

Portomesentric Vein Thrombosis: Our Experience of 3527 Laparoscopic Bariatric Surgery Procedures in Qatar

Keywords: Portomesenteric vein thrombosis; Bariatric complications; Hypercoagulability; Laparoscopic bariatric surgery

Abstract

Background: Portomesenteric vein thrombosis (PMVT) is a rare but potentially serious and life threatening complication that may happen following laparoscopic bariatric surgery (LBS). Multifactorial local and systemic prothrombotic factors have been identified for PMVT development. In this study we intend to assess: 1) Incidence of PMVT post LBS in our patients' population; 2) Our protocol for DVT prophylaxis with applying the enhanced recovery after surgery (ERAS) protocol and application of certain intra-operative techniques in PMVT prevention following LBS.

Methods: This is a retrospective review of patients who underwent LBS between February 2011 and December 2016, who developed PMVT following LBS. Patients' charts were reviewed and data were extracted (patients' demographics, associated comorbidities, potential PMVT risk factors, LBS procedure undertaken, peri-operative details). In addition, the clinical features were also retrieved (PMVT presenting symptoms, management, blood investigations and thrombophilia screening).

Results: Out of 3527 patients who underwent LBS in our center during this period, 4 patients developed PMVT comprising an incidence of 0.1%. Thrombophilic work up of patients who developed PMVT revealed that all had thrombosis risk factors (hyper-homocysteinemia, anti-nuclear antibody, SLE, mutant Factor V Leiden, Protein C, Protein S deficiency). All managed medically and none required surgical intervention. There was no mortality.

Conclusion: Though no consensus concerning PMVT prophylaxis in LBS patients exists, we propose that application of a standard multimodal thromboembolic prophylaxis mainly mechanical prophylaxis, intra-operative techniques and ERAS principles as showed, were major reasons for low PMVT incidence.

All patient who developed PMVT post LBS had positive diagnostic thrombophilia work up. Following standardized non-pharmacological thromboembolic prophylaxis in LBS patients questions the benefit of pharmacological thromboembolic prophylaxis.

Abbreviations

PMVT: Portomesenteric Vein Thrombosis; LBS: Laparoscopic Bariatric Surgery; LRYGB: Laparoscopic Roux-en-Y Gastric Bypass; GERD: Gastroesophageal Reflux Disease; OA: Osteoarthritis; LMWH: Low-Molecular-Weight Heparin; IAP: Intra-Abdominal Pressure; OR: Operation Room; LOS: Length of Hospital Stay; DVT: Deep Venous Thrombosis; PE: Pulmonary Embolism; UFH: Unfractionated Heparin; SMV: Superior Mesenteric Vein; VTE: Venous thromboembolism; IVC: Inferior Vena Cava; MVT: Mesenteric Venous Thrombosis; EGD: Esophagogastroduodenoscopy



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Moamena Ahmed El-Matbouly^{1*}, Nesreen Khidir², Davit Sargsyan¹, Walid El Ansari³, Moataz Bashah¹ and Mohammed Al-Kuwari¹

¹Department of General Surgery, Hamad Medical Corporation, Doha, Qatar

²Department of bariatric and metabolic Surgery, Hamad Medical Corporation, Doha, Qatar

³Department of Surgery, Hamad Medical Corporation, Doha, Qatar

*Address for Correspondence

Moamena Ahmed El-Matbouly, Department of General Surgery, Hamad Medical Corporation, Doha, Qatar, Tel: +974-55910445; E-mail: momenaematbouly@gmail.com

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Background

Thrombosis of portal vein and mesenteric vein (Porto Mesenteric Venous Thrombosis, PMVT) is fortunately a relatively uncommon complication in laparoscopic bariatric surgery (LBS) patients [1]. However, PMVT requires prompt attention as delayed diagnosis could have potentially life-threatening consequences. For instance, PMVT accounts for 5-15% of reported cases of acute mesenteric ischemia, which could progress to bowel necrosis and sepsis, ultimately leading to death [2]. PMVT has been observed after surgical procedures, but the inflammatory and hypercoagulable obese state, laparoscopy, and manipulation of the portomesenteric venous system contribute to the increased risk of PMVT after LBS [3,4].

PMVT post laparoscopic Roux-en-Y gastric bypass (LRYGB) was first reported in 2004 and post laparoscopic sleeve gastrectomy (LSG) in 2009 [5]. Diagnosing PMVT is challenging in the obese patients post LBS period, with vague and non-specific symptoms and complaints [6]. Multiple diagnostic modalities are usually employed to establish the diagnosis e.g. CT scan, doppler ultrasound and MRI [7]. Currently with the advancement in testing for hypercoagulable states, patients diagnosed with PMVT can undergo additional investigations to identify possible predisposing risk factors to thrombus formation. Such risk factors include factor V Leiden mutation, prothrombin G20210A mutation, protein S deficiency, protein C deficiency, antithrombin-III deficiency, activated protein C resistance and antiphospholipid syndrome which have all been linked to increased splanchnic thrombosis [8].

