

# Superior Mesenteric Vein Thrombosis Following Laparoscopic Nissen Fundoplication

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## ABSTRACT

This report describes the second case of a superior mesenteric and portal vein thrombosis following an uneventful laparoscopic Nissen fundoplication. The patient presented on postoperative day 10 with acute onset of abdominal pain and inability to tolerate oral food. A computed tomography (CT) scan revealed superior mesenteric and portal venous thrombosis with questionable viability of the proximal small bowel. He was heparinized and taken for emergent exploratory laparotomy. At surgery and at a planned re-exploration the following day, the bowel was viable and no resection was needed. Despite continuation on anticoagulation therapy, he developed a pulmonary embolism. A hypercoagulable workup was normal. After continued anticoagulation therapy and supportive care, a duplex ultrasound 2 months after the event showed normal flow in both the superior mesenteric and portal veins. Possible mechanisms are discussed along with a review of the pertinent literature.

**Key Words:** Fundoplication, Laparoscopy, Mesenteric vein thrombosis.

## INTRODUCTION

Laparoscopic Nissen fundoplication is a common procedure performed in many patients experiencing symptomatic gastric esophageal reflux. Common complications associated with the procedure include pain, bleeding, infection, failure of the procedure, and gas-bloat syndrome. As part of the technique, carbon dioxide is used to create a pneumoperitoneum. This has been described to cause reduction in portal venous flow. In addition, this has been associated with superior mesenteric and portal vein thrombosis in certain laparoscopic-assisted surgeries. To our knowledge, this is the second case of a superior mesenteric and portal vein thrombosis following laparoscopic Nissen fundoplication.

## CASE REPORT

A 36-year-old otherwise healthy male presented with 2 years of symptomatic reflux disease, nonresponsive to proton pump inhibitors. A preoperative esophagogastroduodenoscopy showed mild esophageal reflux, manometry was normal, and 24-hour pH study demonstrated a DeMeester score of 83 (normal less than 14.72). His past medical history was unremarkable. The patient had no family or personal history of stroke, deep venous thrombosis, or bleeding disorders. He did smoke approximately 1/2 to 1 pack of cigarettes per day. He was admitted to the hospital and underwent an uncomplicated elective laparoscopic Nissen fundoplication. Perioperatively, he was not given subcutaneous heparin or low-molecular weight heparin. He did have sequential compression devices on during surgery. The total operative time was 92 minutes with peak intraabdominal pneumoperitoneum pressures at 15 mm Hg. He was discharged home on postoperative day 2 after an uncomplicated hospital stay.

He presented to an outside clinic on postoperative day 10 with complaints of diarrhea and some nausea. He was afebrile with a normal white blood cell count and liver function tests. An acute abdominal test series was unremarkable. He was rehydrated with 2 liters of normal saline, and after feeling better, discharged home. He re-presented the following morning to our hospital emergency department with diffuse abdominal pain and loose

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stools. Vital signs and laboratory examinations were normal. Abdominal films showed a dilated colon with colonic air-fluid levels. He was observed overnight and started empirically on flagyl for presumed *C. difficile* colitis. The following morning, he remained in considerable pain, but with normal vital signs and laboratory evaluation. An abdominal computed tomography (CT) scan demonstrated superior mesenteric and portal venous thrombosis with questionable viability of the proximal small bowel (**Figures 1 and 2**). He was started on heparin and taken for emergent exploratory celiotomy. The small bowel was thickened, but viable, and an appendectomy with manual decompression of the bowel was performed. The patient was continued on heparin overnight, and the scheduled re-exploration laparotomy the following morning again demonstrated viable bowel.

He was continued on heparin, and extubated on postoperative day 3. He did well until the morning of postoperative day 5 when he was noted to be acutely tachypneic with oxygen desaturation and tachycardic. A duplex ultrasound of the bilateral upper extremity and lower extremity showed only a clot surrounding his left internal jugular line. He was intubated and a chest CT scan demonstrated bilateral lower lobe infiltrates. A pulmonary angiogram demonstrated a right lower lobe pulmonary emboli extending into the medial basal branch of the right pulmonary artery. Over the next week, he was resuscitated, heparinized, and treated for concomitant pneumonia and pancreatitis. He eventually did well and was discharged 1 week later on oral anticoagulation. The follow-up extremity duplex at 1 month showed no upper extremity or lower extremity venous thrombosis, as well as a re-canalized left internal jugular vein with a small amount of residual thrombosis.

