BRIEF REPORT

Hepatic Abscess Presenting as Severe Fatigue and Anemia

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We describe a 54-year-old woman who had severe anemia as the initial presentation of a pyogenic hepatic abscess. She was afebrile and denied any gastrointestinal symptoms other than anorexia. We discovered her hepatic abscess when we evaluated her for an occult malignancy as the cause of her anemia. She was treated with percutaneous drainage of her abscess and parenteral antibiotic therapy. We searched MEDLINE, a computerized database, to find other patients whose hepatic abscesses presented as anemia. Although mild anemia is a common accompaniment of pyogenic hepatic abscesses, we found no reports of patients who presented with fatigue and anemia without any of the more common symptoms of hepatic abscess, such as fever, right upper quadrant pain, malaise, or nausea. We conclude that anemia without fever or abdominal symptoms is a rare presentation of pyogenic hepatic abscess. *(Arch Fam Med. 1993;2:189-192)*

In the preantibiotic era, pyogenic liver abscess had a uniformly fatal outcome if left untreated.[1] Despite diagnostic and therapeutic advances, mortality now ranges from 2% to 32%.[2-7] The prognosis depends in part on the rapidity with which the diagnosis is made and treatment is begun.

Patients with pyogenic hepatic abscess usually report fever and chills, right upper quadrant abdominal pain, anorexia, and malaise.[3,4] The most common physical findings are a temperature of greater than 37.8°C, hepatomegaly, right upper quadrant tenderness, weight loss, jaundice, and ascites.[3,4] The following case illustrates that fatigue and severe anemia can be the initial presentation of pyogenic liver abscess.

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REPORT OF A CASE

A 54-year-old black woman presented with a 6-week history of anorexia, fatigue, dizziness, and a 6.3-kg weight loss. She denied fever, nausea, vomiting, difficulty swallowing, change in bowel habits, abdominal pain, hematemesis, hematochezia, melena, diarrhea, or constipation. The patient drank about half a pint of wine a day for about 10 years and smoked one to two packs of cigarettes per day, but stopped both habits during her illness. There was no history of liver, biliary tract, or diverticular disease. An episode of appendicitis resulted in appendectomy years before. There was no history of anemia, exposure to tuberculosis, current medication use, or allergy. The patient estimated that her menopause had occurred at least 20 years earlier. She worked as a housekeeper in a nursing home.

Physical examination was unrevealing except for severe gingivitis and a soft systolic heart murmur; specifically, vital signs were normal, her abdomen was nontender, and there was no hepatospleno-

megaly or jaundice. Her hemoglobin level was 78 g/L and her hematocrit was 0.25. The mean corpuscular volume was 78.6 fL (reference range, 80 to 100 fL), the mean corpuscular hemoglobin was 24.3 pg (reference range, 27 to 34 pg), and the mean corpuscular hemoglobin concentration was 0.31 (reference, 0.32 to 0.36). The white blood cell count was 173×10^9/L with a slight left shift. Liver function test results were elevated, with an alkaline phosphatase level of 295 U/L, an aspartate aminotransferase level of 96 U/L, and an alanine aminotransferase level of 67 U/L. Total bilirubin level was 27.36 μmol/L (1.6 mg/dL). The carcinoembryonic antigen level was 1.6 μg/L. A chest roentgenogram showed only a slightly elevated right hemidiaphragm.

Examination disclosed *Gardnerella vaginalis* vaginitis, and metronidazole was prescribed. Stool guaiac was negative. Repeat complete blood cell count yielded similar results. Although the patient's anemia was microcytic, causes besides iron deficiency were considered because there was no evidence of gastrointestinal or menstrual blood loss. Her results showed a serum iron level of 9.85 μmol/L (55 μg/dL), a ferritin level of greater than 800 μg/L, an iron-binding capacity of 21.3 μmol/L (119 μg/dL), a serum vitamin B_{12} level of 1135 pmol/L, and a serum folate level of 35.35 nmol/L. There was no evidence of intravascular hemolysis. Hemoglobin electrophoresis results, including hemoglobin A_2, were normal. Hepatitis A and B screens were negative.

Three sets of blood cultures were drawn. Because the patient refused dental referral for her gingivitis, she was given a course of amoxicillin trihydrate in an attempt to improve her appetite. Subsequently, an echocardiogram demonstrated no vegetations and the blood cultures were negative.

