A retrospective comparative study of portomesenteric venous thrombosis after laparoscopic sleeve gastrectomy in morbidly obese patients with low BMI with and without anticoagulation

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Received: 25 June 2022
Revised: 14 July 2022
Accepted: 02 August 2022
Published: 05 April 2023

The Egyptian Journal of Surgery 2023, 41:1035–1039

Background
Portomesenteric venous thrombosis (PMVT) is a life-threatening rare postoperative complication after laparoscopic sleeve gastrectomy (LSG). Thromboprophylaxis is essential to guard against venous thrombosis after laparoscopic surgery. This study points out the benefits of postoperative use of anticoagulation to prevent PMVT after LSG in morbidly obese patients with BMI of 35–45 kg/m^2.
Patients and methods
A retrospective comparative pilot study was conducted that included 60 patients with;Deg;BM;Deg;I between 35 and 45 kg/m^2 who underwent LSG between January 2020 and January 2021 at El-Demerdash Hospital. Half of the patients received antithromboembolic dosage scheme of 0.5 mg/kg/day with the induction of anesthesia and for 14 days postoperatively (group A), whereas the other half had no postoperative anticoagulation prophylaxis (group B).
Results
Patients were followed up with lower limb venous duplex at 2-week, 3-, and 6-month interval from the operation or on appearance of symptoms of PMVT. No patient had PMVT in group A, whereas four patients had PMVT in group B confirmed by venous duplex.
Conclusion
The anticoagulated group showed better outcomes regarding the prevention of PMVT after surgery. Yet, the incidence of PMVT after LSG in patients with BMI of 35–45 kg/m^2 was not affected only by anticoagulation. Evidence about PMVT prevention and thromboprophylaxis plan is still lacking, so we recommend further large prospective trials.

Keywords:
anticoagulation, laparoscopic sleeve gastrectomy, morbidly obese, portomesenteric venous thrombosis

Egyptian J Surgery 2023, 41:1035–1039

1110-1121

Introduction

Postoperative portomesenteric venous thrombosis (PMVT) is a life-threatening complication reported after bariatric laparoscopic sleeve gastrectomy (LSG), with an incidence rate of 0.3–1% [1].

Presenting symptom can be nausea, vomiting, or abdominal pain that could be central or epigastric or even diffuse, so a high index of suspicion is important for diagnosis [2].

Hereditary and acquired thrombophilia, trauma to portal venous system or abdominal inflammation, decreased portal flow as in reversed Trendelenburg position or increased intra-abdominal pressure, direct trauma to portomesenteric system or tributary ligation (splenic or short gastric), and postoperative dehydration are all possible causes of PMVT after LSG [3,4].

Once PMVT is suspected, diagnosis is confirmed by contrast computed tomography (CT) abdomen with mesenteric angiography [5,6]. Doppler ultrasound is recommended as well [3,4].

After establishment of diagnosis, treatment is mandatory either conservative with anticoagulation or surgical if progressed to bowel ischemia or peritonitis develops [7,8].

No appropriate regimen or protocol has been agreed upon yet regarding the use of prophylactic thromboprophylaxis after discharge of LSG patients for prevention of postoperative PMVT [9].

In this study, we aimed to elucidate the preference of postoperative prophylaxis with anticoagulation to prevent postoperative PMVT in morbidly obese patients undergoing LSG with BMI of 35–45 kg/m^2.

Patients and methods

Our study included 60 morbidly obese patients who underwent LSG over 12 months from January 2020 to January 2021. This research was performed at the Department of General Surgery, Ain Shams University Hospitals. Ethical Committee approval and written, informed consent were obtained from all participants.

The age of the patients was more than or equal to 19 years with a preoperative BMI of 35 or 35–40 kg/m^2 with comorbidities (hypertension, dyslipidemia, type 2 diabetes mellitus, sleep apnea, etc.) or with a preoperative BMI of 40–45 kg/m^2 with or without comorbidities.

Then, the study population was divided into two groups: group B was not subjected to any chemical or mechanical thromboprophylaxis and early ambulation was encouraged, and group A was subjected to only chemical thromboprophylaxis in the form of low-molecular-weight heparin (LMWH) (0.5 mg/kg IU/day) subcutaneously postoperatively for 2 weeks and early ambulation was encouraged.

