

## Case Report

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# Splanchnic Vein Thrombosis – an Uncommon Complication after Laparoscopic Sleeve Gastrectomy

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### Key Words

Obesity · Laparoscopic sleeve gastrectomy · Thrombosis

### Abstract

**Background:** Laparoscopic sleeve gastrectomy (LSG) is an innovative and relatively safe surgical approach for weight reduction in morbidly obese people. Splanchnic vein thrombosis (SVT) is an extremely rare complication of LSG and, if not recognized, carries a high mortality rate. This paper highlights a potentially lethal condition of SVT after LSG. **Case Report:** A 37-year-old morbidly obese woman was referred to our institution for LSG. Three weeks after the intervention, she was readmitted with abdominal pain, vomiting, nausea, diarrhea, and fever with positive family anamnesis to viral disease. Abdominal X-ray as well as ultrasonography were both normal, and no X-ray contrast medium leakage was observed. One week later, she was readmitted with septic condition. An abdominal computed tomography scan diagnosed lienal vein thrombosis along its whole length and partial thrombosis of the superior mesenteric vein. **Conclusion:** SVT presents very heterogeneously, which makes it extremely challenging to diagnose and to make an appropriate treatment decision. With regard to the high prevalence of obesity and the increasing frequency of LSG, prompt diagnosis and management are crucial.

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### Introduction

Morbid obesity, defined as a BMI  $\geq 40$  kg/m<sup>2</sup> or a BMI  $\geq 35$  kg/m<sup>2</sup> in the presence of high-risk comorbid conditions [1], is one of the leading health problems worldwide. It increases the risk of type 2 diabetes, hypertension, cardiovascular disease, dyslipidemia, arthritis,

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nonalcoholic steatohepatitis, gallbladder disease, sleep apnea syndrome, and several types of cancer [2].

Laparoscopic sleeve gastrectomy (LSG) is rapidly gaining momentum in bariatric surgery, having the advantages of being less complex than other bariatric procedures and being associated with less malnutritive effects as well as comparable weight loss and resolution of comorbidities as Roux-en-Y gastric bypass [3].

Commonly reported complications of LSG include staple-line leak, respiratory insufficiency, pulmonary embolism, hemorrhage, stricture, and splenic injury [4]. Splanchnic vein thrombosis (SVT) is an unusual complication of LSG, which occurs in approximately 1% of cases [5, 6], and is, to our knowledge, described in only two case reports [7, 8]. The aim of this paper is to present a unique case of SVT after SLG in a morbidly obese woman which was complicated by septic condition.

### Case Report

A 37-year-old woman with a baseline BMI of 50.2 kg/m<sup>2</sup> and preoperative BMI 42.2 kg/m<sup>2</sup> as well as comorbidities (arterial hypertension, chronic obstructive pulmonary disease, dilatative cardiomyopathy, status post cholecystectomy) and amoxicillin-induced hypersensitivity was referred to our institution for LSG.

The standard procedure lasted 45 min under an insufflation pressure of 14 mm Hg, and the patient tolerated it well. A subtherapeutic dose of 9,500 IU anti-Xa/1,0 ml was used to prevent thromboembolic episodes. The postoperative course was uncomplicated, and the patient was discharged on the 4th postoperative day with structured instructions for postoperative food, proton pump inhibitors (PPI), polyvitamin supplements, and physical activity recommendations.

Three weeks later, she was readmitted due to the diffuse and cramp-like abdominal pain, vomiting, nausea, diarrhea, and fever with positive family history to acute abdominal viral disease. Physical examination revealed afebrile, distended, soft abdomen painless to palpation, absence of organomegaly or other masses, presence of bowel sounds, and healed trocar incision sites per primam. Complete blood count revealed increases in C-reactive protein (CRP) as well as in creatinine, urea, and aminotransferases (AST, ALT). Abdominal X-ray and ultrasonography (US) were both normal, and no X-ray contrast medium leakage was observed. The patient was treated with intravenous glucosaline infusions, analgetics (paracetamol), PPI (omeprazol), and antibiotic (ertapenem) in standard dosage of 1 g/day. In the following days, she remained asymptomatic and afebrile, with no other complications, and was discharged on hospital day 3.