The patient had no other risk factors for hypercoagulability except for his recent laparoscopic surgery and history of tobacco use. Hematological studies that included antiphospholipid antibody, lupus anticoagulant, antithrombin III, protein C, protein S, anticardiolipin antibody, homocysteine, APC-Resistant factor V, PT 20210, and factor V Leyden were all normal.

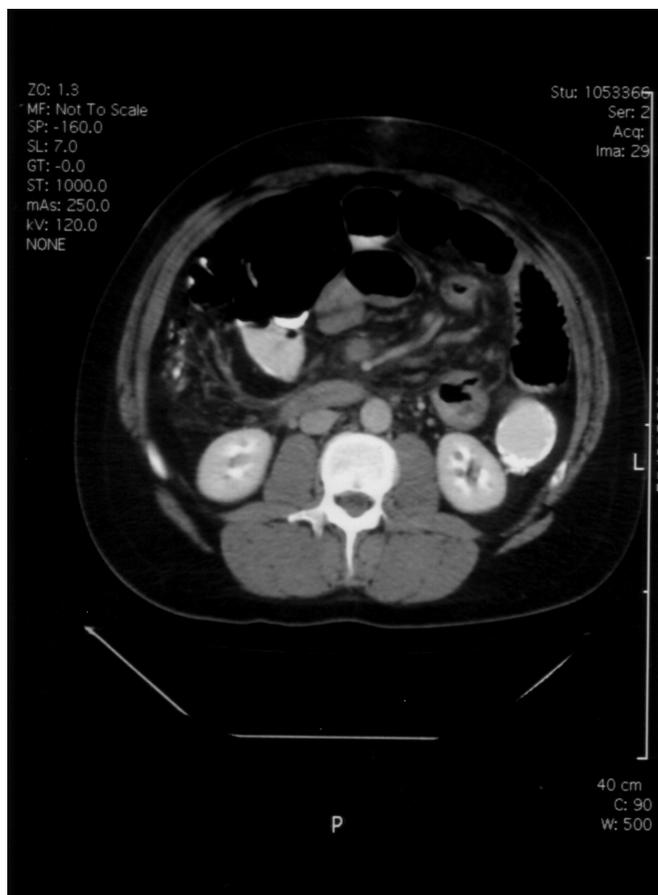
The patient was continued on oral anticoagulation as an outpatient, and a follow-up duplex ultrasound at 2 months demonstrated normal flow in the superior mesenteric and portal vein with no sign of thrombus. The patient remained clinically asymptomatic.



**Figure 1.** CT of the abdomen demonstrating superior mesenteric vein thrombosis.

## DISCUSSION

Gastroesophageal reflux disease (GERD) has gone from a little known disorder in the 1930s to one of the most common presenting complaints today to both primary care physicians and surgeons.<sup>1,2</sup> Similarly, antireflux surgery has seen an exponential rise over the past few decades. Laparoscopic Nissen fundoplication is one of the mainstays for treatment of this disease, used in approximately 75% to 80% of most laparoscopic antireflux surgery. In comparison with medical therapy, it has been shown to be both cost-effective, as well as achieving symptomatic control in over 90% of patients.<sup>3,4</sup> It has the additional benefit of allowing patients the ability to avoid or reduce daily medications.



**Figure 2.** CT of the abdomen demonstrating thickened duodenum and diffuse fat stranding.

This surgery has a low morbidity rate of 4% to 15%.<sup>5</sup> There is a 0.5% rate of early re-operation, and persistent postoperative dysphagia of 3% to 5%.<sup>6</sup> Less commonly occurring complications associated with the surgery include gas bloat syndrome, “slipping” of the wrap, and esophageal or stomach perforation, or both of these.<sup>7</sup> The Nissen fundoplication is almost always completed as a laparoscopic operation, with a conversion rate of 3% to 5.2%.<sup>2</sup> Laparoscopy presents additional potential complications, such as trocar injury, pneumothorax (1%), and pneumomediastinum.<sup>8</sup> The effects of the carbon dioxide-induced pneumoperitoneum have also been shown to decrease the rate of splanchnic blood flow.<sup>9,10</sup> Insufflation pressures of as little as 14 mm Hg have been shown to reduce portal venous flow by 53%.<sup>11</sup> Possible mechanisms of this include mechanical compression of

the mesenteric vessels, carbon dioxide-induced vasospasm, and systemic hypercapnia leading to the decrease in portal flow.<sup>10</sup> Thrombosis in the portal and superior mesenteric vein due to this reduction in flow have been described in other laparoscopic cases, such as laparoscopic right hemicolectomy.<sup>12</sup>

In only one other case have the effects of pneumoperitoneum associated with laparoscopic Nissen fundoplication been associated with a superior mesenteric and portal venous thrombosis.<sup>13</sup> In that case, however, the patient had had a deep venous thrombosis 13 years prior to the operation, and a subsequent normal hypercoagulable workup. He also presented with insidious onset of abdominal pain 2 months after the procedure. A CT scan demonstrated the thrombus in the superior mesenteric and portal veins. That patient was treated with coumadin for 3 months. A follow-up duplex showed normal flow and he remained asymptomatic. Our patient presented with acute onset of abdominal pain and a thrombosis with diffuse vascular engorgement with dilated loops of thickened small bowel and fat stranding. Heparinization was immediately started, and he was taken for laparotomy due to the question of bowel viability with scheduled re-exploration in 24 hours.