Review of the literature revealed a limited number of case reports and series describing PMVT following laparoscopic bariatric surgery as an uncommon complication [4]. A retrospective analysis of 1713 patients who have undergone LSG showed an incidence of 1% PMVT [1]. In our study we present the cases of PMVT following LBS performed at our Metabolic and Bariatric Surgery Center to assess our

incidence of PMVT post LBS; the patient and the procedure-related thrombosis risk factors; and the effect of employing the multimodal thromboembolism prophylaxis, ERAS protocol, as well as application of certain intra-operative techniques on PMVT incidence post-LBS.

Materials and Methods

A retrospective review of all patients who underwent elective LBS in our center between February 2011-December 2016 and subsequently developed symptomatic PMVT.

For each identified PMVT patient, the following variables were extracted from the records: age, gender, initial body mass index (BMI), risk factors for thrombosis (patient history, family history, and medication history), surgical technique used and its details, PMVT presenting clinical features and management. The mean follow up duration was 12 months for the entire study group. For the patients who were diagnosed with PMVT they were followed for three months initially and then at 6 months.

Application of multimodal thromboembolic prophylaxis, ERAS principles and certain intra-operative techniques

In our practice, the ERAS protocol includes peri-operative and post-operative techniques like; mechanical thromboembolic prophylaxis consisting of peri-operative compression stockings and early post-operative ambulation.

Pharmacological thromboembolism prophylaxis: unfractionated heparin (UFH) and low-molecular-weight heparin (LMWH) was limited in our practice to the high risk group (according to our assessment), e.g. those with BMI>50, bed or wheelchair bound patients, with history of previous thromboembolic events or known familial thrombophilic diseases and patients on hormonal or contraceptive medications. Those selected patients received LMWH (dalteparin sodium injection 5000 IU) started 6 hours post-operatively and continued to a total of 14 days after discharge.

All patients who presented with symptomatic PMVT were closely monitored and observed clinically (symptoms, signs and blood works) and if needed radiologically. CT, U/S and Thrombophilia workup was conducted for all PMVT patients as well as follow up CT scan on outpatient basis. A follow up upper gastrointestinal endoscopy (UGE) was performed in all PMVT patients at 1 year post diagnosis, to identify signs of portal hypertension.

Results

the 3527 patients who underwent LBS, mean BMI was 44.2±8.8, 5 kg/m² with 99% (CI 43.8-44.6). Pharmacological thromboembolism prophylaxis was given to 627 (19%) patients; 97% of these patients had BMI>50, and 19 (3%) patients had venous thrombosis risk factors; known family or personal history of DVT, bed or wheelchair bound and patients on hormonal contraceptive therapy. While 81% of our LBS patients did not receive pharmacological thromboembolism prophylaxis. Four of these patients (0.1%) developed PMVT and 25 (0.9%) developed post-op bleeding that required laparoscopic intervention and blood transfusion.

The PMVT patients’ mean age was 44.8 (±8.6 years) and three patients were women. The average BMI was 40.3 (±0.5 kg/m²). PMVT Patients’ characteristics are summarized in Table 1. None of the PMVT patients received pre or post-op pharmacological thromboembolism prophylaxis. Mean length of surgery was 92.5 (±78.5 min). No intra-operative or immediate post-operative complications occurred. The peri-operative details for the PMVT patients including intraoperative and post-operative course are summarized in Table 2. Patients’ mean length of hospital stay was 3±0.8 SD days.

All PMVT cases started to have symptoms after discharge, with a mean duration of symptoms of 3 days (range 2-4 days). Patients were re-admitted after M=17 days (range 5-30 days) post their initial surgery, The PMVT readmission characteristics are shown in Table 3. All the patients had chief complaint of vague abdominal pain mainly

Table 1: PMVT Patient’s demographics, associated comorbidities and potential risk factors (N=4).

