Treatment with Antibiotics Alone for Perforated Diverticulitis Complicated by Pylephlebitis and Mesenteric Venous Thrombosis

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Pylephlebitis is defined as septic thrombophlebitis of the portal vein and its branches. Although rare, portal mesenteric venous thrombosis and pylephlebitis can remain as potentially life-threatening sequelae of ruptured diverticulitis. Treatment recommendations from recent reports have included urgent exploratory laparotomy, prolonged intravenous antibiotic therapy, and anticoagulation for up to a year. In this paper, we describe a 67-year-old male who had fever, right upper abdominal pain and low back pain associated with shortness of breath and tea color urine at the time of admission. Laboratory test showed leukocytosis and jaundice. Computed tomography revealed perforated diverticulitis with pericolic abscess. Mesenteric venous thrombosis and pylephlebitis were present. Broad-spectrum intravenous antibiotics were administered promptly as the sole form of treatment for two weeks, after which time the patient was discharged without evidence of adverse sequelae. Follow-up computed tomography three months later revealed total resolution of pericolic abscess and regressive portal venous thrombosis. Our report emphasizes that for patients with pylephlebitis and intra-abdominal infections, prompt administration of antibiotics alone is an acceptable alternative to surgery and prolonged anticoagulation therapy.

Septic thrombophlebitis of the portal vein and its branches is termed pylephlebitis and is frequently associated with an intra-abdominal inflammatory process. In the preantibiotic era, pylephlebitis, or septic thrombophlebitis of the portal venous system, was a familiar, usually lethal consequence of intra-abdominal sepsis. It is encountered today as a rare complication of diverticulitis, appendicitis, malignancy, inflammatory bowel disease, and vasculitis. Pylephlebitis is often not suspected clinically and may be an incidental finding on abdominal CT. Mortality is still substantial, however, at up to 32% [1, 2].

Herein we report an unusual case with jaundice, intrahepatic portal venous thrombosis, mesenteric venous thrombosis and pylephlebitis due to perforated diverticulitis. After administering antibiotics as the sole form of treatment for two weeks, the patient was discharged without evidence of adverse sequelae. Prompt administration of antibiotics alone is an acceptable alternative to surgery and long-term anticoagulation therapy for patients with this rare presentation.

CASE REPORT

A 67-year-old male with a history of chronic low back pain and lumbar spondylolisthesis was treated at the outpatient department. The patient was otherwise healthy until he was admitted with fever, right upper abdominal pain and lower back pain. At the time of admission, shortness of breath and tea color urine were noted. Physical examination dem-
Treatment with antibiotics alone for pylephlebitis

mstrated right upper quadrant tenderness without Murphy sign or rebounding pain. Mild tenderness at the left lower abdomen was also noted. Laboratory test showed white blood cell count of 17.3 x10^9/L (neutrophils 87%), aspartate amino transferase (AST) 36 U/L, amino alanine transferase (ALT) 20 U/L, alkaline phosphatase 201 U/L, gamma glutamyltransferase 66 U/L and total bilirubin 7.6 mg/dL (direct bilirubin 6 mg/dL). C-reactive protein concentration was 21.2 mg/dL.

Contrast-enhanced computed tomography (CT) scan of the abdomen showed several diverticula in the transverse, descending and sigmoid colon. Wall thickening and pericolic dirty fat stranding at distal descending colon and sigmoid colon were noted, associated with a pericolic abscess about 5cm in size (Fig. 1a). The inferior mesenteric vein was poorly enhanced by the contrast medium. Gas density was present in the inferior mesenteric vein (Fig. 1b). Obliteration of part of the intrahepatic portal veins, mainly in the right anterior and posterior segment, was associated with geographic and patchy inhomoge-

Figure 1. Pylephlebitis and mesenteric venous thrombosis complicating ruptured diverticulitis in a 67-year-old male. Contrast-enhanced CT scan of the abdomen showed a, wall thickening at distal descending colon with diverticula (arrowhead), associated with pericolic dirty fat stranding and pericolic abscess formation (arrows). b, Coronal CT images showed the inferior mesenteric vein was poorly enhanced (arrows) and gas density was present in the inferior mesenteric vein (arrowhead). Thickened colic wall (star) and pericolic abscess (curved arrow) were demonstrated. c, Obliterated right anterior and posterior branch of portal vein (arrows), associated with geographic and patchy inhomogeneous enhancement of the liver were noted, corresponding to infective suppurative thrombosis of the portal vein. Increased hepatic arterial blood flow to compensate the diminished portal flow was the cause of inhomogeneous hepatic enhancement.
Treatment with antibiotics alone for pylephlebitis

Homogeneous enhancement of the liver (Fig. 1c). Perfusion alterations were produced by increases in intrahepatic arterial flow to compensate for the diminished portal venous flow. There was no CT evidence of hepatic abscess, choledocholithiasis or biliary tree dilatation. Image findings were compatible with perforated diverticulitis complicated by mesenteric venous thrombosis and infective suppurative thrombosis of the portal vein.

