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Portomesenteric vein thrombosis after laparoscopic sleeve gastrectomy

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Abstract
Introduction Portal and mesenteric vein thrombosis are relatively uncommon surgical complications, with difficult diagnosis and potentially severe consequences due to higher risk of bowel infarction. The purpose of this study was to present a series of patients who developed postoperative portal vein thrombosis after laparoscopic sleeve gastrectomy.
Methods This is a retrospective analysis of patients who underwent sleeve gastrectomy between June 2005 and June 2011 who developed portal vein thrombosis. Demographic data, personal risk factors, family history of thrombosis, and postoperative results of thrombophilia study were analyzed in this study.
Results A total of 1,713 laparoscopic sleeve gastrectomies were performed. Seventeen patients (1 %) developed portal vein thrombosis after surgery. Of the 17 patients, 16 were women, 8 had a history of smoking, 7 used oral contraceptives, and 2 had a family history of deep vein thrombosis of the lower limbs. All patients were discharged on the third day of surgery with no immediate complications. Symptoms presented at a median of 15 (range, 8–43) days after surgery with abdominal pain in most cases. One case required emergency laparotomy and splenectomy because of an active bleeding hematoma with massive portomesenteric vein thrombosis. In 11 cases, a thrombosis of the main portal vein was identified, in 15 the right portal branch was compromised, and in 10 the left portal branch. Eleven patients presented thrombosis of the superior mesenteric vein, and ten patients presented a concomitant thrombosis of the splenic vein. A massive PMVT was presented in six cases. Seven patients had a positive thrombophilia study.
Conclusions Portal vein thrombosis and/or mesenteric thrombosis are relatively uncommon complications in patients undergoing bariatric surgery. In this series, the portomesenteric vein thrombosis was the most common complication after LSG in a high-volume center.

Keywords Bariatric · Sleeve gastrectomy · Thrombosis · Portal · Mesenteric

Portal (PVT) and mesenteric vein thrombosis (MVT) are rare but potentially severe surgical complications, due to high risk of bowel infarction [1, 2]. The first is described in liver cirrhosis, hypercoagulability states, neoplasms, intra-abdominal sepsis, pancreatitis, and after surgical procedures [1, 2]. The MVT is mainly observed after liver transplantation, splenectomy, and other surgeries, including bariatric procedures [3–13].

Despite the fact that a prothrombotic state is a well-known issue in morbidly obese patients and that deep venous thrombosis (DVT) is a complication clearly described in this group of patients [14, 15], only a few publications of portal-mesenteric thrombosis after bariatric surgery can be found [16–24].

Laparoscopic sleeve gastrectomy is a procedure originally designed as the restrictive element of the biliopancreatic diversion with duodenal switch (BPD-DS) [25, 26]. Afterwards it was incorporated as the first stage in a two-stage procedure in order to complete a Roux-en-Y gastric bypass (RYGBP) or a BPD-DS in mega obese patients, [27,

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28] to decrease morbidity of the procedure performed in a single stage. LSG's good results regarding weight lost and low morbidity in this group of patients, promoted its incorporation as a definitive bariatric procedure in recent years [29, 30].

The most frequently described complications in this procedure are staple line leak and hemoperitoneum [31].

The objectives of this paper are to determine the incidence of PVT and MVT in a series of patients who underwent a LSG, analyze the characteristics of those patients, and evaluate the frequency of this complication.

Patients and methods

A retrospective study was conducted upon a prospective database, where all patients who presented a PVT or another vessel of the splanchnic territory after a LSG were identified. Demographic data, postoperative evolution, personal, and family risk factors for venous thrombosis, anticoagulant therapy outcomes, results from complete thrombophilia study, and late complications secondary to thrombosis were registered. Incidence of PVT in patients that underwent a LSG was analyzed.