Patient	Age	Sex	BMI	Associated Comorbidities	Potential risk factors		
					Hormonal contraception	Smoking history	Thrombosis, hypercoagulable states or thrombophilia*
1	47	F	40	OA	Y	No	Not diagnosed pre-operatively
2	32	F	40	None	No	No	Not diagnosed pre-operatively
3	50	F	41	Asthma, OA	No	No	Not diagnosed pre-operatively
4	50	M	40	GERD	No	No	Not diagnosed pre-operatively

*Thrombosis history; Hypercoagulable states or Thrombophilia family history; Y: the patient gave history of receiving long-term contraceptive injection Depo-Provera, which she failed to report pre-operatively; F: Female; M: Male; GERD: Gastroesophageal Reflux Disease; OA: Osteoarthritis.

Table 2: Peri-operative details of PMVT patients.

Patient	Procedure	Chemoprophylaxis*		OR time (min)	IAP>14 mm Hg	LOS (days)
		Doses (mg/day)	Duration (days)			
1	LSG	No	No	50	No	2
2	LSG	No	No	50	No	3
3	Removal of band+LRYGB	No	No	210	No	4
4	LSG	No	No	60	No	3

LMWH: Low-Molecular-Weight Heparin; IAP: Intra-Abdominal Pressure; OR: Operation Room; LOS: Length of Hospital Stay; Chemoprophylaxis: Pre or Post-op Unfractionated Heparin (UFH) or LMWH.

Table 3: Readmission characteristics: PMVT symptoms, management, blood investigations and thrombophilia screening.

Patient	1	2	3	4
Symptoms				
Fever	No	No	No	Yes
Nausea	Yes	No	No	Yes
Pain location	E, LH	E	E	E, RH
Readmission details				
Readmission*	14	7	30	8
Duration of symptoms**	3	3	4	2
Blood tests				
Hemoglobin (g/dL)	11.1	13.6	12.1	12.8
Hematocrit (%)	35.9	45.9	36.9	38.4
PT/APTT	14.5/30	10.5/33.3	10.9/31.8	14.2/27.6
INR	1.2	1.1	1.2	
Total Bilirubin	20	7	6	19
ALT/AST	10/11	24/25	280/253	46/38
Procalcitonin (ng/ml)	0.19	-	-	-
CRP (mg/L)	220	-	-	-
Lactic Acid (mmol/L)	1.96	0.9	0.96	-
Thrombophilia Work up	SLE, Mutant Factor V Leiden	Protein S Def	Lupus anticoagulant	Protein C and S Def

F: Fever; N: Nausea; RH: Right Hypochondrial; LH: Left Hypochondrial; E: Epigastric; *: Duration (Days) after Initial LBS; **: Duration (Days) of Symptoms before Readmission; PT: Prothrombin Time; APTT: Activated Partial Thromboplastin Time; LMWH: Low Molecular Weight Heparin; Def: Deficit; ANA: Anti-Nuclear Antibody; SLE: Systemic Lupus Erythematosus

in the epigastrium with nausea. All cases were diagnosed by abdominal and pelvic CT enhanced with oral and intravenous contrast. One patient had partial thrombosis in the superior mesenteric vein (SMV) only, while the remaining three had partial thrombosis in the SMV and portal vein. Patients were then subsequently assessed for bowel ischemia and/or signs of sepsis and none of them required surgical intervention. All the four patients had successful medical management for their PMVT (anti-coagulation, low molecular weight heparin and warfarin). None of the patients required percutaneous trans-hepatic thrombolytic therapy.

Discussion

All patients undergoing bariatric surgery BMI≥35 are considered of moderate to high risk of post-operative PMVT, deep venous thrombosis (DVT) and pulmonary embolism (PE) [9,10].

The occurrence of PMVT post LBS may be attributed to the following factors:

1. Hypercoagulable state associated with obesity;
2. Injury to the porto-mesenteric vessels; mechanical or thermal effect during dissection of the greater curvature on the left gastroepiploic and short gastric vessels, direct injury with the splenic or superior mesenteric vein (SMV);
3. The inflammatory response to surgery;
4. Laparoscopy causing a thrombogenic effect due to increased intraabdominal pressure with reduction in venous return from the extremities (especially in the prolonged reverse Trendelenburg position used in LBS) and splanchnic system;
5. Blood stasis within the liver caused by prolonged positioning of the liver retractor, leading to formation of a retrograde thrombosis is also proposed;

6. Dehydration; decreased fluid intake after LBS [6,10]

Currently, there are no available accurate evidence-based risk assessment tools for VTE in bariatric patients. The published literature varies widely on optimal guidelines for the prevention of VTE following LBS. The major accepted forms of prophylaxis range from mechanical compression devices and early ambulation alone, to the addition of chemoprophylaxis and the use of IVC filters [11].

