CASE REPORT JSLS

Portal Vein Thrombosis Following Laparoscopic Sleeve Gastrectomy for Morbid Obesity

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ABSTRACT

Introduction: Portal vein thrombosis has been documented after laparoscopic general surgery and has been uncommonly observed after laparoscopic bariatric surgery. Among bariatric operations, the sleeve gastrectomy is being performed with ever-increasing frequency. Here we report the case of a man who presented with portal vein thrombosis after laparoscopic sleeve gastrectomy.

Case Description: A 41-y-old man underwent an uneventful laparoscopic sleeve gastrectomy for the treatment of morbid obesity, and presented on postoperative day 10 with nonfocal abdominal pain, nausea, vomiting, and leukocytosis. Computed tomography revealed portal vein thrombosis, which was found in the setting of *Clostridium difficile* colitis.

Discussion: Portal vein thrombosis may be identified with increasing frequency as the number of laparoscopic bariatric operations continues to increase. A high index of suspicion is necessary to diagnose this rare, but potentially lethal, complication.

Key Words: Bariatric surgery, Portal Vein thrombosis, Sleeve gastrectomy, Complications.

INTRODUCTION

Portal venous thrombosis (PVT) is a potentially life-threatening condition with multiple etiologies, both local and systemic. Important local predisposing factors include cirrhosis (especially in the setting of hepatic dysfunction), infection, inflammation (such as diverticulitis, pancreatitis, cholecystitis, inflammatory bowel disease), and malignancy (hepatocellular, gastric, pancreatic cancers). Systemic factors include genetic deficiencies affecting coagulation homeostasis, and acquired conditions, such as myeloproliferative disorders, pregnancy, and oral contraceptives.[1] Postoperative PVT has been known to occur following procedures that involve manipulation of major portomesenteric veins, such as splenectomy.[2] However, with the wide use of laparoscopic techniques in general surgery over the past 20 y, reports have emerged of PVT, absent of direct manipulation of the portal or major mesenteric vessels. Included among the laparoscopic procedures with reported postoperative PVT are resections of the appendix, gallbladder, and colon, as well as Nissen fundoplication for gastroesophageal reflux disease, and bariatric surgery for morbid obesity.[3–8] Although the cause of PVT after laparoscopy is unknown, it is established that increased intraabdominal pressure with pneumoperitoneum results in decreased portal venous blood flow, which may lead to a relative prothrombotic environment.[9,10]

The laparoscopic sleeve gastrectomy, first described as a modification of the biliopancreatic diversion, is emerging as a popular single-stage operation for the treatment of morbid obesity, with acceptable morbidity and long-term weight loss, as compared with the Roux-en-Y gastric bypass.[11–13] To our knowledge, this is the second report in the literature of a PVT after an uneventful laparoscopic sleeve gastrectomy in a morbidly obese man.

CASE REPORT

Of 55 patients who underwent laparoscopic sleeve gastrectomy for morbid obesity at our institution, one developed acute PVT in the early postoperative period. He was a 41-y-old man with a body mass index of 42 kg/m^2 and associated comorbidities including hypertension, hyper-

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lipidemia, and obstructive sleep apnea. After an extensive multidisciplinary, he presented for a laparoscopic sleeve gastrectomy. The patient was given a single intravenous dose of a first-generation cephalosporin and 5,000U of heparin subcutaneously prior to incision. The operation was performed using a 5-port technique. The greater curvature was mobilized using a LigaSure device (Valleylab, North Haven, CT), and the gastric sleeve was created using the Echelon stapler (Johnson and Johnson, New Brunswick, NJ) buttressed with SeamGuard (Gore, Flagstaff, AZ). A 36-Fr endoluminal endoscope was used to size the lumen and perform a leak test intraoperatively, and a Jackson Pratt drain was placed along the staple line at the conclusion of the operation. The procedure lasted 100 min and was performed under a carbon dioxide insufflation pressure of 15 mm Hg. During dissection of the lesser sac and posterior stomach, the portomesenteric circulation was not visualized or manipulated.

