Case Report

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A Rare Postoperative Complication of Acute Appendicitis: Portal Vein Thrombosis Required Small Intestine Resection

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Abstract

This case report aims to present the diagnosis and treatment process of portal vein thrombosis, which occurred one week after laparoscopic appendectomy and required small bowel resection. A thirty-eight-year-old man was admitted with abdominal pain in the periumbilical and epigastric regions. He had a history of appendectomy and occlusive cerebrovascular disease. In the physical examination of the abdomen, tenderness was detected in the epigastric region on deep palpation. Leucocytosis, increased levels of alanine transaminase level, aspartate transaminase, gamma-glutamyl transferase, lactate dehydrogenase, c-reactive protein, and D-dimer were detected in laboratory analyses. A computed tomography scan revealed total thrombus in the portal vein, oedema in the segment of approximately 10 cm in the distal ileum, and free fluid in the pelvic region. Enoxaparin sodium was started. During follow-up, widespread defence and rebound in all quadrants of the abdomen occurred. 20 cm ileal resection with end ileostomy was performed. Enoxaparin sodium treatment was continued. On the 6th day of the service follow-up, the patient had left leg pain, and a subacute thrombus was detected in the common femoral, superficial femoral and deep femoral veins on doppler ultrasonography. Edoxaban tosylate 60 mg tablet every 24 hours started as an anti-coagulant treatment, and the patient was discharged without complications on the 18th day of hospitalisation.

Keywords: Appendectomies, enoxaparine, portal vein, thromboses.

Introduction

Acute appendicitis (AA) is the most common cause of acute abdomen in patients of all ages presenting to the emergency department (1). Portal vein thrombosis (PVT) is a rare but important cause of abdominal pain that should be quickly diagnosed and treated (2). It has been reported that the lifetime risk of developing PVT is 1% (3). Intra-abdominal infections, liver diseases, hypercoagulability, and abdominal surgery predispose to PVT (4). PVT usually occurs during clinical signs of acute appendicitis, rarely at the onset of inflammation or after appendectomy in perforated cases as a severe complication.

Early diagnosis of PVT is essential in preventing complications such as gastrointestinal bleeding and mesenteric ischemia. The specificity and sensitivity of ultrasonography (USG), usually chosen for diagnosis, are between 80-100% (5). Computed tomography (CT) shows intraluminal material and helps reveal the possible cause of thrombosis or complications such as perforation and bowel ischemia (6). Anticoagulant therapy, surgical thrombectomy, endovascular thrombectomy and thrombolytic therapy options are treatment methods in patients with PVT. In patients with PVT, it has been reported that the thrombus is recanalised in more than 80% of patients with anticoagulant therapy (7). However, imaging tools indicate diagnostic surgeries (laparoscopy/laparotomy) in cases with severe abdominal pain and suspected ischemia/necrosis.

This case report aims to present the diagnosis and treatment process of portal vein thrombosis, which occurred one week after laparoscopic appendectomy and required small bowel resection.

Case Report

A thirty-eight-year-old man was admitted to the tertiary health centre emergency department with abdominal pain lasting about two days in May 2022. From the beginning, the pain was in the periumbilical and epigastric region and did not show displacement. He underwent a laparoscopic appendectomy one week ago. In addition, he had a history of occlusive cerebrovascular disease about ten years ago, and he was followed up with warfarin about six years ago. The patient with a familial disorder has never had a genetic test before.

On physical examination on admission, his vital findings were as follows: blood pressure: 118/52 mm Hg, pulse rate: 107 beats per minute, oxygen saturation on room air: 97%, and body temperature: 37.3o Celsius. In the physical

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examination of the abdomen, tenderness was detected in the epigastric region on deep palpation. Leucocytosis (12.70x103/mm3), increased levels of alanine transaminase level (118 U/L), aspartate transaminase (83 U/L), gamma-glutamyl transferase (252 IU/L), lactate dehydrogenase (255 U/L), c-reactive protein (17 mg/L), and D-dimer (4548 ng/mL) were detected at laboratory analyses. Other laboratory parameters were unremarkable, including the lactate level on blood gas (1.6 mmol/L). On the USG evaluation of the abdomen, only minimal fluid was found in the pelvic cavity. CT with intravenous contrast showed increased heterogeneous linear density in the mesenteric fat planes with free fluid in the peri-intestinal area (postoperative changes). The patient was hospitalised in the service. Oral intake stopped, and intravenous fluid replacement started. Ceftriaxone 1 gr vial every 12 hours and metronidazole 500 mg/100 mL every 8 hours started for prophylaxis. On the first day of the follow-up, control USG was obtained, and free fluid with a depth of 50 mm was observed in the pelvic area. In control CT, there was a total thrombus in the portal vein (Figure 1), an oedematous bowel loop in the distal ileum (Figure 2), and free fluid in the pelvic region.