Becattini C et al. mentioned a 2% incidence of bleeding complications associated with chemoprophylaxis that raised concerns regarding administering anticoagulation post operatively [12]. Several studies have examined the use of mechanical compression only in bariatric surgery patients [12]. A retrospective study of 1692 patients reported DVT and PE rates of 1.6% and 1.1% respectively, in patients who received LMWH and the sequential compression devices compared with a 0.4% DVT rate and no PEs in the patients who receive mechanical prophylaxis and early ambulation only. The incidence of PMVT was not reported in the study [11]. The incidence of post-operative VTE complications, either symptomatic or asymptomatic seems to be relatively low <1%, and the benefit of weight-adjusted heparin prophylaxis remains controversial. The incidence of symptomatic VTE in the 2 large studies with mechanical prophylaxis appeared to be similar to that observed with heparin prophylaxis [12].

In this study we present our incidence of PMVT over LBS population who received only mechanical prophylaxis. We stress on the same point by Becattini C et al. who suggested that mechanical prophylaxis in addition to the application of enhanced recovery after surgery is sufficient for patients without personal or strong family history of VTE events or known hypercoagulable state [12]. In order to prevent blood stasis peri-operatively, we systematically use a valveless trocar system to control intra-abdominal pressure since a 50% reduction in portal blood flow has been demonstrated when intra-abdominal pressure exceeds 14 mm Hg [13]. We suggest

that the collective application of these techniques comprise major contributing factors in reducing the operative time, venous stasis and blood re-distribution in the splanchnic circulation leading to the decreased PMVT incidence that we observed among our LBS population.

We determined our incidence of PMVT post LBS among 3551 various elective LBS procedures' to be (incidence=0.1%). Few published studies are available on the topic with which we can directly contrast our findings. A research by Salinas and others, resulted in 1% PMVT incidence following 1713 LSG procedures [1]; and the lowest published PMVT incidence after LBS was 0.3% [10]. It should also be noted that the PMVT cases reported here were based on symptomatic patients who underwent diagnostic imaging and no routine imaging or screening was performed for asymptomatic patients.

Regarding patient-related predisposing PMVT risk factors; all our PMVT had positive thrombophilia workup. This finding contrasts with others who reported that out of 17 patients who developed post-LBS PMVT, only four patients exhibited ≥1 of these risk factor/s [10]. In a recent review, 8 out of 36 cases of post LBS-PMVT had a positive thrombophilia workup [14]. The four patients who had PMVT had missed family history that was positive for thrombophilia.

Regarding procedure-related predisposing PMVT risk factors, we observed that three of our four PMVT patients were post LSG, in support of a very recent review that found that the majority of PMVT cases were after LSG, suggesting that it is procedure specific [14]. Others reported that of 5706 LBS patients, 17 (0.3%) had PMVT, 16 of which were post-LSG [10]. Many propositions have been put forward for the high PMVT rate after LSG making it procedure specific. For instance, the intra-operative manipulation of the splanchnic vessels along the stomach's greater curvature as well as the thermal injury to the short gastric vessels when sealed with energy devices [14].

The current literature has evaluated the effectiveness of ERAS principles in improving post operative recovery and in-hospital stay in LBS [15]. To the best of our knowledge, there seems no existing

evaluations of the impact of the use ERAS principles on post operative morbidity and mortality in LBS [15]. An exception is Grantcharov H et al. who examined the value of an accelerated recovery program in elective primary laparoscopic gastric resection [16]. Their prospective study demonstrated that the successful clinical application of ERAS principles led to decrease in post-operative complications [16]. The current study reports the effect of application ERAS principles as a major contribution in achieving a low rate of post-operative incidence of PMVT.

Our study is an observational retrospective study that looked at the patients who had LBS and we are reporting our incidence of PMVT as well as the protocol that we used for DVT prophylaxis. One of the limitations of the study is the lack of comparison between mechanical and pharmacological prophylaxis. In our study we are reporting our finding of observing our population post LBS and stating the protocol that we followed in our institution. Future studies should be done to look into the effectiveness of pharmacological DVT prophylaxis and its impact on the incidence of PMVT post LBS.

Diagnosis and treatment

The clinical diagnosis of PMVT is challenging post LBS. High index of suspicion is required in patients presented with vague abdominal pain post operatively. Contrast CT abdomen has a sensitivity of 95% [17].

Close observation and frequent monitoring of these patients are mandatory to avoid unnecessary surgical interventions considering a high index of suspicion of thrombosis progressing to mesenteric ischemia, sepsis and bowel infarction. Any delay in diagnosing intestinal infarction worsens the prognosis; thus, making early diagnosis a key factor in improving the patient's outcome. In our practice once a suspicion of bowel ischemia or sepsis is raised, we consider measuring sepsis biomarkers; procalcitonin, CRP and lactic acid as effective tools to guide our clinical and therapeutic decisions. Procalcitonin is more sensitive and specific diagnostic parameter for sepsis and a better predictor of mortality [18].