The patient had an unremarkable postoperative course; no additional antibiotics were administered. Water was introduced orally on postoperative day 1 after an upper gastrointestinal contrast study demonstrated no leak from the staple line. On postoperative day 2, oral caloric liquids were introduced, as per our protocol. He was discharged to home on postoperative day 4 with a percutaneous drain, due to persistent serous drainage > 150 mL daily. Amylase levels were checked in the drainage fluid and were found to be normal. Drainage progressively decreased to minimal, and the drain was removed on postoperative day 6. Throughout his hospital course he was treated with subcutaneous injection of heparin 5,000U, 3 times daily. He was ambulatory and was discharged to home without venous thrombosis prophylaxis.

The patient was readmitted on postoperative day 10 with a 2-d history of nausea, vomiting, diarrhea, and diffuse abdominal pain. A computed tomography (CT) scan with oral and intravenous contrast demonstrated an intact gastrectomy, ascites, and PVT **(Figure 1)**. No intestinal ischemia was present. Laboratory tests revealed a leukocytosis to 20,000/mm^3, mildly elevated liver function tests, and positive fecal *Clostridium difficile* toxin. Upon diagnosis he was immediately treated with oral Vancomycin and therapeutic infusion of heparin. Clinical examination and laboratory tests improved, and he was sent home on a soft solid diet, warfarin, and antibiotics. Three months later, he was seen in follow-up. He was feeling very well, follow-

