Porto-Mesenteric Venous Thrombosis (PMVT) Following Laparoscopic Bariatric Surgery: Incidence, Clinical Presentation, Analysis of the etiology and Management in 1434 Cases

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Background: Porto-mesenteric venous thrombosis (PMVT) is a rare but severe surgical complication developing in patients who underwent laparoscopic bariatric surgery; with difficult diagnosis and potentially severe consequences due to higher risk of bowel infarction. Its clinical presentation, management, and sequelae remain poorly understood. We aimed to describe the incidence, clinical features, management and outcome, in patients with PMVT after laparoscopic bariatric surgery.

Patients and methods: This was a retrospective analysis of patients who underwent laparoscopic bariatric surgery for morbid obesity between October 2014 and September 2016 who developed PMVT. Age, sex, body mass index(BMI), personal risk factors for thrombosis, family history of thrombosis, surgical technique, thrombo-embolic prophylaxis, primary surgery outcomes, clinical features and long term postoperative follow up findings were analyzed in this study. The diagnosis was established with an abdominal computed tomography (CT) scan as well as duplex ultrasound of the portal venous system. All patients received long-term anticoagulation.

Results: Of 1434 patients who underwent laparoscopic bariatric surgery; 947 patients underwent laparoscope sleeve gastrectomy (LSG) while 487 underwent laparoscopic mini-gastric bypass (LMGBP). 4 patients of those who underwent LSG (0.42%) developed PMVT. On the other hand no patients experienced PMVT following LMGBP. Three patients were males, the mean age was 34 years, and the mean body mass index was 43 kg/m^2. The time of onset of symptoms was within one to two weeks post operatively in 3 cases while one case (the female patient) presented late after 86 days and the presentation was atypical and rapidly progressive and the patient died within two weeks. New-onset epigastric pain was present in all patients, while other signs and symptoms were variable. Ultrasonography and computed tomography scan were performed and were diagnostic in all cases. The decision regarding the type of drug and the duration of anticoagulation therapy was based on hematology consultant assessment as regards patients' clinical course, result of thrombophilic evaluation, presence of other thrombotic risk factors and follow-up Doppler studies. One patient underwent surgical intervention: laparotomy with splenectomy and necrotic small-bowl resection. One patient died.

Conclusion: PMVT is a rare but serious complication after LSG. Familiarity with this dangerous entity is important. It requires early diagnosis and management as these cases carry significant morbidity and mortality. Prompt diagnosis and anti- coagulation therapy led to favorable outcomes in most cases. Significantly lower rates of thrombosis were found in patients who received an extended course of anticoagulation.

Key words: Bariatric surgery; laparoscopic; portomesenteric vein thrombosis, sleeve gastrectomy, complications.

Introduction

Portomesenteric venous thrombosis (PMVT) is an infrequent rare event usually associated with higher rates of morbidity (mesenteric ischemia in 5–15% of cases) and mortality (20–50%).[1] PMVT has been implicated after laparoscopic surgery.[2] Patients undergoing bariatric surgery are at a higher risk for venous thromboembolism (VTE) due to the underlying inflammatory and hypercoagulable states. Also metabolic syndrome may predispose patients to VTE.[3] It has been suggested that venous stasis secondary to enhanced intra-abdominal pressure and operative intervention in splanchnic vasculature are the possible causative factors for PMVT. Moreover, the diagnosis of PMVT after laparoscopic sleeve gastrectomy (LSG) requires a high index of suspicion as patients usually present with vague symptoms and signs that are difficult to elicit early.[1]

Patients and methods

This was a retrospective analysis of patients who underwent laparoscopic bariatric surgery for morbid obesity between October 2014 and November 2016 who developed porto-mesenteric venous thrombosis as a procedure related complication.

The following variables were collected; patients demographic characteristics (Age, sex), body mass index(BMI), personal risk factors for thrombosis, family history of thrombosis, surgical technique, thrombo-embolic prophylaxis, primary surgery outcomes, surgical events if any, surgery duration, clinical features and long term postoperative follow up findings were analyzed in this study. The diagnosis was established with an abdominal computed tomography (CT) scan as well as duplex ultrasound of the portal venous system. All patients received long-term anticoagulation. In our study, we identified a limited number of cases describing PMVT following laparoscopic bariatric surgery as an uncommon complication.

