

Case Report

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Acute mesenteric venous thrombosis after laparoscopic sleeve gastrectomy: a case report and a literature review

Alaa Sedik^{1*}, Fawaz Aleashed², Ahmed Fathi¹

¹Department of General Surgery, King Khalid Hospital Hail, Kingdom of Saudi Arabia

²Department of Surgery, Hail University, Kingdom of Saudi Arabia

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***Correspondence:**

Dr. Alaa Sedik,

E-mail: asedik59@yahoo.com

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ABSTRACT

Over the last decade, laparoscopic sleeve gastrectomy has become an increasingly popular surgical procedure for morbid obesity. Moreover, post-laparoscopic sleeve gastrectomy mesenteric venous thrombosis is very rarely reported. The presentation varies from mild non-specific abdominal symptoms to life threatening surgical emergency. Superior mesenteric vein thrombosis after bariatric surgery is a difficult diagnosis that needs a high index of suspicion. We report a case of thrombosis of the superior mesenteric vein, who presented as acute emergency to emergency room, shortly after sleeve gastrectomy. Patient underwent laparotomy and offered a resection of 60 cm of infarcted proximal jejunum with restoration of bowel continuity later. Patient subsequently recovered and followed several times in our outpatient and was free of symptoms.

Keywords: Mesenteric thrombosis, Laparoscopic sleeve gastrectomy, Acute

INTRODUCTION

The first case of superior mesenteric venous ischemia due to thrombosis was reported by Warren and Eberhard in 1935. This type of venous ischemia represents 5-15% of all mesenteric ischemia.^{1,2} Clinical picture varies from mild non-specific abdominal symptoms to life threatening surgical emergency. Management is primarily medical through anticoagulation, yet surgery is indicated if bowel infarction occurs. A multidisciplinary long term management of such cases from many specialties, including: hematology, gastroenterology, internal medicine, endocrinology, general or plastic surgery is important both to diagnose, ensure patient's recovery, and also in follow up after discharge.

CASE REPORT

A 29 years old morbidly obese (basal metabolic index-42) Saudi male, known to have type 2 diabetes mellitus

on oral and insulin therapy was presented to emergency room, 5 days post laparoscopic sleeve gastrectomy with confusion and shock. He deteriorated rapidly despite resuscitation and became comatose. He was resuscitated and intubated. Labworks showed a picture of severe diabetic ketoacidosis with RBS: 22 mmol/l, urine ketones +++, PH 7.1, and WBC 25,000, renal and liver impairments reviewing the perioperative data revealed a straightforward procedure and patient was prepared as usual where he was seen preoperatively by endocrinologist and gastroenterologist as he had poor control of blood sugar. He received preoperative and postoperative enoxaparin, according to the hospital policy.

Patient was admitted in ICU and was prepared for contrast enhanced computed tomography of the abdomen (CECT) that failed to demonstrate leakage from the sleeve with a mild free pelvic and perihepatic fluid, that was aspirated under ultrasound guidance. A diagnostic laparoscopy was arranged, revealing an intact sleeve and

an infarcted (60 cm) segment of proximal jejunal segment – less than 5 cm from duodenojejunal junction.

A midline laparotomy was done. The infarcted bowel was resected and both ends stapled and left in the abdomen (Figure 1). The skin was closed with a running prolene No1 suture. A second look laparotomy was done 36 hours later, where the proximal jejunal end was refreshed and resection of further distal 10 cm segment with questionable vascularity. An end to end jejunoojejunostomy was done using interrupted full thickness stitches of 2/0 vicryl. A jejunal loop was chosen, then a decompression retrograde nasogastric tube was passed through an entrostomy about 30 cm distal to the anastomosis, where the tube is passed up to the duodenum and another entrostomy 5 cm distal to the first one was made for a feeding jejunostomy using a (F18) Foley 's catheter. The jejunal loop, carrying both entrostomies, was fixed to the peritoneum with interrupted vicryl 2/0 stitches, then both tubes brought separately through the skin (Figure 2).



Figure 1: (A) Intra-operative, gangrenous proximal jejunal loop; juxta to the duodenojejunal junction and (B) resected jejunal specimen, stapled bowel ends as a damage control procedure.

The abdomen was temporarily closed as fascia couldn't be approximated at that time. He was offered TPN and jejunal feeding started gradually, but unfortunately stopped after 7 days due to ileus. A CECT was arranged, revealing a 10×5 cm collection at the site of jejunal loop attachment to the abdominal wall on the left side, minor leak from the distal entrostomy site, and mild free intraperitoneal fluid.

