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An Unusual Case of Pyogenic Liver Abscess Caused by Community-acquired Methicillin-Resistant *Staphylococcus aureus*

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Abstract Hepatic abscesses are the most common type of visceral abscesses. The annual incidence is estimated at 2.3 cases per 100,000 people and is more common in men than women. A considerable proportion of pyogenic liver abscesses are caused by hepatobiliary pathologies. However, it may also result from hematogenous seeding from the systemic circulation. Most pyogenic liver abscesses are polymicrobial in nature consisting of anaerobic and enteric gram-negative bacilli species. Less than 10% of cases are caused by *Staphylococcus aureus*. Of these, few are caused by Methicillin-resistant *Staphylococcus aureus* (MRSA) and even fewer from the community-acquired strain. The clinical manifestations of pyogenic liver abscesses usually include fever, abdominal pain and non-specific symptoms like nausea, vomiting, malaise, anorexia and weight loss. Treatment of pyogenic liver abscesses consists of source control with drainage and antibiotic therapy tailored to the isolated causative organism. We present a case of a 51-year-old Caucasian man who presented with severe right upper quadrant abdominal pain of 5 days in duration. Abdominal Computed Tomography (CT) showed a 7.7 by 6.0 centimeter multi-loculated mass in the right hepatic lobe with other scattered foci throughout the liver. The abscess was aspirated with a pigtail drain placed under CT guidance. The aspirated culture grew MRSA sensitive to Doxycycline and Trimethoprim/Sulfamethoxazole.

Keywords: *MRSA, abscess, liver, hepatic, community-acquired, Pyogenic*

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1. Introduction

Pyogenic hepatic abscesses are mostly caused by intra-abdominal pathology. However, other causes such as hematogenous spread are possible. Previously, appendicitis and diverticulitis were the leading causes of pyogenic hepatic abscesses but now, nearly 50% of pyogenic hepatic abscesses are caused by hepatobiliary pathology. Pyogenic hepatic abscesses carry a high mortality rate if they are not diagnosed and treated in a timely fashion; which can be difficult as they can present with a vague set of symptoms. Even with appropriate antibiotics and surgical interventions, mortality is greater than 10%. Pyogenic hepatic abscesses are usually polymicrobial in nature consisting of gram-negative and anaerobic organisms, but isolated gram-positive infections are possible via hematogenous spread, as is the case in our patient. We present a case of pyogenic hepatic abscess caused by community-acquired Methicillin-Resistant *Staphylococcus aureus* (MRSA) via hematogenous spread from skin/soft tissue infection.

2. Case Presentation

The patient is a 51-year-old homeless man who presented to our Emergency Department (ED) with complaints of severe right upper quadrant abdominal pain of 5 days in duration, "stabbing" in nature, made worse with deep inspiration and movement with no relieving factors. He has had subjective fevers and malaise. About 3 weeks prior, the patient had an abscess on his right upper extremity for which he sought treatment at an urgent care center. The abscess was incised and drained, and he was given Clindamycin. Unfortunately, cultures from the abscess later grew Methicillin-Resistant *Staphylococcus aureus*, which was resistant to Clindamycin. Two days prior to admission, he was seen in our Emergency Department for this complaint for which blood cultures were done, but the patient left against medical advice before more definitive management could be done. The patient has a past medical history of essential hypertension with no relevant surgical history. He is a cigarette smoker of 1 pack per day and he occasionally abuses

methamphetamines via smoking. He vehemently denies injection drug abuse. He also denies recent travel. He has a penicillin allergy which he claims to develop "anaphylaxis" to, however, he has tolerated cephalosporins in the past. On examination, his vital signs show afebrile temperature, pulse of 82 beats per minute, blood pressure of 161/95 millimeters of mercury, respiratory rate of 16 breaths per minute and oxygen saturation of 95% on room air. General examination reveals a disheveled man in discomfort but not in apparent distress. He had no scleral icterus and no lymphadenopathies palpated. His heart exam was unremarkable, but his lung exam revealed poor inspiratory effort due to pain. His abdominal exam revealed severe tenderness to palpation in the right upper quadrant but non-tender elsewhere. Neurologically, he had no deficits and was fully oriented to all spheres. Laboratory work-up reveals leukocytosis with 16,400 White Blood Cells per cubic millimeter of blood and mild normocytic anemia of 10.1 grams of hemoglobin per deciliter. The comprehensive metabolic panel was unremarkable with normal Liver Function Tests. Interestingly, the patient's blood cultures from the ED visit 2 days ago grew *Staphylococcus hominis hominis*. On imaging, a Computed Topography (CT) of the abdomen with intravenous contrast revealed a 7.7 by 6.0-centimeter multi-loculated mass in the right hepatic lobe with other scattered foci throughout the liver as well (Figure 1).

The patient was admitted to the hospital and empirically started on broad spectrum antibiotics with Vancomycin, Cefepime and Metronidazole. Interventional radiology was consulted and subsequently, the abscess was aspirated with a drain placed under CT guidance. Cultures from the drain grew *Staphylococcus aureus* that was Methicillin-Resistant sensitive to Doxycycline and Trimethoprim/Sulfamethoxazole. Blood cultures from this admission were negative. Furthermore, a chest x-ray did not reveal any intrathoracic abnormalities. A transthoracic echocardiogram was also done that did not show any intracardiac vegetations. The patient remained afebrile during his hospitalization and requested discharge on the fourth day of hospitalization. The drain was left in place and he was discharged on oral Trimethoprim/Sulfamethoxazole for 14 days.