Surgical technique

The patient is placed in a supine position with intermittent pneumatic compression during surgery. The surgeon stands at the right side of the patient and an assistant at the left side. Surgery is performed with a five trocar technique. The peritoneal cavity is accessed with a supraumbilical optical trocar. The pneumoperitoneum is insufflated to 15 mmHg. The gastric greater curvature is dissected with an ultrasound scissor Harmonic Ace™ (Ethicon Endo-Surgery, Guaynabo, Puerto Rico) or Ligasure Atlas™ (Tyco Healthcare, USA), starting 5 cm from the pylorus, ensuring preservation of the gastric antrum. A medial or lateral technique is used according to the surgeon preference. To calibrate the gastrectomy, a bougie ranging from 34 to 60 Fr, depending on surgeon preference, is placed along the lesser curve. The gastric section is performed with gastrointestinal staplers Echelon 60™ (Ethicon, Endo-Surgery, Guaynabo, Puerto Rico) o Endo Gia Universal™ (Autosuture, Tyco Healthcare) directed to the angle of His. The staple line is reinforced with a 2-0 Vicryl™ (Ethicon, Brasil) or 3-0 Monocryl™ (Ethicon, Brasil) continuous suture. The resected stomach is extracted through an enlargement of the left flank port side, inside an extraction Endobag™ (Autosuture, Tyco Healthcare).

Postoperative care

Patients remain the first 24 h with intermittent pneumatic compression and, according to protocol, with prophylaxis with low molecular weight heparins (LMW-H). Compression stockings are used during the entire hospitalization. At the first postoperative day, patients start walking and they start progressive oral intake with a liquid diet. If patients have a good evolution, they are discharged on the second or third day.

Postoperative complications were defined as those immediately after surgery and during the entire hospitalization.

Results

A total of 1,713 LSG were performed between June 2005 and June 2011. The most common complications were PMVT (1 %), staple line leaks (0.7 %), hemoperitoneum (0.4 %), and abdominal abscess (0.4 %). Of the 17 patients (1 %) who presented a portal-mesenteric thrombosis after LSG, 16 were women. Eight of the previous presented a smoking history, seven used oral contraceptives, and two had a family history of deep vein thrombosis of the lower limbs. The mean age and BMI in the patients with PVT were 38.1 ± 9.4 years and 37.4 ± 1.5 kg/m^2 respectively. The median surgical time was 75 (range, 60–130) min and hospital stay had a median of 3 (range, 2–5) days. In one case, a laparoscopic cholecystectomy was simultaneously performed. No conversion to open technique was necessary. All patients received Enoxaparin 40 mg subcutaneously daily starting 12 h after surgery until discharge. Patient characteristics are summarized in Table 1.

Clinical presentation was abdominal pain in 16 patients, malaise in 5 patients, and back pain in 3 patients. One patient was asymptomatic with a thrombus in the right branch of the portal vein that was found incidentally on an abdominal computed tomography (CT) scan 1 year after LSG. No patient presented signs of peritoneal compromise at physical examination. One patient required an emergency laparotomy and splenectomy because of an active bleeding splenic hematoma and massive PMVT [32]. The symptoms presented at a median of 15 (range, 8–43) days postoperative. All of these patients were evaluated with inflammatory parameters, liver, and pancreatic functional tests, all of which resulted normal.

All cases were diagnosed with an abdomen and pelvis CT with oral and intravenous contrast identifying a portal-mesenteric thrombosis of variable extension. In 11 cases, a thrombosis of the main portal vein was identified (Fig. 1A); in 15 the right portal vein branch was

