

CASE REPORT

A Rare Complication of Acute Appendicitis: Superior Mesenteric Vein Thrombosis

Hendra Koncoro*, Rafael Eddy Setijoso*, Maisie ME Johan**

*Department of Internal Medicine, Sint Carolus Hospital, Jakarta

**Department of Radiology, Sint Carolus Hospital, Jakarta

Corresponding author:

Hendra Koncoro. Department of Internal Medicine, Sint Carolus Hospital. Jl. Salemba Raya No. 41 Jakarta Indonesia. Phone/facsimile: +62-21-3904441. E-mail: hendra_koncoro@yahoo.com.

ABSTRACT

Superior mesenteric vein (SMV) thrombosis caused by acute appendicitis is quite rare nowadays. These conditions occurs secondary to infection in the region drained by the portal venous system. In this case, we report a successfully treated case of SMV thrombosis and liver abscess associated with appendicitis with antibiotics and anticoagulant. Early diagnosis and prompt treatment are basic to a favorable clinical course.

Keywords: superior mesenteric vein thrombosis, pyogenic liver abscess, appendicitis

ABSTRAK

Trombosis vena mesenterika superior yang disebabkan oleh apendisitis akut sangatlah jarang dewasa ini. Kondisi ini terjadi karena infeksi di daerah yang didrainase oleh sistem vena portal. Dalam kasus ini, kami melaporkan suatu kasus trombosis vena mesenterika superior dan abses hati yang disebabkan oleh apendisitis dengan menggunakan antibiotika dan antikoagulan. Diagnosis dini dan terapi segera merupakan dasar terhadap perbaikan keadaan klinis.

Kata kunci: trombosis vena mesenterika superior, abses hati piogenik, apendisitis

INTRODUCTION

Acute appendicitis is the commonest surgical emergency in adult. In United States, there were 250,000 cases annually.¹ If untreated, acute appendicitis can have complications such as abscess formation, perforation and peritonitis.²

Traditional complications such as abscess or peritonitis are relatively easy to detect while rare complications such as mesenteric vein thrombosis and pyogenic liver abscess are more difficult to recognize. Superior mesenteric vein (SMV) thrombosis and pyogenic liver abscess caused by appendicitis is very rare now, owing to improved antibiotic therapy. However, the diagnosis of SMV thrombosis and

pyogenic liver abscess is often missed due to its non-specific clinical presentation, such as abdominal pain, fever, chills, fatigue, nausea, and vomiting. Caution need to be taken because mortality rate in SMV thrombosis and pyogenic liver abscess associated with intra-abdominal infection can reach 25-50%.^{2,3}

Rare complications in acute appendicitis were scarcely found. Therefore clinicians need to increase awareness to decrease mortality. The aim of presenting this case is to discuss the management of this rare complication of appendicitis.

CASE ILLUSTRATION

A 17-year-old man presented to the St. Carolus hospital emergency department with a 3-day history of fever, chills, right lower quadrant abdominal pain and jaundice. He was previously healthy until one week prior to presentation. It was initially a dull central lower abdominal pain that developed into a sharp intense pain associated with rigors. Nausea accompanied his complaints. The patient was previously went to surgery clinic six months ago with right lower quadrant abdominal pain and has been diagnosed with appendicitis and advised to be done appendectomy, however he refused. The patient is a student and denied any recent travel or sick contacts. He had neither alcohol intake nor smokes.