At the intensive care unit, empiric broad-spectrum intravenous antibiotics (Meropenem) were administered. Surgical intervention was reserved when medical conservative therapy failed. No anticoagulant therapy was given. The patient’s fever and abdominal pain gradually subsided within one week. His white blood count dropped from 17.3 $\times 10^9$/L to 8.9 $\times 10^9$/L four days after antibiotics treatment. Decreased total bilirubin 1.94 mg/dL (direct bilirubin 1.29 mg/dL) was noted after one week of antibiotic administration. Multiple repeat blood cultures were negative. After one week of treatment, the patient’s fever subsided and his clinical condition became stable. He was transferred from the intensive care unit to the ward. After two weeks of IV antibiotics, his consciousness became more clear with good oral intake. He was then discharged in stable condition. Discharge medications included broad-spectrum oral antibiotics (Augmentin). During a 2-month follow-up visit, colonoscopy revealed diverticulosis 25-50 cm from anal verge. Follow-up computed tomography three months later revealed total resolution of pericolic abscess and regressive portal venous thrombosis (Fig. 2). Laboratory test results showed a white blood cell count of 6.1 $\times 10^9$/L, AST 17 U/L, ALT 20 U/L, alkaline phosphatase 84 U/L, gamma glutamyltranspeptidase 24 U/L and total bilirubin 0.6 mg/dL (direct bilirubin 0.1 mg/dL). The patient was healthy without evidence of adverse sequelae.

DIsCUSSION

Pylephlebitis is defined as septic thrombophlebitis of the portal vein and its branches. It is a rare, life-threatening complication of intra-abdominal infections [3, 4]. Pylephlebitis may be associated with appendicitis and diverticulitis [5], but it has also been described in necrotising pancreatitis, inflammatory bowel disease, haemorrhoidal disease, foreign body perforation, acute cholecystitis, Behcet’s disease, and amoebic colitis [6].

The usual clinical findings of pylephlebitis include fever and right upper quadrant pain. Jaundice is rare, except in cases complicated by liver abscess [6]. In our case, the patient did not have liver abscess or biliary dilatation. Jaundice was still noted, however, which is unusual. There were two causes for the patient’s jaundice. One cause was ischemic change of the bile ducts adjacent to the thrombosed portal branches in the portal tracts. The other was the profound sepsis of this patient. However, jaundice subsided quickly after administration of antibiotics.

Figure 2. Total resolution of pericolic abscess and regressive portal venous thrombosis after three months follow-up. Contrast-enhanced CT of the abdomen showed a, pericolic dirty fat stranding without pericolic abscess formation (arrows). b. Obliterated right posterior branch of portal vein with regressive interval change (arrow).
Since thrombophlebitis of mesenteric veins tends to present with non-specific signs and symptoms (fever, diffuse abdominal pain, nausea), imaging findings are essential in its diagnosis. Doppler ultrasonography, contrast CT, and MRI are all considered sensitive imaging techniques in diagnosing pylephlebitis, although no comparative evidence exists. The presence of an intraluminal echogenic thrombus provides the best ultrasonographic evidence of portal vein thrombosis. Additional signs include dilation of vessels proximal to the occlusion, the presence of collateral vessels, and the absence of an identifiable portal vein [7]. Ultrasonographic examination using a duplex Doppler technique is helpful in confirming the diagnosis of portal vein thrombosis and cavernous transformation of the portal vein, a possible complication of chronic obstruction of the portal venous system leading to portal hypertension [7].