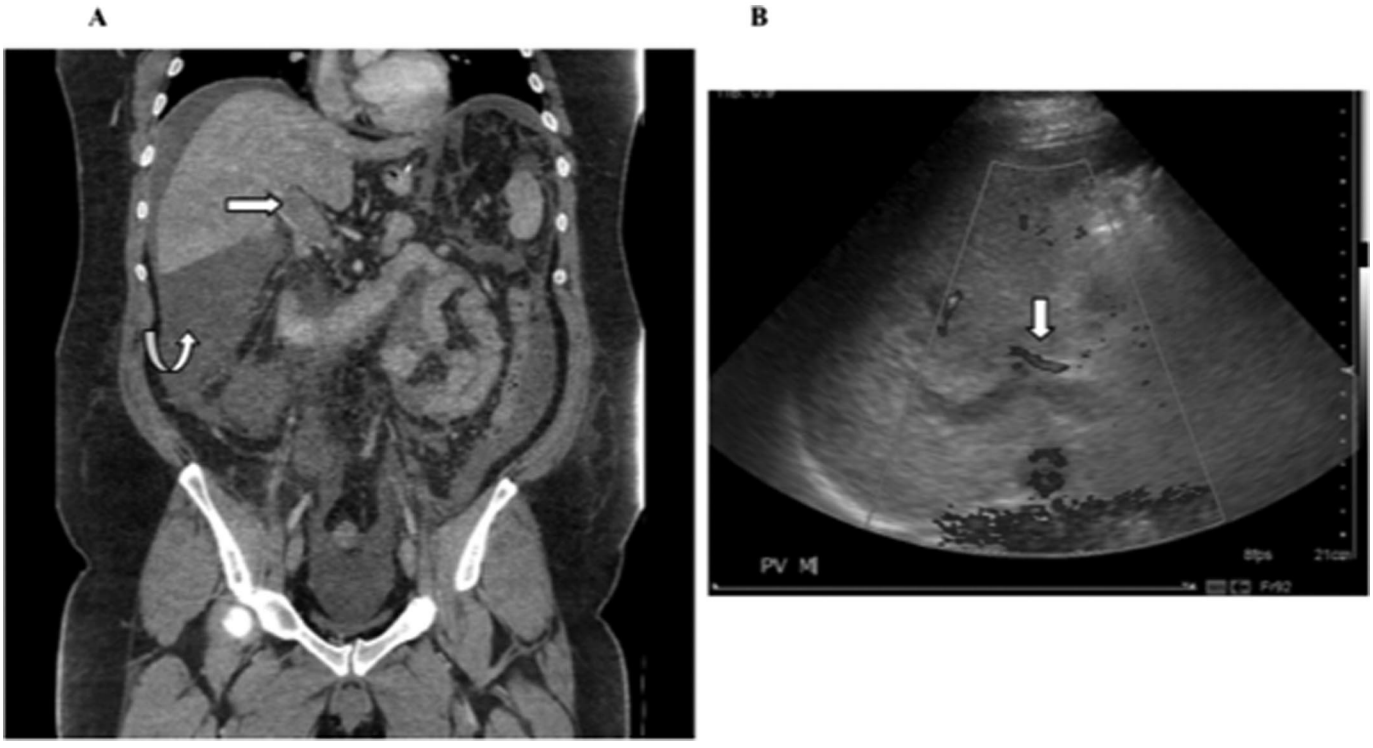

Figure 1. (A) Contrast-enhanced coronal reconstructed computed tomographic image demonstrating PVT (arrow) and ascites (curved arrow). (B) Doppler ultrasound demonstrating no flow in the portal vein. Flow is seen in the adjacent hepatic artery (arrow).

ing the diet as per our protocol, and exercising regularly. At 6 mo postoperatively he will undergo a repeat CT scan to evaluate the resolution of the PVT.

DISCUSSION

Portal vein thrombosis is a well-described, infrequent complication of operations that involve the portal or mesenteric veins. It is a rare complication of laparoscopic general surgery that may be potentially life threatening due to mesenteric ischemia or infarction.[3–7] In a literature search, spanning a 17-y time period, James et al.[3] identified a total of 18 case reports of portomesenteric vein thrombosis after laparoscopic surgery, excluding operations involving splenectomy.

Case reports of PVT in the bariatric population have emerged as the prevalence of laparoscopic weight loss surgery has increased over the past decade. Portomesenteric thrombosis has been noted after laparoscopic Roux-en-Y gastric bypass and laparoscopic adjustable gastric banding.[14–19] While commonly reported complications after laparoscopic sleeve gastrectomy include staple-line leak, respiratory insufficiency, pulmonary embolism, hemorrhage, stricture, and splenic injury,[11] to our knowledge this is the second known report of a PVT after sleeve gastrectomy.

Clinical presentation may be subtle and requires a high index of suspicion. In retrospect, larger than expected drainage fluid in the postoperative period in this case may have represented early ascites and PVT in our patient. Most commonly, patients present approximately 2 wk postoperatively with nonfocal abdominal pain, nausea, vomiting, and low-grade fever.[1,3] Laboratory values are typically within normal limits; however, leukocytosis and mild elevation of liver function tests can also be seen. Thus, physical examination findings can be normal, or alternatively, if associated with bowel ischemia, patients could present with peritonitis and septic shock. In this case, our patient returned to the hospital with a typical clinical presentation of PVT. However, this presentation is nonspecific, especially since vague abdominal pain, nausea, and vomiting can also be seen for a few weeks in the normal postoperative course of sleeve gastrectomy. In addition, our patient presented with ascites, which was identified on CT. Ascites is found in approximately 1/3 of patients who present with PVT, and may be associated with delayed initiation of anticoagulation treatment or thrombus resistant to recanalization.[20]

In noncirrhotic patients, the etiology of PVT is generally divided into local and systemic causes.[1] Local surgical factors known to predispose to venous thrombosis include direct surgical manipulation of portomesenteric vessels, as in splenectomy. Portal vein thrombosis is known after splenectomy, where ligation of the splenic vein incites endothelial damage that in turn increases thrombogenicity of the vein. Whereas gastric bypass involves some transection of the gastric and mesenteric veins causing endothelial damage, the sleeve gastrectomy involves transection of the short gastric veins alone, and is thus less likely to cause PVT. In these cases, other causes of PVT are considered. Genetic coagulopathies that have been associated with PVT include factor V Leiden, the prothrombin G20210A mutation, protein C and S deficiency, antithrombin III deficiency, the homozygous MTHFR mutation, and hyperhomocysteinemia.[21] Our patient had no known prothrombotic hematologic condition and no family history suggesting a genetic coagulopathy.