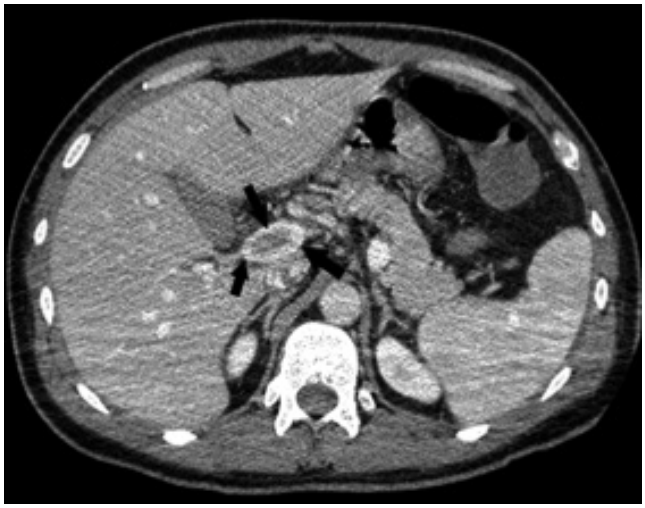

Figure 1. On CT scan, total thrombus in the portal vein is indicated with black arrows.

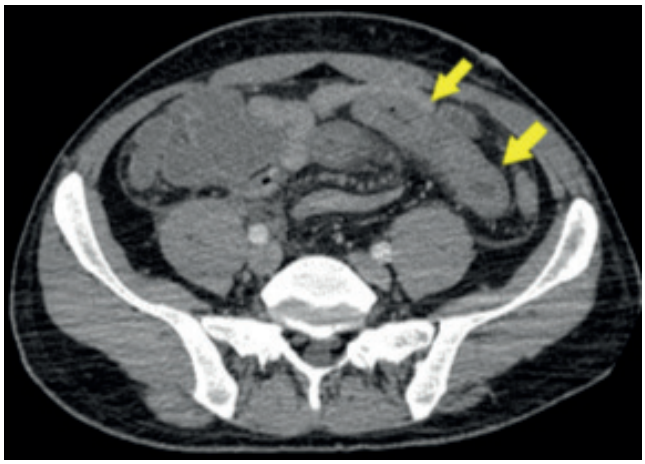

Figure 2. On the CT scan, the oedematous bowel loop in the distal ileum is shown with yellow arrows.

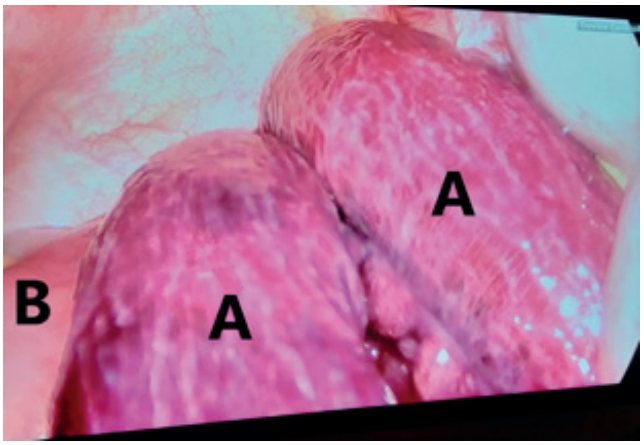

Figure 3. During laparoscopy, there was an oedematous intestinal loop with necrosis in the distal ileum (A: oedematous bowel loop with insufficient blood supply; B: oedematous bowel loop with regular blood supply).

