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CASE REPORT

Portomesenteric Vein Thrombosis after Laparoscopic Sleeve Gastrectomy: A Case Report

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Portomesenteric vein thrombosis is an uncommon but potentially life-threatening complication associated with laparoscopic sleeve gastrectomy. We present the case of a 26-year-old male who underwent an uneventful laparoscopic sleeve gastrectomy and presented on postoperative day 14 with portomesenteric vein thrombosis. The patient was treated conservatively with IV heparinization, followed by an oral anticoagulant agent. He was discharged in stable condition without further problems. A high index of suspicion for the disease is required not to miss or delay the diagnosis of portomesenteric vein thrombosis which could lead to a fatal outcome. All patients should be screened beforehand for underlying hypercoagulability before surgery.

Key Words: Portomesenteric vein thrombosis, Laparoscopic sleeve gastrectomy, Bariatric surgery, PMVT, LSG

INTRODUCTION

Bariatric surgery has become one of the standard treatment methods for morbid obesity worldwide. It has already proved to be effective in losing weight and managing obesity-related co-morbidities [1]. In 2019, as Korean national health care insurance started to cover the cost of the procedure, the number of patients undergoing this procedure began to increase.

Although laparoscopic bariatric surgery provides favorable results, it has inherent complications such as bleeding, stapled-stump leakage, stricture, internal herniation, anastomotic stenosis, gastric erosion, intestinal small bowel obstruction, deep vein thrombosis (DVT), pulmonary embolism (PE), and portomesenteric vein thrombosis (PMVT) among others [2].

Specifically, PMVT is a rare complication that may occur after bariatric surgery and is associated with a potentially life-threatening outcome. The reported incidence of PMVT ranges from 0.07 to 1% [3-6]. It is assumed to be implicated both by systemic and local factors. The diagnosis of PMVT requires a high index of suspicion because signs and symptoms are generally vague. The majority of the disease has been managed successfully with conservative anticoagulation [4,6,7]. However, there have also been cases requiring emergent surgical exploration and mechanical thrombectomy or invasive thrombolysis, some of which end in mortality [7]. Thus, it is important for present bariatric clinicians to understand the nature and cause of this entity to allow for proper management.

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Here we present the case of a patient who was diagnosed with PMVT following laparoscopic sleeve gastrectomy (LSG) and was treated successfully with intravenous (IV) heparinization, which was bridged to oral rivaroxaban afterward. This was our first experience of a complication after LSG, and to our best knowledge, it has not yet been reported in Korea.

CASE REPORT

The patient was a 26-year-old male with a Body Mass Index of 35.4 kg/m^2 and an insignificant past medical history. He was an ex-smoker and had no personal or family history of thromboembolic diseases. He underwent LSG at a private bariatric clinic.

LSG was performed with the patient in the reverse Trendelenburg position using five laparoscopic ports under a 15 mmHg carboperitoneum. The greater omentum was dissected using a vessel-sealing system from 4 cm oral to the pyloric ring to the angle of His. After a 35 Fr bougie was put in the lesser curvature of the stomach, a creation of the sleeve was done with a linear stapler. Following the completion of the stapling, 50 cc of indigo carmine was introduced through the bougie while compressing the duodenum for 10 seconds to check for leakage. The procedure, the carboperitoneum, and the anesthesia lasted 100 min, 70 min and 150 min, respectively. Endoscopy was performed under general anesthesia before the operation. For the next 24 hours after operation, a total of 4500 cc of the crystalloid was infused. Intermittent, sequential pneumatic compression and a graduated compression stocking were used starting when anesthesia was administered. The gastrografin-using upper gastrointestinal series was performed on postoperative day 1 to check for the presence of a leakage or obstruction. On postoperative day 2, the patient started to drink water and was discharged that afternoon without any complications. Prophylactic anticoagulation was not used.