Inclusion criteria

The inclusion criteria were morbidly obese patients with BMI from 35 to 45 kg/m^2 who underwent LSG at Ain Shams University hospitals aged 19–55 years old with no past medical history, patients who were not currently or recently had any anticoagulation treatment, patients who were radiologically free preoperatively as confirmed by Doppler abdominal ultrasound and portal venous duplex.

Exclusion criteria

Patients with autoimmune disease or genetic hereditary diseases (protein C and S deficiency, factor V Leiden, etc.), hematologic disease, neoplasms, and chronic renal or hepatic disease; all were excluded by history and clinical evaluation. Patients with history of thrombotic or thromboembolic events, patients with hemophilia, and patients who were at risk of bleeding or thrombophilia were excluded as well.

All participants included in the study were subjected to the following.

Preoperative assessment was as follows:

(1) History taking, with a focus on risk factors of venous thrombosis, for example, diabetes mellitus, hypertension, ischemic heart disease, hyperlipidemia, and old Cardiovascular system (CVS).
(2) Preoperative routine investigations such as Chest x-ray (CXR), pelvi abdominal ultrasound (PAUS), respiratory function tests, complete blood count (CBC), prothrombin time (PT), international normalized ratio (INR), partial thromboplastin time (PTT) liver profile, kidney profile, and thyroid profile.
(3) Preoperative Doppler abdominal ultrasound and portal venous duplex being free of abnormalities.

Surgery

All of the patients underwent LSG under general anesthesia and complete aseptic conditions, and operative time was obtained as well as good intraoperative hydration with intravenous fluids.

Postoperative assessments were as follows:

(1) All patients were encouraged early ambulation within two hours after recovery.
(2) All patients were subjected to good hydration during the hospital stay and after discharge.
(3) All patients were discharged on clear fluid intake for a 10-day duration.
(4) Documentation of discharge time.
(5) Documentation of early postoperative major complications, for example, leakage and bleeding.
(6) All patients were followed up postoperative by history taking of any complaints, for example, abdominal pain, nausea, vomiting, and gastrointestinal bleeding.
(7) The postoperative period was assessed for anticoagulation intake, early ambulation after surgery, sufficient fluid intake, and symptoms of PMVT in the 6–12-month follow-up.
(8) Postoperative duplex was done showing portomesenteric venous abnormalities.

Surgical and clinical data collection from patients' files

Data of enrolled patients from both groups were collected from hospital files in the form of sex, BMI, age, preoperative laboratory investigations, preoperative RBS, preoperative cortisol, liver function tests (LFTs), kidney function tests (KFTs), coagulation profile, thyroid profile, and preoperative hemoglobin. Moreover, preoperative radiology, CXR, PAUS, and portal vein duplex were done.

Patient questioning postoperatively

All of the questioned patients were discharged on the second postoperative day with a liquid diet advised for 10 days. Oral clear liquids were allowed on the second day of surgery; 30 ml was given every hour in the early

morning, and if the patient was tolerating it, only clear fluids were administered in the evening for 10 days. On the first or second postoperative day, the patient was discharged after removal of the intraperitoneal drain.

At the outpatient clinic follow-up after surgery, patients were questioned about postoperative anticoagulation intake (if yes, type and duration), postoperative dehydration, postoperative hemorrhage, postoperative leakage (fever, tachycardia, and tachypnea with elevated heart rate), postoperative stricture (dysphagia, nausea, and vomiting), and postoperative PMVT symptoms (abdominal pain, nausea, vomiting, and upper or lower gastrointestinal bleeding).

Statistical analysis

Data were collected, revised, coded, and entered into the Statistical Package for the Social Sciences (IBM SPSS, statistics for windows, Armonk, NY: IBM Corp), version 23. The quantitative data were presented as mean, SD, and ranges when parametric and median and interquartile range when data were found nonparametric. Moreover, qualitative variables were presented as numbers and percentages.

The comparison between groups regarding qualitative data was done using the χ^2 test and/or Fisher exact test when the expected count in any cell was found less than 5.

The comparison between two groups regarding quantitative data and parametric distribution was done using an independent *t* test, whereas assessment of nonparametric distribution was done using the Mann–Whitney test.