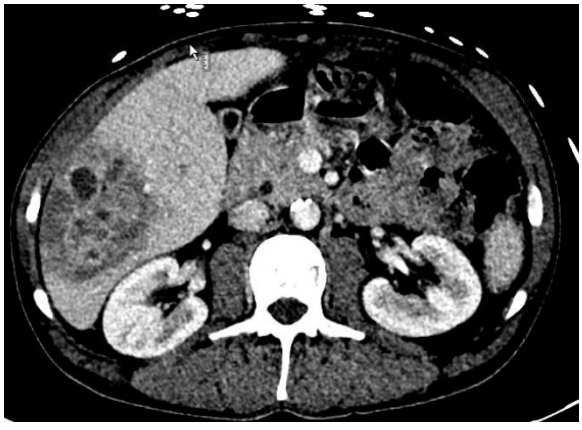

Figure 1. CT showing a 7.7 X 6.0 cm abscess in the right hepatic lobe

3. Discussion

The incidence of liver abscesses is fairly low at 2.3 cases per 100,000 hospitalizations in North America [1]. Mortality was as high as 75-80% in the early 1990's but has markedly decreased to about 10-40% today [2]. Infectious liver abscesses develop through contiguous spread from infection of neighboring tissues, from blunt or penetrating trauma or through hematogenous spread most commonly due to systemic bacteremia or in intra-abdominal infections [3]. The causative bacteria enter the liver from adjacent organs, through the portal venous system or the arterial flow. The most common route of infection is from the biliary tree responsible for 30-50%. Bacteremia, however, is only detectable in 43% of cases of liver abscesses [4].

Depending on the source of infection, liver abscesses can be poly-microbial in up to 55% of cases [5]. Organisms commonly isolated include *Escherichia coli*, *Klebsiella*, *Proteus*, *Pseudomonas*, *Streptococcus* species, *Bacteroides fragilis* and *Fusobacterium necrophorum*. *Staphylococcus aureus* is rare and represent less than 10% of pyogenic liver abscesses[6]. In addition, even fewer are caused by methicillin-resistant strains of *Staphylococcus aureus* (MRSA), especially community-acquired strains [7].

Pyogenic liver abscesses usually present with right upper quadrant abdominal pain, fever and chills with laboratory work-up showing leukocytosis, elevated alkaline phosphatase and low albumin [4]. Imaging with contrast-enhanced CT will lead to the diagnosis. In our patient, no other intra-abdominal focus of infection was found. He also did not have the common predisposing conditions such as underlying gastrointestinal malignancy, previous biliary surgery or endoscopy, immunosuppression, nor diabetes mellitus [1]. Additionally, he had no recent hospitalizations leading up to this admission. However, our patient did have a skin abscess 3 weeks prior to presentation with incised cultures growing MRSA that was inadequately treated with Clindamycin. Though there was no documented bacteremia with MRSA, we propose that the pathophysiology of his pyogenic liver abscesses was most likely from hematogenous spread from transient bacteremia caused by his soft-tissue abscess. Interestingly, he had blood cultures growing *Staphylococcus hominis hominis* several days prior to his admission during an ED visit. His blood cultures at this admission, however, showed no growth. *Staphylococcus hominis* subspecies, along with *staphylococcus epidermidis* and *staphylococcus haemolyticus* species account for up to 89% of contaminants [8]. Therefore, we believe that the blood cultures containing *staphylococcus hominis hominis* were likely contaminants and a red herring.

Drainage of pyogenic liver abscesses remain the standard of care and can be achieved percutaneously [9]. Percutaneous catheter drainage has been shown to be the most effective in terms of success rate, clinical improvement and time to achieve 50% reduction in size of cavity [9]. Surgical drainage is sometimes needed for abscesses that are difficult to access via percutaneous approach. After percutaneous catheter drainage, our patient continued intravenous antibiotics until cultures from the aspirate returned. He was then transitioned from Vancomycin to oral antibiotic with Trimethoprim/Sulfamethoxazole to target the community-acquired MRSA to facilitate discharge from the hospital. The duration of antibiotic therapy after adequate drainage is usually 14 to 42 days

depending on clinical response, though no randomized controlled trials have studied the optimal length of therapy [10]. Of note, many experts recommend colonoscopy evaluation in cases of cryptogenic pyogenic liver abscess to detect hidden colonic malignant lesions [11]. However, given our patient's age and the recent history of MRSA soft tissue abscess, colonic malignancy was deemed to be highly unlikely to be the etiology of his pyogenic liver abscess.

4. Conclusion

Pyogenic liver abscesses most commonly occur following peritonitis due to leakage of intraabdominal bowel contents with subsequent spread through the portal circulation or via direct spread in the setting of biliary infection. They may also result from hematogenous seeding in the setting of systemic infection. Most pyogenic liver abscesses are polymicrobial, caused by anaerobic and enteric gram-negative bacilli species. Less than 10% of cases are caused by Staphylococcus aureus. Of these cases, few are caused by methicillin-resistant Staphylococcus aureus (MRSA) and fewer still by a community-acquired strain. The clinical manifestations of pyogenic liver abscess usually include fever and abdominal pain in addition to other nonspecific symptoms like nausea, vomiting, malaise, anorexia and weight loss. Evaluation of suspected pyogenic liver abscess includes imaging, blood cultures, followed by aspiration and culture of the abscess material. The mainstay of treatment of a liver abscess is drainage and appropriate systemic antibiotics. In the case of MRSA causing a liver abscess, the selection of an appropriate antibiotic is critical to decrease morbidity and mortality.

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