CASE REPORT DOI: https://doi.org/10.18502/fem.v9i1.18071 Liver abscess due to appendicitis: a case report

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Abstract: Pyogenic liver abscess (PLA) is a rare but serious condition that can be life-threatening. Recent developments in diagnostic techniques and treatment have resulted in earlier detection and intervention and hence reduction in mortality rates. Despite these improvements, PLA remains a clinical challenge due to its hidden symptoms and associated health risks. PLA may be caused by seeding from appendicitis.

We present a 52-year-old woman presented with PLA due to appendicitis. Upon physical examination, laboratory study and imaging, an acute appendicitis with pyogenic liver abscess were found. The patient underwent percutaneous drainage of the abscesses with a catheter and an interval appendectomy.

Keywords: Appendicitis; Pyogenic Liver Abscess; Percutaneous Drainage

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1. Introduction

Liver abscess can be classified into three main types: pyogenic, amoebic, and fungal (1). Although the most common type, pyogenic liver abscess (PLA) is an uncommon condition that can be caused by various factors, such as biliary issues, gastrointestinal perforation, seeding from other infections, or liver injury (2). PLA is more common in men and people over 65, especially those with a history of malignancy, alcoholism, diabetes, liver transplants, and autoimmune conditions like rheumatoid arthritis and systemic lupus erythematosus (2).

In a few cases, like the one we describe, pyogenic liver abscesses have been linked to seeding from appendicitis. A study with a population-based active-surveillance design has found that appendicitis only causes about 1% of pyogenic liver abscesses (2).

In this report we discuss a case of a 52-year-old woman who developed a PLA secondary to appendicitis. This case report aims to review this unusual condition and enhance the diagnostic criteria for pyogenic liver abscesses by considering appendicitis as a potential complication.

2. Case presentation

A 52-year-old woman presented to emergency department (ED) with a constant abdominal pain for 3 weeks. Her pain was in the right upper quadrant of abdomen, without any radiation or nausea and vomiting. She revealed that eating meals worsens her pain. She denied having fever and chills and she was a known case of hypothyroidism. She also complained of low appetite, but she didn't notice any changes in her bowel habits. In the ED, she was in pain and distress, mildly tachycardic with heart rate of 108 beats/min. She was normothermic and her blood pressure was 110/51 mmHg.



Figure 1 CT scan with IV contrast showing a hypodense lesion with gas focuses in favor of liver abscess (star)

She had mild epigastric and right upper quadrant abdominal tenderness without rebound tenderness or guarding, also her abdomen was mildly distended, but Murphy's sign was negative on physical examination. We also found diminished breath sounds in her right lower chest auscultation.

Laboratory results revealed platelet count of $695 \times 10^3 / \mu L$ (normal range: $150-450 \times 10^3 / \mu L$), without leukocytosis (WBC count: $8.18 \times 10^3 / \mu L$ with normal range of $4-10.5 \times 10^3 / \mu L$). C-reactive protein (81.9 mg/L), (normal range: 0-8 mg/L) and ESR (98 mm/h), (normal range: 0-20 mm/h) were elevated, and liver enzymes were in normal range. The ultrasonography (US) showed a 100 by 60 mm subcapsular collection with gas focuses on the right lobe of liver, which warranted further investigation by computer tomography (CT) imaging. The patient underwent contrast-enhanced computed tomography (CECT) (Figures 1 and 2). The CECT

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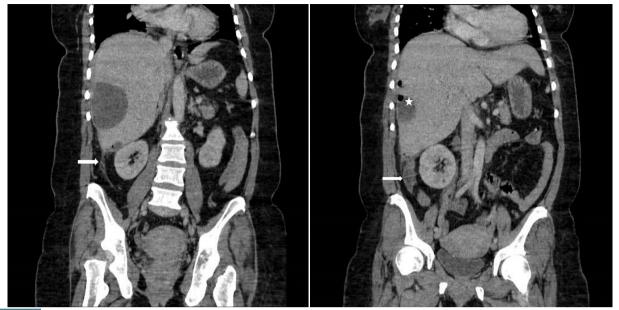


Figure 2 CT scan with IV contrast (coronal reconstruction slices) showing inflamed appendicitis in the inferior border of liver with fat stranding (arrows), as a cause of liver abscess which is a hypodense lesion with air focuses (star)

showed two hypodense lesions (measuring 26 by16 mm and 107 by100 mm) with air bubbles in favor of liver abscess. Interestingly, an inflamed appendicular loop measuring 8 mm lying on the inferior border of the liver with mesenteric fat standings was present. The diagnosis of liver abscess secondary to a long-lasting appendicitis was made. CECT also revealed a mild right-side pleural effusion.

The patient underwent percutaneous drainage of her liver abscess under ultrasonography guidance. She also received broad spectrum intravenous antibiotics (ceftriaxone and metronidazole). She underwent an interval appendectomy after 6 weeks of considering the successful abscess drainage and antibiotic therapy. The patient had an uneventful course.

