Portal thrombosis after Laparoscopic Bariatric Surgery. Description of 3 cases and Systematic Literature Review

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ABSTRACT: Background: Portal vein thrombosis (PVT) after Laparoscopic Bariatric Surgery (LBS) is an uncommon complication. However it is a potentially life-threatening condition reported after gastric sleeve, gastric bypass and adjustable gastric banding. Clinical symptoms may be insidious, and progression can lead to intestinal infarction and portal hypertension. Treatment, and outcomes remain poorly understood. Possible etiologic factors include venous stasis from increased intra-abdominal pressure, intraoperative manipulation, or damage to the splanchic endothelium and systemic thrombophilic states.

Main Outcome Measures: Systematic review of the literature on PVT after LBS and the 3 reported cases encountered at our institution

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INTRODUCTION

Portal Vein Thrombosis (PVT) refers to an obstruction in the trunk of the portal vein. It can, however extend downstream to the portal branches, or upstream to the splenic and/or the mesenteric veins, with different clinical features according to the site and extension of the obstruction in the portal venous system. It is a relatively rare condition in patients with a previously healthy liver, at least in developed countries. 1 Its pathogenetic factors are the same as those long recognized for venous thromboembolism: damage to the vessel wall, slowing of blood flow, and hypercoagulability.

In partial PVT, symptoms are fewer or absent. On the other hand, if the superior mesenteric vein is involved, symptoms are more severe with colicky abdominal pain and diarrhea. 4 When the extension of the thrombosis reaches the proximal mesenteric venous arches, severe abdominal pain, often radiating to the back, and ileus, due to intestinal ischemia, ensue. Signs of progression to intestinal infarction include: hematochezia, ascites, metabolic acidosis and renal or respiratory failure. Intestinal infarction carries a high mortality and a severe morbidity rate in surviving patients, even if surgical resection of the affected bowel is promptly accomplished.

Patients and Methods: We have used PubMed to search MEDLINE for articles published between January 1, 1990, and May 31, 2010, using the search terms portal vein thrombosis, mesenteric venous thrombosis, laparoscopic surgery and laparoscopy. Additional articles culled from references were obtained. The inclusion criteria were documented PVT by imaging studies such as angiography, ultrasonography, computed tomography [CT], or magnetic resonance imaging (MRI) or surgery following LBS. We include 3 cases (2 after gastric sleeve and 1 after gastric bypass) from our institution and 13 cases of PVT – 3 were Vertical Gastrectomy (LVG), 1 Adjustable Gastric Band (ABG) and 9 Roux-en-Y gastric bypass (LRNYGBP). Results: One developed a chronic cavernoma, a second had bowel resection and the third recovered. Conclusions: Laparoscopic surgeons should be aware of the risk of PVT, and it should be suspected in cases with an atypical outcome after LBS. Once PVT is diagnosed, prompt anticoagulation therapy may resolve the thrombotic event.

KEY WORDS: Portal vein. Thrombosis

Case Report #1

A 42-year old woman with obesity and nephrotic syndrome, BMI-35.5 had a Laparoscopic Vertical Gastrectomy (LVG) lasting 60’ under an insufflation pressure of 15 mm Hg. The peritoneal adhesions were mild and easily dissected. During the dissection of the posterior aspect of the stomach through the lesser sac, neither the portal nor hepatic artery was seen. Stapling of the stomach was carried out using 5 cartridges of 60 mm linear cutting stapler. The patient responded well to the procedure. Her immediate postoperative course was without complications. The patient was discharged home on POD1 with low-molecular-weight heparin for 8 days.

She was readmitted 2 weeks later because of nausea associated with diffuse mild abdominal pains. On physical examination she was afibrile, her abdomen was diffusely tender, there was no guarding or rebound tenderness, and bowel sound were present. CBC, serum amylase, liver function tests, and abdominal ultrasonography were normal. The patient was kept NPO and given intravenous fluids. 12 hours later, the patient remained asymptomatic, with no other complication and was discharged having tolerated diet and passed flatus.

She was re-admitted 3 days later with right side abdominal pain, vomiting and fever. Abdominal and
Pelvic CT scan, enhanced with intravenous contrast, revealed the lack of opacification of the PV with prominent liver. These observations were consistent with the diagnosis of PVT (Fig 1). A few hours later, she was asymptomatic and did not receive specific treatment. She was discharged home on hospital day 3, tolerating a clear liquid diet.

A 35-year-old woman with BMI-39 had hypercholesterolemia. A LVG was done. The procedure lasted 37’ and the course was uneventful, and she was discharged home on POD1 with enoxaparine 40 mg SC for 8 days. Two weeks later, she was readmitted because nausea and mild abdominal pain, and her CBC revealed increase in white blood cell and the CT scan showed lack of opacification of the portal vein, without any sign of intestinal ischaemia. These observations were consistent with the diagnosis of PVT (Fig 3) and the patient was immediately hospitalized and was started on anticoagulation with low molecular weight heparin. She recovered completely.

