Postoperative mesenteric venous thrombosis: Potential complication related to minimal access surgery in a patient with undiagnosed hypercoagulability

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Abstract

Context: Mesenteric venous thrombosis is a rare but potentially fatal complication associated with laparoscopy which has now become common practice and gold standard for many procedures in general surgery. There are only few scattered case reports in the literature describing this postoperative thrombotic event. Case Report: In the present study, we describe a patient presenting with severe abdominal pain at 25 days following an uneventful laparoscopic paraesophageal hernia (PEH) repair and nissen fundoplication. Exploratory laparotomy revealed an extensive small bowel ischemia requiring bowel resection followed by a second look laparotomy. Retrospectively performed hematologic workup revealed a genetic mutation associated with hyperhomocysteinemia in addition to her hyperfibrinogenemia. Previously published data were collected and discussed. Conclusions: Mesenteric venous thrombosis is a rare but potentially serious complication after laparoscopic surgery especially in patients with underlying hypercoagulability. High index of suspicion is important in early diagnosis and subsequent treatment.

Keywords: Mesenteric venous thrombosis, laparoscopy, hypercoagulability.

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Introduction

Peritoneal insufflation during laparoscopic surgery may carry significant morbidity of thrombosis in the mesenteric venous system. Its clinical presentations vary from a nonspecific abdominal discomfort to an acute abdomen mandating an exploratory laparotomy. Warren and Eberhard in 1935 reported 34% mortality rate [1]. In more modern day however, prompt diagnosis followed by an aggressive use of anticoagulation, thrombolytic agents and relatively new endovascular techniques seem to improve outcomes. Previously published reports have described MVT in association with roux-en-y gastric bypass, splenectomy, colectomy, cholecystectomy and appendectomy. In this article, we report a patient who developed an extensive small bowel infarction due to mesenteric venous thrombosis (MVT) following a laparoscopic paraesophageal hernia repair and nissen fundoplication.

A 61 year old female with history of hypertension and gastroesophageal reflux was referred to our facility for a repair of type III paraesophageal hernia with 75% intrathoracic stomach. She then underwent an uneventful laparoscopic repair and nissen fundoplication. Her postoperative course was unremarkable and she was discharged home on postoperative day (POD) 2. On POD 9, she presented to another institution with fever and urinary symptoms. After appropriate investigation, she was released on an oral antibiotic for urinary tract
infection. During follow up in our clinic 2 weeks postoperatively she complained of nonspecific abdominal pain and moderate gas bloatedness consistent with common complaints following nissen fundoplication. The remainder of her examination were unremarkable.

On POD 25 she represented to our facility with acute frank peritonitis. Computed tomography (CT) scan demonstrated thickened small bowel loops consistent with mural ischemia (Fig. 1) and nearly complete filling defects in the superior mesenteric/portal veins (Fig. 2). Laboratory findings showed leukocytosis of 25,000/mm³ and elevated lactic acid of 9.4 mg/dl.

Patient was immediately heparinized and taken to the operating room for an exploratory laparotomy when 110 cm of ischemic jejunum with complete thrombosis of the superior mesenteric vein (SMV) were found. Jejunal resection with primary anastomosis was performed and the abdomen was left open. During a second look laparotomy on the following day, dusky terminal ileum was again seen with adequately perfused surrounding small bowel and cecum mandating an a dditional 10 cm terminal ileum resection. The abdomen was closed and an end-ileostomy was created. Postoperative course was complicated by small bowel leak and enterocutaneous fistulas requiring reoperations. Comprehensive hematologic workup revealed a C-677-T point mutation associated with methylenetetrahydrofolate reductase (MTHFR) deficiency leading to an elevated level of homocysteine [2]. Hyperfibrinogenemia was also identified.

Mesenteric venous thrombosis was first described by Elliot in 1895 [3]. It accounts for 5-15% of reported cases of acute mesenteric ischemia [4, 5]. Previously reported cases demonstrated mean age of 43 without significant difference in gender distribution. It is postulated that MVT is caused by physiologic derangements associated with laparoscopy since it occurs at a much lower incidence following open surgery. An early prospective study conducted by Masataka Ikeda et al on patients undergoing splenectomy revealed that portal venous system thrombosis occurred at a significantly higher rate in laparoscopic group versus open group, 55% and 19% respectively [6]. Twenty four previously reported cases of MVT that we have collected along with our current case are displayed in Table 1.

A variety of factors associated with laparoscopy such as pneumoperitoneum induced hemodynamic changes, hypercapnia induced mesenteric vasoconstriction, and coagulation impairment may contribute to the development of MVT. Takagi et al showed that portal venous trunk diameter and blood flow were significantly decreased with intraperitoneal pressure elevation above 10 mmHg [7]. Quantitatively measured, elevation of intraperitoneal pressure during laparoscopy to 7 and 14 mmHg is associated with 37% and 53% reduction in mean portal blood flow respectively. Majority of splanchnic vein thrombosis developed within the widely accepted insufflation pressure of 12 to 15 mm Hg. Elevated portal venous pressure during laparoscopy normalizes after abdominal desufflation [8].

