CASE REPORT

Portomesenteric venous thrombosis complicated by a haemorrhagic shock: a rare complication of laparoscopic gastrectomy

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SUMMARY

Portomesenteric venous thrombosis is a rare complication reported in only a few cases involving laparoscopic bariatric surgery. We report a case of a 44-year-old woman who presented 14 days after recent laparoscopic sleeve gastrectomy with the chief complaint of abdominal pain and associated nausea. Abdominal CT demonstrated thrombi in her superior mesenteric, portal and splenic veins. She was initiated on therapeutic heparin but developed haemorrhagic shock shortly afterwards. Subsequent CT angiogram failed to localise the source of her haemorrhage. Her haemodynamic instability improved following a 6-day intensive care unit stay requiring vasopressive agents and blood transfusions. Further hypercoagulable workup revealed that she was a heterozygous carrier of the prothrombin gene mutation, and thus started on lifelong oral anticoagulation.

BACKGROUND

We present a case of a middle-aged woman presenting with portomesenteric venous thrombosis (PMVT), a rare but life-threatening complication of laparoscopic surgery. Portal vein thrombosis (PVT) is more commonly seen in patients with end-stage liver disease or malignancies, predisposing risk factors not seen in our case.¹ However, our patient's recent bariatric procedure, severe obesity and heterozygous prothrombin gene mutation likely increased her risk of developing a PMVT. Haemorrhagic shock complicated her hospital stay due to a supratherapeutic anticoagulation treatment with heparin for her PMVT, from which she successfully recovered and was subsequently placed on lifelong oral anticoagulation.

CASE PRESENTATION

A 44-year-old obese woman with a body mass index of 38 kg/m² presented a 14-day status post laparoscopic sleeve gastrectomy (LSG) with the chief complaint of abdominal pain for 2 days accompanied with occasional nausea. Her abdominal pain was gradually worsening, being sharp and localised to the right lower quadrant. She denied any other gastrointestinal symptoms including episodes of vomiting, constipation, diarrhoea, change in stool patterns and haematochezia.

She had a medical history of depression and gastro-oesophageal reflux disease. Aside from her

recent bariatric procedure, she had two caesarean sections in the past. During her bariatric procedure, the dissection of the lesser sac and the posterior stomach, the portomesenteric circulation was not visualised or manipulated. The procedure and postoperative course leading to her presenting symptoms were otherwise unremarkable. Postoperatively she was taking ferrous sulfate, omeprazole and hydrocodone/acetaminophen one to two times daily for pain control.

In the emergency department, she was given an antiemetic for nausea and vomiting. A CT scan of the abdomen was performed which demonstrated extensive acute thrombi of the superior mesenteric vein, left splenic vein and portal vein (figure 1). The proximal segmented branches of the right and left portal vein showed a right greater than left obstruction with splenomegaly. This finding was consistent with a PMVT and mesenteric ischaemia. She was immediately initiated on a therapeutic heparin nomogram and admitted to intensive care unit for further evaluation and management.

INVESTIGATIONS

On admission, her initial haemoglobin was 128 g/L and her activated partial thromboplastin time (aPTT) was 38 s. Shortly afterwards she became hypotensive and tachycardic with a blood pressure of 73/59 mm Hg and heart rate of 150 beats per minute. She received continuous boluses of intravenous fluid resuscitation and had a central venous catheter placed in her right internal jugular vein. Repeat laboratory studies showed a sharp drop in her haemoglobin to 4.1 mg/dL and aPTT >277 s. Heparin therapy was discontinued, and she was transfused with 7 units of packed red blood cells (PRBCs) on hospital day 1. Additional laboratory results revealed an elevated white blood cell count of $16.1 \times 10^3/\mu\text{L}$ with no bandaemia and a lactate level of 59 mg/dL likely due to haemorrhagic shock and mesenteric ischaemia. She became lethargic and hypoxic (blood oxygen saturation 87%) and was promptly intubated after her arterial blood gas demonstrated profound acidemia (pH 7.26, partial pressure of carbon dioxide 54 mm Hg and a partial pressure of oxygen 84 mm Hg on room air).