Surgical technique:
In our study all the patient received a prophylactic dose of low molecular weight heparin (LMWH) 12 hours prior to surgery and broad spectrum antibiotic was given with induction of anesthesia. Adequate hydration intra and postoperatively was done. Elastic stockings were used before induction and continued during the entire hospitalization.

Under general anesthesia all patients underwent an uncomplicated LSG in reverse Trendelenburg supine position. A 5 port technique was used. Pneumoperitoneum was created using the closed access technique by a Veress needle, with a maximum preset pressure of 15mmHg. A liver retractor was placed through a 5-mm incision in the epigastrium. The gastrocolic omentum and gastrosplenic ligament were divided using a vessel sealer and divider (LigaSure Atlas®; Covidien Ltd., Norwalk, CT, USA). Gastric resection was performed using a gastrointestinal flexible endo-stapler (EndoGIARoticulator®; Covidien Ltd., Norwalk, CT, USA). The resected stomach was removed through the port site. Each operation was performed over a 36F bougie catheter, beginning the sleeve 2 cm proximal to the pylorus.

The surgical technique of laparoscopic mini-gastric bypass (LMGBP) involves placement of five laparoscopic ports. The stomach is divided at the junction of the body and the antrum at the level of the crow's foot with 45-mm Endo-GIA stapler to get the longest possible gastric pouch. A lesser curvature-based tube of stomach is constructed with a 60-mm linear stapler using 3.5- mm blue cartridges (EndoGIARoticulator®; Covidien Ltd., Norwalk, CT, USA) around an orogastric tube of 36 Fr size. A jejunal loop, 200 cm distal to the ligament of Treitz, was then brought up antecolic and anastomosed to the stomach tube with 45-mm Endo-GIA stapler. The common stapling defect was closed over the nasogastric tube with two layers of No 2-0 absorbable V-LocTM suture (Autosuture Division of Covidien, USA) in a running fashion.The anastomosis was then tested with methylene blue injected through the nasogastric tube.

Patients received postoperative fluids to maintain a urine output > 50 ml/hr. Early ambulation was required. Patients were discharged by the first post- operative day taking adequate oral fluids (>2 L/d). Treatment with the low-molecular-weight heparin (LMWH) enoxaparin (Clexane®; Sanofi-Aventis, Paris, France) at 40 mg per day was initiated 12 hours preoperatively and continued daily for 7 days. Patients started walking within 4–6 hours of surgery with assistance. All Patients who developed PMVT underwent follow-up computed tomography (CT).

Results
Among the 1434 laparoscopic bariatric surgeries performed, 947 (66%) patients underwent LSG between October 2014 and September 2016; four (0.42%) of these patients developed PMVT. While those who underwent laparoscopic mini-gastric bypass (LMGBP); 487 (34%) did not develop PMVT. The mean age was 34 years (range 32–37 years). Three patients were men. The mean BMI was 43 kg/m2 (range 41–45 kg/m2). Two patients had a history of smoking, the female patient was on hormonal contraceptives pills, and none had a history of previous thrombosis or family thrombophilia records. The median surgical time was 58 minutes (range 43–67 min) with a hospital stay of 1 day. During the LSG procedure for these 4 patients, no complications were observed, and no conversion to open technique was necessary. The patient characteristics are summarized in **Table 1**.

Mechanical and pharmacologic thromboprophylaxis was provided for all patients. Routine prophylaxis included the application of a thigh-length elastic stocking at the time of surgery and until the patient recovered. Our protocol also applied early ambulation; patients started walking within 4–6 hours of surgery with assistance. Patients were discharged by the second post- operative day taking adequate oral fluids (>2 L/d). Treatment with the low-molecular-weight heparin (LMWH) enoxaparin (Clexane®; Sanofi-Aventis, Paris, France) at 40 mg per day was initiated 12 hours preoperatively and continued daily for 7 days. No complications were observed during hospitalization.