Mesenteric venous thrombosis is a relatively uncommon clinical entity, encompassing 5% to 15% of mesenteric vascular occlusions.<sup>14</sup> First described by Elliott<sup>15</sup> in 1895, symptoms can range from an insidious onset of low grade abdominal pain to acute onset of pain, bowel ischemia, infarction or perforation requiring urgent exploratory surgery. Classically, mesenteric venous thrombosis remains a diagnostic dilemma. History and physical examination are often unremarkable, making the clinical diagnosis difficult. Additionally, this happens in young patients with no predisposing risk factors. The most common presenting complaint is acute onset of abdominal pain with progressive signs of bowel ischemia. Other patients present with a more indolent pain occurring over the course of weeks to months.<sup>16</sup> Pain is characteristically out of proportion to the findings of the physical examination. Other presenting complaints include anorexia, diarrhea, nausea and emesis, constipation, and GI bleeding.<sup>17</sup> CT scan and other radiological imaging, such as ultrasound or angiography, are important in helping make this diagnosis. The CT scan remains the optimal examination, and reveals the thrombus, collateral circulation (when onset is subacute), as well as any bowel wall changes.<sup>17,18</sup>

Patients often have a history of an abdominal inflammatory process, or a subsequent hypercoagulable workup is positive. Protein S deficiency,<sup>14</sup> Protein C deficiency,<sup>19</sup> primary antiphospholipid antibody,<sup>20</sup> factor VII abnormalities,<sup>21</sup> anticardiolipin antibody,<sup>22</sup> myeloproliferative disease,<sup>23</sup> factor IX deficiency,<sup>24</sup> and antithrombin III deficiency<sup>25</sup> have all been documented in association with the development of superior mesenteric and portal vein thrombosis. Some other risk factors associated with the development of superior mesenteric or portal vein thrombosis include cirrhosis and abdominal inflammatory states like pancreatitis,<sup>26</sup> appendicitis, or peritonitis,<sup>18</sup> a delayed diagnosis of malrotation,<sup>27</sup> infections with *Yersinia*,<sup>28</sup> and even young women taking oral contraceptives.<sup>29</sup> In most patients, a delay in diagnosis can range from weeks to years.

Treatment of mesenteric thrombosis depends on the patient's clinical state. Treatment can range from anticoagulation therapy to exploratory laparotomy should signs of intestinal infarction be present. Surgical thrombectomy,<sup>30</sup> percutaneous transhepatic thrombectomy,<sup>31</sup> and thrombolytics (urokinase and streptokinase)<sup>12,32</sup> have also been used successfully. As in this case, patients who present with signs consistent with intestinal infarction should have a laparotomy with resection as necessary, followed by planned re-exploration 24 hours later and anticoagulation.<sup>17</sup>

Possible causes of superior mesenteric vein thrombosis in our patient include surgery and effects of the pneumoperitoneum. Additionally, he did have a smoking history, which is known to predispose one to vascular thrombosis.<sup>29</sup> He had no other predisposing conditions. Although an unknown hypercoagulable disorder may be present, his initial hematological workup was normal. Additional screening in the future may be warranted. No trauma occurred during surgery or kinking of vessels that have been described in other cases as possible causes.<sup>12</sup> Finally, no indications were present of an inflammatory process, such as pancreatitis, as the inciting event. It is important to keep in mind that although older studies<sup>33</sup> showed no predisposing factors in 55% of patients, more recent analyses have shown over 80% of patients have contributing events.<sup>34</sup>

## CONCLUSION

Although this case marks only the second documented instance of superior mesenteric and portal vein thrombo-

sis following a laparoscopic Nissen fundoplication, as long-term data begin to grow, the incidence of this potential complication may increase. Patients who present postoperatively with unusual abdominal pain and otherwise normal vital signs and laboratory examinations should be evaluated with this potential complication in mind. In addition, in all cases involving a pneumoperitoneum, insufflation pressures should be kept at the lowest level while still providing optimal visualization.

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