Table 1 Patient characteristics

Case	Age (year)	Sex	BMI (kg/m^2)	Symptoms	Days from surgery	Comorbidities	Habits	Oral contraceptive	Thrombophilia
1	36	F	35.2	Vague abdominal pain	15	IR, HYP	None	No	Protein C deficiency
2	45	F	35	General malaise	9	IR, DLP, HYP	Tobacco use, occasional alcohol	Yes	Protein C deficiency + 20210a mutation
3	42	F	35	General					malaise + vomiting
21	HTN,		HYP	Tobacco use, occasional alcohol	Yes	No			
4	30	F	34.8	Epigastric pain	1	None	Tobacco use, alcohol	Yes	No
5	34	F	36.6	Vague abdominal pain + vomiting	10	IR, HYP	Occasional alcohol	No	No
6	45	F	35.1	Abdominal pain + bloating	11	HTN, HYP	Former tobacco use, occasional alcohol	No	No
7	26	F	32.5	Low back pain	6	IR, DLP	None	Yes	Protein C and S deficiency
8	44	F	36	Late vague abdominal pain	415	None	None	No	No
9	35	M	35.6	Epigastric pain	17	IR. DLP	Tobacco use	–	No
10	29	F	36.1	Vague abdominal pain	12	IR, HTN	Tobacco use, occasional alcohol	Yes	No
11	19	F	36	Epigastric pain	4	IR	None	Yes	Protein S deficiency
12	56	F	33.2	Right hypochondrium pain	2	IR, HYP, FL	None	No	No
13	46	F	36.8	Abdominal pain + bloating	14	IR, DLP, HTN	None	No	No
14	44	F	31.8	Back pain	4	IR, DLP, HTN, FL	Alcohol	No	No
15	28	F	33.2	Vague abdominal pain	18	T2DM	Tobacco use, alcohol	Yes	No
16	37	F	33.7	Vague abdominal pain	1	None	None	No	Protein C deficiency + 20210a mutation
17	51	F	30.5	Vague abdominal pain	11	IR, DLP, FL	Tobacco use	No	20210a mutation

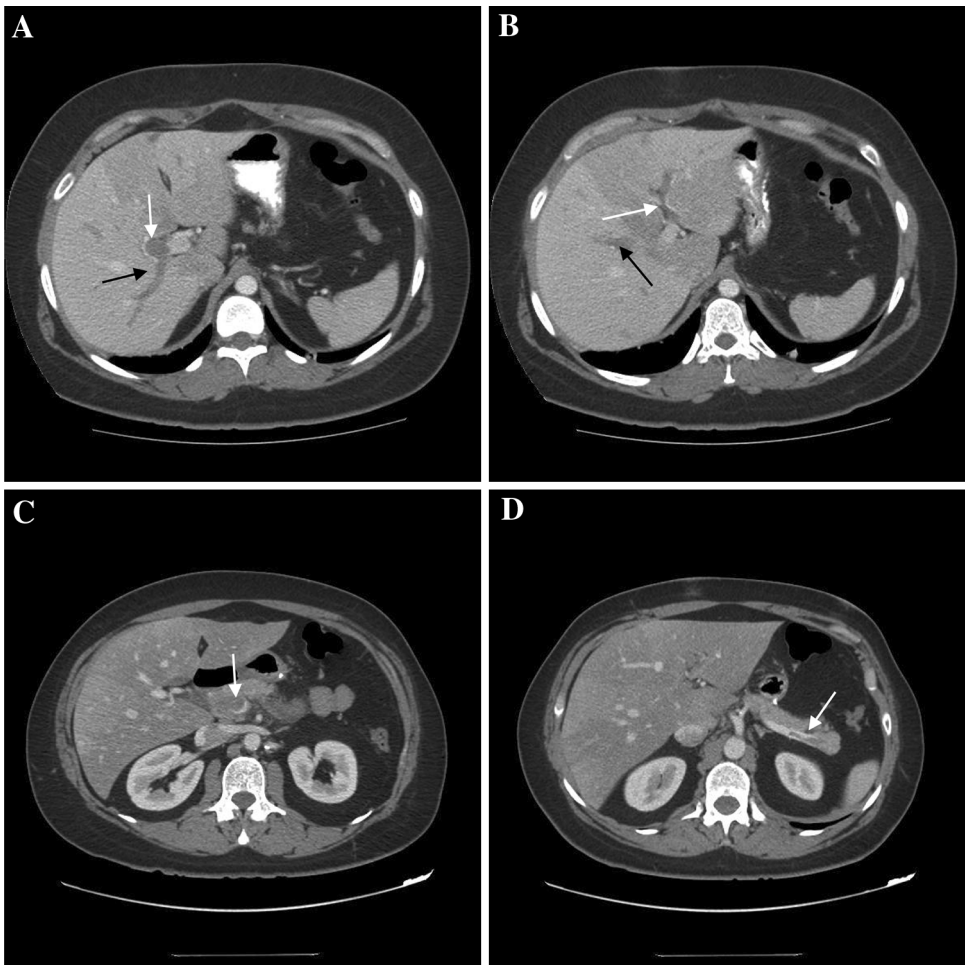

Fig. 1 Contrast-enhanced abdominal CT of patients with portal or mesenteric vein thrombosis. **A** *White arrow* indicates thrombosis of main portal vein and *black arrow* right portal branch. **B** *White arrow* indicates thrombosis of left portal branch, and *black arrow* portal branch thrombosis of the right anterior segment. **C** *White arrow* indicates superior mesenteric vein thrombosis. **D** *White arrow* indicates splenic vein thrombosis