On examination, his blood pressure was 138/70 mmHg with a pulse rate of 100/min. He had a temperature of 38.1 °C, with an oxygen saturation of 99% on air. Eyes were icteric. The abdomen was soft but there was evidence of a hepatomegaly. Maximum tenderness was elicited in the right upper quadrant of the abdomen. The spleen were non-palpable. Bowel sounds were slightly increased and minimal lower quadrant tenderness was presented. Peripheral white blood cell count was 22,750/mm³ (segmen neutrophil 88.7%). Hematocrit was 37.6% and platelet count was 85,000/mm³. Other values were AST 38 IU/L, ALT 52 IU/L, gamma-GT 160 u/L, alkaline phosphatase 202 IU/L, total bilirubin 9.90 mg/dL, direct bilirubin 8.70 mg/dL, protein 5.20 g/dL, albumin 2.04 g/dL, INR was 1.12, and d-dimer 4650 ng/mL. Abdominal ultrasonography revealed hepatomegaly with multiple hypoechoicheterogen masses with largest mass was 45 x 35 x 46 mm, suggestive of abscess and minimal sludge in gall bladder. The pancreas and kidneys appeared normal (Figure 1).



Figure 1. Ultrasonography revealed multiple hypoechoicheterogen masses, suggestive of liver abscess

Streptococcus agalactiae was grown from blood culture. Since intra-abdominal septic foci were suspected, abdominopelvic computerized tomography (CT) scan was taken. A contrast-enhanced CT scan revealed a thrombus within portal vein that extended throughout superior mesenteric vein (Figure 2).

Abnormal multiple focal densities located in the liver were multiple, pyogenic liver abscess. Appendix was retrocaecal, swelling with periappendiceal haziness suggestive of appendicitis. A small amount of abnormal fluid was seen around the liver, right paracolic gutter and in the pelvis. On the basis of the microbiological data, treatment was done with ceftriaxone 3 g IV q 24 hours and metronidazole 500 mg IV q 8 hours. On the 7th hospital day, the patient's condition continued to improve and he was discharged 2 weeks later. After 14 days from treatment with ceftriaxone and metronidazole, his bilirubin level was lowered to total bilirubin 4.90 mg/dL, direct bilirubin 3.43 mg/dL. Further treatment was not needed. We decided to treat the appendicitis conservatively with antibiotics and SMV thrombosis with therapeutic dose of heparin after discussion with a haematologist. After two weeks of antibiotic therapy, the patient showed improvement in clinical and biochemical signs. This patient was planned to done interval appendectomy, however after careful treatment which showed improvement, he denied further appendectomy and discharged home.

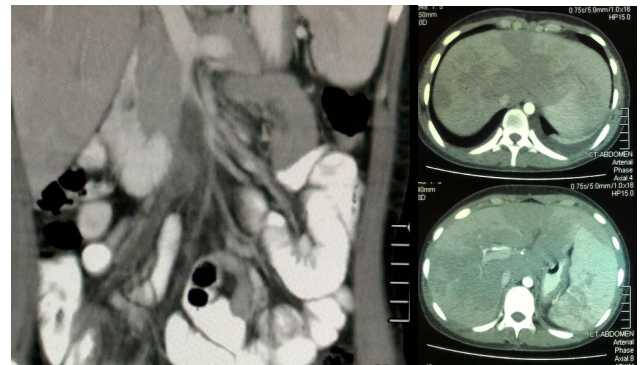


Figure 2. A contrast-enhanced CT scan confirmed the portal vein thrombosis and also demonstrated superior mesenteric vein thrombosis. Abnormal multiple focal densities located in the liver were multiple, pyogenic liver abscess

DISCUSSION

Appendicitis is one of the most common surgical diagnosis. Appendicitis usually complicated as perforation, peritonitis and abscess formation. Some reports stated that appendicitis can result in portal vein thrombosis.³ Inflamed thrombosis of the portal vein is called pylephlebitis. Pylephlebitis occurs as a result of an abdominal infection draining into the portal venous system.¹ The thrombus began from the small veins of the affected area to larger veins, leading to septic thrombophlebitis of the mesenteric vein and, eventually, of the portal vein. Swelling of the intestine and ischemia may occur due to SMV thrombosis which

lead to high morbidity and mortality.⁴ SMV thrombosis caused by appendicitis itself is not common, with incidence rate of 0.4% before 1950 and became extremely rare afterwards due to adequate antibiotic use. However, when occurred the mortality rate become 80% in the past and has decreased to 30-50% nowadays.⁵

