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Surgery for Obesity and Related Diseases 14 (2018) 271–276

SURGERY FOR OBESITY AND RELATED DISEASES

Original article

Portomesenteric vein thrombosis in sleeve gastrectomy: a 10-year review

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Received September 11, 2017; accepted December 8, 2017

Abstract

Background: Worldwide, the laparoscopic sleeve gastrectomy (LSG) is becoming the dominant bariatric procedure due to its reliable weight loss and low complication rate. Portomesenteric vein thrombosis (PVT) is an uncommon complication of LSG with an incidence of .3% to 1% and can lead to serious consequences, such as bowel ischemia and death.

Objectives: This paper will present the presentation, risk factors, treatment, and long-term outcomes of patients who had PVT post-LSG.

Setting: Five bariatric centers in a private setting in Australia.

Methods: Retrospective data were collected from 5 bariatric centers across Australia from 2007 to 2016.

Results: Across 5 centers, 5951 patients underwent LSG; 18 had recognized PVT (.3%). The mean body mass index was 41.8. Of patients, 39% had a history or family history of deep vein thrombosis. The average time to diagnosis was 13 days (range, 5–25). Treatment was nonoperative with anticoagulation in 94%. One patient required operative management with bowel resection. All patients were discharged on therapeutic anticoagulation. Mean total weight loss was 27.7% (14.8%–66.3%). Mean follow-up was 10 months. There were no mortalities. Given the low number of patients, no statistically significant data could be derived.

Conclusion: PVT is difficult to diagnose, with significant consequences. The presenting symptoms are nonspecific, and a high index of suspicion needs to be maintained. Cross-sectional imaging with computed tomography of the abdomen is recommended. Patients with PVT post-LSG without previous risk factors can be anticoagulated for 3 to 6 months with an international normalized ratio of 2 to 3. (Surg Obes Relat Dis 2018;14:271–276.)

Keywords: Portomesenteric vein thrombosis; Sleeve gastrectomy; Bariatric

The proportion of overweight and obese people in Australia is approximately 60% [1]. Bariatric surgery is becoming a recognized, well-established modality in the treatment of both obesity and its associated co-morbidities. Sleeve gastrectomy (SG) was the second most common

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https://doi.org/10.1016/j.soard.2017.12.010

bariatric procedure performed in Australia and New Zealand in 2011 [2], and with its rapidly growing popularity, it is expected to soon surpass the other bariatric procedures in world prevalence [3].

Portomesenteric vein thrombosis (PVT) is a recognized complication of SG with an incidence rate of .3% to 1% [4–6] and a total population incidence of 1.1% [7]. Its presenting symptoms can be nonspecific, making it difficult to diagnose. However, it has severe consequences, such as ascites (62%), esophageal varices (58%), terminal gastroesophageal bleeding (47%) [7], and bowel infarction, which is an important cause of mortality [8].

The aim of this study was to document the incidence, risk factors, presenting symptoms, mainstays of treatment, and outcomes in the largest recorded case series involving patients with PVT after laparoscopic SG (LSG).

Methods

This study included a multicenter retrospective analysis of all patients who underwent LSG complicated by PVT. It involved 5 bariatric centers across Australia from February 2007 to August 2016. Patients were identified using surgeons' personal records.

Study inclusion criteria were patients who had undergone an elective LSG for the purpose of weight loss, who went on to have a PVT. The term portal vein thrombosis included thrombus located in the portal vein, superior mesenteric vein, and/or splenic vein.

Patient demographic characteristics, complications throughout their elective admission, risk factors for thrombosis, thrombophilia results, anticoagulation regimens, final outcome, and final weight loss were recorded.

Ethics approval was granted with approval number 17/38.

Results

Of the 5951 patients who underwent an elective SG, 18 had recognized PVT, equating to an incidence of .3%.

Table 1 provides the baseline characteristics of the patients. All patients had at least one systemic predisposition toward venous thrombosis. These included prothrombotic factors, such as morbid obesity (n = 9), personal or family history of deep vein thrombosis (DVT; n = 7), current smoker (n = 4), or being on hormone replacement therapy or oral contraceptive pill (n = 3). One patient was diagnosed with Factor V Leiden deficiency.