On hospital day 2, a CT angiogram of the chest, abdomen and pelvis was performed to identify the source of bleed; however, imaging only showed

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Figure 1 Initial CTA showing thrombosis in splenic and portal veins. CTA, CT angiography.

evidence of the haemoperitoneum without any active area of extravasation (figure 2). Despite aggressive fluid resuscitation, her mean arterial pressure was <65 mm Hg, and vasopressive agents were started. On hospital day 3, her haemodynamic status improved and was titrated off vasopressors. By hospital day 5, after transfusion of a total of 17 units of PRBCs, her haemoglobin

Figure 2 Repeat CTA demonstrating intraperitoneal haemorrhage. CTA, CT angiography.

level was stable at 91 g/dL. Her respiratory status also showed marked improvement and was subsequently extubated without complication. On hospital day 6, she was successfully initiated on oral anticoagulation.

On hospital day 7, she was transferred to the general medical service. In addition, her acquired and inherited hypercoagulable work up showed she was a heterozygous carrier of the prothrombin G20210A mutation and was placed on lifelong oral anticoagulation. She was eventually discharged to a rehabilitation centre without further complications.

TREATMENT

In this circumstance, she was anticoagulated on a heparin nomogram.

OUTCOME AND FOLLOW-UP

After she had become supratherapeutic, she was switched to a weight-based heparin nomogram, and her haemorrhaging gradually subsided. It was also soon discovered that this patient was heterozygous for prothrombin gene mutation and is now on lifelong anticoagulation. At 6 months, postoperatively, repeat CT imaging demonstrated resolution of the PMVT.

DISCUSSION

The reported incidence of PMVT from LSG ranges from 0.3% to 1.0%.^{2,3} Clinically, PMVT can be particularly challenging to diagnose as patients present with vague, non-specific symptoms such as non-localised abdominal pain, nausea and fever.⁴ Table 1 shows a literature review of clinical presentation, management and outcome of reported cases of PMVT after LSG. The presentation of PMVT between the cirrhotic and non-cirrhotic patients has two different presentations. In the non-cirrhotic patient with PMVT, the patient will present with abdominal pain due to bowel ischaemia with portal hypertension. Varices and hepatic encephalopathy will also be present. However, no ascites will be present.⁵

In comparison, the presentation of PMVT in the cirrhotic patient will also present with abdominal pain due to bowel ischaemia with portal hypertension, varices and hepatic encephalopathy. However, ascites will not be present.⁵ Considering the abdominal pain, the patient will have an abdominal imaging study where the thrombosis of the vessels is then detected. In many non-cirrhotic patients, there is usually a prothrombotic disorder. The most common disorders include factor 5 Leiden mutation, lupus anticoagulant, protein C/protein S deficiency, underlying myeloproliferative disorders or various mutations in the thrombin gene.⁵

As the number of laparoscopic bariatric surgeries performed continue to rise, clinicians will need to maintain a high index of suspicion for PMVT and subsequent mesenteric ischaemia, which has a mortality rate of 10% to 20%.⁶

Even without direct manipulation of the portomesenteric vasculature, laparoscopic abdominal surgery, excluding splenectomy, has shown not only to be a risk factor for PMVT but its reported prevalence has been steadily increasing within the last decade.² Some studies have shown that the risk of thromboembolism continues long after bariatric surgery and postoperative discharge and suggest continued prophylaxis after discharge.⁷

In a prior retrospective study, it was proposed that the duration of anticoagulation treatment for PMVT be 6–12 months for non-surgical patients who developed acute PVT.⁸ It was concluded that anticoagulant therapy decreased the rate of recurrence or extension of PMVT by two-thirds and without

Table 1 Literature review of reported cases of portomesenteric venous thrombosis secondary to laparoscopic gastrectomy

Referenced reported cases	Clinical presentation	Management	Outcome
Pigeyre <i>et al</i> ¹	Nausea, vomiting diarrhoea and abdominal pain	Therapeutic heparin during hospitalisation, warfarin outpatient for 6 months	Not disclosed
Goitein <i>et al</i> ²	Severe epigastric pain, nausea and vomiting	Therapeutic heparin during hospitalisation, warfarin outpatient for 6–12 months	Not disclosed
Muneer <i>et al</i> ¹⁴	Vague abdominal pain, severe nausea, vomiting, fever and diffuse abdominal tenderness	Therapeutic heparin during hospitalisation, warfarin outpatient for 6 months	Survived
Muneer <i>et al</i> ¹⁴	Vague abdominal pain, severe nausea, vomiting, fever and diffuse abdominal tenderness	Surgical bowel resection	Survived
Bhatia <i>et al</i> ¹⁵	Progressive central abdominal pain and nausea	Therapeutic heparin during hospitalisation, warfarin outpatient for 3 months	Survived
Abu Jkeim <i>et al</i> ¹⁶	Epigastric pain and nausea	Therapeutic heparin during hospitalisation, warfarin outpatient for 6 months	Survived
Baba <i>et al</i> ¹⁷	Sudden severe abdominal pain	Therapeutic heparin during hospitalisation, lifelong warfarin anticoagulation due to positive prothrombin gene mutation	Survived
Baba <i>et al</i> ¹⁷	Sharp abdominal pain in the epigastric area with radiation to whole abdomen, physical examination showed abdominal tenderness on palpation with voluntary guarding	Therapeutic heparin during hospitalisation, lifelong warfarin anticoagulation due to positive prothrombin gene mutation	Survived