Table 1: The characteristics of the patients who developed PMVT. HTN: hypertension, OSA: obstructive sleep apnea, OCP: oral contraceptive pills, OR: operation room

Case	Age	Sex	BMI	Comorbidities	Thrombosis history	Thrombotic risk factor	Family history	OR time	Post operative stay
1	33	Male	41	HTN, fatty liver, dyslipidemia	Negative	Non	Negative	43	1 day
2	37	Male	45	HTN, faty liver, dyslipidemia, OSA	Negative	Smooking	Negative	65	1 day
3	32	Male	44	faty liver, dyslipidemia, OSA	Negative	Smooking	Negative	57	1 day
4	34	Female	42	faty liver, dyslipidemia	Negative	OCP	Negative	67	1 day

The time to PSMVT diagnosis after surgery was variable (Range, 7–86 days). All of the patients with PMVT presented with acute conditions. Presenting symptoms included new onset epigastric pain (4 patients, 100%), nausea, vomiting, anorexia, fever (2 patients, 50%) and dysentery (1 patient, 25%). On examination, abdominal tenderness was noted in all patients. Ultrasonography was used for the four patients, and positive results were found for all of these patients. Portal and mesenteric CT scan was performed and was diagnostic in all cases. The clinical features are summarized in **Table 2**. Patients were treated by anticoagulation with either heparin infusion for 5 to 7 days or subcutaneous treatment with the low-molecular-weight heparin (LMWH) enoxaparin at 1 mg/ kg twice daily then we shift to oral thrombolytic medication. The treatment details for PMVT and its outcome are summarized in **Table 3**. The decision regarding the type of drug and the duration of anticoagulation therapy was based on hematology consultant assessment as regard patients' clinical course, result of thrombophilic evaluation, presence of other thrombotic risk factors and follow-up Doppler studies.

Case 1: Patient presented 12 days after LSG with severe abdominal pain. Pain did not improve with analgesics even morphia. Pelviabdomial CT and CT portal and mesenteric angiography were done. There was portal vein thrombus extending to superior mesenteric vein. We started treatment with LMWH 1 mg/ kg twice daily. The patient was discharged on Warfarin.

Table 2: The clinical features of the patients who developed PMVT.WBC: white blood cell count, CRP: C reactive protein, LFT: liver function test, PVT: portal vein thrombosis, SMVT: superior mesenteric vein thrombosis, SVT: splenic vein thrombosis, CT: computed tomography, U/S: ultrasonography

Case	Time of onset of symptoms	Presentation	Wbc	CRP	LFT	U/S & DUPLEX	CT
1	12	Agonizing epigastric pain	7 300	48	Normal	PVT	PVT, SMVT
2	10	Severe abdominal pain, fever and hypovolemic shock (Hg % 8 gm)	18 000	156	Elevated	PVT & ruptured splenic hematoma	PVT, SMVT, SVT, ruptured splenic hematoma
3	7	Mild abdominal pain, dysentery and mild bleeding per rectum	9 500	60	Normal	PVT	PVT, SMVT
4	86	Mild epigastric pain, fever 40 c and irreversible septic shock	11 400	261	Elevated	Splenic abscess 4x3.5 cm	PVT, splenic abcess

Table 3: The treatment details and outcome of PMVT.LMWH: low molecular weight heparin, ICU: intensive care unit

Case	Anticoagulation therapy	Length of stay	Duration of antithrombolytic therapy	Thrombophilia screening	ICU admission	Radio intervention	Surgical intervention	Survival
1	LMWH	7 Days	6 Months	Negative	No	No	No	Alive
2	LMWH	33 Days	12 Months	Negative	Yes	No	Exploration spleenectomy, resection anastomosis	Alive
3	LMWH	9 Days	6 Months	Protein c Defficiency	Yes	No	No	Alive
4	Heparin infusion	10 Days	Non	Negative	Yes	U/S guided drainage of splenic abscess	No	Died

Case 2: Patient presented 10 days after LSG with severe abdominal pain and hypovolemic shock (Hg % 8 gm). Patient was admitted to ICU. Patient was resuscitated and pelviabdomial CT scan was done. CT scan showed ruptured spleen. Splenectomy was done. There was large ruptured subcapsular hematoma at the upper pole of the spleen **(Figure 1)**. CT portal and mesenteric angiography scan revealed portal vein thrombus extending to superior mesenteric and splenic veins. We started treatment with LMWH 48 hours after splenectomy. We noticed greenish discharge from the drain 6 days after splenectomy. Abdominal exploration was done. We resected 1 meter of gangrenous intestine and intestinal anastomosis was done **(Figure 2)**. Seven days later, there was greenish discharge at single point of the abdominal wound. There was no fever. Pelviabdomial CT scan showed no abdominal or pelvic collection. Discharge stopped after ten days and patient was discharged on Xarelto.

