CASE REPORT

Portomesenteric vein thrombosis after laparoscopic sleeve gastrectomy for morbid obesity

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SUMMARY

Portomesenteric vein thrombosis (PMVT) is a rare, life-threatening pathology associated with increased prevalence in laparoscopic sleeve gastrectomy (LSG) versus other gastric bypass procedures. Gaining popularity, LSG is becoming the first choice of patients and physicians alike due to its low complication profile compared to the gastric bypass procedure. We present the case of a 34-year-old woman who underwent an uncomplicated LSG for the treatment of morbid obesity, and presented on postoperative day 13 with PMVT. The patient underwent mechanical thrombectomy and thrombolytic therapy. After 2 days, patency was restored and the patient was discharged in stable condition. A high index of suspicion for PMVT should be considered in patients reporting diffuse abdominal pain after LSG. Owing to its lethality, upon confirmation of PMVT, therapy should begin immediately along with extended anticoagulation therapy on discharge.

BACKGROUND

Portomesenteric vein thrombosis (PMVT) is a rare, but potentially life-threatening pathology, which may present after laparoscopic bariatric surgery.¹ Although the condition has been known to occur in the postoperative period after a laparoscopic Roux-en-Y gastric bypass and laparoscopic gastric banding procedures, PMVT has been noted with increased incidence in patients recovering from laparoscopic sleeve gastrectomy (LSG).² PMVT is rooted within multiple aetiologies, local and systemic.³ Patients who elect to undergo bariatric surgery, generally those who fall under the category of morbid obesity (MO) are more likely to be predisposed to metabolic syndrome, to begin with.⁴ These individuals present with underlying proinflammatory mediators and hypercoagulability, thereby further increasing predisposition to venous thromboembolism (VTE).⁵

Even with the advent of minimally invasive surgery, the presentation of PMVT within the past two decades has continued to emerge.⁶ Certain laparoscopic operations still report thrombotic events with regularity: appendectomy, cholecystectomy and colectomy, as well as Nissen fundoplication and LSG for MO. The cause of PMVT in these cases has yet to be confirmed, but recent analysis of thrombotic events post-LSG indicate specific contributions which may catalyse their occurrence post-operatively: increased portal venous blood pressure and decreased blood flow due to pneumoperitoneum, mechanical and thermal effect on splenic vasculature and vessels of the greater curvature of

the stomach, blood flow pattern alteration due to intraoperative ligation of the short gastric vessels, and restriction of liquid intake after surgery are all factors leading to thrombosis.^{1 3 4 6 7}

LSG has gained popularity as an effective weight-loss procedure for all age groups. Although it is a single-stage operation, the results of LSG are long-term and avoid potential nutritional deficits often liable to other gastric bypass procedures.⁸ Although a relatively rare complication, PMVT has lethal implications, which warrant vigilant patient observation following discharge. Here we report a severe case of PMVT presenting 13 days after LSG.

CASE PRESENTATION

A 34 year-old woman with a medical history of asthma, gastroesophageal reflux disease and a body mass index (BMI) of 43 kg/m² was preoperatively evaluated and found to be a candidate for LSG. General endotracheal anaesthesia was induced along with the administration of perioperative intravenous antibiotics (cefazolin 4 g) and heparin 5000 units subcutaneously. Sequential compression devices were employed. LSG was performed without incident, and the patient tolerated the procedure without difficulty. Operative time was 60 min with 50 mL of blood loss. She was awakened from anaesthesia and transferred to the post anaesthesia care unit on extubation. Her post-operative course was uncomplicated. She was started on clear liquid diet on POD 0 and maintained on normal saline at 125 mL/hour. Heparin 5000 units subcutaneously was administered three times daily and she was given oxycodone 5 mg PO every 4 hours as needed for pain. The patient was discharged home on POD 2.

INVESTIGATIONS

The patient presented to the bariatric clinic on POD 13 with symptoms of hypogastric pain, nausea, and vomiting and constipation. According to the patient, her symptoms started a week ago and have been continuous. She reports 2–3 episodes of vomiting clear fluids daily. She has flatus but her last bowel movement was 5 days ago. Intake by mouth was poor and <1500 L water recommended on discharge. Her vital signs were normal (heart rate 98 bpm). Physical examination revealed mild tenderness to palpation diffusely over the abdomen without rebound pain, guarding or rigidity. Laboratory tests were within the normal limits, except for mild leukocytosis (11 100/mm³), and an elevated lipase level (602 U/L) consistent with acute pancreatitis. The patient also presented

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in metabolic acidosis with an elevated anion gap of 18. Abdominal radiographs were ordered to further evaluate the patient. A right upper quadrant ultrasound (US) scan was performed, which indicated occlusive thrombosis of the splenic vein (SV), portal vein confluence (PVC) and main portal vein (MPV) (figure 1). US findings were confirmed via CT of the abdomen and pelvis with intravenous contrast, which demonstrated thrombosis with distension of the SV, MPV, as well as right and left portal veins (figure 2). Acute pancreatitis, extensive peripancreatic and mesenteric root oedema were also noted. Bowel compromise was not appreciated. Haematology work-up for hypercoagulable disorder consisted of anticardiolipin antibodies, factor V Leiden, antithrombin level and protein C and S levels was negative. The work-up was conducted on blood samples from the emergency department, prior to the patient being started on a heparin drip.