The patient developed vague abdominal pain and nausea on postoperative day 10 without improvement for the next 4 days. He presented to the clinic and was promptly referred to our center. Upon arrival, the physical exam showed direct tenderness on the left upper quadrant area of the abdomen but no sign of peritoneal irritation. Initial blood pressure was 123/83 mmHg, heart rate was 83 beats/minute, and body temperature was 36.5 degrees Celsius. The laboratory tests revealed leukocytosis (12,600/μl), elevated C-reactive protein (15.53 mg/dl), and lactic acid (4.5 mmoL/L). Liver function tests (LFT) and platelet count were within normal range. The computed tomography scan with IV contrast demonstrated diffuse thromboses from the left and main portal vein to the main

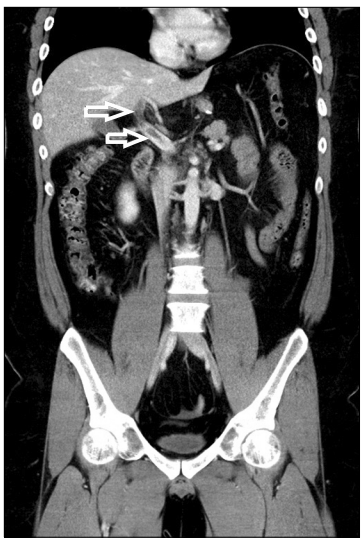

Fig. 1. Computed tomographic image showing Lt and main portal vein thrombosis (white arrow).

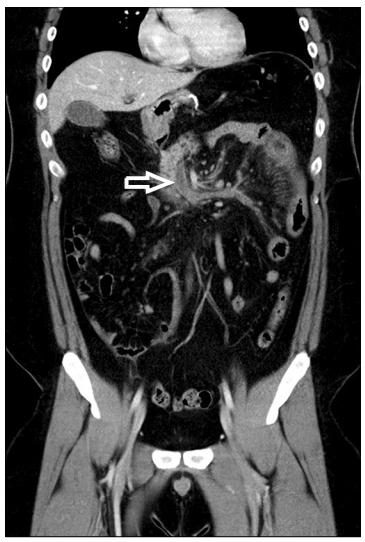

Fig. 2. Computed tomographic image showing superior mesenteric vein thrombosis (white arrow).

branch of the superior mesenteric vein (SMV) and edematous small bowel (Figs. 1, 2). Upon diagnosis, IV heparin was immediately infused, aiming for 50–75 sec of activated partial thromboplastin time (aPTT) and prophylactic antibiotics (Ceftriaxone+Metronidazole) were started. The patient was hospitalized and fasted to rest the bowels.

Abdominal pain disappeared on the 3rd day and nausea lasted one day longer, vanishing after discontinuing antibiotics. On the 4th day, heparin was replaced with oral administration of rivaroxaban (20 mg/day) and a liquid diet was commenced. Although a mild elevation of LFT was seen on the 6th day, the patient was discharged with no complaints the following day, after ensuring there was no deterioration of aspartate transaminase (AST) and alanine transaminase (ALT). At the outpatient clinic 1 week later, blood test results showed improvement in LFT and the patient did not reveal any discomfort.

DISCUSSION

PMVT is a rare but potentially lethal complication following LSG. It has been reported to increase in frequency over the last decade and many clinicians have tried to understand the nature of this complication.

PMVT is more often associated with LSG compared to other gastric bypass surgeries, although the cases for this are unknown. Considering that LSG has become the most popular choice due to its technical easiness, low complication profile, and avoidance of nutritional deficits compared to other bypass methods, there could be more patients suffering from LSG-related complications in the future [8].

1. Etiology

PMVT is speculated to be influenced by multiple factors such as genetic predisposition (Factor V Leiden deficiency, protein-C and -S deficiency, antithrombin III deficiency, G20210A prothrombin mutation, hyperhomocysteinemia, etc.) and acquired prothrombotic state (malignancy, atrial fibrillation, oral contraceptive use, myeloproliferative disorders, obesity). Abdominal operations, especially those involving ligation of major portal tributaries such as liver transplantation and splenectomy , trauma to the portal venous system, and cirrhosis, which decreases portal blood flow, are also recognized as PMVT-inducing events.

In the review by Karaman et al. [9], the rate of coagulopathy disorders was up to 28.8%. Morbid obesity itself is a well-proved thromboembolic predisposing factor, as several procoagulant factors such as fibrinogen, factor VIII, and von Willebrand factor have been discovered to be elevated in obese patients, which inevitably creates a pro-thrombotic state. It is also associated with a chronic inflammatory state and venous stasis in the lower limbs. Regarding these factors, screening of the underlying thrombogenic state, which is not routinely performed in most centers, should be considered before proceeding with LSG.