Advanced imaging techniques such as CT scan have increased the sensitivity for the detection of portomesenteric vein gas [1, 8], and are the most useful radiographic signs to diagnose this entity. CT abnormalities can be divided into three categories: 1) findings of a primary source of infection; 2) involvement of the mesenteric branches, intrahepatic branches, or the main portal vein; and 3) intrahepatic abnormalities [9]. CT scan detects an inflammatory reaction or an air-filled or fluid-filled abscess at the site of the primary inflammation. Primary sources of infection are usually located in the right lower quadrant (appendix, cecal diverticulitis) or left lower quadrant (sigmoid diverticulitis). Pyogenic extension into the draining mesenteric veins is indicated on CT images by the presence of intravascular air or thrombi in the periphery or central portal system. These findings may be present throughout the portal system adjacent to the primary source of infection and centrally involving the superior or inferior mesenteric veins, main portal vein, or intrahepatic branches of the portal vein [9]. Intravascular air may be the initial finding, with thrombi developing a few days after the initial onset of the disease. The early hepatic abnormalities consist of unopacified intrahepatic branches of the portal vein and central or peripheral zones of low attenuation, secondary to decreased portal venous flow and increased compensated intrahepatic arterial flow. CT scan provides a useful tool to allow the clinical physician to understand the patient’s “whole picture” even though other clinical clues are not evident. This modality reveals the involvement of the mesenteric branches, intrahepatic branches, or the main portal vein. Intrahepatic abnormalities such as biliary obstruction and hepatic abscess can be identified. Since pylephlebitis is a serious and potentially lethal abdominal septic complication, early recognition with prompt treatment is mandatory.

On MRI images, portal vein thrombosis causes an area of abnormal signal within the lumen [10]. MR angiography has also been shown to be of value in the evaluation of flow and patency within the portal system as well as in diagnosing cavernous transformation of the portal vein [11].

In patients with bacterial infections, B. fragilis is the most frequently isolated organism. Gram-negative organisms (e.g., Escherichia coli, Klebsiella, Proteus or Shigella), anaerobes (e.g., Clostridium or Peptostreptococcus), and other streptococci have also been reported [6]. The treatment of pylephlebitis is based on the use of broad-spectrum antibacterials as soon as the infection is suspected. The initial empiric treatment antibacterial choice should include coverage for streptococci, anaerobes, and enteric gram-negative bacteria [6]. Empiric therapy should be tailored to local sensitivity and antimicrobial availability patterns and adjusted according to the culture results. In patients in whom the diagnosis is made early and the underlying infection does not require surgery, antibacterial therapy until complete resolution or cavernous transformation of the thrombus, as confirmed by serial CT scans, has been suggested [12, 13]; this usually occurs within 4–6 weeks. In our case, several blood cultures before and after antibiotic treatment were negative. This indicates that prompt empiric therapy with broad-spectrum antibacterials is mandatory in patients with pylephlebitis. In addition, no anticoagulant therapy was given in our case. The role of anticoagulation is controversial, because no randomized studies addressing this issue exist [6].

Prompt surgical intervention is required only if there is: 1) evidence of synchronous liver abscesses; 2) complication of diverticulitis (perforation, bleeding); 3) intestinal ischaemia; 4) mesenteric ischaemia (based on clinical findings and laboratory parameters); 5) when medical conservative therapy fails [3].

A high index of suspicion for pylephlebitis is required in patients with any septic abdominal event and right upper quadrant pain, even in the absence of clinical signs from the primary site of infection (i.e., perforated diverticulitis). Modern imaging techniques facilitate early diagnosis. To the best of
our knowledge, this is the first reported case of conservative management of perforated diverticulitis complicated by jaundice, pylephlebitis, intrahepatic portal venous thrombosis and mesenteric venous thrombosis. Based on our experience we propose that, for patients with this rare presentation, early administration of broad-spectrum antibacterials alone is an acceptable alternative to emergent exploratory laparotomy and long-term anticoagulation. Prompt administration of antibiotics in this situation should be considered life saving.

REFERENCES

單用抗生素治療破裂的大腸炎併發腸靜脈栓塞和門靜脈炎

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門靜脈炎是指門靜脈和其分支所發生的血栓性靜脈炎。雖然很少見，破裂的大腸炎可能會造成致命的腸靜脈血栓和門靜脈炎。目前治療的建議包括緊急的剖腹手術，長期的使用靜脈注射抗生素，以及使用抗凝血劑達一年的時間。在這篇病例報告裡，我們描述一位六十七歲的男性，他有發燒，右上腹痛及下背痛，在住院時同時有氣促及茶色小便的情形。實驗室檢查顯示有白血球上升及黃膽。電腦斷層攝影顯示有破裂的大腸炎以及大腸旁的膿瘍，同時有腸靜脈血栓和門靜脈炎。廣效性的靜脈抗生素馬上給予，治療了二週後，病人順利的出院，無後遺症產生。三個月後的電腦斷層攝影顯示大腸旁的膿瘍已經消失，門靜脈栓塞獲得改善。我們的報告強調對於有門靜脈炎及腹腔內感染的病人，僅即時的給予抗生素是手術和長期使用抗凝血劑之外的替代療法。