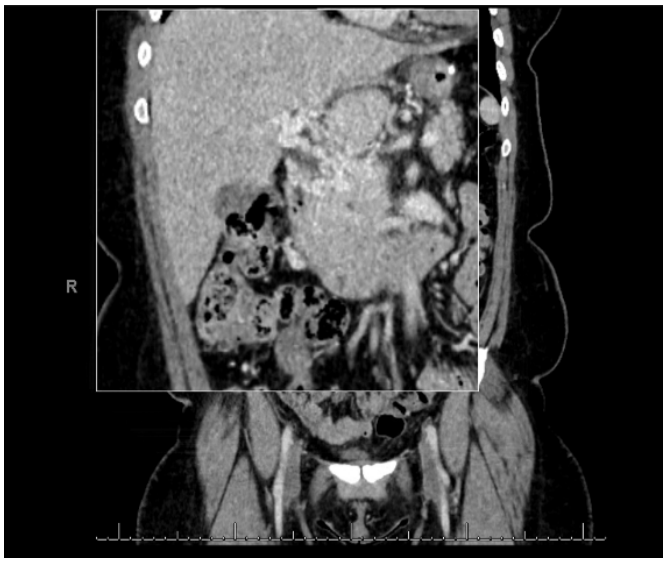

Figure 6. Repeat CT at six-month follow-up shows serpiginous vessels highly suggestive of portal cavernoma.

Discussion

The incidence of PVT after laparoscopic sleeve gastrectomy has been reported to be between 0.3 and 1 percent.[1,2] PVT occurs more commonly after LSG than any other bariatric procedure.[1–4] There are only a few reported cases of PMVT following bariatric surgery in the existing literature[5–13]; the first LSG-related PMVT was reported by Berthet et al. in 2009.[14] The majority of patients who do develop PVT following bariatric surgery have a positive thrombophilia study with evidence of some genetic coagulopathy.[15–17] However, morbid obesity in and of itself is a predisposing factor for post-operative venous thrombosis. According to the Modified Caprini Risk Assessment tool for assessment of VTE risk, virtually all bariatric patients are at moderate risk (three percent risk without mechanical or chemical prophylaxis) for VTE.[18] To mitigate this risk, the journal *Surgery for Obesity and Related Diseases* recommend unfractionated heparin or low-molecular weight heparin preoperatively plus the use of sequential compression devices during the recovery period.[19] The patient received these precautions. However, the guidelines are unclear about anticoagulation after patients are discharged from the hospital.

Multiple suggestions have been offered to explain the pathogenesis of PMVT preferentially following LSG as opposed to other laparoscopic bariatric procedures. These include (1) splenic ischemia from ligation of the short gastric vessels; (2) endothelial damage from thermal effect on the left gastroepiploic vessel during skeletonization of the greater curvature; and (3) hepato-splanchnic congestion from liver retraction for a prolonged period of time.[1–3,15,20] Furthermore, the author noted that ligation of the short gastric veins as well as skeletonization of the greater curvature is expected to result in (1) a reduction in the respective pressure differentials between the splenic vein to the portal vein and gastro-epiploic vein to the portal vein in the direction of venous flow as well as (2) an increase in resistance, thereby slowing venous flow according to Ohm's law (flow = pressure differential/total resistance). In a patient undergoing laparoscopic surgery already being exposed to endothelial damage by thermal ligation, an underlying genetic coagulopathy completes Virchow's triad for thrombosis: flow stasis, endothelial damage, and hypercoagulability.

The treatment of PMVT in our patient required careful consideration of three management options: conservative anticoagulation therapy, catheter-directed thrombolysis, and thrombectomy.[21–23] Without prompt initial anticoagulation, risks include intestinal ischemia, infarction, and ultimately necrosis and/or acute pylephlebitis.[24] Asymptomatic PVT may remain undetected until it becomes chronic and causes portal hypertension evidenced by the development of venous collaterals, or "cavernous portal transformation." Thrombectomy is generally reserved for severe cases that necessitate taking the patient back to the operating room (intestinal necrosis).

Catheter-directed thrombolysis has been emerging as a practical option to recanalization of acute PVT without intestinal infarction.[22,23] Thrombolysis can be performed percutaneously through various approaches: transjugular, transhepatic, transsplenic, transileocolic, and omental vein.[25–28] In the transjugular approach, the portal vein is accessed from one of the hepatic veins or the inferior vena cava. Transhepatic access carries an increased risk of bleeding in the setting of thrombolytic therapy. The transileocolic approach requires surgical access. For this patient with evidence suggestive of diffuse mesenteric venous

Table 2. Cases of appendicitis resulting in lower extremity abscess

thrombosis, indirect access through the superior mesenteric artery via the common femoral artery was the preferred approach.[29]

Recanalization for the patient was successfully achieved by catheter-directed lysis with tissue plasminogen activator, and similar successful lysis of acute PVT has been reported elsewhere.[30,31] However, given that the patient did not develop ischemia but did develop portal cavernous transformation at six months, there is a question of whether enough recanalization was achieved. This patient will now need to be followed closely to screen for varices or other consequences of portal hypertension as well as malignant transformations. Further exploration of this management approach with controlled prospective trials are needed to determine its efficacy in comparison to therapeutic low-molecular weight heparin alone.

Furthermore, this case invokes the utility of VTE prophylaxis, defined as prophylactic anticoagulation for one to four weeks postoperatively. The patient's clot developed well into the postoperative period, and although there seems to be a consensus on preventing perioperative thrombosis, there is minimal level I evidence to recommend a dose or duration of therapy for extended VTE prophylaxis after discharge.[2,19,32] The patient had no history of previous thrombotic event, no known risk factors, and did not have a previously diagnosed coagulopathy; she was therefore not given postdischarge prophylaxis. Thrombophilia workup was only positive for a mild elevation in apolipoprotein A that has been associated with prothrombotic fibrin clot phenotype contributing to arterial disease[33] but has not been proven to increase venous thrombosis risk.[34,35]

For this patient, the authors recommended long-term anticoagulation with follow-up imaging to avoid significantly increasing her risk for major bleeding. The rate of major bleeding with indefinite anticoagulation was estimated to be 2.7 per 100 patient-years in one meta-analysis.[36] In general, it is not recommended to initiate indefinite anticoagulation in patients with a first-time provoked VTE due to a transient risk factor such as surgery. For such patients, the VTE recurrence risk without anticoagulation is estimated to be one percent at one year and three percent at five years.[37–39] Current evidence suggests it is generally acceptable to anti-coagulate for three to six months and repeat imaging studies in a patient similarly at minimal risk for major bleeding and does not have indications for lifelong therapy (e.g., genetic coagulopathy, recurrent VTE, unprovoked VTE).[19,21]

Conclusion

Virtually all bariatric patients are at least at moderate risk for developing venous thrombosis during the perioperative period. As the symptoms of portomesenteric venous thrombosis are nonspecific, a high index of suspicion is necessary for diagnosis during the extended postoperative period. This case highlights the need for prospective controlled trials to identify those patients who might benefit from extended VTE prophylaxis, and to study the efficacy of catheter-directed thrombolysis.

Lessons Learned

Postoperative portomesenteric venous thrombosis is a rare complication associated with laparoscopic sleeve gastrectomy, more so than with other bariatric procedures. There is currently minimal evidence to recommend which patients may benefit from continuing chemical thromboprophylaxis postdischarge to prevent this condition. A high index of suspicion is necessary for diagnosis as the symptoms are nonspecific.

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