One week after her initial presentation, the patient's appetite had not returned. Her hemoglobin level and hematocrit had declined to 63 g/L and 0.20, respectively, and her reticulocyte count was 0.02. An abdominal computed tomographic scan revealed the presence of a mass in the right lobe of the liver with "daughter" lesions. On needle aspiration, this was found to be an abscess. A percutaneous catheter was placed, and approximately 600 mL of pus was removed. Smears of drainage materials showed sheets of gram-positive cocci. Drainage materials were cultured for aerobic, anaerobic, fungal, acid-fast bacteria, protozoa, and amebic organisms.

The patient was admitted to the hospital for treatment with intravenous imipenem-cilastatin sodium (Primaxin) and further drainage. All cultures of drainage materials remained sterile. Stool cultures yielded normal flora, and no parasites were identified. Her hemoglobin level and hematocrit stabilized at 103 g/L and 0.31.

An endoscopic retrograde cholangiopancreatogram revealed several cystic duct stones and mild dilation of the extrahepatic common bile duct with suggestion of a stone distally. A communication was found between the abscess cavity and the biliary system. A 1-cm sphincterotomy was performed with passing of a 10-mm balloon through the common bile duct. No stone passage was seen. The consulting gastroenterologist recommended an elective cholecystectomy after the patient had recuperated and she was discharged home. Two months after discharge, the drainage stopped and the catheter was removed.

Six months after her initial presentation the patient had returned to her premorbid weight and had no complaints. Her hemoglobin level and hematocrit were normal. She underwent laparoscopic cholecystectomy uneventfully. No evidence of the former abscess cavity was found, but pathologic examination showed chronic cholecystitis.

COMMENT

Pyogenic liver abscesses are relatively rare lesions, accounting for only six to 10 of every 100 000 hospital admissions.[3] Most patients with liver abscesses report fever, right upper quadrant pain, and malaise, although fewer than one third of the 73 patients reviewed by Branum et al[3] had the complete triad. Anemia, usually attributed to chronic disease, is frequent also, occurring in two thirds of the patients described by Branum et al,[3] and in 70% of the patients in Bissada and Bateman's series.[4]

Using the search terms *hepatic abscess* together with *anemia* we searched MEDLINE from August 1992 to 1966 to determine if other patients with hepatic abscesses who presented with anemia have been described. We were unable to find another patient with this degree of anemia who did not also have at least one of the more usual symptoms of a pyogenic liver abscess. A 61-year-old man with a pyogenic hepatic abscess and a hemoglobin level of 69 g/L was described, but he also had fever and jaundice.[8]

The cause of the hepatic abscess in our patient was not clear, but biliary tract disease, possibly an occult episode of cholecystitis, may have been the cause. In the past, the appendix and the colon have been considered the major sources of pyogenic liver abscesses, seeding the liver via the portal vein,[1] but biliary tract disease is now the most common cause.[4,6,7,9,10] In about 25% of patients no source can be identified.[4] These so-called cryptogenic abscesses are thought to be caused by infection of infarcted portions of the liver.[11] Patients with sickle-cell disease also may develop hepatic abscesses in infarcts of the liver,[12,13] and a pyogenic hepatic abscess has been reported as the initial presentation of sickle-cell disease.[13] Abscesses may also result from complications of liver transplantations or from chronic granulomatous diseases.[14] Other sources can be infection in a contiguous structure, such as the gallbladder, infected foci anywhere in the body spread via the hepatic artery, or an infection secondary to a penetrating wound to the liver.[15,16]

We did not find any organisms in either the blood or abscess cultures from our patient. This may be because the patient received courses of amoxicillin and metro-

Clues to the Diagnosis of Pyogenic Hepatic Abscess

History
Fever for several days
Night sweats
Right upper quadrant or other abdominal pain
Malaise
Nausea or anorexia
Weight loss
Physical findings
Temperature greater than 37.8°C
Right upper quadrant tenderness
Hepatomegaly
Laboratory findings
Elevated white blood cell count
Anemia
Elevated liver function test results, especially alkaline phosphatase level
Elevated right hemidiaphragm on chest roentgenogram

nidazole during her evaluation, although the blood cultures were drawn prior to administration of amoxicillin. In a series of 54 patients with pyogenic hepatic abscesses reported by Stain et al,[2] only three patients had both sterile blood and abscess cultures.[2] Blood cultures and cultures of the abscess drainage should always be obtained from these patients.

