Superior Mesenteric and Portal Vein Thrombosis Following Laparoscopic-Assisted Right Hemicolectomy

Report of a Case

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PURPOSE: This article describes a case of superior mesenteric and portal vein thrombosis following laparoscopic-assisted right hemicolectomy. METHODS: A retrospective case review was performed. RESULTS: Data continue to grow regarding safety and technical feasibility of laparoscopic-assisted colectomy. As this minimally invasive alternative to open colonic resection becomes more popular, it is inevitable that information on benefits and complications associated with it will continue to expand. We report a case of superior mesenteric and portal vein thrombosis following laparoscopic-assisted colon resection. To our knowledge, this represents a complication of laparoscopic colon resection not previously reported in literature. CONCLUSION: Careful patient selection for this procedure is important. Additionally, the incision for extracorporeal resection and anastomosis in laparoscopic-assisted colectomy must be planned appropriately and carefully monitored intraoperatively to avoid potential complication of vascular trauma leading to mesenteric vein thrombosis. [Key words: Laparoscopy; Laparoscopic colectomy; Complications; Laparoscopic-assisted colectomy]

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Laparoscopic-assisted colectomy has been shown to be technically feasible for a number of colonic pathologic conditions, including colorectal polyps, cancer, diverticular disease, colonic lipomas, rectal prolapse, intestinal stomas for diversion, cecal or sigmoid volvulus, bleeding, and colitis.[1, 2] As this minimally invasive alternative to open colonic resection becomes more popular, it is inevitable that complications of this approach will be described. A case of superior mesenteric and portal vein thrombosis following a laparoscopic-assisted hemicolectomy is reported. To our knowledge, this is a complication of laparascopic colon resection that has not been previously reported in the literature.

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REPORT OF A CASE

A 63-year-old man was found to have occult blood in his stool on routine physical examination. Past medical history was significant for hypertension and gout, for which he takes Hytrin® (Abbott Laboratories, Abbott Park, IL) and Benemid® (Merck, West Point, PA). Past surgical history includes an inguinal hernia repair and bilateral hip replacements. Physical examination was otherwise unremarkable.

The patient underwent colonoscopy for work-up of guaiac-positive stool, which revealed three polyps. A 1.5-cm pedunculated rectal polyp and 1-cm pedunculated sigmoid polyp were removed with a snare. A 1.5-cm sessile polyp at the ileocecal valve was identified. Polypectomy was not believed to be safe, and a biopsy was taken. Pathologic examination of the three specimens revealed benign adenomatous tissue.

The patient subsequently underwent a laparoscopic-assisted right hemicolectomy. The procedure was performed using four trocars for dissecting and retracting. The right colon, including the hepatic flexure, were mobilized. A 7-cm transverse incision was made in the right mid abdomen. The right colon and terminal ileum were brought through this muscle-splitting incision. A limited right colon resection was

performed, and the specimen was sent for a frozen section. The lesion was a flat, 1.5-cm tubular adenoma. A functional end-to-end stapled anastomosis was performed. The procedure took approximately 2.5 hours, and estimated blood loss was 100 ml.

The patient's immediate postoperative course was uneventful. He was started on a liquid diet on postoperative day 3. This was advanced to a general diet the following day, and he was discharged to home with a prescription for pain medication on the same day.

On postoperative day 10, the patient experienced severe abdominal pain, which woke him from sleep. He was tolerating his diet and had no other gastrointestinal complaints. On physical examination, his abdomen showed no signs of distention or tenderness. His Tylenol® (McNeil Consumer Products, Fort Washington, PA) with codeine prescription was renewed, and he was sent home.

The patient was seen in the emergency room on postoperative day 17 with persistent abdominal pain that increased in severity with meals. He was having normal bowel movements and denied nausea or vomiting. On physical examination, he had no fever and abdominal examination was unremarkable. Laboratory studies were remarkable only for mild elevation of transaminases and specifically revealed a normal white blood cell count and amylase. At this time, it was believed that he may have gastritis or peptic ulcer disease. He was then started on an H2 blocker and sent home.

He continued to have pain, which was increasing in duration and severity. On postoperative day 21, the patient underwent a computed tomography (CT) scan of the abdomen. This revealed a thrombus within the superior mesenteric (Fig. 1) and portal veins (Fig. 2). At this point he was admitted to the hospital.

The patient was initially started on a heparin drip. A hematology consultation was obtained, and hypercoagulable states such as protein C, protein S, and antithrombin III deficiencies were excluded. Besides recent surgery, he had no other predisposing conditions to have caused mesenteric and portal vein thrombosis.

During his hospital stay, he continued to have pain without any signs of intestinal necrosis. Because the pain was not improving, the heparin drip was discontinued and thrombolytic therapy was started on postoperative day 22. Streptokinase therapy was initiated at 250,000 units for 30 minutes followed by 100,000 units per hour. Over the next four days, the patient improved significantly. He was then placed on a heparin drip, which was converted to Coumadin® (DuPont Pharma, Wilmington, DE).

