

Portal Vein Thrombosis Resulted from Delay Diagnosis of Ruptured Appendicitis

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ABSTRACT

Background: Portal vein thrombosis may result from different etiologies, and the clinical presentation is changeable. Septic thrombo-phlebitis of portal vein may be resulted from intra-abdominal inflammatory process such as appendicitis. Portal venous thrombosis accounts for a mortality rate of 5 - 20%. Early diagnosis, rapid usage of antibiotics and early anti-coagulation could decrease the complication.

Case presentation: We report a case of 63-year-old female who experienced fever and abdominal pain in right lower quadrant. She was referred to our emergency department with signs of septic shock and jaundice. Tenderness was complained in the peri-umbilical area and right iliac fossa. Icteric sclera was also noted in emergency department. Laboratory examination revealed a raised total leukocyte count (predominantly neutrophilic). Non-contrasted CT scan abdomen found swelling of appendix with peri-focal fatty stranding. Conservative treatment was arranged due to the suspicion of sepsis from cholangitis instead of appendicitis. Appendectomy was done 2 days after diagnosis and pathology proves the diagnosis of ruptured appendicitis. However, progressive elevation in bilirubin was noted for 2 days after operation. Abdomen CT with contrast was arranged and showed portal vein thrombosis. Intravenous anti-coagulation was prescribed and patient was discharged eventually.

Conclusion: Symptoms of portal vein thrombosis may be diverse and atypical, so the diagnosis is often delayed. Highly clinical suspicion should keep in mind for patient with risk. This report described a rare complication of portal vein thrombosis in acute appendicitis. Once identified, broad-spectrum of antibiotics and anti-coagulation should be prescribed.

Keywords

Appendicitis, Portal vein, Anti-coagulation.

Abbreviations

ALT: Alanine aminotransferase; AST: Aspartate aminotransferase; CT: Computed tomography; SMV: Superior mesenteric vein.

Background

Portal vein thrombosis may occur in 10-25% of patients with cirrhosis and other risk factors include oral contraceptive pills, pregnancy, chronic inflammatory diseases, inflammation or injury portal venous system (including surgery) and malignancies. Septic thrombophlebitis of portal vein may be resulted from

intra-abdominal inflammatory process such as appendicitis, diverticulitis or peritonitis, particularly when anaerobic organisms are involved. Portal venous thrombosis accounts for a mortality rate of 5-20%. Early diagnosis, use of antibiotics, and early anti-coagulation should be managed.

Case Presentation

A 63-year female presented with 5-day history of right-sided lower abdominal pain accompany with fever and tea-color urine for 2 days. She visited local medical department 2 times but condition deteriorate so she was referred to our emergency department (ED) for further management. She did not have any prior significant medical or surgical history. Vitals were pulse rate of 123 /minute,

blood pressure of 89/ 57 mmHg, respiratory rate of 18/minute, and oxygen saturation (SO₂) of 98% on room air. Physical examination showed tenderness in the peri-umbilical area and right lower abdominal area. Rebound tenderness was present in right iliac fossa. Bowel sounds were audible. Rest of the examination was unremarkable. Investigations revealed a low hemoglobin (Hb 10.5 g/dL) and platelet count (3320,000 /uL), but raised total leucocyte count of 17,010/mm³. Differential leucocyte count showed 94% neutrophils, 1.5% lymphocytes. Liver function test showed elevated in Bilirubin and AST (AST: 72 U/L; Direct Bilirubin: 4.6 mg/dl; Total Bilirubin: 5.6 mg/dl) and renal impairment were noted (BUN: 72.1 mg/dl; Creatinine: 3.0 mg/dl), and Lactate was 11.8 mg/dl. Hepatitis B and C serology was negative. Urine routine examination was also unremarkable. Abdominal ultrasound scan showed diffuse liver parenchymal disease.

Hepatic hypoechoic lesions, favor fat focal sparing and gallbladder sludge. CT scan abdomen without contrast was arranged due to poor renal function and showed swelling of appendix with perifocal fatty stranding, acute appendicitis first (Figures 1 and 2). General surgeon was consulted and appendectomy was not performed immediately because the suspension of septic shock due to cholangitis. The patient was then admitted to intensive care unit with conservative treatment. Operation was then performed 2 days after admission due to persisted peritoneal sign in RLQ area. Operative finding found some serous ascites in whole abdomen about 300ml with abscess formation between cecum, appendix, terminal ileum, sigmoid colon, and uterus area. Ruptured appendix was noted with tip rupture and pus coating. However, progressive elevation in bilirubin was noted for 2 days after operation (Direct Bilirubin: 6.6 mg/dl; Total Bilirubin: 8.1 mg/dl) with normal liver enzyme (AST 26 U/L, ALT29 U/L). Abdomen CT with and without contrast was arranged and showed portal vein thrombosis, including superior mesenteric vein (SMV), main portal vein and intrahepatic portal vein, appendicitis related considered. Anticoagulation with intravenous heparin to keep APTT above normal range 1.5 folds was commenced and eventually switched over to warfarin to be used for 6 months with weekly check on INR. Inherited pro-thrombotic conditions was surveyed with no significant findings (protein S, protein C). The patient made a remarkable recovery and was discharged on oral warfarin.