DISCUSSION

The PV accounts for 75% of the blood supply to the liver. It is an 8-cm, valveless conduit originating from the confluence of the superior mesenteric and splenic veins posterior to the neck of the pancreas. In the porta hepatis, the portal vein divides into right and left branches that continue to their respective hepatic lobes, ultimately emptying into hepatic sinusoids. Portal vein thrombosis is defined as a condition resulting from formation of a blood clot in the extrahepatic portion of the portal vein.

PVT has been reported after blunt abdominal trauma and various types of open or laparoscopic GBP, VG. The mechanism by which laparoscopic surgery increases the risk of development of splanchnic vessels thrombosis remains unclear 7, 8, 9, 10, 11, 37. The role of the

Fig 1. PVT with hepatic low perfusion in hepatic spaces

Fig.2. Porta cavernous degeneration and hepato-spleno megaly

She was readmitted 3 months later, for left side abdominal pain. CT scan of the abdomen and pelvis was repeated and showed cavernous transformation of the portal vein, with signs and symptoms of portal hypertension including ascites and hipersplenism (Fig 2)

Case Report #2

A 55-year-old woman with morbid obesity, BMI-46 and significant Arterial Hypertension had medical and surgical evaluation, and a Laparoscopic Roux-en-Y gastric bypass (LRNYGBP) was performed. The procedure lasted 76’ under an insufflation pressure of 15 mmHg. An antecolic antegastric 150-cm Roux limb was created. The patient tolerated the procedure well. Her immediate postoperative course was uncomplicated, and was discharged home on POD2 with subcutaneous Fraxiparine 0.6 ml for 8 days.

One week later, she was readmitted because of nausea and vomiting associated with diffuse severe abdominal pains, afebrile, and absent bowel sounds. Exploratory laparotomy found sero-sanguineous peritoneal fluid and small bowel edematous with and a necrotic bowel segment (30 cm), without arterial pulse, and thrombosed veins were seen. The bowel was resected

Case Report #3

A 35-year-old woman with BMI-39 had hypercholesterolemia. A LVG was done. The procedure lasted 37’ and the course was uneventful, and she was discharged home on POD1 with enoxaparine 40 mg SC for 8 days. Two weeks later, she was readmitted because nausea and mild abdominal pain, and her CBC revealed increase in white blood cell and the CT scan showed lack of opacification of the portal vein, without any sign of intestinal ischaemia. These observations were consistent with the diagnosis of PVT (Fig 3) and the patient was immediately hospitalized and was started on anticoagulation with low molecular weight heparin. She recovered completely.

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surgical approach- open or laparoscopic- in the development of PVT is not yet clear. Although results from some studies indicate no influence of surgical technique on the incidence of PVT, specifically splenectomy 12, 13, others report a significantly higher incidence after laparoscopic procedure, from 8% to 52% (14, 15) compared with around 10% after open procedure 16.

Thrombosis related to capnoperitoneum may be caused by complex interactions of mechanical and systemic effects leading to a variety of changes in coagulation and cardiovascular and splanchnic hemodynamics. Many years ago, Kotzampassi 17 demonstrated decreases in jejunal mucosal blood flow and mucosal pH with a sustained pressure of 14 mmHg, further suggesting that clinically significant reductions in splanchnic circulation might be produced via capnoperitoneum, and at pressures lower than previously thought. The hypercarbia induced by CO2 insufflation causes sympathetic vasoconstriction, thereby increasing peripheral resistance, mean arterial blood pressure, pulmonary artery pressure and pulmonary capillary wedge pressure 11. There are reports that demonstrated an inverse relationship between intra-abdominal pressure and portal venous flow in porcine models, with a gradual decrease in cardiac index with concomitant elevation of systemic and splanchnic vascular resistance and reduction in portal venous flow to 70% of baseline as intra-abdominal pressure was increased in a stepwise fashion from 5 mmHg to 25 mmHg 18.

PVT is a rare condition, comprising 5-15% of all mesenteric ischemic events, and it is a very rare complication when related to laparoscopic surgery. It has been suggested that PVT occurs only when several predisposing factors are combined 8, 19. Another consideration specific to Gastric Bypass is trauma to the vessel during the closure of Petersen defect 11.

Although both local and systemic prothrombotic factors are implicated in causing portal vein thrombosis, cirrhosis of the liver is considered an important predisposing condition. In patients with well-compensated cirrhosis, the incidence of thrombosis reportedly varies between 0.6% and 16% 20.