TREATMENT

Anticoagulation therapy with unfractionated heparin drip was initiated, immediately (loading dose of 60 units per kilogram and maintenance dose of 12 units per kilogram per hour). She was aggressively volume replaced with crystalloids and her nausea was controlled with an antiemetic. On HD 2, her acidosis had resolved, her lipase was 220 U/L and white cell count was normal. Her haemodynamics remained stable and she was tolerating a clear liquid diet. She then underwent ultrasound-guided transhepatic portomesenteric venography, which showed complete occlusion of the central segment of the SMV and the MPV (figure 3). Pharmacomechanical MPV and SMV thrombectomy was performed using tissue plasminogen activator (tPA) and AngioJet with pulse spray and ultra thrombectomy techniques. This was followed by angioplasty of the SMV and MPV. A multisided infusion catheter was positioned across the thrombosed MPV/SMV to facilitate intravenous thrombolytic therapy (figure 3). Follow-up portal venography was performed 38 hours later, showing partial resolution of thrombus in the portal system. However, there was still mesenteric root oedema, slightly less than that on presentation (figure 4). Further mechanical thrombectomy was performed on the MPV and SMV resulting in complete patency with no significant residual thrombus (figure 5). Thrombolytic therapy using tPA was continued.

Follow-up SMV/portal venography was performed 50 hours later, showing overall improvement with patency of the SV,

Figure 1 US scan demonstrating a thrombosis in the MPV. MPV, main portal vein.

Figure 2 CT scan showing thrombosis of the MPV, SPV and SMV. MPV, main portal vein; SPV, splenic vein; SMV, superior mesenteric vein.

Figure 3 Transhepatic portomesenteric venogram with IC placement. Note the collateral flow into the IVC. IC, infusion catheter; IVC, inferior vena cava.

MPV and SMV. Collateral drainage to the inferior vena cava was stable, and thrombolytic therapy was terminated (figures 6 and 7). In addition, the mesenteric root oedema disappeared, and there was no bowel compromise.

OUTCOME AND FOLLOW-UP

She experienced significant deconditioning during her visit, and physical therapy was recommended as well as ambulation

Figure 4 Cannulation and partial patency of the MPV, SPV and SMV. MPV, main portal vein; SPV, splenic vein; SMV, superior mesentery vein.

Figure 6 Patent MPV, SMV and SPV. MPV, main portal veins; SMV, superior mesentery veins; SPV, splenic vein.

Figure 5 Improved patency of the MPV, LMV, RMV, SMV and SPV. MPV, main portal veins; LMV, left portal vein; RMV, right portal vein; SMV, superior mesentery veins; SPV, splenic vein.

assistance with a walker. At the time of discharge, she was tolerating intake by mouth, was pain free and her constipation, nausea and vomiting had completely resolved. Apixaban was initiated for 3 months. Her follow-up radiograph at 1-month

Figure 7 Re-demonstration of patent MPV, SMV and SPV. MPV, main portal veins; SMV, superior mesentery veins; SPV, splenic vein.

post discharge displayed continued portal venous system patency.

DISCUSSION

PMVT are well described, albeit rare complications of laparoscopic procedures. As the popularity of LSG continues to rise as a long-term response in controlling MO, PMVT as a potentially life-threatening complication merits consideration. According to Villagrán *et al*, in a complete series of 1578 bariatric surgeries, no PMVT was reported outside of the LSG group.⁴ Overall, large cohorts have reported portal–splenic–mesenteric venous thrombosis at an incidence of 0.3–1% after LSG.⁷ Other commonly encountered postoperative complications include staple-line leak, atelectasis, pulmonary embolism, haemorrhage and splenic injury.⁶

It is widely agreed that presentation of PMVT after LSG is multifactorial in origin. Genetic predispositions of hypercoagulability include mutations in prothrombin and factor V Leiden, as well as hyperhomocysteinemia.^{1 7 8} Acquired thrombophilia as a result of obesity predisposes an individual to metabolic syndrome, and therefore a systemic state of chronic inflammation and increased abdominal pressure. Additionally, sedentary lifestyle leads to blood flow reduction in the lower extremities and susceptibility to VTE. Therefore, all patients undergoing a bariatric procedure are at high-risk of thromboembolism.⁹ Villagrán *et al* also stated that smoking is the predominant risk factor for postoperative PMVT.⁴ On diagnosis of PMVT, a thorough hypercoagulability work-up should be ordered to rule out the possibility of inherited thrombophilia as the aetiology.

