Transdiaphragmatic Liver Abscess Secondary to Streptococcus milleri in an Immunocompetent Host

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Background
Pyogenic liver abscesses due to hematogenous spread present infrequently, resulting in uncommon patient presentations and microbiological profiles.

Summary
A healthy 44-year-old male presented with cough, hemoptysis, dyspnea, night sweats, right upper quadrant abdominal pain, nausea, and vomiting. Multiple imaging modalities of the abdomen demonstrated a necrotic right hepatic dome lesion extending into the right diaphragm and lower lobe of the lung consistent with an abscess. *Streptococcus milleri* (*S. milleri*) was isolated from aspirate. Infectious workup demonstrated two dental caries and no other potential sources of infection. Abscess treatment was successful with antibiotics and surgical debridement. While pyogenic liver abscesses are rare, they are commonly caused by *Klebsiella pneumoniae* (*K. pneumoniae*) or *Entamoeba histolytica* (*E. histolytica*). Here we present a case of penetrating pyogenic liver abscess uncommonly caused by *S. milleri* in an immunocompetent patient.

Conclusion
While *S. milleri* can cause pyogenic abscesses, they often occur locally and in immunocompromised patients. This case report is a rare case of penetrating pyogenic liver abscess caused by *S. milleri*, demonstrating that unlikely causes of advanced penetrating liver abscesses should be expected in the immunocompetent host. A complete workup should be performed on patients with symptoms of a liver abscess to ensure early detection and treatment.

Keywords
Pyogenic liver abscess; *Streptococcus milleri*; immunocompetence; transdiaphragmatic

Disclosure:
The authors have no conflicts of interest to disclose.

To Cite:
Case Description

The majority of liver abscesses occur secondary to intraabdominal infections. Thus, pyogenic liver abscesses (PLA) typically are caused by organisms commonly found in the bowel, including Klebsiella pneumoniae (K. pneumoniae), Escherichia coli (E. coli), bacteroides, and enterococci. Other causes of PLAs include hematogenous spread, direct extension, and hepatic trauma, and are associated with other microorganisms, including Staphylococcus and Streptococcus spp. Additionally, immnosuppression increases the risk of fungal and opportunistic organisms. Here, we present an unusual case of a penetrating liver abscess due to Streptococcus milleri (S. milleri) in an immunocompetent host.

A 44-year-old Caucasian male presented to the emergency department with a one-day history of small-volume hemoptysis with dyspnea and vomiting. Three months prior, he was treated with a seven-day course of ciprofloxacin for fevers, paroxysmal cough, night sweats, and a 30-pound unintentional weight loss, which mostly resolved. He endorsed use of chewing tobacco, but otherwise denied other substance use, recent travel, and a history of cancer or abdominal surgery. Initial exam demonstrated tachycardia, tachypnea, diaphoresis, right lower lobe coarse wheezes, and mild right upper quadrant (RUQ) tenderness. Lab results included: white blood count 18.46 K/µL with neutrophilic predominance, albumin 3.0 g/dL, serum bilirubin 1.3 mg/dL, alkaline phosphatase 289 U/L, AST 23 U/L, and ALT 44 U/L. Electrolytes were unremarkable. Influenza, HIV, and hepatitis panel were negative.

Chest computed tomography images demonstrated complete obstruction of the right lower lobe bronchus with lymphadenopathy with an incidental 7.1 x 9.3 cm heterogeneous right liver mass (Figure 1).