The confidence interval was set to 95%, and the margin of error accepted was set to 5%. So, the *P* value was considered significant as follows:

P value more than 0.05: nonsignificant.
P value less than 0.05: significant.
P value less than 0.01: highly significant.

Results

The is a comparative retrospective pilot study that included 60 patients who were divided into two groups: group A included 30 patients and group B included 30 patients. Group B had not taken postoperative thromboprophylaxis. Group A was subjected to postoperative chemical thromboprophylaxis in the form of LMWH according to body weight. All patients had the same average operative time, early ambulation, and good hydration. Data were presented, and suitable analysis was done according to the type of data obtained for each parameter (Tables 1–3).

Discussion

There are numerous theories regarding why patients develop PMVT in general. Local and systemic factors include cirrhosis (28%), primary hepatobiliary cancer (23%), and secondary malignancy of the hepatobiliary region (44%). Local factors include pancreatitis and postsurgical (e.g. liver transplant or splenectomy) or portal vein compression by nodes. Laparoscopic surgery is also a risk factor, with the suggested contributing factors of venous stasis from increased intra-abdominal pressure, intraoperative manipulation of splanchnic vasculature, and systemic hypercoagulable states [3].

Several theories explain the increased risk of a PVT associated with LSG. These can be divided into intraoperative factors and postoperative factors. Intraoperative factors include ligation of the right gastroepiploic vessels with energy devices near the splenic vein. The mechanical or thermal effect can potentially cause thrombosis. Ligation of short gastric vessels changes the venous return from the stomach. This may be a factor in PMVT formation and has previously been suggested as the causative factor in the formation of PMVT after laparoscopic fundoplication [9].

Prolonged liver retraction could cause congestion and stasis within the liver, causing a clot to form.

Table 1 Demographic data

	N=60
Sex [*n* (%)]	
Female	45 (75.0)
Male	15 (25.0)
Age	
Mean±SD	40.15 ± 11.44
Range	23–65
BMI	
Mean±SD	39.62 ± 3.13
Range	35–45

Table 2 Comparison between two groups in anticoagulation and portomesenteric venous thrombosis

	N=60
Anticoagulation [*n* (%)]	
Yes (LMWH)	30 (50.0)
No anticoagulation	30 (50.0)
PMVT [*n* (%)]	
No	56 (93.3)
Yes	4 (6.7)

LMWH, low-molecular-weight heparin; PMVT, portomesenteric venous thrombosis.

Table 3 Comparison between two groups in anticoagulation and portomesenteric venous thrombosis incidence

	Anticoagulation		Test value*	*P* value	Significance
	Yes (LMWH) *N*=30	No anticoagulation *N*=30			
PMVT [*n* (%)]					
No	30 (100.0)	26 (86.7)	4.286	0.038	S
Yes	0	4 (13.3)			

LMWH, low-molecular-weight heparin; PMVT, portomesenteric venous thrombosis; S, significant. *χ^2 test. *P* value more than 0.05: nonsignificant; *P* value less than 0.05: significant; *P* value less than 0.01: highly significant.

Postoperative factors include PMVT, usually presenting after the patient has been discharged. This may in part be owing to dehydration after discharge. Hypovolemia is a known risk factor for developing thrombosis, including Deep venous thrombosis (DVT), pulmonary embolism (PE), and PMVT. The clinical features of acute PMVT are poorly defined in the literature. Throughout, the presentation of PMVT has been described as vague, with typical symptoms, such as abdominal pain, nausea, and fever. The severity of symptoms varies significantly and may be associated with the extent of mesenteric venous thrombosis because of bowel ischemia [10].

The broad spectrum of clinical presentations of PMVT ranges from incidental findings, in an asymptomatic patient, to life-threatening bowel infarction. Leukocytosis and mild elevation of liver function tests are also observed. Thus, physical examination can be normal, or patients could present with peritonitis and septic shock if associated with bowel ischemia. CT enhancement with oral and intravenous contrast has been reported in published studies to diagnose and monitor the patient's course with a sensitivity of 90% [9].