3. Discussion

Appendicitis is a rare cause of PLA. It is believed that this happens through the transfer of infection via portal vein to the liver. While this is not common, it is important to be aware of the possibility of developing hepatic abscess as a complication of appendicitis.

PLAs are the most common visceral abscess and account for 48 % of the cases. The incidence of PLA is higher among men (3.3 per 100,000 versus 1.3 per 100,000) (2). There are several risk factors for developing PLA like diabetes mellitus, pancreatic or hepatobiliary disease, use of proton pump inhibitors, and liver transplant (2). The formation of PLAs can occur through three main mechanisms. One involves the spread of bacteria from an infection in the intra-abdominal area or from a source drained by the portal venous system, leading to the bacteria being transferred to the liver. Another mechanism is biliary tract diseases like strictures, malignancy, or gallstones, which can cause biliary obstruction

and allow infectious material to extend directly to the liver, resulting in abscess formation. Lastly, systemic bacteria can spread through the arterial bloodstream, often due to conditions like bacterial endocarditis or periodontal infections, which can also lead to the development of PLA (3). Bacterial infections are the most common cause of PLA, and nearly half of these are polymicrobial with the most common organisms identified were Streptococcus milleri group, Klebsiella species, and anaerobes (4).

The most common presenting symptoms in patients with PLA are right upper quadrant pain, fever, rigors, malaise, nausea, vomiting, loss of appetite, vague abdominal pain and jaundice (1). Hepatitis of any cause, right lower lobe pneumonia, cholangitis, cholecystitis, and noninfectious etiologies like tumor or collection of bile are among the most important differential diagnosis of PLA. Diagnostic laboratory studies include a complete blood count with differentials, liver enzymes, liver synthetic function tests (prealbumin and INR), alkaline phosphatase, CRP, ESR, and blood cultures (5). Diagnostic imaging modalities have a significant role in suspected cases of PLAs. CECT and US are primary diagnostic tools with sensitivities for diagnosis of PLA are 85% and 97%, respectively (6). On ultrasound PLAs are shown as hypoechoic to hyperechoic lesions, and on CECT scan the most typical finding is a round with central hypoattenuation lesion (7).

Similar to our case, liver abscess due to appendicitis happens due to hematogenous spread of intestine organisms via the mesenteric venous drainage entering the portal venous system and later seeding into the liver. It is important to note that without proper imaging, the true cause of the abscess could have easily been missed, especially given the negative signs and symptoms of appendicitis like this case. In Wich-

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mann et al. study about patients with liver abscess 9.4% of all abscesses were caused by an acute appendicitis. Diagnosis of appendicitis as cause of PLA was made during surgery for liver resections in three out of five patients. The most common symptoms in these patients were fever and right upper quadrant tenderness similar to other cases of PLA (8).

Principles of treatment are drainage and antibiotic therapy. Drainage options are percutaneous (needle aspiration versus catheter placement) and surgical (open versus laparoscopic or by endoscopic retrograde cholangiopancreatography (ERCP)). Single unilocular abscesses are best drained percutaneously, while placing the catheter is preferred especially if the abscess size is larger than 5 centimeters. Percutaneous drainage procedures are typically guided by a CT scan or ultrasound (9). The decision on drainage approach in multiple or multiloculated liver abscesses is surgical approach traditionally, while they may be successfully managed percutaneously specially if they are small and accessible. Surgical drainage is indicated in cases of inadequate response to percutaneous drainage (10). Also, surgical drainage is a proper approach when the underlying cause warrant surgical management. Antibiotic therapy should be based upon local bacterial resistance patterns and once blood and abscess specimen cultures are available the regimen can be adjusted.

The mortality rate among patients with PLA ranges from 4.2% to 11.7% (11). Yu Jan SU et al. found that patients who are diagnosed earlier have a greater chance of being treated by parenteral antibiotics. In contrast, patients who underwent surgical intervention encountered shock more commonly during their clinical course (35.7% versus 14.35%, P value=0.007) (12). Need for open surgical treatment, anaerobic infection, larger abscesses, sepsis, and malignancy are among independent risk factors for mortality among these patients (13).

4. Conclusion

There are numerous reasons for PLA, but the primary cause is typically the infection of the biliary tract. It is believed that appendicitis can lead to the spreading of bowel bacteria through the portal vein, resulting in the seeding of the liver. While this is not common, it is important to be aware of the possibility of developing hepatic abscess as a complication of appendicitis. Recent advances in testing and treatment have improved early detection and reduced mortality rates. The most effective management and treatment of PLA involves a combination of antimicrobial therapy and percutaneous drainage of the abscess. However, in older or immunocompromised patients, or if treatment is delayed, complications such as peritonitis can occur.

5. Declarations

5.1. Acknowledgement

None.

5.2. Authors' contribution

Both authors contributed on the manuscript equally.

5.3. Conflict of interest

None.

5.4. Funding

None.

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