compromised (1A), in 10 the left portal vein branch was altered (B), 11 patients presented thrombosis of the superior mesenteric vein (C), and 10 patients presented a concomitant thrombosis of the splenic vein (D). A massive PMVT was presented in 6 cases.

Patient management consisted of hospitalization, electrolyte replacement, intestinal rest, and anticoagulant treatment with intravenous unfractionated heparin before switching to oral anticoagulation with acenocoumarol when anticoagulation rank was reached. All patients evolved with remission of the symptoms within the first 48 h and oral intake was resumed progressively. All patients were discharged asymptomatic, after a mean hospitalization period of 6 (range, 5–7) days.

Thrombophilia study was completed in all patients at the moment of PVT diagnosis, with positive findings in only six patients. Two patients had a prothrombin gene mutation G20210A with Protein C/S deficiency, one patient had a protein C deficiency, one patient had a prothrombin gene mutation G20210A, one patient had a protein S deficiency, and one patient had a protein C and S deficiency.

At a median follow-up of 2.7 years the patients with a positive thrombophilia study, were on a lifelong anticoagulation regime. Five patients had portal cavernomatosis, one patient had portal cavernomatosis with portal hypertension signs, and one patient had partial recanalization. At a median follow-up of 3 years, the remaining ten patients were treated with oral anticoagulation for 1–1.5 years and currently six are completely recanalized, two have portal cavernomatosis with portal hypertension, one has portal cavernomatosis, and one has partial recanalization.

Discussion

There is abundant evidence about obesity as a risk factor for venous thrombosis, especially DVT of lower limbs [33], being patients undergoing bariatric surgery a high-risk group. The reported incidence of deep vein thrombosis and pulmonary embolism after undergoing bariatric surgery varies between 1.2 and 1.6 % and from 0.8 to 3.2 %, respectively [34]. However, PVT after bariatric

surgery is a very rare but potentially serious complication, especially considering its nonspecific clinical presentation and the high index of suspicion required for its diagnosis.

The etiology of this complication after bariatric surgery is probably multifactorial, adding elements, such as metabolic syndrome, undiagnosed thrombophilia, oral contraceptive use, and increased intra-abdominal pressure in laparoscopic procedures, among others. These factors may play a role in its genesis.

The MVT can be classified into primary and secondary according to its etiology. In primary cases, it is not possible to identify its cause. In secondary cases, there is an underlying etiology and currently, with the use of tests that evaluate hypercoagulable states, many of the causes until now unknown can be identified [18].

Metabolic syndrome, which is present in a significant proportion of patients undergoing bariatric surgery, generates changes in the coagulation pathway that increase the risk of developing clots. On one hand, it is associated with increased plasmatic levels of fibrinogen and factors VI and VII, which leads to a potential hypercoagulable state. On the other hand, it promotes increased levels of plasminogen activator inhibitor (PAI-1), which reduces the conversion of plasminogen into plasmin, resulting in a hypofibrinolysis state [35, 36]. All these factors could explain some reasons of the increased thrombotic events in obese patients.

The CO_2 insufflation and the secondarily increased intra-abdominal pressure during laparoscopic procedures produce different hemodynamic changes, among which is included the reduction of the splanchnic irrigation and portal venous flow, which promotes venous thrombotic events in splanchnic territory [37–39]. If the previous factors are added to the use of oral contraceptives, the presence of undiagnosed thrombophilias, and other factors, these could all combine to promote a splanchnic thrombotic event.

Multiple hypotheses have been put forward to explain this complication; perhaps the liver separation may cause blood stasis within the liver or the presence of a retrograde thrombosis might be another underlying factor. Other possible explanation could be a surgical trauma to the SMV, but dissection is performed adjacent to the greater curvature of the stomach and there is no direct trauma to the SMV. On the counterpart, we believe that the mechanical or thermal effect on the left gastroepiploic arcade or the short vessels during the skeletonization of the greater curvature could be a contributing factor.