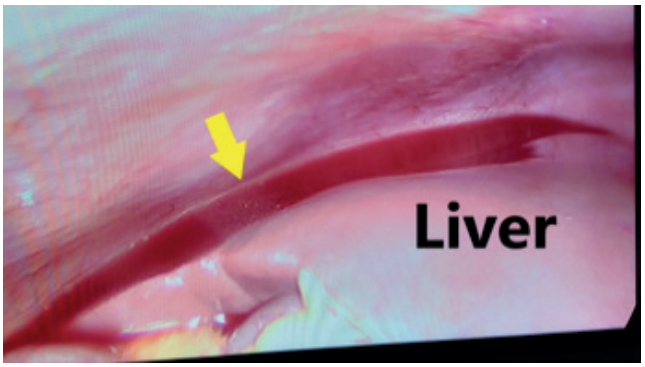

Figure 4. The yellow arrow shows haemorrhagic fluid on the perihepatic area during laparoscopy.

Enoxaparin sodium 6000 U subcutaneously every 12 hours was started. On the 3rd day of the patient's follow-up, there was widespread defence and rebound in all quadrants of the abdomen in the abdominal examination. Emergency laparoscopy was planned. On exploration, there was oedema in the intestinal loops and necrotic intestinal loop in the distal ileum with haemorrhagic fluid in all abdominal cavities (Figures 3 and 4), and laparotomy with midline incision was performed. There was a 5 cm necrotic area, 15 cm ischemic area, and a demarcation line. 20 cm resection and end ileostomy were performed (Figure 5). The patient was taken to the intensive care unit during the postoperative period, and enoxaparin sodium treatment was continued on the 1st postoperative day. Small bowel contents came from the ostomy on the 2nd postoperative day, and the patient was transferred to the service on the 3rd day. On the 6th day of the service follow-up, the patient had left leg pain, and a subacute thrombus was detected in the common femoral, superficial femoral and deep femoral veins on doppler USG. Edoxaban tosylate 60 mg tablet every 24 hours started as an anti-coagulant treatment. The patient was discharged without complications on the 18th day of hospitalisation. Genetic examination of the patient had homozygous polymorphism of both MTHFR A1298C and Plasminogen activator inhibitor (PAI) 4G/4G.

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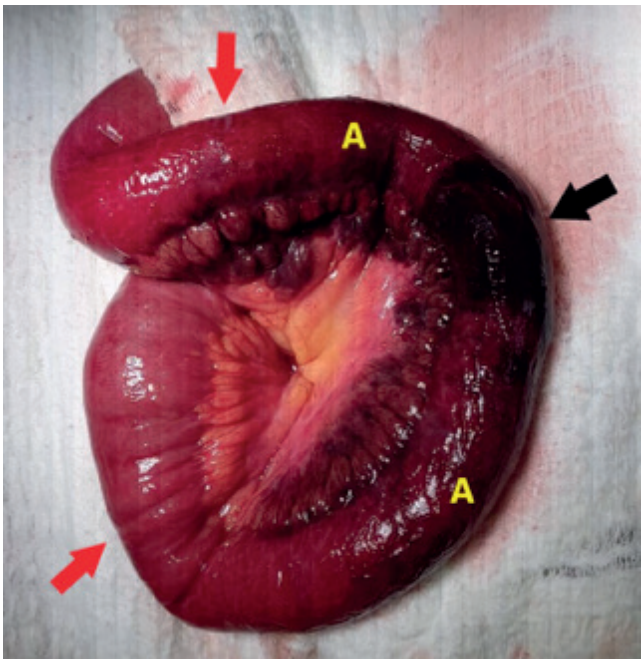

Figure 5. Intraoperative image of the resection material (A: pre-ischemic areas, red arrows show the demarcation lines, and black arrow shows necrotic intestinal loop).

Discussion

Appendectomy is the most commonly applied surgical procedure in emergency conditions at all ages. Among the causes of abdominal pain, portal vein thrombosis (PVT) is rare and often overlooked (8). PVT can be seen in cases of abdominal inflammation such as appendicitis, diverticulitis, inflammatory bowel diseases, pancreatitis, cholecystitis, hepatic abscess and cholangitis, liver cirrhosis, malignancies and hypercoagulability (9). Aetiology includes liver cirrhosis in 24-32% of patients, malignancies in 21-24%, myeloproliferative diseases and coagulation disorders in 10-12% (5). No etiologic cause was found in 8-15% of the patients (10).