The initial admission for LSG was uncomplicated for all patients except one, who had PVT diagnosed during the initial admission. The average time of operation, as a surrogate for time of liver retraction, was 51 minutes (range, 40–85). The standard intra-abdominal pressure used by all surgeons was 15 mm Hg. Mean length of stay was 3.9 days. All patients included in this study received thromboprophylaxis during admission for SG. The standard protocol differed among centers and consisted of either prophylactic doses of low molecular weight heparin (LMWH) or unfractionated heparin with mechanical thromboprophylaxis. All surgeons involved routinely discharged their high-risk patients on thromboprophylaxis. None of the patients with recognized PVT had a prior admission for dehydration or difficulty with oral intake before the PVT was diagnosed.

Table 1
Patient baseline characteristics

Baseline characteristics	N (%) or mean (range)
Age	44 (27–60)
Sex	
Male	7 (39)
Female	11 (61)
Baseline BMI	41.8 (33.3–54.3)
Personal/Family history of clotting disorder	7 (39)
Smoker	
Current smoker	4 (22)
Ex-smoker	5 (28)
Never smoked	9 (50)
Thrombophilia screen positive	1 (6)
Oral contraceptive/HRT for females	3 (27)

BMI = body mass index; HRT = hormone replacement therapy.

The average time to diagnosis was 13 days (5–25), and patients most commonly presented with abdominal pain (77%) and nausea and vomiting (33%). Other, less common, presenting symptoms included shoulder tip pain alone, inability to tolerate fluids, constipation, and diarrhea.

Mean white cell count on diagnosis was 11 (range, 3.2–22.5). Mean C-reactive protein was 135, ranging from 12 to 267. Of patients, 83% had C-reactive protein and white cell count performed on admission.

Patients were most commonly diagnosed with PVT by computed tomography (CT) alone (56%), followed by a combination of CT and duplex ultrasound (DUS) (32%) and DUS alone (11%). When DUS was performed in conjunction with CT, it was most often performed after the CT to either confirm the diagnosis or to determine the extent of thrombosis.

Treatment varied depending on hospital site: 44% of patients received therapeutic LMWH, another 44% were initiated on a heparin infusion, and 12% were started on other forms of anticoagulation including riveroxiban and warfarin. Of the patients started on LMWH, 50% were converted to warfarin, 37.5% continued to have LMWH for the entirely of their treatment, and 12.5% were converted to riveroxiban. Of the patients on heparin infusions, all except one were discharged on warfarin with LMWH bridging. The one patient developed heparin-induced thrombocytopenia and had to be converted to a danaparoid infusion; this patient was subsequently discharged on warfarin. Of patients, 66% were kept on warfarin for between 3 and 6 months. Of patients, 11% stayed on warfarin for life given their history of DVT and pulmonary embolism (PE). No patient in this series received thrombolysis.

Only one patient required operative management. This patient's medical history was significant for nonalcoholic fatty liver disease. Admission for SG was unremarkable, except that the patient was noted to have a fatty liver with slight nodularity intraoperatively. The patient presented 2 weeks later with generalized abdominal pain and was tachycardic and febrile with a raised white cell count. CRP on admission was 76. CT on arrival demonstrated PVT with small bowel edema. The patient was started on a heparin infusion but deteriorated overnight, requiring an intensive care unit admission. Laparoscopy demonstrated venous ischemia of the small bowel. Because it was not frankly necrotic, no small bowel was resected. Postoperatively, the patient improved temporarily but then began to deteriorate, requiring inotropic support and showing signs of liver failure. The patient was diagnosed with heparin-induced thrombocytopenia 1 week postadmission and was converted to a danaparoid infusion. Three additional laparotomies took place with progressive small bowel resection, leaving the patient with 50 cm of terminal ileum with a few centimeters of proximal jejunum, an intact stomach, duodenum, colon, and ileo-caecal valve. The patient initially recovered, passing 1 bowel motion per day, and was discharged on warfarin. Five months postoperatively, the patient showed signs of nutritional failure with progressive weight loss, requiring a 6-week admission with total parenteral nutrition. At 1-year follow-up, total parenteral nutrition had been ceased and oral diet was being tolerated, maintaining a stable weight.