Another issue to be considered is thrombosis prophylaxis. All of our patients are encouraged to ambulate early and a physical therapist supervises that this is done. On the first postoperative day patients wear an intermittent compression device and compression stockings on the following days. They also receive enoxaparin 40 mg daily 12 h after surgery. With these measures, in our series of 1,713 sleeve gastrectomies only 2 patients had DVT (0.11 %). We believe that these measures are adequate in preventing vein thrombosis. There must be other factors that determine the high incidence of PMVT. The genesis of this disease must be different of that of DVT.

The incidence of PMVT in our series is much higher than the published literature, but our surgical technique and patient care do not significantly differ from the published description by other authors. We do not have a convincing explanation about this matter, and there may be other unknown factors in our population to explain this incidence.

Early detection with high index of suspicion is crucial to treat this complication, because symptoms are generally vague and physical examination findings are nonspecific, presenting peritonitis and shock only in a minor frequency. The most common symptom is abdominal pain associated with nausea and vomiting; vital signs and laboratory tests often are normal. Symptoms usually presents within the first 45 days of surgery [18, 21–24]. The extension of the thrombosis into the superior mesenteric vein is not uncommon and if not detected and treated early it can lead to intestinal infarction, perforation, and secondary peritonitis.

A late detection and treatment may condition an organization of the thrombus, with a secondary portal cavernomatosis. The latter would result in all the potential complications of portal hypertension, mainly in variceal gastrointestinal bleeding [35]. The imaging method of choice for diagnosing this entity is the contrasted abdomen CT with a sensitivity of 90 % [18, 40, 41]. An acute thrombosis appears as a central opacity in the mesenteric vein as seen in most of our patients. A bowel dilatation and edema or thickening of the fat, although not conclusive, should put in consideration the diagnosis of MVT in absence of other obvious clinical etiology [18]. Other diagnostic modalities include magnetic resonance imaging (MRI), mesenteric angiography, and Doppler ultrasound [21]. MRI has excellent sensitivity and specificity but is not always available and requires time for its completion; the mesenteric angiography is not helpful in the diagnosis, because it requires delayed images [18].

Once diagnosis is made, treatment should be started immediately. If it is done during surgery, proper resection of compromised intestines followed by anticoagulation is the recommended alternative, and a second look at 24–48 h also is suggested if resection is done [23]. In case of no evident ischemic compromise of the bowel and if the patient is in a stable condition, the anticoagulation may be the only necessary treatment, reserving surgery for patients who develop signs or symptoms of acute abdomen or

progression to transmural necrosis or perforation [18, 22, 23]. Even when diagnosis is made by imaging methods, the importance of laparoscopy should not be underestimated. In addition to confirming the diagnosis of intestinal infarction, it also can determine the extent of the ischemia and the need for resection, allowing the surgeon to perform a second look, and guiding the decision between continuing observation and anticoagulation versus surgical intervention [21, 23]. In our experience, no patient developed signs or symptoms of acute abdomen and all cases remained hemodynamically stable. There were no signs of ischemic compromise in the abdominal CT. This is why we adopted a treatment of observation, bowel rest, and heparin anticoagulation in all patients, with the only exception of a patient operated because of an active bleeding hematoma.

In our series, all cases corresponded to an acute PVT or MVT. Although spontaneous thrombus recanalization has been reported in cases like this, it is very rare, and all patients should be treated with anticoagulation therapy. The limited available evidence suggests that long-term treatment with anticoagulant therapy could achieve permeability in more than 80 % of the cases. Experts' recommendation is to maintain treatment for 6 months if there is no identifiable thrombophilia [42] and keep it indefinitely in case no treatable thrombophilia is identified. This conduct has been followed in this case series.

Conclusions

The portal and/or mesenteric thrombosis are the most common complications in our patients after laparoscopic sleeve gastrectomy. Early diagnosis with contrasted CT and immediate anticoagulation in stable patients without peritoneal compromise or image findings suggestive of bowel ischemia could prevent acute complications secondary to thrombosis, such as intestinal infarction.

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