

CASE REPORT

복강경하 맹장절제술 후 재발한 복통

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Recurrent Abdominal Pain after Laparoscopic Appendectomy

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Laparoscopic surgical approaches, compared with open surgical approaches, provide comparable clinical outcomes, but lower complications. Unfortunately, a rare complication—portomesenteric vein thrombosis—had been reported after laparoscopic surgery. A 42-year-old woman was referred our hospital for recurrent abdominal pain after laparoscopic appendectomy from acute appendicitis. It was determined that abdominal pain was due to postoperative superior mesenteric vein thrombus. A six-month anti-coagulation therapy is an excellent treatment for superior mesenteric vein thrombus. Therefore, physicians should be aware of portomesenteric vein thrombosis in patients with newly developed abdominal pain after successful laparoscopic surgical management. (Korean J Gastroenterol 2017;69:187-190)

Key Words: Mesenteric veins; Thrombus; Appendectomy; Laparoscopy; Abdominal pain

INTRODUCTION

Laparoscopic surgical approaches, when compared with open surgical approaches, provide similar clinical outcomes, but lower complications, less postoperative pains, including shorter hospital stays.¹ However, laparoscopic surgery has been suggested to cause portomesenteric vein thrombosis (PVT) attributed to decreased portomesenteric blood flow due to increased abdominal pressure and hypercapnia.² PVT after laparoscopic appendectomy is an uncommon complication. Its clinical signs vary, and include abdominal pain with diverse distribution and severity, nausea, vomiting, diarrhea, and fever. Therefore, diagnosis of PVT is often delayed.³ We report a patient with recurrent abdominal pain after laparoscopic appendectomy due to a new onset of superior mesen-

teric vein (SMV) thrombus, who was successfully treated with anticoagulation therapy.

CASE REPORT

A 42-year-old woman (height, 167 cm; weight, 91 kg; body mass index, 32.6) was transferred from a local hospital to evaluate sustained, vague abdominal pain on the periumbilical area. About two weeks prior to her visit, she had an uneventful laparoscopic appendectomy due to acute appendicitis at the same hospital. Except for her appendix, her initial diagnostic abdominal computed tomography (CT) scan showed no abnormal findings of other organs, including mesenteric vessels (Fig. 1). On the fifth postoperative day, she again experienced a vague postprandial abdominal pain.

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Fig. 1. Initial diagnostic abdominal CT scan. No abnormal findings of superior mesenteric vein. CT, computed tomography.



Fig. 2. Follow-up abdominal CT scan on the fourteenth post-operative day. Nearly total occlusion of superior mesenteric vein by thrombus. CT, computed tomography.

However, the pain was initially diagnosed as benign post-operative abdominal discomfort. On the fourteenth post-operative day, she had severe sustained abdominal pain on the periumbilical area without fever. Therefore, she was transferred to the emergency department of a tertiary hospital.

On initial physical examination, she had a blood pressure of 140/90 mmHg, pulse rate of 60 per minute, respiratory rate of 20 per minute, and body temperature of 37.2 °C. The abdomen was soft and flat. However, mild tenderness in the left upper quadrant area was identified. Laboratory test showed a white blood cell count of $3.77 \times 10^3/\text{mm}^3$ (polymorphonuclear neutrophils, 46.2%; lymphocytes, 38.2%; eosinophils, 6.0%), platelet count of 246,000/uL, C-reactive protein level of 1.89 mg/dL, erythrocyte sedimentation rate of 49 mm/hr, and prothrombin time of 12.8 seconds (international normalized ratio 0.97). Renal function and electrolytes tests were normal. The result of abdominal CT scan showed nearly total occlusion of SMV by thrombus without any malignant findings in the abdominal organs (Fig. 2).

To exclude general risk factors that may cause thrombosis, additional laboratory and malignancy screening tests were performed. Pregnancy test was negative. She had no specific past medical history and family history of coagulation disorders. Chest CT scan did not show any abnormal findings

in the lung parenchyma and mediastinum. The fibrinogen degradation product level was 5.68 ug/mL (normal <5), and D-dimer level was 1.80 mg/L (normal <0.5). Antithrombin III level was 86% (normal 80-120%), protein C activity was 99% (normal 70-130), and protein S activity was 68% (normal 58.7-119.2). The lupus anticoagulant screening test was negative, and the levels of anti-cardiolipin antibodies were less than the normal range (IgG 5.0 GPL U/mL, normal <23.0; IgM 6.0 MPL/mL, normal <11.0).

Consequently, she was diagnosed with acute superior mesenteric thrombosis caused by laparoscopic appendectomy. On the first day of admission, empirical systemic antibiotic agents (ciprofloxacin and metronidazole) and intravenous heparin, which was changed to warfarin, were administered. There was no evidence of infection, and the empirical antibiotics soon stopped. Abdominal pain subsided after 10 days of anticoagulation therapy. The patient was discharged after showing 21 days without complications. On the 3rd month of anticoagulation therapy, a follow-up abdominal CT scan was performed to evaluate the efficacy of treatment, which showed no remained thrombus in the SMV (Fig. 3). The oral anticoagulation agent had been administered for 6 months, and there was no bleeding during the use of oral anticoagulation.



Fig. 3. Follow-up abdominal CT scan on the third month of anti-coagulation therapy. No remained thrombus in the superior mesenteric vein. CT, computed tomography.

