# **CASE REPORTS**

# Pylephlebitis caused by a liver abscess

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#### ABSTRACT

Pylephlebitis is a rare complication associated with an intra-abdominal septic process in the portal venous system. It is defined as thrombophlebitis of the portal vein and is often reported in association with appendicitis and diverticulitis. We present a 64-year-old female who presented with fever, chills, myalgia, and loss of appetite. A computerized tomography (CT) chest/abdomen/pelvis was performed and the patient was found to have a low-density lesion within the left lobe of the liver suspicious for a hepatic abscess and a suspected left segmental plyephlebitis. The diagnosis of pylephlebitis can be challenging as there is a broad differential diagnosis to consider. When considering pylephlebitis, empiric antibiotic coverage for poly-microbial infection targeting both gram-negative aerobes and anaerobes should be initiated. Antimicrobial therapy is modified according to blood culture results and treatment can be extended for 4 to 6 weeks. To our knowledge, there are only a few cases identifying liver abscesses as an etiology for pylephlebitis. This case was atypical compared to other cases in that the diagnosis of pylephlebitis was incidental.

Key Words: Pylephlebitis, Liver abscess, Anticoagulation, Broad-spectrum antibiotics

#### **1. INTRODUCTION**

Pylephlebitis is a rare complication associated with an intraabdominal septic process, specifically from a region drained by the portal venous system. It is defined as thrombophlebitis of the portal vein and is often reported in association with appendicitis and diverticulitis.<sup>[1,2]</sup> It is believed to occur as a result of the spread of the infection into the small vessels, further draining into the portal system.<sup>[3]</sup> Although the occurrence of this condition is now rare because of the advent of antibiotics, high clinical suspicion should still be emphasized in patients with an underlying abdominal source of sepsis presenting with fever and abdominal pain. In our case, we describe a case of pylephlebitis that was caused by a left sided liver abscess.

#### 2. CASE PRESENTATION

A 64-year-old female with type 2 diabetes mellitus presented to the hospital with fevers, chills, myalgia, and loss of ap-

petite. The patient had recently returned from a trip from Mexico. Upon arrival, the patient was found to have a lowgrade fever of 100.3 Fahrenheit (F), a pulse of 103 beats per minute (BPM), and a blood pressure of 119/70 millimeters mercury (mmHg). The physical exam was relatively within normal limits. Hematology and chemistry panel was performed and the only abnormalities included a leukocytosis of 11.8 kilo per microliter (K/UL) as well as an alkaline phosphatase elevation of 339 international unit per liter (IU/L). The patient was inevitably admitted to the hospital for observation for suspected viral illness and was provided supportive therapy. Overnight the patient continued to develop febrile episodes. Repeat blood cultures were drawn and broad-spectrum antibiotics (Vancomycin and Zosyn) were initiated. The Infectious Disease (ID)service was consulted for further recommendations and they recommended performing a CT chest/abdomen/pelvis. The CT scan showed low-density lesions within the left lobe of the liver suspicious

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for hepatic abscess, pneumobilia, and suspected infected thrombus (see Figure 1). Intravenous Metronidazole was added to treat the liver abscess. An amebiasis antibody was checked but was negative. The patient underwent CT guided aspiration for which 100 milliliters (ml) of fluid was aspirated

and was sent for cytology and culture. Magnetic Resonance Cholangiopancreatography (MRCP) was performed and an acute left portal vein thrombus was noted consistent with pylephlebitis. The patient was inevitably discharged home with long-term antibiotics as well as anticoagulation.



Figure 1. CT abd/pel. Red arrow: liver abscess, Green arrow: suspected infected thrombus

# 3. DISCUSSION

The diagnosis of pylephlebitis is unfortunately challenging but important to highlight due to the mortality associated with this disease. A single center retrospective study that reviewed outcomes of 95 patients over ten years reported an 11% mortality1. Patients with pylephlebitis have non-specific clinical manifestations such as abdominal pain, nausea, vomiting, and fever.<sup>[4]</sup> Imaging via CT scan (95%) and/or ultrasound (51%) helps confirm the diagnosis but has limitations in their specificity and sensitivity. Due to the rarity of this complication, physicians may easily attribute a patient's gastrointestinal symptoms to the more obvious underlying infectious process and miss the complication of thrombus formation. Common sites of the thrombus formation include the right portal vein (33%), main portal vein (32%) and the superior mesenteric vein (31%).<sup>[1]</sup> When considering pylephlebitis, empiric antibiotic coverage for polymicrobial

infection, targeting gram-negative aerobes and anaerobes should be initiated. Appropriate broad-spectrum antibiotics include Metronidazole and a third-generation Cephalosporin or Fluoroquinolones.<sup>[5]</sup> This therapy is modified according to blood culture results and treatment can be extended for 4 to 6 weeks. Intravenous antibiotics are given until a satisfactory clinical response is achieved.<sup>[6]</sup> When it comes to anticoagulation therapy, there is no consensus due to lack of prospective randomized trials in favor of or against their use. Anticoagulation aims to prevent thrombus extension and further complications. Multiple studies have demonstrated superior outcomes and decreased mortality in patients that were treated with anticoagulation and antibiotics versus patients that were treated with antibiotics alone.<sup>[7]</sup> Anticoagulation may not be necessary for patients that have normal clotting function or an isolated portal vein thrombus. It should be considered when there is involvement of the mesenteric veins

due to the risk of complicating bowel ischemia, or if the patient has a known hypercoagulable state.<sup>[7]</sup> Anticoagulant therapy can be used for 3 to 6 months if there is no underlying thrombotic condition.<sup>[8]</sup> Currently there are no clear recommendations for repeating radiographic testing to document resolution of thrombus. The most recent pylephlebitis case reported by Wong et al. reported colonic perforation complicated by the occurrence of multiple hepatic abscesses. Here they mention that liver abscesses can complicate up to 50% of the pylephlebitis cases and are associated with an increased mortality.<sup>[9]</sup> To our knowledge, there are only a few cases of liver abscesses as the primary etiology for pylephlebitis.<sup>[1,10–12]</sup> Contrary to our case, other case reports have reported pylephlebitis causing the formation of a liver abscess through hematogenous spread and in most of these cases, patients have a positive blood culture.<sup>[13]</sup> Rahmati et al. reported a case of an hepatic abscess secondary to Fusobacterium infection, causing pylephlebitis.<sup>[14]</sup> In our case, we believe the liver abscesses preceded the development of the pylephlebitis of the left portal vein due to negative blood

cultures. Previous cases have been reported on potential local seeding of infection such as in a case presented by Tandon et al. identified pylephlebitis complicating a liver biopsy.<sup>[15]</sup> In our case, drainage of the abscess could have created seeding of the portal system inevitably causing left portal vein pylephlebitis.

## 4. CONCLUSION

This case was atypical compared to other cases in that the diagnosis of pylephlebitis was encountered incidentally and challenging with absence of abnormalities in her liver enzymes, and negative blood and abscess fluid cultures. It is important to consider pylephlebitis in the setting of any intraabdominal process as it requires a different therapeutic management. Adequate treatment with anticoagulation and appropriate prolonged antibiotic therapy is important to prevent long term complications.

### **CONFLICTS OF INTEREST DISCLOSURE**

The author declares no conflict of interest

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