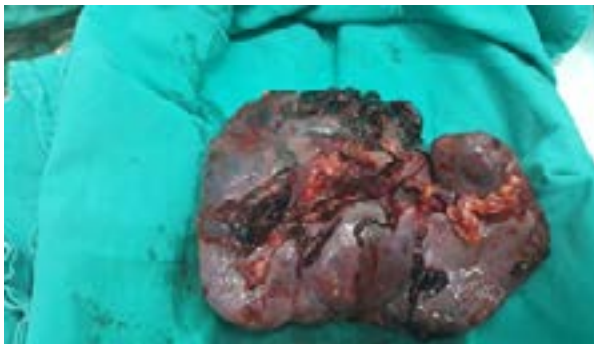

Fig 1: Upon exploration of case 1; the patient had a large ruptured subcapsular hematoma at the upper pole of the spleen.

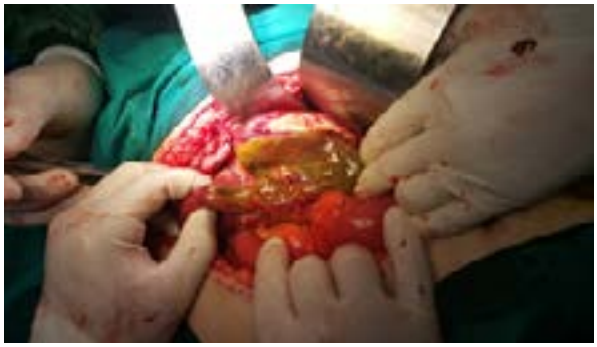

Fig 2: Re-exploration revealed gangerenous small bowel loops. Resection anastomosis was done.

Case 3: Patient presented seven days after LSG with recurrent mild abdominal pain, dysentery and mild bleeding per rectum. Pelviabdomial CT and CT portal and mesenteric angiography were done. There was portal vein thrombus extending to superior mesenteric. We started treatment with LMWH 1 mg/ kg twice daily. The patient was discharged on Warfarin.

Case 4: Patient presented 2 months post sleeve gastrectomy with low grade fever, cough and expectoration, underwent full assessment including CBC (TLC 11000), CRP (markedly elevated), pelviabdominal ultrasound was normal, urine analysis (pus cells over 100), blood and urine culture were positive for MRSA. The patient received antibiotic coverage till the cultures were negative. Two weeks later the patient developed mild epigastric pain, fever reaching 40 ºC and fits for which she was admitted to the ICU. The patient underwent pelviabdominal ultrasound which revealed a splenic abscess then CT pelviabdomen with IV and oral contrast was done showing a splenic abscess, portal vein thrombosis **(Figure 3)** and left sided lung collapse with encysted empyema of the lower lobe of the lung. The patient developed disturbed conscious level, underwent ultrasound guided aspiration and pigtail tube insertion for the splenic abscess and was kept on heparin infusion and broad spectrum antibiotic coverage. The follow up results were unfavorable as the patient developed irreversible septic shock, arrested and died.

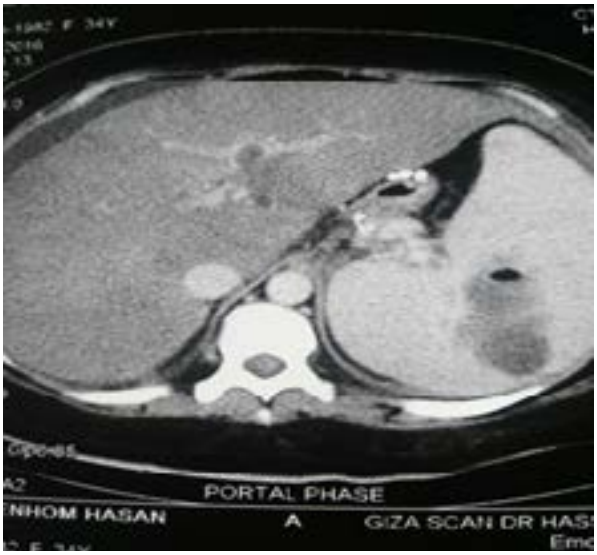

Fig 3: CT pelviabdomen with IV and oral contrast revealed splenic liver abscess and portal vein thrombosis Figure 3: CT pelviabdomen with IV and oral contrast revealed splenic liver abscess and portal vein thrombosis.