Postdischarge, the mean percentage of total weight loss was 27.7% (range, 14.8%–66.3%). Mean loss in body mass index was 11.6 (range, 5–30.5). Mean percentage of excess weight loss was 25% (range, 15%–45%). Mean follow-up was 10 months. Of patients, 66% were followed up for >6 months. There were no mortalities.

Discussion

There are numerous theories regarding why patients develop PVT in general. These can be divided into local and systemic factors [9]:

1. Systemic causes include inherited prothrombotic disorders, acquired hematologic disorders associated with thrombosis, drugs (e.g., oral contraceptive pill), and miscellaneous factors including sepsis [9]. There is an association with intra-abdominal or systemic sepsis in nearly 40% of patients with PVT [10]. Other risk factors include cirrhosis (28%), primary hepatobiliary (HPB) cancer (23%), and secondary malignancy of HPB region (44%) [7].
2. Local factors include pancreatitis and postsurgical (e.g., liver transplant or splenectomy) or portal vein compression by nodes [9]. 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 [11].

The incidence of PVT post-LSG found in this study is very similar to that of others [4] but does not quite reach the 1% mentioned elsewhere in the literature [5]. It appears that the incidence of PVT in LSG is higher than that with other bariatric operations, with Goitein et al. [4] having 0 from 966 PVT in laparoscopic Roux-en-Y gastric bypasses and 0 from 142 biliary pancreatic diversions. It is noted that 7 cases of PVT post–laparoscopic Roux-en-Y gastric bypass have been reported elsewhere in the literature [11]; however, the denominator in this case is unknown, making the incidence unclear.

A number of theories have been put forward to explain the increased risk of a PVT associated with LSG [4]. These can be divided into intraoperative factors and postoperative factors.

Intraoperative factors include

1. Ligation of the right gastroepipolic vessels with energy devices is in close proximity to the splenic vein. The mechanical or thermal effect can potentially cause thrombosis [5].
2. Ligation of short gastric vessels changes the venous return from the stomach. This may be a factor in PVT formation and has previously been suggested as the causative factor in the formation of PVT after laparoscopic fundoplication [11–13].
3. Prolonged liver retraction could potentially cause congestion and stasis within the liver, causing a clot to form [14].

Postoperative factors include PVT usually presenting after the patient has been discharged. This may in part be due to dehydration postdischarge. Hypovolemia is a known risk factor for developing thrombosis, including DVT, PE, and PVT [15–18].

The clinical features of acute PVT are poorly defined in the literature [19]. Throughout, presentation of PVT has been described as vague, with typical symptoms, such as abdominal pain, nausea, and fever [4–6,11,19–21]. The severity of symptoms varies significantly and may be associated with the extent of mesenteric venous thrombosis because of bowel ischemia [20]. After laparoscopic surgery, the literature suggests that symptoms appeared on average 12 to 15 days postoperatively, in keeping with our findings [4,5,11].

Diagnosis can be made with a combination of color Doppler ultrasound, contrast-enhanced CT, or magnetic resonance angiography [22]. The literature is divided about whether Doppler ultrasound should be first line [23,24] or whether CT should be first line [22,25,26].

Doppler ultrasound allows direct evaluation of mesenteric and portal veins, provides semiquantitative flow information, and permits Doppler wave form 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" [22]. At the present time, this modality is less sensitive than CT and magnetic resonance for the detection of splanchnic vein thrombosis, particularly the splenic and superior mesenteric veins [22]. We agree with the recommendation made by Bradbury et al. [22], especially as patients often present with such vague symptoms, that cross-sectional imaging can be far more useful than that of a targeted ultrasound.

Other methods of diagnosis have also been investigated with linear endoscopic ultrasound having a sensitivity of 81% and a specificity of 93% in a nonblinded, small study [27]. Because of the CT sensitivity of 90% [25], in addition to the fact that endoscopic ultrasound is invasive and usually an in-hours service, CT was still thought to be superior.

