Pyogenic Hepatic Abscess

Presented is a case of spontaneous pyogenic hepatic abscess in a previously healthy young man without associated risk factors. This disease entity has a low incidence, however, it is associated with significant morbidity and mortality if diagnosis and treatment are delayed. [Vukmir RB: Pyogenic hepatic abscess. Ann Emerg Med April 1991;20:421-423.]

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INTRODUCTION

Since antiquity, hepatic abscess has been suggested as a disease entity associated with significant morbidity and mortality. In 1886, Fitz reported pyogenic liver abscess as a complication occurring in young adults secondary to appendicitis, termed "Le Foie Appendiculare."[1,2]

Analysis of recent disease trends reveals stable incidence, morbidity, and mortality rates, even with the advent of modern diagnostic and treatment modalities.[3-5] However, pyogenic hepatic abscess today is most often a disease of middle age occurring as a complication of biliary disease.[6] The most significant prognostic factor determining patient outcome is delay in diagnosis. Fever of unknown origin and cholangitis are most often suggested as the primary findings at clinical presentation.[5]

CASE REPORT

A previously healthy, 21-year-old man presented for evaluation of a recent illness with fever to 40.3 C accompanied by rigor, malaise, nausea, and emesis. The patient was evaluated at three different health care facilities during a four-day period. He was diagnosed sequentially as suffering from 1) a viral syndrome, which was treated conservatively; 2) a urinary tract infection based on sterile pyuria found on urinalysis; and 3) a prostatitis, which was treated with trimethoprim/sulfamethoxazole.

The patient presented for evaluation after his symptoms worsened. An outpatient blood culture, obtained on a previous visit, also was positive for anaerobic *Streptococcus.* Pertinent medical history included no allergy, drug or alcohol use, travel, or occupational, wildlife, or contagious disease exposure. He had, however, been treated with isotretinoin for acne vulgaris. Review of systems was significant for gastrointestinal complaints but negative for symptoms of urinary tract infection or upper respiratory infection. Physical examination found a toxic-appearing young man with blood pressure of 130/70 mm Hg; pulse, 92; respirations, 18; and temperature, 38.1 C. Head and neck, cardiorespiratory, and neurologic examinations were unremarkable except for a chronic left ptosis. Abdominal examination revealed diffuse tenderness without rigidity. There were no dermatologic or musculoskeletal abnormalities noted, and lymphadenopathy was absent.

Laboratory evaluation was remarkable for leukocytosis with WBCs of 12,600 and a left shift (24 bands); slight anemia with a hemoglobin of 13.7 g/100 mL and a hematocrit of 40.3 mL/100 mL and normal indexes; respiratory alkalosis with pH 7.54; P_{CO_2}, 24 mm Hg; P_{O_2}, 64 mm Hg; HCO_3, 24 mEq/L; sterile pyuria with 10 to 15 WBCs/high power field; and an elevated erythrocyte sedimentation rate of 40 mm/hr. Liver function tests were most notable for nonspecific elevation with SGOT of 176; SGPT, 197; LDH, 398; and alkaline phosphatase of 208 IU as well as total bilirubin of 2.8 mg/dL and direct 1.6 mg/dL with normal ammonia, prothrombin time,

and partial thromboplastin time.

A chest radiograph demonstrated an elevated right hemidiaphragm, and an abdominal radiograph revealed hepatomegaly causing an abnormal bowel gas pattern (Figure 1). Screening toxicologic, serologic, bacterial, viral, and fungal studies were subsequently negative. However, the initial blood culture isolate was identified as nonhemolytic anaerobic *Streptococcus mitior-milleri*. An abdominal computed tomography scan was ordered, and the patient was transferred to the ICU for stabilization with a presumptive diagnosis of hepatic abscess rather than hepatitis, secondary to infectious or toxin (isotretinoin) exposure.

The patient's hospital course began with a hepatobiliary scan being substituted as a screening examination. This was normal. In light of the unusual pathogen, the patient was then diagnosed as having acute bacterial endocarditis and treated with high-dose penicillin followed by gentamycin. There was no clinical improvement by hospital day 5, and an echocardiogram, IV pyelogram, and indium autologous leukocyte scan were also normal.

The patient subsequently developed a right pleural effusion. At this time, an abdominal computed tomography scan revealed a solitary (15 cm) right hepatic lobe abscess (Figure 2). This pyogenic hepatic abscess required percutaneous drainage followed by open surgical drainage, which resulted in satisfactory patient recovery.