Discussion

Portomesenteric vein thrombosis (PMVT) is a rare but potentially fatal clinical complication that may lead to intestinal ischemia and infarction.[3] So it must be identified and managed promptly. The mortality rate of acute mesenteric thrombosis can range from 20% to 50%.[4]

Bariatric surgery is the only evidence based treatment of morbid obesity with proven and sustained weight loss and improvement in

comorbidities.[5] Recently, laparoscopic sleeve gastrectomy (LSG) has been considered as an effective approach for management of morbid obesity. It is considered easier, faster, and less traumatic to perform for surgeons than laparoscopic Roux-en-Y gastric bypass (LRYGB).[6]

Our Current literature reports that LSG is associated with an increased risk of PMVT compared to other abdominal procedures in general and; specifically, to other bariatric procedures. The incidence of PMVT in our series is 0.42% (we identified 4 cases out of 947 cases who underwent LSG), with a range of 0.3%–1% reported in other literature.[7-9]

The development of venous thrombosis is generally considered to involve a combination of locoregional and systemic prothrombogenic factors.[3] Obesity induces venous thrombosis by elevation of clotting factor levels, reduction of fibrinolysis and release of pro-inflammatory mediators.[10] Other risk factors for venous thrombo-embolism (VTE) development include abdominal surgeries, smoking, varicose veins, use of oral contraceptives, previous history of venous thrombo-embolism, age greater than 60 years old, and venous insufficiency.[11] In our study one patient was on oral contraceptive pills, while 2 patients were smokers.

The etiology of PMVT after bariatric surgery is probably multifactorial. The association of VTE and obesity is well established and is attributed to several factors, including chronic inflammatory state, the metabolic syndrome, increased intra-abdominal pressure, and venous stasis in the lower limbs. Moreover, increases in several procoagulant factors such as fibrinogen, factor VIII, and von Willebrand factor have been documented in obese patients.[12]

Other suggested etiologies for the development of PMVT include genetic deficiencies (Protein S, Protein C, Antithrombin III, and Factor V Leiden deficiency), prothrombotic states (sepsis, pregnancy, obesity, use of oral contraceptive, liver cirrhosis, lupus anticoagulant, polycythemia vera, heparin-induced thrombocytopenia, and malignancy or myeloproliferative disorders), iatrogenic causes (trauma to the portal venous system, abdominal wall inflamation), neoplastic disorders or decreased portal flow.[7,13]

Several mechanisms related to the laparoscopic surgical technique have been involved in the development of PMVT. The flow in the splanchnic and portal vessels has been found to be inversely proportional to capnopneumoperitoneum, and intra-abdominal pressure >14 mmHg results in >50% reduction in portal blood flow.[14] In addition, hypercapnia induced by CO2 insufflation causes vasoconstriction of the splanchnic system, with further reduction in blood flow.[15]

Other potentially contributing factors include the negative fluid balance after surgery, intraoperative vasopressin release, and reverse Trendelenburg position, which is used during laparoscopic surgeries.[16-18]

Goitein et al proposed 3 possible mechanisms by which LBS might result in PMVT, in addition to the rare, sporadic cases encountered in general laparoscopic surgery:

1. The change in blood flow (venous return from the stomach) occurs after LSG because of the division of the short gastric vessels. The altered flow may be a factor in PMVT promotion. It is interesting to note that, in a large cohort of patients who underwent LBS by Goitein et al, all cases of PMVT except one occurred after LSG (the most common procedure in this group), with none after LRYGB or biliopancreatic diversion. Although biliopancreatic diversion was infrequently used, LRYGB contributed almost 1000 cases in which PMVT was not encountered. This difference in proportions was not statistically significant.