Treatment

Studies have shown that anticoagulation may result in recanalization in >80% of cases [19]. There is no consensus regarding duration and extent of anticoagulation [6]. We advocate for the guidelines set out by Webster et al. [9] for 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., postsurgery), a 3- to 6-month course of warfarin with an international normalized ratio of 2 to 3 is reasonable. Patients with prothrombotic tendencies, ongoing local predispositions to PVT, or extensive thrombosis may warrant long-term anticoagulation.

There is some evidence to suggest treating acute PVT with early thrombolysis, as there are higher rates of recanalization compared with either conservative management or heparin infusion [9,28,29]. These studies are not specifically looking at postoperative patients, but rather at patients with cirrhosis and malignancies.

There has been 1 study in which a successful thrombolysis occurred in a postoperative patient [4]. Upon laparotomy, the patient was found to have edematous, ischemic bowel. The patient underwent percutaneous transhepatic thrombolysis of the portal vein with a continuous infusion for 2 days. On second and third relook laparotomies, the patient had viable bowel, and no resection had to be performed.

In our view, in the postoperative setting, thrombolytic therapy must be considered very carefully before being initiated, as there is a possibility of further surgery with the potential for bowel resection if the thrombolysis is unsuccessful. Further research in this area is recommended.

Prevention

There is very little in the literature regarding prevention and prophylaxis of PVT after LSG. However, similar to the guidelines on treatment, prophylaxis would likely be guided by that of deep venous thrombosis.

DVT and PE remain among the leading causes of mortality after bariatric procedures, with evidence of DVT in 1% to 3% of patients and PE in .3% to 2% [15]. The mortality of bariatric patients with PE has been reported to be as high as 30% [17,30–32]. 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 to have died from PE, up to 80% of patients had microscopic evidence of pulmonary emboli, despite being on appropriate prophylaxis [32].

In a survey published in 2009 [15], approximately 50% of bariatric surgeons reported at least 1 fatality because of DVT complications. In-hospital thromboprophylaxis has been recognized to reduce the incidence of DVT, however, this raises the question of postdischarge thromboprophylaxis.

Although suggested by many papers [16,33,34], there is currently no evidence on whether home thromboprophylaxis changes DVT outcomes. However, this issue has been extensively studied in another high-risk thrombus group—cancer patients. Prophylaxis for 2 to 3 weeks with LMWH after discharge appears to reduce the incidence of asymptomatic DVT in cancer surgery patients [35]. A double blinded multicenter randomized control trial comparing LMWH for 3 weeks postdischarge with receiving thromboprophylaxis in hospital only demonstrated that discharging with prophylaxis was superior, with the rate of Venous thromboembolism of 12% in the placebo group and 4.8% in the treatment group ($P = .02$) [36].

At this point, there is inadequate evidence regarding the utility of prophylactic thromboprophylaxis after discharge for prevention of PVT. Prospective, controlled trials are recommended to assist in developing guidelines.

One of the limitations of this study is that patients were retrospectively identified, potentially causing recall bias. However, patients were identified through audits and mandatory in-hospital morbidity and mortality meetings. In addition, all involved surgeons had electronic records in which patient complications were prospectively recorded.

A further limitation of this study was patient follow-up. Standard practice is that all patients were contacted within 1 to 2 weeks by the practice nurse, reducing any bias. All patients were reviewed by the surgeon within 6 weeks of surgery. As this study has demonstrated, the majority of patients present with symptoms within the first fortnight; therefore, we are confident the majority of patients would have been identified, but the risk is that this could potentially cause an underestimation of the true incidence of PVT in LSG.

Conclusion

PVT in LSG is a rare complication, but it can have disastrous effects. Early detection is self-evidently optimal. The presenting symptoms are nonspecific, but it can be diagnosed on cross-sectional imaging. Anticoagulation for 3 to 6 months with a target international normalized ratio of 2 to 3 is recommended unless the patient has additional risk factors and therefore indicated for longer treatment. Further research into thrombolysis and anticoagulation postdischarge is recommended.

Disclosures

The authors have no commercial associations that might be a conflict of interest in relation to this article.

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