increasing the rate of gastrointestinal tract bleeding.⁸ However, there is no consensus regarding aggressive postoperative prophylaxis, and thus was not considered a standard of care in our case.⁹

Morbid obesity has also shown association with low-grade systemic inflammation. It has been postulated that leptin and adiponectin, released from adipocytes, are key mediators of inflammation and atherosclerosis in morbidly obese individuals.⁶ Leptin supports human platelet aggregation by increasing the average platelet response to its agonist ADP and thrombin.⁷ This response is the suggested mechanism for thrombotic events in the obese. The protein adiponectin is expressed in adipocytes and accounts for 0.01% of total plasma protein.¹⁰ In obesity, adiponectin levels are inversely correlated with the measure of obesity.¹⁰ Also in correlation, low levels of adiponectin are associated with, but not limited to, congestive heart disease, glucose intolerance, type 2 diabetes and hypertriglyceridaemia.¹⁰ Although PMVT is an infrequent postoperatively complication, our patient has several predisposing risk factors.^{7–9} The most significant of the predisposing factors is that the patient tested positive as a heterozygous carrier for the prothrombin G20210A mutation. The prothrombin G20210A mutation falls second place after factor V Leiden mutation as the most common inherited thrombophilia.^{11–12} The gene that makes factor V is the *F5* gene; this gene is affected in the factor V Leiden mutation. Activated protein C (APC) degrades coagulation factor V, through which negative feedback slows down the clotting mechanism and prevents clots from becoming too large.¹² However, in factor V Leiden mutation, coagulation factor V cannot be degraded by APC. In return, the clotting process is prolonged and incidence of clots being formed is increased.¹²

In prothrombin G20210A mutation, prothrombin (factor II; F2) is the precursor to thrombin in the anticoagulation cascade.¹³ A fibrin clot is formed by cleaving fibrinogen to fibrin via thrombin.¹³ This fibre clot was formed in the portomesenteric vein in our patient. Conversely, the risk of a venous thrombotic event (VTE) in an individual with prothrombin G20210A heterozygotes has been estimated to be relatively increased by threefold to fourfold compared with the general population without this mutation.¹³ Homozygotes are at a higher risk of acquiring a VTE than heterozygotes. The overall likelihood of a deep vein thrombosis and pulmonary embolism

is increased overall in both heterozygote and homozygote carriers of the prothrombin G20210A gene.¹³

In this case, patients may also have VTE at an atypical site such as portal veins. Treatment is with anticoagulation for at least 3–6 months. The clinical decision to continue anticoagulation beyond this time is dependent on whether the thrombosis was provoked or unprovoked.

PMVT is a rare but lethal complication that may occur after LSG. The clinical suspicion needs to remain high in patients with poor PO (oral) intake, and ambulation several days postoperative who present with severe acute onset of abdominal pain. Once PMVT is determined, immediate anticoagulation with hypercoagulable studies must be ordered. The determination of a provoked versus unprovoked would elicit a longer anticoagulation period for an unprovoked VTE.

Learning points

- ▶ Portomesenteric venous thrombosis (PMVT) is a rare but life-threatening complication of laparoscopic sleeve gastrectomy (LSG).
- ▶ The incidence of postoperative PMVT in LSG ranges from 0.3% to 1.0% but is likely under-reported.
- ▶ Due to vague presenting symptoms, a high clinical suspicion for PMVT is needed for diagnosis.
- ▶ Acute PMVT treatment is based on severity and includes immediate supportive care, anticoagulation and possible laparoscopy or laparotomy.
- ▶ Acquired and inherited hypercoagulable conditions should be considered as predisposing factors for PMVT and require lifelong anticoagulation.

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