Table 4: Shows interpretation of the radiological signs of bowel ischemia.

Radiological signs	P' (1)	P' (2)	P' (3)	P' (4)
Mural signs				
Bowel wall thickening	(+)	(-)	(-)	(+)
Intramural edema	(-)	(-)	(-)	(-)
Intramural hemorrhage	(-)	(-)	(-)	(-)
Abnormal wall enhancement	(-)	(-)	(-)	(+)
Halo sign/target sign	(-)	(-)	(-)	(-)
Pneumatosis intestinalis	(-)	(-)	(-)	(-)
Vascular signs				
Venous filling defect	Near complete	Partial	Partial	Complete
Vein enlargement	(-)	(-)	(-)	(+)
Venous engorgement	(+)	(-)	(-)	(-)
Venous collateral circulation	(-)	(-)	(-)	(-)
Mesenteric venous gas	(-)	(-)	(-)	(-)
Extramural non vascular signs				
Mesenteric fat edema	(+)	(+)	(+)	(+)
Ascites	(+)	(+)	(+)	(+)
Bowel dilatation	(-)	(-)	(-)	(+)
Free-intraperitoneal air	(-)	(+)	(-)	(-)

In this article, we provide an overview of the diverse radiological signs of CT imaging, as an important factor in predicting the occurrence of subsequent bowel ischemia and infarction. Subdividing them into categories facilitates comprehension; and decision making regarding progression to the operating room and exploration. Initial CT findings of our PMVT patients are shown in Table 4. Sometimes serial CT imaging was required [17].

The radiological signs of bowel ischemia can be divided into mural, vascular, and extramural signs. Circumferential bowel wall thickening, caused by intramural hemorrhage and edema is the most commonly reported sign in acute venous bowel ischemia. It is more pronounced with venous congestion due to mesenteric venous thrombosis (MVT) than with arterial thrombosis. Decreased bowel wall enhancement has been reported as highly specific for bowel infarction in patients with MVT [17]. Signs of mucosal edema-“halo sign” (mural stratification into two layers). Stratification into three layers is also called the “target sign”), which is composed of three rings: outer high-attenuation muscularis propria, a middle ring of gray attenuation, and an inner ring of high attenuation (mucosa) [19]. Venous filling defect may be partial or complete. Vein caliber enlargement is an indicative of an acute thrombosis. Extramural non vascular signs such as pneumatosis intestinalis and portomesenteric venous gas, when present transmural bowel infarction was seen in 91% of patients [19]. Mesenteric fat edema is related to the underlying inflammatory processes. Bowel dilatation results from interrupted intestinal peristalsis in response to ischemic injury and bowel infarction. Free intraperitoneal air results from perforation of an infarcted bowel segment. Presence of free intraperitoneal air in our patient number 2, was correlated to the retained CO₂ used for pneumoperitoneum; CT was done on day 7 post surgery.

We stress on the point that mural and extramural-nonvascular CT signs of bowel ischemia are nonspecific. However, if CT findings are suspicious and not conclusive or if the patient shows signs of deterioration into sepsis, laparoscopic exploration is highly indicated.

Once PMVT is diagnosed; full anticoagulation with either subcutaneous LMWH or intravenous unfractionated heparin was applied. This treatment was continued and changed to oral anticoagulation (target international normalized ratio, 2.5-3), which continued for 6 months. Early diagnosis and treatment with prompt anticoagulant therapy could lead to a dramatic decrease in the incidence of prehepatic portal hypertension in the near future. In contrast, late detection and treatment may result in prehepatic portal hypertension with the associated sequelae and portal cavernomatosis; resulting in portal hypertension. A screening esophagogastroduodenoscopy (EGD) for esophageal varices was done for post-PMVT patients at one year and was negative for all patients.

Conclusion

Although there is no consensus concerning PMVT prophylaxis in LBS patients, we propose that application of a standard multimodal thromboembolism prophylaxis, intra-operative techniques and ERAS principles as described, were major reasons for low PMVT incidence in our series.

All the patient who developed PMVT post LBS had positive diagnostic thrombophilia work up. Following standardized non-pharmacological thromboembolic prophylaxis in LBS patients questions the benefit of pharmacological thromboembolic prophylaxis. Careful clinical monitoring of the patient and proper comprehension of the CT signs of bowel ischemia can avoid further aggressive interventions and exploration. Proper comparative study between mechanical and pharmacological DVT prophylaxis should be done to assess its impact on the incidence of PMVT post LBS.

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