The presenting symptoms in PVT depend on the degree of thrombus (partial/total). Although acute thrombus cases are clinically asymptomatic, symptoms such as abdominal pain, distention, diarrhoea, nausea, vomiting and bleeding occur as the thrombus duration increases. PVT does not have a specific laboratory finding and is usually expected without liver disease. However, leucocytosis, a decrease in prothrombin time and other coagulation parameters increase in D-dimer level may occur (11). In addition, etiological causes should be investigated in patients with a confirmed diagnosis of PVT. Prothrombotic events, polycythemia vera, factor V Leiden mutation, prothrombin gene mutation, antithrombin III, and protein C/S levels should be investigated. In patients with a disease prone to thrombosis, such severe PVT can be observed even after an appendectomy, which is frequently performed in general surgery practice, and the diagnosis of PVT should be kept in mind by both emergency physicians and general surgeons in this patient population. In our case, the initial symptom was abdominal pain in the periumbilical and epigastric regions that continued for about two days, and tenderness was detected in the epigastric region on deep palpation. Laboratory analyses revealed increased levels of alanine transaminase, aspartate transaminase, gamma-glutamyl transferase, lactate dehydrogenase, c-reactive protein, and D-dimer with leucocytosis. In addition, the patient had homozygous polymorphism of both MTHFR A1298C and Plasminogen activator inhibitor (PAI) 4G/4G at the genetic examination.

In cases where the diagnosis cannot be made, mesenteric ischemia, liver abscess, septic shock, and pulmonary embolism may develop. Therefore, early diagnosis of PVT is essential to reduce morbidity and mortality. Doppler USG, CT, and magnetic resonance (MR) angiography are diagnostic methods that help detect PVT early. USG is the first method used in diagnosis because it is cheaper and non-invasive. Endo-USG can give more detail about small PVT than routine USG. CT shows intraluminal material. It helps reveal the possible cause of thrombosis or complications such as perforation and bowel ischemia (6), while MR angiography can provide information about thrombus localisation and blood flow (2). In our case, no thrombus was seen at USG and CT on admission, but total portal vein thrombosis was seen at the control CT scan.

PVT treatment aims to prevent the progression of thrombosis, ensure the patency of the portal vein and prevent the development of serious complications. Traditional treatment options are antibiotic therapy, anticoagulant therapy, surgery and the endovascular thrombolytic method. Antibiotic therapy is essential for infection control. Antibiotic therapy, recommended for 4-6 weeks, should initially be an empirical treatment for the common microorganisms and then continue with the appropriate antibiotic according to the blood culture results. Today, anticoagulant therapy is the best way to prevent thrombus progression and provide portal vein recanalisation. Initiation of anticoagulant treatment as soon as the diagnosis is made is one of the most critical factors affecting the healing process. While the rate of recanalisation is 69% in those who start anticoagulant therapy in the first week, this rate drops to 25% with the initiation of treatment in the second week (11). However, it has been reported that 10% of the patients are resistant to anticoagulant therapy, and relapse occurs in 6-40% of those who terminate the treatment early (12). It is recommended to continue oral anticoagulant therapy for at least 3-6 months and evaluate thrombus resolution with intermittent MR angiography or CT (13). In the cases where there is non-responsiveness to medical treatment and diagnoses of persistent abdominal pain, peritonitis, intestinal ischemia, and necrosis, diagnostic laparotomy should be made immediately (14). After our patient was diagnosed with

PVT, subcutaneous anticoagulant therapy with enoxaparin sodium was started, but diagnostic laparoscopy was planned because an acute abdomen developed under anticoagulant treatment. Laparotomic bowel resection was performed due to the necrotic bowel loop observed in laparoscopy.

Conclusion

Portal vein thrombosis (PVT) is a rare but important cause of abdominal pain that should be quickly diagnosed and treated. PVT usually occurs during clinical signs of acute appendicitis, rarely at the onset of inflammation or after appendectomy in perforated cases as a severe complication. However, it can also be seen as a complication of appendectomy. Anticoagulant therapy should be started as soon as possible in patients without persistent abdominal pain, peritonitis, intestinal ischemia and necrosis. On the other hand, diagnostic surgeries (laparoscopy/laparotomy) should be considered in the first-line treatment in patients with the indicated symptoms and signs.

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