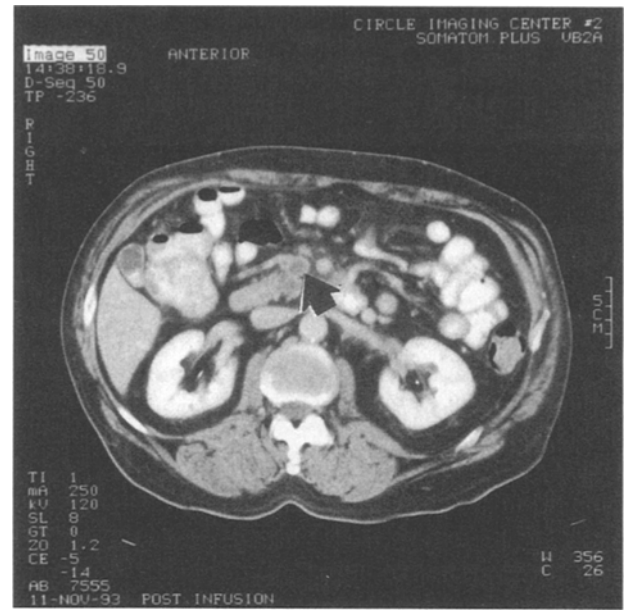

Figure 1. Thrombus in superior mesenteric vein (arrow).

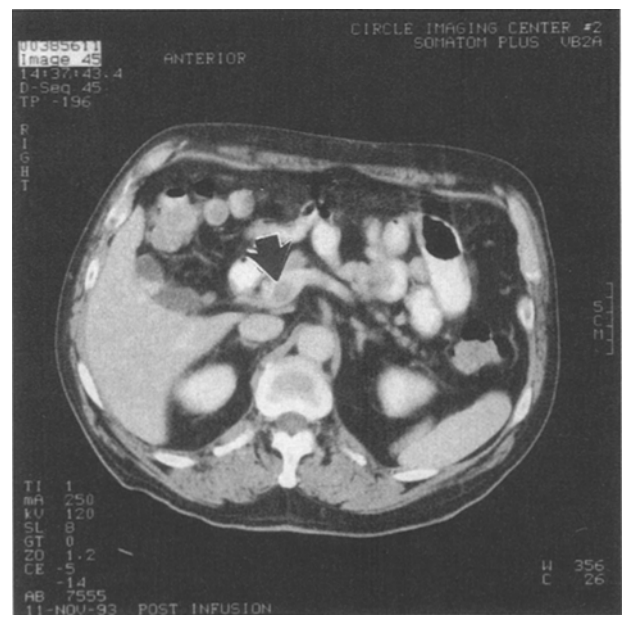

Figure 2. Thrombus in portal vein (arrow).

The patient resumed his diet and was pain-free after three days of thrombolytic therapy. A computed tomography scan was performed on postoperative day 32, which showed near resolution of the thrombus within the superior mesenteric vein (Fig. 3) and no evidence of thrombus in the portal vein (Fig. 4). He was discharged on the following day still receiving Coumadin®. There have been no further symptoms reported.

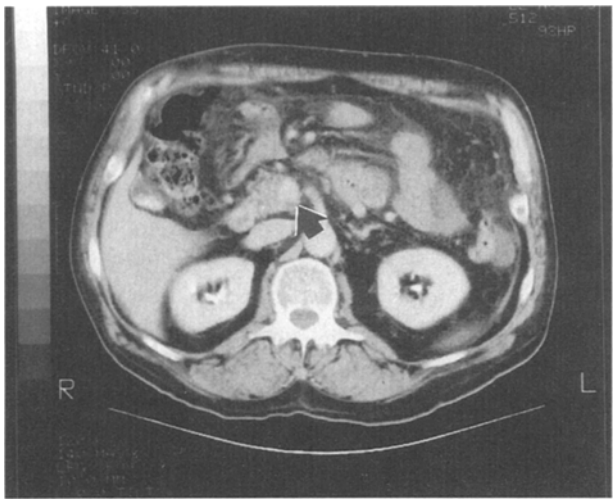

Figure 3. Near complete resolution of thrombus in superior mesenteric vein (arrow).