Pathogenesis of SMV thrombosis are thrombophlebitis resulting in thrombosis and can be explained as follows: hypercoagulability state occurred due to abscesses in the mesoappendix; thrombi are formed locally. Bacteria infiltrate into the SMV, cause inflammation such as portal vein phlebitis, and induce thrombus formation. Bacteria spread into tissues around the veins, cause periphlebitis along the vessels, and as inflammation extends into the vascular lumens, coagulation cascade is promoted, leading to thrombus formation.⁶ Coagulation was facilitated via its surface and capsular components. The surface component accelerates fibrin cross-linking and the capsular polysaccharides initiate the clotting cascade by activating macrophages.¹

It is difficult to establish diagnosis only from clinical findings. The symptoms of SMV thrombosis are non-specific. High suspicion should be assumed in patients who present with abdominal pain, fever, and signs of sepsis, as well as leukocytosis and elevated liver enzymes. Other clinical features are as follows: fatigue, malaise, chills, nausea, vomiting, diarrhea, and weight loss. Hepatomegaly and jaundice are other clinical findings usually seen. SMV thrombosis can be diagnosed via abdominal ultrasonography showing a thrombus in the portal vein.⁴ An abdominal CT-scan is less operator-dependent and is more widely used because of its ability to detect other sources of infection in the abdomen. CT imaging can diagnose this complication at an early stage.⁷

In our case, appendicitis was the cause of SMV thrombosis. Diagnosis of SMV thrombosis are made by clinical signs and symptoms, laboratory examination, blood culture and imaging modalities. Clinical signs and symptoms are fever, right upper quadrant pain, jaundice and hepatomegaly. Pylephlebitis can be caused by ascending *Escherichia coli*, *Proteus mirabilis*, *Klebsiella pneumonia*, *Enterobacter* species, *Pseudomonas* species and gram-positive cocci (*Staphylococcus aureus*, *Streptococcus* species).⁸ In our case, *Streptococcus agalactiae* was grown from blood.

Modern imaging techniques provide supportive diagnostic evidence. In the setting of probable

intra-abdominal infection, CT scanning may be the most reasonable initial choice for imaging, given its proven ability to detect not only thrombi but infection foci as well. CT-scan also less operator dependent compared to ultrasonography. Thus, CT scan is the most reliable initial diagnostic choice. Early phase of this intraabdominal infection, hepatic infection are microscopic which coalesce to form macroabscess.⁸

On the basis of treatments, it appears that empirical antibiotic therapy for a patient with suspected SMV thrombosis should include broad coverage for gram-negative bacilli and agents active against anaerobes, and coverage for aerobic *Streptococcus* species. Patients with demonstrated macroscopic liver abscess complicating SMV thrombosis should probably receive at least 6 weeks of antibiotic therapy, with or without drainage.^{1,8} In our case, ceftriaxone (3 g/ day) and metronidazole (1.5 g/day) was performed for 2 weeks and resulted in complete resolution of hepatic lesions. Further appendectomy was planned, however due to condition improvement, he denied any surgical procedure.

The role of anticoagulation in the treatment of SMV thrombosis is controversial. Several reports recommended using anticoagulation therapy to reduce recurrence rate and mortality rate.⁹ No formal study of anticoagulation in SMV thrombosis has ever been done.¹⁰ Despite such evidence that heparin may not be critical for the survival of patients with SMV thrombosis, the possibility exists that anticoagulation might benefit some patients by decreasing the chance of septic embolization to the liver from infected portal thrombi and pulmonary emboli. In our patient, a previously healthy patient presented with nonspecific symptoms and fever. *Streptococcus agalactiae* was grown from blood, which clearly suggested an intra-abdominal origin. Infection was most likely caused by appendicitis, confirmed by abdominal CT, which seeding to the mesenteric vein and liver. Septic conditions were improved with early initiation of broad-spectrum antibiotics. Surgical intervention were denied due to improvement of condition.⁸

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