Several factors have been found to be associated with an elevated risk of developing PMVT. The pneumoperitoneum on laparoscopy causes elevated intra-abdominal pressure via insufflation of CO₂.¹ Inversely proportionate between the two, as abdominal pressure increases, portal and splanchnic venous flow will decrease. As reported by Rottenstreich *et al*, intra-abdominal pressures >14 mm Hg result in >50% reduction of portal blood flow.⁷ These reductions are due to direct compression. Additionally, hypercapnia as a result of the carbon dioxide insufflation will further cause inhibition of blood flow due to its vasoconstrictive action on the splanchnic and mesenteric region. Furthermore, several authors have noted that steep reverse Trendelenburg positioning may also cause additional stress to the patient's portal venous system, thus reducing blood flow and increasing portal pressure further.^{3 4 6 7} Distinctive from other gastric bypass procedures, LSG requires mechanical manipulation of the surrounding short gastric vessels of the stomach, as well as the left gastroepiploic arcade during skeletonisation of the greater curvature.^{4 8 10} Damage to the splanchnic and portal endothelium may increase local agents of thrombosis.¹¹ Lateral thermal effect on the region via monopolar energy, such as that emitted from this case's Harmonic scalpel, may also have played a role in thrombus propagation.¹² Interestingly, direct SV contact during surgery, also common to LSG, has also been proposed as another catalyst of thrombosis.^{4 6} Common to all bariatric procedures, water intake postoperatively is limited, thereby predisposing the patient to dehydration and systemic thrombosis formation.^{4 5}

Most commonly, patients present within 45 days after surgery with an unrevealing physical examination: nausea, diffuse abdominal pain, vomiting and low-grade fever.^{1 7} Laboratory findings are generally normal; however, leukocytosis, metabolic acidosis, as well as elevated liver function tests may also be observed.^{4 6 8} Although not seen in this case, one-third of patients with PMVT present with ascites.¹³ Clinical suspicion of PMVT is crucial, and a definitive diagnosis requires non-invasive radiograph imaging. CT with intravenous contrast media is the gold standard for confirming PMVT with a sensitivity of 90%.¹ Although Doppler US has the lowest sensitivity out of all imaging modalities; it is often useful in patient follow-up and observing the restoration of blood flow.

Once a confirmed diagnosis of PMVT has been made, therapy should start immediately; however, treatment must be tailored specifically to the patient; patient condition and the occlusive nature of the thrombus observed are major criteria in deciding treatment modality.^{1 5} Initial treatment of a stable patient with non-occlusive thrombus consists of therapeutic anticoagulation with generally successful outcomes.^{3 5} However, as in the patient described in this case, severe occurrences of PMVT may require pharmacomechanical thrombectomy and

transhepatic venous thrombolysis.^{2 5} It has been shown that direct thrombolysis has had a high success rate in patients who remain unresponsive to anticoagulation therapy alone. Moreover, direct perfusion of tPA may be continued postoperatively without increasing the patient's bleeding rate.⁵

Without proper treatment, persistent thrombosis may go on to cause bowel ischaemia, peritonitis and septic shock via extra-hepatic portal hypertension.^{6 14} Prompt detection of high-risk, postoperative bariatric patients is paramount. Perioperative anticoagulation, although routinely administered, has not been sufficient in preventing all cases of PMVT. Although there is no consensus concerning long-term prophylaxis for patients who have undergone LSG, as of 2013, the opinion of the American Society of Metabolic and Bariatric Surgeons states that extended anticoagulation therapy is recommended for all bariatric procedures on discharge.⁷ Protocol regarding specific dose and duration of therapy has yet to be determined; however, several sources state that anticoagulation prophylaxis—which correlates with reduced recurrence rates—should be continued for at least 2 weeks after surgery, and up to 6 months postoperatively (longer courses may be necessary for patients known to be inherently susceptible to VTE).^{1 4 7} In an attempt to enhance blood flow and prevent a sedentary state, early ambulation and physical therapy is also recommended throughout patient recovery.¹

Learning points

- ▶ Portomesenteric vein thrombosis (PMVT) is a rare, and potentially lethal, complication associated in 0.3–1% of patients who have undergone laparoscopic sleeve gastrectomy (LSG).
- ▶ Presentation of symptoms is most likely to occur within 45 days postoperatively, and treatment should be started promptly.
- ▶ In cases of persistent occlusion with considerable mesenteric and bowel oedema and/or ischaemia, thrombolytic therapy and mechanical thrombectomy may be necessary to return vessel patency.

Contributors LSA is responsible for planning, conduct, reporting, conception and design, acquisition of data or analysis and interpretation of data, writing and reviewing the manuscript. RF is responsible for design, acquisition of data or analysis and interpretation of data, writing and reviewing the manuscript. MA is responsible for writing and reviewing the manuscript. PR is responsible for writing and reviewing the manuscript.

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