In absence of percutaneous drainage option of this intraperitoneal collection in our hospital, patient was taken to theatre and drainage was done. He was put on TPN and jejunal feeding resumed again. He recovered slowly and extubated after 16 days of intubation. Patient was shifted to high dependency unit where He started oral feeding and tolerated well. Feeding tube removed on 32th day. All drains and catheters removed, and finally, he was discharged free of symptoms. He was seen in outpatient surgery clinic and was free of complaint He is not interested to have elective repair of the incisional

hernia. He was seen also by gastroenterology, medical, and nutritional teams and his diabetic status improved and he lost 50% of his preoperative weight in 6 months.

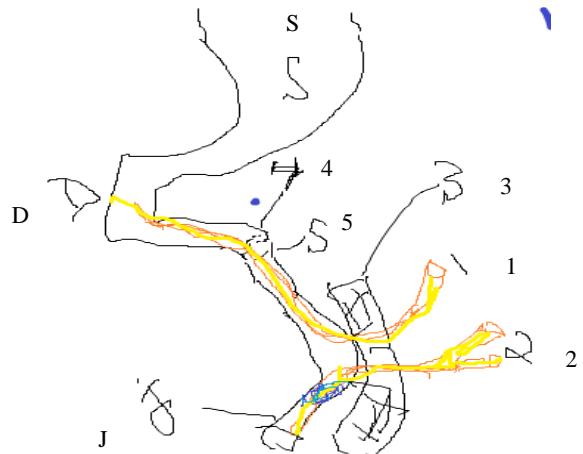


Figure 2: The jejuno-jejunal anastomosis (5), with placement of retrograde decompression NG tube across the anastomosis (1) and a distal feeding jejunostomy (2), the place of duodenojejunal junction (4) and adjacent abdominal wall (3), S, D, J stands for stomach, duodenum and jejunum respectively.

DISCUSSION

Weight loss surgery include gastric restrictive procedures (e.g., adjustable gastric banding and sleeve gastrectomy), malabsorptive procedures (e.g., Roux-en Y gastric bypass), and biliopancreatic diversion), or a combination of these surgeries.^{3,5} Portomesenteric venous thrombosis (PMVT) is a rare complication and is associated with high morbidity (mesenteric ischemia in 5–15% of cases) and mortality (20–50%).^{4,6,7} Venous stasis secondary to enhanced intra-abdominal pressure above 14 mmHg is enough to reduce the portal venous flow by 50%, and also CO₂ itself causes splanchnic vasoconstriction through release of vasopressors which reduces venous flow, operative intervention in splanchnic vasculature, and hypercoagulable state with increased release of fibrinogen and coagulation factors are the possible factors for PMVT following bariatric surgery.^{3,4,8-11} Intraabdominal Inflammation, portal hypertension, and neoplastic diseases are other etiologies. The clinical diagnosis of PMVT is difficult, and diagnosis is usually confirmed by CECT abdomen, with a sensitivity of 90%.^{4,7}

In addition, the prolonged reverse Trendelenburg position is considered as another possible factor for the development of PMVT. Protein C and S deficiency, homocysteinemia, and prior abdominal surgery are other etiological factors.^{8,10}

Most patients with mild PMVT can be successfully treated conservatively, if no evidence of bowel infarction, with low molecular weight heparin alone to recanalize the

portal venous system and to reduce the risk of further thrombotic events.^{12,13} Treatment with anticoagulation should continue for at least 3–6 months, but can be extended further if the signs and symptoms persist. However, patients with a systemic etiology are required to be on lifelong anticoagulation therapy. Supportive measures that could also be used to supplement anticoagulation therapy include bowel rest, fluid resuscitation, and nasogastric suction for conservative management.^{14,15}

No protocols or guidelines are available that are specific for prevention of PMVT in patients who underwent bariatric surgery, due to its rarity or reporting of such complications. Therefore, the general standard prophylactic measures that apply to obese and post-laparoscopic patients in general surgery are currently applied for bariatric patients. As bariatric surgery grows in popularity, patients should receive more attention, screening, and management preoperatively.^{15,16}

Diagnostic laparoscopy for the diagnosis of PMVT remains controversial, as it may be done if the CECT is nonconclusive and patient showed signs of clinical deterioration (as in our case). Others support the use of diagnostic laparoscopic exploration in all suspected cases regardless of the clinical or radiological findings.¹⁷

CONCLUSION

Although PVT is a rare occurrence after laparoscopic sleeve gastrectomy, it is important to have a high index of suspicion for diagnosing it promptly diagnosis to improve the outcome of this potentially fatal condition. Conservative management through anticoagulants or thrombolytics is considered as the primary treatment option. Also, we recommend that bariatric surgery patients should be maintained postoperatively on anticoagulants for at least 2 weeks.

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