**Pediatric Pyogenic Liver Abscess**

**Rafi Israeli, MD,* Jose Ernesto Jule, MD,† and Jeffrey Hom, MD, MPH‡**

The triage vital signs were oral temperature of 103°F, blood pressure 105/64 mm Hg, heart rate 124 beats/min, and respiratory rate 24 breaths/min. Physical examination revealed a well-developed male child in mild distress. Pertinent physical findings included anicteric sclera, mildly erythematous pharynx, as well as a supple neck without lymphadenopathy. The cardiopulmonary examination revealed tachycardia but no murmurs. Abdominal examination revealed normal bowel sounds. The abdominal examination demonstrated right upper and lower quadrant tenderness to deep palpation. There were no masses noted on examination. The genitourinary examination demonstrated normal external genitalia with bilateral descended testes. No hernia, masses, or tenderness were noted. Rectal examination was heme negative and without palpable masses or tenderness.

Laboratory studies yield the following significant results: white blood cell count, 20,640/µL, with toxic granulations; hematocrit, 32.1 g/dL; albumin, 3.8 g/dL; aspartate aminotransferase, 19 U/L; alanine aminotransferase, 26 U/L; alkaline phosphatase, 217 U/L; total bilirubin, 0.9 mg/dL; and prothrombin time/thromboplastin time, 13.8/42.1 seconds with international normalized ratio of 1.3. The urine analysis was negative. The chest radiograph (posteroanterior and lateral) was unremarkable.

Because of the RUQ physical examination findings, along with negative chest radiograph for pneumonia and leukocytosis with toxic granules, additional diagnostic imaging, that is, computed tomography (CT) of the abdomen and pelvis, was performed. A CT of the abdomen and pelvis showed a pyogenic liver abscess (PLA), 7.4 cm (transverse) × 6.8 cm (anterior-posterior) × 6.0 cm (craniocaudal) in the posterior aspect of the right hepatic lobe, extending from the hepatic dome to the midportion of the liver (Fig. 1). A course of antibiotics including ampicillin with sulbactam and metronidazole was initiated in the ED.

On hospital day 4, the coagulation factors, prothrombin time/thromboplastin time, were prolonged to 15.4/3.9 seconds; international normalized ratio was 1.5. Additional abnormal values with the clotting factors were fibrinogen of 1492.4 mg/dL, D-dimer of 2.37, and fibrin split products of less than 5 µg/mL. These abnormal coagulation values raised concerns about extensive liver involvement. One unit of fresh frozen plasma was administered, and interventional radiology for percutaneous drainage with placement of a catheter was performed. One hundred milliliters of pus was drained. The initial Gram stain showed no organisms and had moderate white blood cell count. Cultures of the fluid were ultimately negative for acid-fast bacilli, as well as for aerobic and anaerobic organisms. In addition, the fluid cytology was negative.

Serologies for amoebiasis and echinococcus were negative as well. A follow-up CT of the abdomen and pelvis, performed on hospital day 7, revealed the PLA reduced in size to 5.6 × 6.0 × 5.6 cm. However, a clinically insignificant right pleural effusion was discovered. The patient continued his antibiotic therapy.

A subsequent follow up CT on hospital day 11 revealed the PLA reduced to 3.6 × 5.1 × 5. cm. However, the right pleural effusion had increased in size. The pulmonary consult service considered it a reactive pleural effusion, with possible...
extension from the liver abscess. On hospital day 15, the effusion was drained via CT-guided thoracentesis with aspiration of approximately 10 mL of serosanguineous fluid. Cultures were negative for aerobic, anaerobic, and acid-fast bacilli. Cytology was negative as well.

The hepatic drain was removed on hospital day 22. On hospital day 25, the patient was switched to oral antibiotics, ciprofloxacin and metronidazole.

The patient was discharged on hospital day 26. Approximately 3 weeks after discharge, the patient had an outpatient RUQ ultrasound, which revealed complete resolution of the abscess.