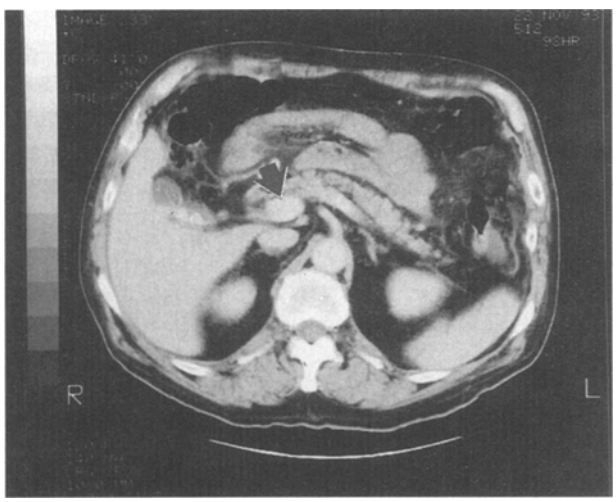

Figure 4. Complete resolution of thrombus in portal vein (arrow).

DISCUSSION

Incidence of mesenteric vein thrombosis (MVT) as a cause of mesenteric ischemia rapidly changed with improved understanding of nonocclusive mesenteric ischemia.[3, 4] It is currently understood to be an uncommon form of intestinal ischemia, comprising approximately 5 to 15 percent of all acute ischemic mesenteric events.[5]

Cause of MVT is described as primary and secondary. In the largest, most recent study,[5] 21 percent had primary, idiopathic MVT, whereas 79 percent had secondary MVT. The most prevalent conditions include previous abdominal procedure, hypercoagulable states, previous MVT, smoking, and history of deep vein thrombosis. Other known conditions associated with MVT include portal hypertension, trauma, and inflammatory disease processes.[3]

Known complications of laparoscopic-assisted colectomy have been well described in numerous sources.[6-9] Intraoperatively, these include enterotomy, perforation of the urinary bladder, ureteral transection, splenic injury, bowel rotation, hemorrhage, atrial fibrillation, and cerebrovascular accidents. Postoperatively, prolonged ileus, atelectasis-pneumonia, urinary tract infection, wound infection, small-bowel obstruction, urinary retention, recurrent prolapse, subfascial abscess, wound infection, anastomotic leak, abdominal wall recurrence of adenocarcinoma, and stress gastritis with bleeding have all been reported. A case report of failure to recognize a synchronous cecal cancer in resection of a near obstructing sigmoid cancer has also been noted.[10] To our knowledge, the case presented in this article is the first case of MVT associated with laparoscopic-assisted colectomy.

Several established factors of laparoscopic surgery could have contributed to mesenteric and portal vein thrombosis in our case with laparoscopic-assisted colectomy. Decreased splanchnic flow with CO_2 pneumoperitoneum has been confirmed experimentally.[11, 12] Berggren *et al.*[12] showed a stepwise reduction in portal blood flow, with stepwise increase in intra-abdominal pressures of 10, 20, and 25 mmHg CO_2. This effect is thought to be the result of the combination of vascular bed vasoconstriction with increase in resistance to blood flow across the liver. Epstein *et al.*[13] revealed that hypercapnia induced by breathing CO_2 at concentrations less that 5 percent will increase portal venous pressure and can cause mesenteric vasoconstriction. In addition, Ishizaki and colleagues[11] postulated that CO_2 pneumoperitoneum produces mechanical compression of vessels, hypercapnia, and elevated vasopressin, which all contribute to decreasing splanchnic blood flow. Mechanical compression of the colon on exteriorizing the area for anastomosis could have contributed significantly to the cause of thrombosis. It was noted during surgery that the incision site through which the colon was exteriorized seemed to be compressing the exteriorized colon along with its mesentery (Fig. 5). Certainly a hypercoagulable state could have played a role. However, this was essentially ruled out by our hematology consult. Levels of protein C, protein S, and antithrombin III were shown to be within normal limits.

The patient certainly displayed characteristics of a subacute superior mesenteric vein thrombosis.[5,14]

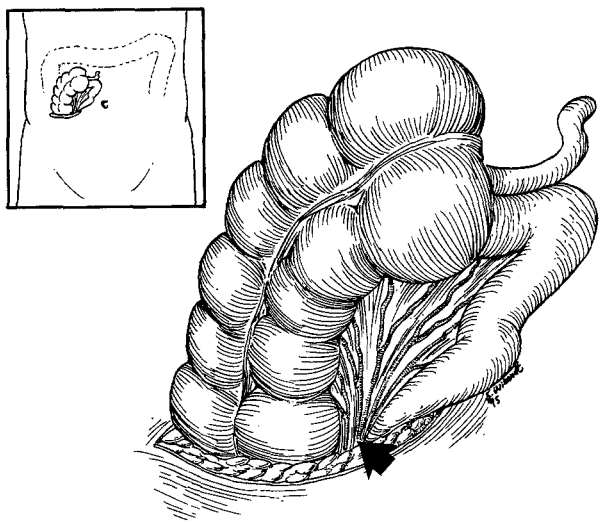

Figure 5. Illustration of mechanical compression of abdominal wall on exteriorized colon with its mesentery.