One week later she represented to our facility with septic condition. On admission, abdominal computed tomography (CT) scan and US revealed lienal vein thrombosis along its whole length, partial thrombosis of the superior mesenteric vein (SMV), several hypoechoic lesions, suggestive of hepatic abscesses – especially the one in the IVb segment (19 × 16 mm in diameter) –, and splenomegaly with small hypoechoic lesions, suggestive of infarcts. Thoracic CT scan was normal, while the echography showed earlier signs of hypertrophic heart and minimal pleural effusion. The patient was immediately heparinized with low-molecular-weight heparine – Fraxiparine<sup>®</sup> 5700 anti-Xa IU/0.6 ml s.c. / 12 h and further on with Fraxiparine 7600 anti-Xa IU/0.8 ml s.c. / 12 h. Also the antibiotics imipenem and cilastatin were given followed by solved amoxicillin p.o., according to a local hospital protocol. Control US revealed patent vena portae in its distal two-thirds, but, on contrary, the distal part of SMV and Vena lienalis could not be assessed. In the following days, clinical examination as well as laboratory tests improved, and the patient was discharged on hospital day 13.

One week later, she was readmitted again with septic condition. Urgent abdominal CT scan showed small hepatic abscess (6 × 4 mm in diameter) in the IVb segment and several similar hepatic hypodense lesions, all of which were smaller than in the previously taken CT scan. Vena lienalis was thrombosed through its whole length, in contrast to patent Vena portae and SMV. US-guided percutaneous drainage procedure was performed to treat the largest hepatic abscess. The patient was treated with Fraxiparine 9500 anti-Xa IU/2 × 1 ml s.c. / 12 h, antibiotics piperacillin (Tazocine<sup>®</sup> 4.5 g / 8 h) and amoxicillin (Amoxiclav<sup>®</sup> 1.2 g / 8 h), antiulcer drug (PPI, pantoprazol) and intravenous glucosaline infusions with polyvalent vitamin formula.

On hospital day 11, she was discharged, but re-administered the day after due to the short episode of hematemesis, for which 4 units of packed red cells were transfused. After several administrations to ambulant antithrombotics and, finally, following the recommended regime with Fraxiparine 9500 anti-Xa IU/1 ml s.c. / 12 h, genital and gastrointestinal bleeding stopped. Gastroscopy showed no traces of blood down to the lower portion of the duodenum, and neither were any potential sources of bleeding detected. Due to clinical suspicion of septic emboli, a transthoracic echocardiogram (TEE) was performed, but no evidence for emboli was found. However, upon consultation with the infectiologists, new antibiotic therapy based on combination of cephalosporine and metronidazole was introduced with subsequent gradual decline in inflammatory markers. Also, analgetics as well as infusion solution of alanylglutamine (Dipeptiven®) and vitamin B12, along with multivitamin supplements, were administered. Control US showed a homogeneous and uniformly enlarged spleen (17 cm in diameter) and complete recanalization in the splanchnic system. The patient was discharged 13 days later in stable condition. An excellent late postoperative course could be observed, with a 5-month weight loss of 40 kg (over 70 EWL%), no metabolic complications, good physical performance, and improvement of muscular length and lean body mass detected in Tanita™ measurement system of body impedance.

## Discussion

SVT is an unusual manifestation of venous thromboembolism which involves one or more abdominal veins (portal, splenic, mesenteric and supra-hepatic veins) [9]. According to the literature, it may be associated with different underlying disorders, either local (abdominal malignancy, liver cirrhosis, pancreatitis, intraabdominal inflammation, surgery), is systemic (myeloproliferative neoplasms, oral contraceptives, increased factor VIII level, hyperhomocysteinemia, prothrombin gene mutation, protein C deficiency) or idiopathic [10].

In patients undergoing bariatric surgery, metabolic syndrome, chronic inflammation, venous stasis, and intraoperative manipulation of splanchnic vasculature are among the suggested causative factors for SVT [11].