2. In patients who undergo LSG direct physical contact with the splenic vein is possible while the surgeon is working in the lesser omental bursa. Intimal damage may occur and cause later thrombosis.

3. After bariatric surgery, patients are usually discharged from the hospital soon after the procedure. Some patients encounter difficulty in reaching the suggested 2-L/d fluid intake and experience various degrees of dehydration. This may put them at a higher risk for thrombotic complications, deep vein thrombosis, pulmonary embolism, and PMVT.[7]

In addition to above; Rodrigo Villagrán et al proposed other 2 possible mechanisms for PMVT post LSG:

1. Mechanical or thermal effect on the left gastroepiploic arcade or short vessels during the skeletonization of the greater curvature. Intraoperative surgical manipulation may damage the splanchnic endothelium and lead to local thrombus formation that may then propagate throughout the portal venous system.

2. Splenic ischemia or infarction. The ligation of the short vessels during LSG may lead to

insufficient perfusion of the upper pole of the spleen, perceptible as a demarcation during the operation. Such a condition, while asymptomatic in most cases, may occasionally lead to the symptomatic development of a splenic infarct and even subsequent abscess, reflecting the release of inflammatory mediators.[3]

Goitien et al., in a multicenter study including 5706 bariatric surgeries, reported 17 (0.3%) cases of PMVT; all cases but one occurred after LSG, and no PMVT occurred in the group of approximately 1000 patients who had undergone laparoscopic Roux-en-Y gastric bypass (LRYGB).[7] Additionally, in our complete series of 1434 surgeries, no PMVT was reported outside of the LSG group.

It was noticed that patients who develop PMVT usually present with non-specific abdominal pain and, less commonly, with nausea, vomiting, diarrhea, or gastrointestinal tract bleeding. Physical findings vary and range from no physical findings to low-grade fever, mild abdominal tenderness, peritoneal signs, or frank shock due to bowel ischemia. It is be mentioned that, after bariatric surgery, some of the patients who experience vague epigastric pain with some degree of dehydration might, in fact, have a mild, more chronic form of PMVT that is never diagnosed and spontaneously resolves.[7]

In our study the presentation was variable like persistent non specificepigastric pain, dysentery, bleeding per rectum or hypovolemic or septic shock. Three patients presented in the first two weeks after surgery; however the fourth patient presented after 86 days.

Prophylaxis against PMVT is used by most bariatric centers. Many different regimens are used with regard to the time of initiation, the specific pharmacologic agent used, the dosage, the length of treatment, and the adjunct appliances used (e.g., pneumatic compression stockings and inferior vena cava filters).[7] To the best of our knowledge, no protocols or guidelines are available that are specific for prevention of PMVT in patients who underwent bariatric surgery, due to the infrequent development or reporting of such complications.[19] 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.[20]

Early ambulation and pneumatic compression are the non-pharmacological measures used in current practice. Low molecular weight heparin (LMWH) and unfractionated heparin (UFH) are helpful to prevent PMVT; however, no clear guidelines about the optimal dose, duration, and type of heparin are currently available. In a cohort study by Birkmeyer et al, they evaluated the effectiveness and safety of various different venous thromboembolism VTE prophylaxis strategies for patients undergoing bariatric surgery. They had patients who used unfractionated heparin preoperatively and postoperative (unfractionated heparin/unfractionated heparin), patients who use unfractionated heparin preoperatively and low molecular weight heparin (LMWH) postoperatively, and finally a third subset of patients who used low molecular weight heparin both preoperatively and postoperatively (LMWH/LMWH). Rates for emboli development were 57% lower for the unfractionated heparin/LMWH group and 66% lower for the LMWH/LMWH group when compared to the unfractionated heparin/unfractionated heparin group. They concluded that low molecular weight heparin was more effective than unfractionated heparin for the prevention of VTE and it did not increase the risk of bleeding in bariatric surgery patients.[21]

Inadequate anticoagulant dosing may lead to venous thromboembolism, especially in obese patients. Frederiksen et al demonstrated that there was a negative correlation with body weight and the anticoagulant effect of a fixed dose of LMWH.[22] The role of extended prophylaxis after discharge is still undetermined. Although guidelines of the American College of Chest Physicians advocate against its use.[23]