This term has been used to describe patients who have abdominal pain without infarction of the intestine for several weeks to months. Pathophysiology of this entity has been explained by extension of thrombosis at a rate fast enough to cause pain but slow enough to allow collaterals to form or acute thrombosis of only enough venous drainage to produce reversible ischemic injury.

Typically, pain is the only symptom of subacute MVT. Some patients also complain of nausea and diarrhea. Physical examination is classic in the sense that the patient's pain is out of proportion to physical findings. Laboratory tests are normal. Although pain is frequently reported to be nonspecific, pain has been reported to be related to meals in a few patients. Other reported symptoms of MVT include anorexia, diarrhea, constipation, nausea and vomiting, and gastrointestinal bleeding. Notable signs of MVT include abdominal tenderness, abdominal distention, decreased bowel sounds, guaiac-positive stools, guarding or rebound tenderness, fever, hypotension, and tachycardia. Significant laboratory abnormalities include leukocytosis, elevated lactate, amylase, and creatinine kinase.[5]

Clinical course of subacute superior mesenteric vein thrombosis has been known to vary. Some patients have developed intestinal infarction. In others, symptoms subside. In the population of patients in which symptoms subside, the patient may develop problems associated with asymptomatic chronic MVT.

Chronic MVT patients may stay asymptomatic or develop bleeding from esophageal or intestinal varices.[15-17] These complications are the result of thrombosis of the portal or splenic veins that always occurs. If only the superior mesenteric veins are involved, there may be abnormal findings, such as bowel wall thickening and retroperitoneal collateral vein formation; however, when these findings are present, they are caused by portal hypertension. Laboratory values may be altered, showing pancytopenia or thrombocytopenia because of secondary hypersplenism in portal or splenic vein involvement.

Variability of clinical course and absence of specific symptoms, signs, or laboratory studies make diagnosis of MVT challenging. In the past, laparotomy was used to make the correct diagnosis in 90 to 95 percent of patients. Currently, CT provides the most sensitive method for diagnosis of MVT. Other methods of demonstrating thrombi in the superior and portal veins include magnetic resonance imaging, angiography, and ultrasonography.

CT will reveal the thrombus, venous collateral circulation, and abnormal segments of intestine.[3] Specific findings include thickening and enhancement of the bowel wall, enlargement of the superior mesenteric vein, the thrombus seen as a central lucency in the lumen of the vein, sharply defined vein wall with a rim of increased density, and dilated collateral vessels in a thickened mesentery.

In our patient, the thrombus was identified on CT in the superior mesenteric and portal veins. Selective mesenteric arteriography was not used because we believe optimum treatment for our patient would not include surgery, and the potential advantage of improved delineation of thrombosed veins and access for administration of intra-arterial vasodilators would not be exercised.

Therapeutic options of MVT is directed at presentation of the patient. In the past, standard treatment of a diagnosed case of MVT required laparotomy. As methods of diagnosis have developed to the level at which patients can be identified before infarction of bowel occurs, nonsurgical treatment methods have become an option. In the group of patients in whom MVT is identified by ultrasound, CT, magnetic resonance imaging, or angiography but no physical findings of intestinal infarction are present, a course of anticoagulant or thrombolytic therapy should be tried. Heparin and streptokinase have been shown to be successful for this purpose.[18-20] Patients who develop signs of intestinal infarction require immediate laparotomy.

Patients who do not fall in the category described above should have prompt laparotomy with resection and anticoagulation followed by a second-look lapa-

rotomy and anticoagulation.[5] Options include mesenteric venous thrombectomy[19] or intra-arterial papaverine infusions through the superior mesenteric artery with an angiographic catheter;[21] however, these have only been used in a limited number of patients. Management algorithms have been created to follow a logical approach with available information.[3]

In our patient, there were no signs of peritonitis or intestinal infarction allowing for a trial of anticoagulation. Our hematologists advised attempting first treatment with heparin alone because this particular clot was already established and collateral circulation was already beginning to form. Anticoagulants alone were attempted to prevent further extension of the thrombus. However, the patient's pain did not improve with this alone and thrombolytic therapy was shown to be necessary to alleviate symptoms. In retrospect, a trial of heparin and streptokinase would probably have decreased the patient's symptoms faster and allowed a faster conversion to oral anticoagulant.

CONCLUSIONS

As data continue to grow on laparoscopic-assisted colectomy, it is inevitable that possible complications associated with this surgery will be better understood. Patients who do develop acute pain after laparoscopic-assisted colectomy should be evaluated with the possibility in mind that mesenteric vein thrombosis has occurred. In addition, careful patient selection for this procedure should be undertaken. Intraoperatively, the surgeon must monitor the incision for extracorporeal resection and anastomosis to avoid the potential complication of vascular trauma leading to mesenteric vein thrombosis.

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