DISCUSSION

PVT is a rare, potentially life threatening condition, which accounts for 5% to 15% of all mesenteric ischemic events.³ The cause of PVT is a combination of both local and general risk factors. About 30% of PVT could be identified with a local risk factor, and 70% of them are identified with having general risk factors. General risk factors include inherited thrombophilias (e.g. antithrombin III deficiency, protein C and S deficiencies, factor V Leiden deficiency, etc.) and acquired prothrombotic conditions (e.g. sepsis, pregnancy, myeloproliferative disorders, etc.).⁴ Malignancy and cirrhosis are the main local risk factors for PVT. In the rest of the PVT patients, the most common local risk factor is inflammation of the abdomen.⁵

The suggested mechanisms for PVT development include endothelial injuries to the portomesenteric venous system (e.g. splenectomy, hepatectomy), endothelial inflammations (e.g. pancreatitis, appendicitis, diverticulitis, inflammatory bowel disease), and decreased velocity of portomesenteric blood flow made by liver cirrhosis or laparoscopic procedure, creating pneumoperitoneum-increasing intra-abdominal pressure. Jakimowicz et al. reported that the portal blood flow, as estimated by intraoperative duplex doppler ultra-

sound, slowed down by 53% during laparoscopic cholecystectomy with insufflations of the abdomen to 14 mmHg.²

Laparoscopic approaches could have locoregional factors contribute to the development of PVT. Insufflation of the abdomen and an increase in the intra-abdominal pressure may compress the portal or mesenteric vessels, leading to a decrease in portomesenteric blood flow, which varies from 35% to 84%.⁶ Moreover, there was a proportional relationship between insufflation pressures and venous stasis.³ In addition, carbon dioxide, which is used during laparoscopic surgery for the maintenance of intra-abdominal pressure, has been suggested to cause a greater decrease in venous flow than other gases.⁷ Insufflated carbon dioxide can diffuse into blood circulation, developing hypercapnia that could be implicated in splanchnic vasoconstriction and decreased portomesenteric venous flow.

PVT after laparoscopic surgery has rarely been; it is potentially a lethal condition. The laparoscopic procedures were diverse, including cholecystectomy, colectomy, Roux-en-Y gastric bypass, Nissen fundoplication, and appendectomy. The most common symptoms were abdominal pain with diverse severity and distribution, as well as nausea, vomiting, diarrhea, and fever. The mean duration of PVT development after surgery was 14 days (median, 12 days; range, 3-42 days), which is similar to our case. In aspect of physician, it is noteworthy that some patients with PVT dose not have any specific symptoms and there were no abnormal physical examination findings initially.³

Treatment of acute PVT after laparoscopic surgery has not been fully elucidated. In general, anticoagulation therapy in acute PVT is recommended for the prevention of bowel ischemia or infarction. According to experts, the optimal duration of treatment is at least 3 months,⁴ usually between 6 and 12 months.³ They reported that earlier the initiation of anti-coagulation therapy, greater recanalization of mesenteric venous thrombus.

Several reports have been published regarding SMV thrombosis or thrombophlebitis caused by acute appendicitis.^{8,9} However, it has previously been reported that laparoscopic appendectomy itself induced SMV thrombosis.¹⁰ In our case, there was no evidence of PVT from a pre-operative abdominal CT scan. On the fourteenth postoperative day, the patient experienced sustained left upper quadrant abdominal pain. The follow-up CT scan revealed a newly developed SMV

thrombus on the sixteenth post-operative day. Considering the lack of systemic or localized infection evidence, the main cause of SMV thrombus in this case was hypothesized to be an endothelial injury by the laparoscopic appendectomy, and not endothelial inflammation by infection. Therefore, the main treatment strategy was focused on long-term systemic anti-coagulation therapy, which started with heparin and then switched to oral vitamin K antagonist (warfarin).

Laparoscopic appendectomy requires relatively a short operation time compared with other laparoscopic surgeries. Consequently, the duration of decreased blood flow and reverse Trendelenburg position¹¹ would be less than other laparoscopic surgeries. Therefore, we suspected that a relatively short operation time would be one of the reasons why PVT may be uncommon after laparoscopic appendectomy. However, PVT development that is related to laparoscopic or robotic surgery, along with its clinical implication, should be investigated in greater detail in a future study.

Recently, it was suggested that one of the leading cause of PVT was central obesity.¹² In our case, she was overweight, with a body mass index of 32.6. Consequently, obesity was considered to be the main cause of PVT. However, the patient developed sudden PVT after surgery, which was cured with an anticoagulant without weight reduction. Therefore, laparoscopic appendectomy was the main reason for PVT in this case, and obesity was thought to have had a synergistic effect.

Unfortunately, this patient underwent laparoscopic appendectomy at a local medical center, and the intra-operative information, such as operation duration, intra-abdominal pressure, and insufflations, gas was not available. These factors were proved to make a significant change in intra-abdominal blood flow, which could have eventually been a cause of PVT.² Therefore, this restricted information may be a limitation of our case report.

Laparoscopic appendectomy can make various complications, such as superficial wound infections, postoperative adhesions, intramural abscesses, and stump appendicitis.¹³ These complications are thought to cause postoperative abdominal pain or discomfort, revealing various severity and distribution. The non-specific symptoms and low index of suspicion would delay the proper diagnosis of abdominal pain after laparoscopic appendectomy. Therefore, physicians need to suspect other rare causes of postoperative abdominal

pain, including PVT. In conclusion, laparoscopic appendectomy can lead to SMV thrombus. Thus, PVT development should be considered in any patients who recently underwent laparoscopic appendectomy with a newly developed abdominal discomfort or pain.

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