The role of inherited thrombophilia (such as Factor V Leiden Mutation and prothrombin 20210 gene mutation) in the pathogenesis remains uncertain 20, 21. Interestingly, in patients with bacteremia from Bacteroides fragilis of an unknown source, there is a higher incidence of portal vein thrombosis, presumably due to the transient appearance of prothrombotic anticardiolipin antibodies 22.

The patient presented in the case report # 1 had hypoproteinemia and severe proteinuria by a nephrotic syndrome accompanied by an obvious hipercoagulable state and portal vein thrombosis; however, PVT has a relatively low incidence in patients with nephrotic syndrome and usually occurs during treatment or recurrence of the condition, not as the first sign. PVT as the first sign of nephrotic syndrome is very rare 23.

**CLINICAL PRESENTATION**

Acute PVT: PVT is considered acute when symptoms develop until 60 days post-surgery and there is no clinical, radiological, or endoscopic evidence of portal hypertension or collateral circulation 20. Symptoms of PVT are nonspecific and include abdominal pain (90%), vomiting (77%), nausea (54%), and diarrhea (36%) 8, 24, 38, 39. When the extent of the thrombosis reaches the proximal mesenteric venous arches, severe abdominal pain, often radiating to the back, and ileus, due to intestinal ischemia, ensue. Signs of progression to intestinal infarction include hematochezia, ascites, metabolic acidosis and renal or respiratory failure. Intestinal infarction carries a high mortality and a severe morbidity rate in surviving patients, even if surgical resection of the affected bowel is promptly accomplished 1.

Chronic PVT: Chronic PVT involves formation of numerous hepatopetal collateral veins around the thrombosed portal vein that allow blood flow from the patent proximal portion of the portal vein to the distal portion- the so- called "portal cavernoma"(Figure 3). Most patients at this stage present signs and symptoms of portal hypertension, including variceal bleeding, ascites, and hypersplenism 20.

**DIAGNOSIS**

For establishing the diagnosis, newer noninvasive modalities such as ultrasonography with Doppler imaging, CT, and MRI of the abdomen have supplanted the traditional invasive tests of portal venography and superior mesenteric arteriography 25. Angiography, traditionally the definitive investigation in portal vein thrombosis, is generally reserved for preoperative assessment if surgical intervention is planned. For obese patients, CT is an excellent means to evaluate gastro-intestinal or non-specific complaints in the post-operative course of surgical procedures 38, 39. Unless there is a high degree of suspicion, as has been described by many authors and intravenous contrast for CT scanning is given with appropriate timing, this potentially lethal problem may not be diagnosed which can result in death.

Gastroesophageal varices may be detectable as early as 1 month after acute PVT. Thus, an endoscopic screening for gastroesophageal varices must be performed within a few months and, if varices are not identified, repeated 6 months later (if portal vein recanalization has not been achieved). The mortality rate from oesophageal bleeding is consistently lower than in patients with cirrhosis 5, 6.

**MANAGEMENT**

Asymptomatic patients with portal vein thrombosis and patients with incomplete occlusions of the portal vein can remain untreated as long as they are followed up regularly (e.g. every 2-4 weeks). All other patients with portal vein thrombosis, however, must be treated.

Acute PVT in patients without concomitant cirrhosis: The goals of therapy for acute portal vein thrombosis are 2-fold: to establish complete patency of the portal
The aims of management include: prevention and the late stage of portal cavernoma, bleeding may occur. retrospective studies. When chronic PVT in patients without concomitant cirrhosis: postoperative complications or underlying disease is around 85% and mortality is mostly related to age, etiology of PVT or unrelated disease, rather than to complications of portal hypertension 30.

CONCLUSION

PVT represents an important and perplexing clinical problem, not infrequently encountered by physicians, given the increased use of radiological investigations. Capnoperitoneum-induced changes in hemodynamic and coagulation states, in combination with other prothrombotic factors, may induce mesenteric or PVT. Although clinical evidence is lacking, symptomatic portal biliopathy can be treated with ursodeoxycholic acid. Jaundice due to bile duct stenosis should be firstly approached by ERCP and biliary stent placement. A persistent relief of biliary symptoms after stent removal can be achieved in approximately 50% of patients 33-34. Liver transplantation may be the last option for severe biliary complications due to portal vein cavernoma 35.

LONG-TERM PROGNOSIS OF CHRONIC PORTAL VEIN THROMBOSIS

Morbidity is mainly related to variceal bleeding, recurrent thrombosis, symptomatic portal biliopathy and hypersplenism 1. Mortality among patients with chronic PVT is low (5-10% at 5 years) and is mainly related to age, etiology of PVT or unrelated disease, rather than to complications of portal hypertension 30.

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REFERENCES