**DISCUSSION**

Pediatric PLAs are difficult to diagnose.1 There is a paucity of signs and symptoms to aid in the diagnosis.2 The classic symptoms of fever, RUQ tenderness, and jaundice are only present in approximately 10% of patients. Other signs and symptoms are tenderness to percussion of the RUQ.3-5 Most diagnoses are made in adult patients of about 50 years of age.1,2 The incidence of diagnosed cases ranges from 8 to 20 cases per 100,000 admissions. Approximately half of these patients have both fever and RUQ pain.1

As with the physical examination, the patient’s history also does not usually identify a specific factor unique to a liver abscess. Some historical elements, such as malaise, fever, fatigue, anorexia, diarrhea, cough, dyspnea, and fever of unknown origin, have been previously cited.1,2 However, medical histories such as protein-energy malnutrition, chronic granulomatous disease, or diabetes have been cited as immunocompromised conditions that may help identify high-risk patients for a PLA.2

Laboratory testing, similar to history and physical examination, does not have the specificity to help diagnose cases. Elevations in liver function tests (alanine aminotransferase, aspartate aminotransferase, bilirubin, and alkaline phosphates) are common, but may also be normal. Leukocytosis and elevated sedimentation rate are other common laboratory features that may be present.1,4 Laboratory testing is mainly used to help confirm the diagnosis of a PLA.

Radiographic imaging is the key element in diagnosing PLA. Both ultrasound and CT offer visual identification and anatomical localization and size of the abscess. Ultrasound is useful in identifying concurrent RUQ pathology, such as biliary disease. Computed tomographic scanning, however, may better visualize the anatomic localization of the abscess. This visualization may help facilitate drainage of the abscess.

Pyogenic liver abscesses typically present as a solitary lesion, most of the time in the right hepatic lobe. Pyogenic liver abscesses are often found in patients with other comorbid conditions, especially immunocompromised states such as human immunodeficiency virus disease or organ transplantation.2 Hepatic invasion of bacteria may come from numerous sources, including the biliary tree, portal vein, hepatic artery, or penetrating trauma or may even be a direct extension from another contiguous focus of infection.1 In adults, the most commonly identifiable cause of PLA is suppurative cholangitis. Commonly isolated organisms are *Escherichia coli* and *Klebsiella pneumoniae*.6,7 Many other etiologic organisms have been identified. These include *Streptococcus viridans*, enterococci, and anaerobes. Microbial infections also exist. This spectrum of microorganism can be attributed to the many etiologic causes of PLA, as well as to inadequate culture techniques in isolating the organism.1,6 An abscess is termed cryptogenic when no identifiable etiologic agent is recovered from the culture.

Treatment begins with recognition of the PLA. The presumed causative agent dictates the antibiotic. Often, the suspected source agents are biliary or colonic organisms. The recommendation for a biliary source is ampicillin + gentamicin ± metronidazole. An alternative treatment is vancomycin + gentamicin ± metronidazole. Imipenem or meropenem may also be used. For a colonic source organism, the recommended antibiotics are third-generation cephalosporin + metronidazole. An alternative regimen is imipenem, meropenem, or antipseudomonal penicillin with a β-lactamase inhibitor. A fluoroquinolone + metronidazole ± amino glycoside may also be used.8

Traditional treatment involves both antibiotic administration and drainage of the abscess. Methods of drainage include an open surgical approach, laparoscopic drainage, radiographically guided percutaneous drainage, percutaneous aspiration, and needle aspiration.8

Pyogenic liver abscess is an uncommon disease in pediatrics. Our patient had the typical solitary node in the right lobe. There were no identifiable causative organisms; thus, the abscess was cryptogenic in origin. Antibiotics and drainage led to resolution of the abscess. Our patient developed a right pleural effusion, which resolved with subsequent drainage.

**REFERENCES**