Pyogenic liver abscesses are frequently polymicrobial. *Escherichia coli*, *Klebsiella* species, or other enteric gram-negative bacilli are cultured from the majority of abscesses.[2,10,15,17,18] The high frequency of sterile abscess cultures noted in the past was probably due to inadequate anaerobic cultivation.[18,19] The specific types of microorganisms that cause liver abscesses probably vary with the underlying disease.[8] *Escherichia coli* is often found in abscesses of biliary origin.[8] Anaerobic organisms are more common in abscesses of cryptogenic and portal origin than in those of gallbladder origin.[20] In other studies, *Staphylococcus aureus* and group A streptococci were isolated from patients who also had bacteremia due to the same organism, suggesting hematogenous spread.[3,4,18,21]

Clinical suspicion based on history, physical findings, and laboratory values, with confirmation from radiologic studies, allows physicians to make the diagnosis of hepatic abscess in most patients (**Table**). In addition to the classic triad of fever, right upper quadrant pain, and malaise, patients with hepatic abscess may complain of night sweats, abdominal pain, weight loss, jaundice, vomiting, or diarrhea.[3] Physical findings are less consistent. Temperatures greater than 37.8°C occur in more than half.[3,4] Right upper quadrant tenderness and hepatomegaly are more variable findings, but occur frequently.

Abnormal laboratory results are common. Depending on the definition of leukocytosis used, elevated white blood cell counts are present in 70% to 85% of patients with pyogenic hepatic abscesses.[2-4] Anemia occurs frequently, but is usually mild. Alkaline phosphatase is elevated in 80% to 90% of patients with pyogenic hepatic abscess, and other liver function test results are frequently abnormal too.[2-4] Both elevated liver function test results and elevated white blood cell counts were found in our patient.

Radiographic diagnostic clues to the presence of an hepatic abscess include elevation and limitation of motion of the right hemidiaphragm (as was seen in our patient), basilar atelectasis, right pleural effusion, or gas within the abscess cavity noted on plain roentgenogram of the abdomen or chest.[22] Ultrasonography and computed tomography have emerged as highly sensitive techniques for the detection of liver abscesses and are the diagnostic procedures of choice.[3,15,23-25] While ultrasound is preferred for initial testing,[2] Bissada and Bateman[4] recommend a computed tomographic scan if ultrasound fails to detect an abscess in highly suspicious cases.[4] In addition, ultrasonography and computed tomographic scan allow accurate placement of percutaneous drainage catheters, which are used both for diagnosis and therapy. These imaging modalities also allow close follow-up of the resolution of the abscess during and after therapy.

Once a diagnosis of liver abscess is confirmed, parenteral broad-spectrum antibiotics directed at both aerobes and anaerobes should be administered.[2] Antibiotic therapy can subsequently be modified depending on the patient's clinical response and the results of blood or abscess cultures.

Previously, all patients with pyogenic hepatic abscesses underwent surgical treatment. During the past decade, however, either percutaneous needle aspiration or catheter drainage has been preferred as initial therapy in uncomplicated cases.[2-4,26] Percutaneous aspiration or catheter drainage may also be considered for patients who are poor surgical risks.[4] Surgical therapy is reserved for patients with multiple or multiloculated abscesses, or for patients in whom surgical intervention is necessary because of coexistent conditions.[3]

Most patients' fevers will subside within 2 weeks of the start of antibiotic therapy and drainage.[17] Patients treated initially with percutaneous drainage or aspiration should be considered for surgery if (1) symptoms and fever persist for more than 2 weeks despite percutaneous drainage and appropriate antibiotic therapy,[17] (2) inadequate drainage occurs,[4] or (3) the patient's clinical condition worsens.[4] Treatment of patients with pyogenic hepatic abscesses with intravenous antibiotics alone has a high failure rate and should be considered a last resort.[3]

Pyogenic liver abscess can present with severe anemia, fatigue, and anorexia without other gastrointestinal symptoms. Liver abscess remains a diagnostic challenge despite the availability of improved diagnostic imaging techniques, and still carries a significant mortality rate. Nevertheless, the patient's history and physical examination, together with the re-

sults of common laboratory tests, can guide the physician to the diagnosis. Percutaneous drainage with antibiotic therapy is preferred for uncomplicated cases.

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Reprints not available.

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Erratum

In the November 1992 issue of the ARCHIVES, page 295, reference 27 should have read as follows: Kellie SE. Measurement of bone density with dual-energy x-ray absorptiometry (DEXA). *JAMA.* 1992;267:286-294.