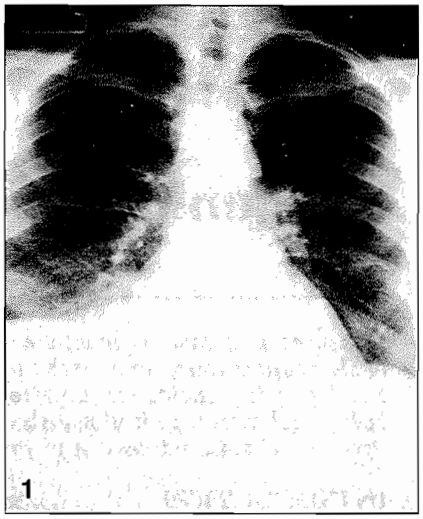
DISCUSSION

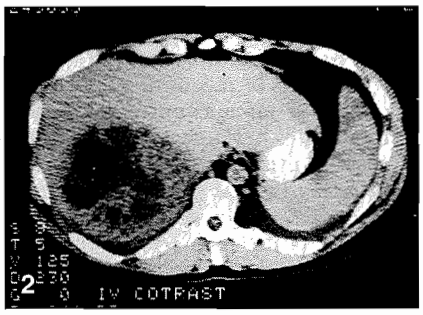
Hepatic abscess is a relatively rare condition with an incidence rate of 0.008% to 0.016% in hospital admissions but 0.29% to 0.54% in patients at autopsy.[4,5,7] Pyogenic abscesses are found in 90% of cases associated with older patients (more than 50 years old), malignancy, jaundice, palpable mass, and a fulminant presentation.[4,8] Amebic abscesses occur in 10% of cases, usually in younger patients (less than 50 years old) with recent travel history, diarrhea, abdominal discomfort, hepatomegaly, and a more insidious presentation.[4,8]

Pyogenic hepatic abscess is most often associated with fever (87% to 100% of cases) as well as abdominal tenderness (right upper quadrant, 71%).[4,9] Nonspecific symptoms and signs include nausea and vomiting, fatigue, weight loss, hepatomegaly, and ascites.[4,5,9]

FIGURE 1. *Chest radiograph featuring elevated hemodiaphragm.*

FIGURE 2. *Abdominal computed tomography scan revealing large, right hepatic lobe abscess.*





Diagnosis begins with laboratory evaluation that reveals leukocytosis with a left shift, anemia, and elevation of liver enzymes, indicative of both parenchymal (SGOT, SGPT, and LDH) and cholestatic (bilirubin and alkaline phosphatase) involvement.[4,5] Initial radiographic assessment includes an abnormal chest radiograph in 53% and an abnormal abdominal radiograph in 19% of cases.[4,5] Secondary radiologic assessment includes ultrasound with a sensitivity of 80%, radionuclide scanning with a sensitivity of 80% to 90%, and computed tomography as the screening procedure of choice with 95% to 100% sensitivity for diagnosis of hepatic abscess.[10,11] Microbiologic evaluation finds positive blood cultures in 50% of the patients, whereas abscess cultures yield a positive result in 73% of cases.[12]

Pathogenesis of hepatic abscess most often involves biliary obstruction (31.6%) and portal vein pyelophlebitis secondary to an intra-abdominal source (21.8%).[7,13] Less frequent etiologies include hematogenous dissemination through the hepatic artery (14.5%), direct extension (5.1%), primary hepatic disease (5.1%), and cryptogenic causes (22%).[7,13]

Microbiologic analysis of pyogenic hepatic abscess suggests that the most frequently encountered organisms are Enterobacteriaceae, specifically *Escherichia coli* (25.5% to 56.3% of patients).[7,13] Additional pathogens include *Streptococcus* in 19%, *Staphylococcus* in 16.4%, and anaerobes, specifically bacteroides, in 8% of patients.[7,13] Recent analysis suggests a changing bacteriologic spectrum with a decrease in incidence of Enterobacteriaceae and anaerobic organisms and prominence of nonhemolytic lancefield group F organisms.[4,14] These streptococci, *Streptococcus sangius, S mitior-milleri, Streptococcus mutans,* and *Streptococcus salivarius,* are normal oral flora and are capable of producing spontaneous pyogenic abscess as well as endocarditis in 5.7% of cases.[5,15]

One study found *S milleri* isolated in ten of 16 cases (62.5%), suggesting this to be the most frequently isolated pathogen in spontaneous disease. This is a significant finding in that *Streptococcus sp* are resistant to metronidazole and other agents directed at anaerobic and enteric organisms.

Therapy of pyogenic hepatic abscess involves both medical and surgical intervention. Initial emergency department management should include obtaining appropriate culture specimens and instituting broad-spectrum antibiotic coverage to include Gram-negative anaerobic and Gram-positive organisms. This is accomplished by therapy with ampicillin, clindamycin, or metronidazole and an aminoglycoside; ampicillin and chloramphenicol; or ampicillin-sulbactam or third-generation cephalosporin combined with an aminoglycoside.[14,16] The inclusion of metronidazole allows treatment of 100%

of amebic pathogens if the diagnosis is unclear.[17]

Admission along with surgical consultation is suggested because most patients require percutaneous or open abscess drainage for full recovery.[12,18] Prognosis is variable and adversely influenced by such factors as age extremes, underlying disease, and multiple abscesses.[4,12,13,19] Mortality was 100% in untreated cases and presently ranges from 25% for uncomplicated cases to 73% for patients having one or more complication such as perforation, septicemia, or recurrence.[3,9,12]

SUMMARY

Pyogenic hepatic abscess is a disease entity infrequently encountered in the ED although it is associated with significant morbidity and mortality. Our patient was unusual because of his good state of health and absence of notable risk factors. An interesting bacteriologic etiology – *S mitior-milleri*, is suggested as an emerging new pathogen in the development of this disease.[14] Furthermore, it is suggested that a high level of suspicion in patients presenting with a prolonged febrile illness is warranted and that early diagnosis before the onset of complications can affect outcome significantly.

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