Portal venous flow is affected by increased intraabdominal pressure with carbon dioxide pneumoperitoneum. It is unclear at what pressure this effect becomes clinically significant; however, it is suspected that a decrease in portal blood flow may contribute to a prothrombotic state in the portomesenteric circulation.[10,22] Controversy exists over how clinically relevant this is, as the changes in blood flow vary between patients, and pneumoperitoneum results in venous thrombosis so infrequently.[23,24] In addition, the steep reverse-Trendelenburg position during laparoscopic bariatric surgery may augment the flow effects of pneumoperitoneum. While bariatric surgery patients are often treated with routine pharmacologic deep vein thrombosis (DVT) prophylaxis, it is yet unclear what role routine pharmacologic DVT prophylaxis has in prevention of PVT.

The definitive diagnosis of PVT is made with noninvasive imagining. More invasive portal venography is typically not necessary. The diagnosis can be established with contrast-enhanced CT or color Doppler ultrasonography.[25,26] Our patient had both studies to confirm the diagnosis, prior to initiation of anticoagulation treatment.

Treatment of PVT is guided by the acuity of the disease and the underlying cause, if known. Therapeutic anticoagulation is recommended in stable, noncirrhotic patients with acute PVT. The suggested duration of treatment is 6 to 12 mo. For patients with known systemic prothrombotic states, treatment may be life long, with the goal of recanalization of the portal vein.[27] In addition, any underlying predisposing condition should be treated as well. We initiated treatment with unfractionated heparin, which was later transitioned to warfarin, immediately after the

diagnosis was made. In addition, treatment for *C. difficile* colitis was started concurrently. Symptoms gradually resolved over the course of 1 wk. It is feasible that the inflammatory response to the *C. difficile* infection contributed to the prothrombotic state. However, inflammatory bowel disease, rather than *C. difficile* colitis, has been associated with PVT.[28,29] We found a single report of mesenteric vein thrombosis (not PVT) in the setting of *C. difficile* colitis, in a patient who also had a urinary tract infection.[30] Clinical improvement in this patient was rapid following the initiation of systemic anticoagulation and specific antibiotic treatment. Follow-up may include imaging studies to confirm recanalization of the portal vein after 3 mo to 6 mo of treatment.

Although still a subject of debate, some studies suggest that the risk of thromboembolism following bariatric surgery extends long after discharge from the hospital, and prophylaxis should therefore be continued for several weeks into the postoperative period.[31] This aggressive approach, however, which could also theoretically lower the risk of PVT, is not considered standard.[32]

In the setting of persistent thrombosis, or with concerns for bowel ischemia, more aggressive therapy may include endovascular thrombolysis or percutaneous thrombectomy.[33,34] No large studies exist to support the routine use of thrombolytics; however, this treatment has been shown to be effective in cases resistant to standard anticoagulation therapy.[35] In addition, this treatment has to be weighed against the potential for life-threatening hemorrhage. In this case, there was no indication to initiate thrombolytic therapy.

As mentioned, acquired thrombophilias may also contribute to PVT, particularly in the bariatric population. Obesity is associated with an increased risk of thromboembolism, and consequently all bariatric surgery patients carry an increased risk.[36] Additionally, the postoperative bariatric patient on a stringent diet may be micronutrient deficient, and vitamin K deficiency in particular could lead to decreased protein C and S concentrations with an increased prothrombin time. Additional acquired thrombophilias including myeloproliferative disorders and antiphospholipid syndrome should be considered. In this case, the cause of PVT was unclear and likely multifactorial. The patient was morbidly obese, underwent laparoscopic surgery in a reverse-Trendelenburg position, and suffered from *C. difficile* colitis, all potential factors contributing to development of PVT in the early postoperative period. As the prevalence of bariatric surgery continues to increase, PVT may become an increasingly identified diagnosis.

CONCLUSION

Here we describe a patient who presented with PVT after laparoscopic sleeve gastrectomy. Portal vein thrombosis is an uncommon complication following laparoscopic bariatric surgery, with potentially life-threatening consequences. Presenting symptoms are often vague, and a high index of suspicion is required to obtain the proper noninvasive imaging studies to confirm the diagnosis and begin prompt treatment.

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