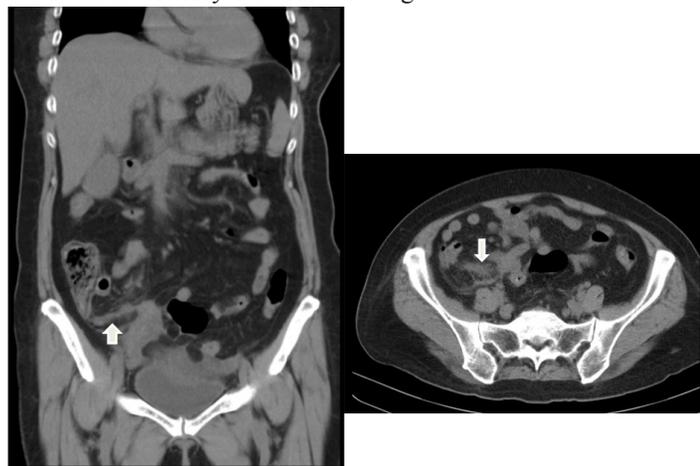


Figure 1: CT scan showed swelling of appendix with perifocal fatty stranding (White arrow).

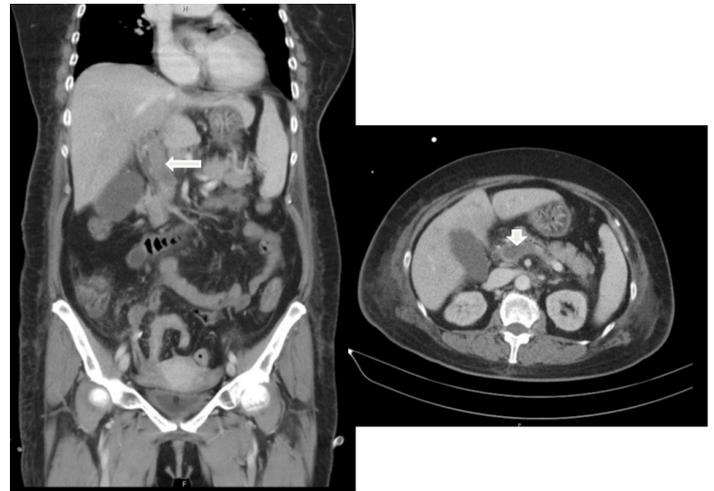


Figure 2: CT scan showed portal vein thrombosis, including SMV, intrahepatic portal vein and main portal vein (White arrow).

Discussion

Portal vein thrombosis may present as in a variety of clinical conditions and the symptoms are nonspecific, so the diagnosis of septic thrombophlebitis of the portal vein and SMV is generally delayed, and the mortality rate is very high. The major cause of non-cirrhotic is portal hypertension. Causes can be one of the followings below: 1. reduced flow or portal hypertension (cirrhosis and hepatobiliary malignancies); 2. Hyper-coagulable state (inherited pro-thrombotic conditions, myeloproliferative disorders); 3. endothelial disturbance (local inflammation and infection).

Initial diagnostic tests portal vein thrombosis can be detected by color doppler ultrasonography and definite diagnosis can be made by contrast-enhanced CT. Our patient received non-contrast CT of abdomen due to impairment of kidney function, so portal vein thrombosis was not detected initially. However, CT with contrast medium post operation was arranged due to persist elevation in bilirubin, with normal GOT, GPT. Liver function test abnormalities are usually present but frank jaundice is uncommon. Whether CT with contrast medium should be arranged for patient with impair kidney function is frequently addressed, the consensus nowadays is that it is safe for patients with creatinine is below 4.0 mg/dl after nephron-protective strategy.

Treatment for infected venous thrombosis involves fluid resuscitation, broad spectrum of intravenous antibiotic therapy, and anticoagulation. Antibiotic therapy should be prolonged because it is difficult to penetrate into infected thrombus; but optimum therapy duration is unclear. Although the usage of anticoagulation is controversial, most reported cases have used intravenous heparin followed by warfarin. Few reports were treated with low-molecular-weight heparin for anticoagulant therapy. Tissue plasminogen activator (TPA) is better avoided owing to increased risk of hemorrhage under the consideration of operation.

Conclusion

The clinical symptoms of portal vein thrombosis may be diverse and atypical, so the diagnosis is commonly delayed. Highly clinical suspicion should keep in mind for patient with risk. This report described a rare complication of portal vein thrombosis in acute appendicitis. Once identified, broad-spectrum of antibiotics and anti-coagulation should be prescribed.

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