On the other hand, this disease has been reported after many other laparoscopic abdominal procedures such as Roux-en-Y gastric bypass, Nissen fundoplication, cholecystectomy, appendectomy, and colectomy [7]. During the procedure, increased intraabdominal pressure reduces portal flow and it is further amplified by hypercapnia induced by CO_2 insufflation, which causes vasoconstriction of the splanchnic system [10]. The reverse Trendelenburg position, which is often used during the operation, also contributes to a lower flow rate [11].

Dehydration, which is enhanced after operation because patients are instructed to consume only a liquid diet, is also one of the factors that induce the disease.

There are a few theories that explain why LSG causes PMVT over other bariatric methods. First, division of the short gastric arteries inducing splenic ischemia or infarction may cause mesenteric thrombus, as it does in splenectomy. Additionally, thermal and mechanical injury on the gastroepiploic vessels during the skeletonization of the greater curvatures is also though to contribute to the development of PMVT [6].

If this hypothesis is correct, using a surgical clip instead of an energy device will reduce the prevalence. However, the usefulness of this attempt should be assessed thoroughly from multiple points of view, as the amount of time for the operation and technical simplicity must be sacrificed.

2. Diagnosis

As many case reports and review journals have described, symptoms and laboratory findings are vague and non-specific; therefore, the diagnosis of the disease could be challenging. Abdominal pain is the most common symptom, characterized by pain out of proportion to physical examination or laboratory findings. Leukocytosis and the elevation of C-reactive protein are seen frequently. Enhanced computed tomography is the first choice modality, with a sensitivity of more than 90%. Some centers also performed US Doppler for initial work-up or follow-up exam. The investigators uniformly have emphasized the index of high suspicion upon this uncommon pathology not to miss or delay the diagnosis.

Some have suggested that diagnostic laparoscopy should be executed [12], even if there is no sign of infarction due to the low sensitivity of the examination and potential lethality of complications. However, in practice, a surgical option is considered only after the ischemia is obvious by the imaging work-up or deterioration of the patient despite conservative treatment.

3. Treatment

In most events, anticoagulation alone was sufficient to treat the disease. Intravenous heparin or low molecular weight heparin should be commenced as soon as the diagnosis is made intraoperatively or in the case of active bleeding, as it has been proved to significantly improve survival [13,14]. After stabilization, heparin would be replaced with an oral agent, including warfarin and rivaroxaban for the next 3-12 months. Patients who were diagnosed with any systemic thrombophilic disease were treated for a lifelong period. It appears to be important to start the treatment as early as possible to avoid bowel infarction. If infarction has already happened, immediate surgical exploration should be prosecuted to resect a damaged section of the intestine. Some clinicians also tried thrombectomy or IV thrombolysis, which led to the reversal of portal flow.

In general, as in our patient, most centers use conservative anticoagulation with satisfying results. However, in severe cases where the situation deteriorates despite routine management, the operation should not be delayed. It is well established that an infarcted bowel should be resected as soon as possible to avoid sepsis. When PMVT is totally occlusive, the treatment tactics should be more thoroughly discussed. Belnap et al. [5] performed IV thrombolysis and mechanical thrombectomy which reversed flow successfully.

4. Prophylaxis

Although bleeding is one of the major complications, prophylactic anticoagulation for the prevention of thromboembolism is given routinely in many abdominal surgeries, and there is no exception in the bariatric field. Both low molecular weight heparin and unfractionated heparin could be used. Nevertheless, to date, there are no widely accepted guidelines regarding the dose and duration of treatment.

Godoroja et al. [3] and Caruso et al. [15] suggested that a prolonged prophylactic regimen of 40 mg sodium enoxaparin qd for 4 weeks may lower the incidence of PMVT.

In the study by Caruso et al. [15], it was shown that applying a prophylactic regimen with a longer duration significantly lowered the incidence of PMVT compared to other centers (1/2853). Thus, due to the well-known high-risk of thromboembolism in morbid obese patients, introduction of an enhanced anti-thrombotic regimen might provide more benefits than risks.

In conclusion, PMVT is a rare but potentially lethal complication with vague presentation. A high index of suspicion is required to diagnose the disease. High-risk patients should be screened preoperatively and may benefit from enhanced prophylaxis.

CONFLICT OF INTEREST

None to declare.

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