The mechanism by which laparoscopic surgery increases the risk of development of splanchnic vessel thrombosis remains unclear [12–17]. However, the main factor that is adversely affected during laparoscopy is venous stasis [18]. Insufflating the abdomen with pressures greater than 14 mm Hg have been reported to reduce the portal blood flow by up to 53% [19, 20]. Insufflating with CO<sub>2</sub> may also contribute to reduced intraoperative splanchnic blood flow [21] by causing the release of vasopressin [16]. In addition, mesenteric vasoconstriction results from hypercapnia related to intraperitoneal CO<sub>2</sub> absorption, as described in experimental animal models [16, 22]. Other factors that favor thrombosis by interrupting flow include the reverse Trendelenburg position [23] due to compressive effect of abdominal viscera on the iliac veins [18]. In our patient, a standardized 14 mm Hg CO<sub>2</sub> insufflation in combination with reverse Trendelenburg position was utilized. Accuracy of surgical technique, reduction of the duration of surgery, and ideal anesthetic protocol using sugammadex for prompt reversal of neuromuscular block all reduce the risk for SVT [24].

Intraoperative surgical manipulation may damage the splanchnic endothelium and lead to local thrombus formation that may then propagate throughout the portal venous system. This may be particularly true for laparoscopic splenectomy, in which ligation of the splenic vein causes endothelial damage in proximity to the portal vein [25], but not in our case. Many other procedures may also lead to some manipulation of the splanchnic vasculature, but in our opinion intraoperative surgical manipulation during sleeve gastrectomy which only involves transection of the short gastric veins is less likely to be the cause of SVT, provided that surgical technique is good and no intraoperative bleeding occurs.

Metabolic syndrome resulting in a hypercoagulable state may also contribute to postoperative venous thrombosis in our patient as obesity promotes thrombosis through a variety of mechanisms, including adipocytokines, depression of the fibrinolytic cascade and augmen-

tation of the coagulation cascade, increased inflammation, and endothelial dysfunction [23, 26].

SVT occurs with broad spectrum of clinical presentations, ranging from incidental findings in an asymptomatic patient to concomitant presence of extensive thrombosis and gastrointestinal bleeding. Clinical presentations of acute SVT are most commonly seen approximately 2 weeks postoperatively. Different underlying factors can trigger SVT – among them also infectious diseases. Like in our case, patients initially complain of nonfocal abdominal pain, nausea, vomiting, and low-grade fever [27]. Laboratory values are typically within normal limits, but leukocytosis and mild elevation of liver function tests can also be seen [7], the former also noted in our patient. 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 [7]. Nonetheless, as shown in our case, SVT is a potentially life-threatening condition, in which an extensive thrombosis and subsequent intestinal infarction led to ascites, gastroesophageal hemorrhage, and septic shock.

For obese patients, CT is an excellent means to evaluate gastrointestinal or nonspecific complaints in the postoperative course of surgical procedures [26, 29]. Invasive procedures are usually not needed, unless there is a high degree of suspicion [30]. In our case, gastroscopy was performed for detection of any potential source of bleeding and exclusion of the presence of peptic ulcer and gastroesophageal varices. Although US is readily available and expedient, it has the lowest specificity for detection of SVT of available imaging techniques [31] and is best used to document restoration of venous flow in a patient with known SVT. Magnetic resonance imaging is highly sensitive and specific for SVT but not widely available [32]. In the absence of major contraindications, anticoagulant therapy is generally recommended for all patients presenting with acute symptomatic SVT, starting with either low-molecular weight or unfractionated heparin and continuing with the vitamin K antagonists in most patients [33, 34]. We began to treat SVT with high therapeutic dose of low-molecular-weight heparin, which was subsequently shifted to half a dose because of bleeding and then replaced by warfarin. Along with specific antibiotic therapy, the patient recovered completely. The recommended duration of anticoagulation treatment is 6–12 months [25]. Some studies suggested that the risk of thromboembolism following bariatric surgery extended long after discharge from the hospital, and prophylaxis should therefore be continued for several weeks into the postoperative period [35]. However, this aggressive approach, which could also theoretically lower the risk of SVT, is not considered standard [36].

## Conclusion

SVT is an uncommon complication of sleeve gastrectomy in morbidly obese patients. Clinical presentations are heterogeneous ranging from asymptomatic findings to bowel infarction and sepsis. As this condition is potentially life-threatening, the diagnosis should be prompt. For acute symptomatic SVT in patients without major contraindications, prompt anticoagulant therapy in therapeutic dose is the current standard. Also, doses of all medications are suggested to be calculated to actual BMI/body weight, especially those with fatty tissue-wide distribution. However, recommendations are derived only from observational studies, and further research is needed for fully delineation on the acute management of SVT after laparoscopic surgery.

## Disclosure Statement

No reported conflicts of interest.

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