Diagnosis can be made using color Doppler ultrasound, contrast-enhanced CT, or magnetic resonance angiography. At present, this modality is less sensitive than CT and magnetic resonance to detect splanchnic vein thrombosis, particularly the splenic and superior mesenteric veins [9].

PMVT in Doppler ultrasound allows direct evaluation of mesenteric and portal veins, provides semiquantitative flow information, and permits Doppler waveform analysis of the visceral vessels but is limited by operator dependency, insensitivity to slow flow, and absence of suitable acoustic window if the overlying bowel gas is present [5].

Studies have shown that anticoagulation may result in recanalization. There is no consensus regarding the duration and extent of anticoagulation. We advocate for the guidelines for the duration and degree of anticoagulation; they suggest it is practicable to adopt the DVT management algorithm. This suggests that where a self-limited cause has been found (i.e. after surgery), a 3–6-month course of warfarin with an international normalized ratio of 2–3 is reasonable [10].

There is very little literature regarding the prevention and prophylaxis of PMVT after LSG. However, like the guidelines on treatment, prophylaxis would likely be guided by that of deep venous thrombosis. Venous thromboembolism remains among the leading causes of mortality after bariatric procedures, with evidence of DVT in 1–3% of patients and PE in 3–2%. The mortality of bariatric patients with PE was as high as 30%. Of concern, in a small series of 10 autopsies performed on patients who died after bariatric procedures, although only 20% of patients were clinically suspected of having died from PE, up to 80% of patients had microscopic evidence of pulmonary emboli, despite being on appropriate prophylaxis [7,11].

A high index of suspicion is maintained after bariatric surgery to detect and promptly treat PMVT. All bariatric surgical patients complaining of nonrevolving abdominal discomfort or pain in the postoperative period should be evaluated for PMVT by noninvasive imaging [2,4].

Once a diagnosis of PMVT is made, treatment should be promptly started. An exploratory laparotomy is required with possible resection of the necrotic bowel in patients presenting with peritonitis or shock. Full anticoagulation with either subcutaneous LMWH or intravenous unfractionated heparin is applied in patients who do not develop bowel ischemia or necrosis. In patients with a recent portal or mesenteric vein thrombosis treated with anticoagulation, follow-up imaging data and clinical outcome appear favorable [7,10].

This treatment is continued and changed to oral anticoagulation (target international normalized ratio, 2.5–3), which should be continued for several months; the duration of this treatment will depend on the coagulation profiles and a hematologic consultation. Following prompt anticoagulant therapy, early diagnosis and treatment of the underlying causal factors (abdominal sepsis and prothrombotic factors)

could lead to a dramatic decrease in the incidence of extrahepatic portal hypertension [8].

As bariatric surgery grows in volume, PMVT may increasingly need better prophylaxis, screening, treatment, and follow-up. Prophylaxis is routinely perioperatively administered as a standard prevention measure for DVT at most bariatric centers, and many different regimens are used regarding the time of initiation (presurgery, anesthesia induction, intraoperatively, and postsurgery), a specific pharmacologic agent used, dosage, and length of treatment (during hospital stay until discharge and up to 2 weeks after surgery). However, there is no consensus concerning PMVT prophylaxis. The current experience is nascent, but growing evidence could lead to the routine surveillance of PMVT in the postbariatric surgical patient. The ideal duration of postevent anticoagulation is yet to be defined [9,10].

PMVT is a rare complication after laparoscopic bariatric surgery. Smoking history was a predominant risk factor for PMVT, and abdominal pain was the main symptom. Early diagnosis and appropriate management are essential in preventing catastrophic complications such as intestinal infarction and corresponding increases in morbidity and mortality.

CT of the abdomen and pelvis is the proven method of choice for PMVT diagnosis. Anticoagulant therapy is generally satisfactory to avoid thrombosis progression and to achieve partial or complete recanalization. However, careful follow-up is necessary to evaluate the effect of PMVT on long-term patient outcomes.

Further research into thrombolysis and anticoagulation after discharge is recommended.

Conclusion

Patients who underwent LSG had low risk for developing PMVT, with average operation time and early ambulation. Anticoagulation with LMWH showed better outcomes in prevention of PMVT after surgery. There is paucity of literature regarding PMVT prevention and postoperative thromboprophylaxis, so we recommend further large prospective trials.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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