Increased serum level of vasopressin related to hypercapnia is a well documented phenomenon that also occurs in response to peritoneal insufflation using carbondioxide (CO2) [9]. Epstein et al discovered that hypercapnia induced by breathing CO2 at a concentration less than 5 percent increases portal venous pressure and results in mesenteric vasoconstriction [10].
Soft tissue trauma associated with surgery releases tissue factors that can lead toward MVT in patients with previously undiagnosed hypercoagulable state. Our literature review showed that 44% of patients have had underlying prothrombotic state which was diagnosed retrospectively only after occurrence of this complication (Table 1). The more likely occurrence of MVT during laparoscopic operations for inflammatory pathologies such as diverticulitis seems to support this theory. In addition, literature has also described a controversy on alteration of coagulation associated with laparoscopy. It was reported that laparoscopy potentially causes a prothrombotic state [11].

No single classic presentation exists for this disease therefore clinical suspicion is crucial. Laboratory abnormalities include leukocytosis, elevated lactate, amylase, and creatinine kinase [12]. The current diagnostic standard is CT scanning with a sensitivity of 90% [13]. Acute MVT presents as a central lucency in mesenteric vein arcades. Dilatation of the SMV with a prominent vessel surrounded by hyperdense tissue is suggestive. Edematous bowel or mesenteric fat stranding, although not conclusive, should also prompt consideration of MVT in the absence of other obvious clinical etiologies. Even though angiography is the gold standard in diagnosing arterial thrombosis, it is less valuable in this situation [14]. Other tests such as transabdominal doppler ultrasound, and magnetic resonance imaging are less accurate and more expensive without additional benefit respectively [15].

Diagnostic laparoscopy is controversial for a suspected MVT because it may compound the primary problem. However, many surgeons prefer to perform diagnostic laparoscopy to exclude bowel ischemia therefore circumventing the need for nontherapeutic laparotomy [16-18]. Not to undermine the superiority of CT scan in diagnosing MVT, it is however notoriously to be inaccurate in determining the extent of bowel ischemia.

All patients with known underlying prothrombotic states should be placed on a preoperative thrombotic prophylaxis regimen. In our opinion, a consultation with a hemato-oncologist is an appropriate step in this situation. When it occurs postoperatively, therapeutic anticoagulation using heparin drip must be immediately started. Aggressive intravenous hydration, bowel rest and total parenteral nutrition are important adjunctive measures to operative intervention. Abu et al demonstrated a 12% reduction in mortality in patients undergoing resection and anticoagulation versus those who underwent resection alone [19]. Less invasive measures such as surgical thrombectomy, percutaneous transhepatic thrombectomy, intra-arterial papaverin infusion through

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### Table 1: Previously published reports on splanchic venous thrombosis following laparoscopic operations

| Authors                  | n | Sex | Age (yr) | Laparoscopic Surgery | Prothrombotic State | Operative time (min) | Intra-abdominal pressure (mmHg) | Onset of Symptoms (POD) | Findings/Treatments |
|--------------------------|---|-----|----------|----------------------|---------------------|----------------------|-------------------------------|------------------------|-------------------|
| Masataka et al [6]       | 12| M:F=2:10 | 44 (18-67) | Splenectomy | Yes | 3/12 | 116 | 10 - 12 | 6.75 | Anticoagulation only |
| Ouania A et al [25]      | 1 | F   | 32       | Cholecystectomy    | Yes | N/A | 15 | 7 | Percutaneous transhepatic thrombectomy |
| Lydia L et al [26]       | 1 | M   | 19       | Appendectomy       | No | N/A | NR | 7 | Anticoagulation only |
| Garcia et al [27]        | 1 | M   | 20       | Nissen Fundoplication | No | 105 | 14 | 7 | Extensive thrombosis on laparotomy, Postop Anticoagulation Negative Laparotomy |
| Millikan KW et al [23]   | 1 | M   | 47       | Nissen Fundoplication | Yes | 143 | 12 to 15 | 42 | Anticoagulation only |
| Garcia Diaz RA et al [27] | 1 | F   | 74       | Sigmoidectomy      | Yes | N/A | NR | 11.6 | 2 Patients required bowel resection, 1 with Negative Laparotomy |
| Current Case             | 1 | M   | 43       | Roux-en-Y Gastric Bypass | Yes | < 60 | NR | 25 | Bowel resection and Anticoagulation |
| Johnson CM et al [10]    | 1 | M   | 63       | Lap assisted R Hemicolectomy | No | 150 | NR | 10 | Streptokinase IV |
| Poultsides GA [11]       | 1 | M   | 48       | Lap assisted R Hemicolectomy | Yes | N/A | 12 - 15 | 8 | Urokinase infusion via SMV Anticoagulation only |
| Baixauli J et al [30]    | 1 | F   | 74       | Sigmoidectomy      | Yes | 120 | 12 | 7 | Anticoagulation only |
| Current Case + Patient expired | | | | | | | | | |
| Swartz DE et al [15]     | 3 | M:F=2:1 | 46.3 (36-53) | Roux-en-Y Gastric Bypass | Yes | N/A | NR | 25 | Bowel resection and Anticoagulation |
| Current Case + Patient expired | | | | | | | | | |

NR= Not Reported
superior mesenteric artery, and thrombolytics (urokinase and streptokinase) have also been successfully used [16, 21-24]. For those with primary coagulation disorder, lifelong anticoagulation is mandatory.

Conclusions
Splanchnic venous thrombosis is a rare but potentially serious complication associated with laparoscopic surgery especially in patients with underlying hypercoagulability disorders. Patient’s medical history is key for its perioperative managements and subsequent prevention. High index of suspicion is needed for early diagnosis and treatment when this condition occurs. Primary care physicians including general surgeons should be well aware of this potential untoward event post laparoscopic operations.

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