A subsequent magnetic resonance image of the abdomen and pelvis revealed the necrotic mass violating the hepatic capsule into the right diaphragm and right lower lobe (Figure 2). With concern for community-acquired pneumonia versus metastasis versus abscess, empiric ceftriaxone 1g and azithromycin 500 mg were started, and he was admitted to the general medical floor.
A bronchoscopy with bronchiolar lavage and lung biopsy was performed. Cytology and surgical pathology were negative, but the bronchiolar lavage specimen grew pan-sensitive *S. milleri*. Serum galactomannan, blood cultures, AFP, CA 19-9, and CEA were negative. Interventional radiology was consulted for aspiration of the presumed liver abscess, which was unsuccessful due to location and viscosity. The purulent aspirate was negative for bacterial and fungal growth and for *E. histolytica* antibodies. Azithromycin was discontinued, ceftriaxone was increased to 2 grams, and metronidazole 500 mg and pharmacy-dosed vancomycin were added. Despite a downtrending leukocytosis, the patient began complaining of right-sided pleuritic chest pain and RUQ pain, with fevers to 104°F and oxygen desaturation. Surgery was consulted at one week following presentation, and the patient was taken to the operating room for an exploratory laparotomy; right hepatic abscess drainage and resection; right diaphragmatic opening with empyema and lung abscess drainage and repair; and placement of a Jackson-Pratt drain and two thoracostomy tubes.

The patient immediately began to improve on postoperative day one. Panorex and echocardiogram were completed as part of the infectious workup, demonstrating two dental caries. Without other infectious sources, the caries were presumed to be the source of the positive bronchiolar lavage cultures, and the patient was placed on four weeks of ceftriaxone and metronidazole. He was discharged on postoperative day six without complication.

**Discussion**

When located adjacent to the diaphragm, PLAs may present with respiratory symptoms, such as pleuritic chest pain, cough, and dyspnea. However, it is rare for primary PLAs to cross the diaphragmatic plane into the pleural space and lung, especially in immunocompetent patients. Previous reports of transdiaphragmatic liver abscesses have been attributed to *K. pneumoniae* and *Entamoeba histolytica (E. histolytica)*.

In this case report, we present a rare advanced transdiaphragmatic PLA secondary to a *S. milleri* dental infection in a non-bacteremic immunocompetent patient.

Members of the *S. milleri* group are commensal organisms of the oropharyngeal, gastrointestinal, and genitourinary tracts known to cause aggressive pyogenic infections, especially abscess formation. Typically, these abscesses form secondary to sepsis and hematogenous spread via the arterial system. In our case, *S. milleri* was presumably the PLA causative organism due to the positive Panorex and a lack of risk factors for primary liver disease or an intraabdominal source. While the consistently negative blood cultures and negative echocardiogram argue against hematogenous spread, the preceding one month antibiotic treatment possibly suggests incomplete treatment of the initial bacteremia that seeded the liver with *S. milleri*. Thus, with time, the PLA locally progressed into the lung and pleural space without bacteremia recurrence.

*S. milleri* infections typically present as monomicrobial non-metastatic localized abscesses of the central nervous system, head and neck, abdomen, and thoracic cavity. This is the first case of a localized *S. milleri* PLA that crossed a fascial plane in an immunocompetent host. Sunwoo and Miller reported the only other *S. milleri* infections that crossed a fascial plane; in this article, the patients were immunocompromised due to advanced age, smoking status, and/or an autoimmune disorder. In our immunocompetent case, the incompletely treated initial infection allowed the infection to progress mostly unnoticed, until the abscess reached a size causing the acute progression of symptoms. Whether this advanced presentation of *S. milleri* is the norm for delayed or incomplete treatment in an immunocompetent host remains to be determined.

In immunocompetent hosts, PLAs have the potential to go unnoticed or be undertreated, resulting in an advanced presentation of penetrating disease. For healthy patients who present with a moderate to severe constellation of symptoms, it is important to perform a thorough examination and to consider all possible sources of infection to ensure early detection and treatment of a PLA.

**Conclusions**

Advanced penetrating PLAs occur, but are often associated with *K. pneumoniae* or *E. histolytica* infection. While *S. milleri* can cause a PLA, they often occur locally and in immunocompromised patients. We present a rare case of penetrating PLA caused by *S. milleri* in an immunocompetent patient. This case demonstrates unlikely causes of advanced PLAs should be expected in the immunocompetent host, necessitating a complete workup to determine the source of infection.
Lessons Learned

In immunocompetent hosts, it is possible for incompletely treated *S. milleri* to cause advanced penetrating liver abscesses. A thorough workup, including Panorex, should be considered for immunocompetent patients presenting with symptoms of a PLA.

References