The updated guidelines of the American Association of Clinical Endocrinologists and the Obesity Society support the use of extended thromboprophylaxis after discharge only in high risk patients, such as those with a history of DVT.[24] The 2013 position statement of the American Society of Metabolic and Bariatric Surgeons concluded that extended post discharge prophylaxis should be considered for all patients, although the specific dose and duration remain unclarified.[25]

The patients in our series received DVT prophylaxis including compression elastic stocking and preoperative prophylactic dose of low-molecular-weight heparin (LMWH) enoxaparin to be continued for 7 days postoperatively, the thrombosis rate was significantly lower in patients who received anticoagulation therapy after discharge. The clinical presentation was nonspecific in these patients but all of them complained mainly of abdominal pain, nausea and malaise consistent with other literatures. As we experienced cases that developed PMVT post sleeve gastrectomy;

therefore, we decided to extend our protocol of prophylactic anticoagulation. We recommend that bariatric surgery patients should be maintained on anticoagulants for at least 2 weeks postoperatively. Longer duration should be considered in patients with a personal or family history of VTE or known thrombophilia. Laboratoty studies of patients with PMVT are usually nonspecific. We think that thrombophilic evaluation should be performed in patients with PMVT. The evaluation of one of our cases showed protein C deficiency.

Computed tomography CT is currently the test of choice for the diagnosis of PSMVT, with a reported accuracy reaching up to 90%. Findings included mesenteric venous lucency, mesenteric fat stranding, splenomegaly, splenic or hepatic infarcts, and edematous small bowel. CT can also reveal the extent of bowel affected and exclude other pathologic conditions (e.g., leaks and abdominal collections).[26] Doppler ultrasonography should not be used in the acute setting for the diagnosis of PSMVT because of its low sensitivity (70%).[9]

PMVT can be managed successfully with a conservative approach if there is no evidence of bowel infarction. Most patients with mild PVT can be successfully treated with LMWH alone. It has been recommended that patients with acute PMVT should be treated with anticoagulation therapy as early as possible. This promotes the recanalization of the portal venous system and decreases the risk of further thrombotic events.[27,28]

However, the optimal duration of treatment with systemic anticoagulation is not well defined. Ghandi et al. recommended 3–6 months of anticoagulation, which can be extended further if the signs and symptoms persist.[29] Another study suggested a longer duration of anticoagulation therapy, ranging from 6 to 12 months.[30] However, patients with a systemic etiology are required to be on lifelong anticoagulation therapy.[31] In this study, patients were treated by anticoagulation with either heparin infusion for 5 to 7 days or subcutaneous treatment with the low-molecular-weight heparin (LMWH) enoxaparin at 1 mg/kg twice daily then we shift to oral thrombolytic medication. The treatment details for PMVT and its outcome are summarized in **Table 3**. The decision regarding the type of drug and the duration of anticoagulation therapy was based on hematology consultant assessment as regard patients' clinical course, result of thrombophilic evaluation, presence of other thrombotic risk factors and follow-up Doppler studies.

Despite the evolving role of invasive therapies for acute PVT, there is still a lack of consistency regarding the utility of thrombolytics because most of the findings are based on either case reports or case series, which needs solid evidence- based management from prospective studies and clinical trials.[1] Several investigators recommended either percutaneous or transhepatic thrombolytic therapy when anticoagulation is not showing progressive response in severe presentations, or is clinically and/or radiologically compatible with bowel ischemia, or otherwise when a non-operative approach is indicated.[7,32,33]

Conclusion

Portomesenteric vein thrombosis is a rare presentation after LSG. However, it is a serious complication that needs a high index of suspicion. Moreover, familiarity with this entity facilitates early diagnosis and management. Early diagnosis is very important for best patient outcomes and essential in preventing catastrophic complications such as intestinal infarction and corresponding increases in morbidity and mortality. Conservative management through anticoagulants and thrombolytics is quite effective, and, if indicated, should always be considered as the primary treatment option. Therefore, we recommend that bariatric surgery patients should be maintained on anti¬coagulants for at least 2 weeks postoperatively.

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