Liver Abscess Caused by *Clostridium bifermentans* following Blunt Abdominal Trauma

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Pyogenic liver abscess is an unusual infection in both the pediatric and adult populations and is reported to occur in 0.016 to 0.03% of all hospital admissions (10). Previous reports have shown a significant mortality rate associated with liver abscesses, ranging from 42% in the pediatric age group (2) to 79% in the elderly (12).

Major conditions leading to the formation of liver abscesses include pyelonephritis complicating appendicitis, biliary infections, bacteremia, parasitic infestations, neoplastic diseases, and, rarely, blunt abdominal trauma (11, 12). In the pediatric age group, liver abscess is also seen as a complication of underlying immunological disorders (2).

The microbiological species most often associated with liver abscesses in pediatric patients include *Staphylococcus aureus*, *Escherichia coli*, *Klebsiella species*, *Enterobacter* species, and *Pseudomonas* species (1, 2). Anaerobes rarely account for these infections. In previous case reports associated with anaerobic infections, blunt trauma has not been reported as a factor (3).

The following case report describes a rapidly developing clostridial liver abscess in a child after blunt trauma that was managed successfully by early surgical intervention and systemic antibiotics and reviews the pertinent literature.

Case report. A 6-year-old previously healthy male was hospitalized after being struck on his right chest and abdomen by an automobile. On admission, his vital signs included the following: temperature, 36°C; pulse rate, 111 beats per min; blood pressure, 122/74 mm Hg; and respiratory rate, 36 breaths per min. There were no abrasions on his body, but he had a laceration over the right temporal area. There was tenderness over the right flank and right upper quadrant of his abdomen. On admission, the chest radiographs showed fractures of the right 10th and 11th ribs and a right lower-lobe pulmonary contusion. A computerized tomography scan of the abdomen revealed a laceration of the right lobe of the liver with some free fluid in the peritoneal cavity. The hematocrit of the patient was 22%, and he was transfused with 250 ml of packed erythrocytes.

On the third hospital day, the patient developed fever to 38.8°C, nausea, vomiting, and abdominal pain and distention. Radiographs at this time showed an elevated right hemidiaphragm with the presence of free air under the right diaphragm and an air fluid level in the liver. There was no free air in the biliary system. Laboratory investigations revealed a leukocyte count of 25,000/mm³ with 85% polymorphonuclear cells and 9% band forms, a hemoglobin level of 12 gm/dl, and a hematocrit of 37%. Liver enzymes were elevated, including a serum glutamic oxaloacetic transaminase level of 801 U/liter, a serum glutamic pyruvic transaminase level of 1,600 U/liter, and a serum alkaline phosphatase level of 336 IU/liter (a normal level for this age is 45 to 273 IU/liter). The total bilirubin was 0.8 mg/dl, and the albumin-to-globulin ratio was 3.8:2.5. Prothrombin time, partial thromboplastin time, and arterial blood gases were normal.

Antimicrobial therapy with intravenous cefoxitin (150 mg/kg per day) was started empirically, and the patient underwent an exploratory laparotomy. By a thoracoabdominal approach, approximately 1 liter of viscous foul-smelling fluid was aspirated from the right pleural space and peritoneum. The posterior inferior segment of the right hepatic lobe was necrotic and covered by a purulent exudate. The liver capsule was intact, and a specimen of the right liver lobe was sent for pathologic review. The blood vessels at the site of the liver laceration had already clotted. The right diaphragm was intact. After a partial right hepatectomy was performed, an omental graft was applied over the raw surface of the liver. Peritoneal fluid, pleural fluid, and liver tissue were collected for aerobic and anaerobic culture (Vacutainer Anaerobic Specimen Collector; Becton Dickenson and Co., Rutherford, N.J.) and transported immediately to the microbiology laboratory. A Gram stain of the liver tissue showed a moderate number of leukocytes but no organisms. All specimens were inoculated to aerobic and anaerobic media and incubated at 35°C aerobically in 5% CO₂ or anaerobically in a GasPak Pouch (BBL Microbiology Systems, Cockeysville, Md.). Blood cultures (BACTEC NR6A and NR7A; Johnston Laboratories, Inc., Towson, Md.) were also obtained. Postoperatively, antimicrobial therapy with ampicillin (150 mg/kg per day), gentamicin (7 mg/kg per day), and clindamycin (45 mg/kg per day) was begun.

Over the next 2 days, both preoperative blood cultures and cultures of all intraoperative specimens grew a gram-positive anaerobic bacillus. The organism produced a transparent spreading colony on the surface of anaerobically incubated blood agar (PRAS Brucella; Anaerobe Systems, Santa Clara, Calif.). The organism, identified as *Clostridium bifermentans*, was indole and lecithinase positive, hydrolyzed gelatin, and produced acid from glucose, maltose, and fructose. Catalase, urease, nitrate, and lipase reactions were

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negative, as were lactose, mannitol, mannose, sucrose, and xylose reactions. Identification as *C. bifermentans* was confirmed by the Anaerobe Laboratory at Virginia Polytechnic Institute and State University, Blacksburg, Va.

A piece of liver tissue fixed in Formalin and measuring 8 by 5 by 2 cm was received by the pathology laboratory for evaluation. Gross examination revealed a blackened capsule with ragged resected margins. Samples were processed for routine light microscopy and stained with both hematoxylin and eosin and Brown-Brenn (tissue Gram) stain. Coagulative necrosis, focally hemorrhagic, of the hepatic parenchyma involved nearly the entire sample. In some areas there was an increase in sinusoidal polymorphonuclear leukocytes. In another area, adjacent to a large recently thrombosed vein, was a cluster of rounded empty spaces (bubbles) consistent with spaces created by gas-forming organisms. The bacterial stain was negative.

The organism was susceptible to chloramphenicol, erythromycin, penicillin G, and tetracycline as determined by the broth-disk elution method (14). Ampicillin was then replaced by penicillin (150 mg/kg per day). The temperature of the patient returned to normal on the 3rd postoperative day, and he was discharged home in good condition on the 17th hospital day. Oral penicillin (75 mg/kg per day) was continued for a total period of 4 weeks. On reevaluation 2 months after discharge, the patient was asymptomatic and had no evidence of pulmonary or hepatic sequelae.

**Discussion.** In the case presented here, the predisposing event appeared to be blunt trauma to the abdomen. Often the trauma can predate the abscess by days to weeks. The pathogenesis of hepatic abscesses following trauma is not known. However, Eng et al. have speculated that hepatic hematomas often result when there is blunt trauma to the right side of the chest and abdomen (4). Organisms from the gastrointestinal tract which travel through the portal circulation settle and grow in the organizing clot. The hematoma provides an optimal medium for the development of anaerobic or mixed bacterial infections.

Recent advances in anaerobic microbiology have resulted in a greater awareness of the role of anaerobic bacteria in the etiology of liver abscesses (10). In the past, when aerobic cultures showed no growth, these abscesses were often called sterile or cryptoanerobic. Anaerobes isolated from liver abscesses include *Bacteroides, Peptostreptococcus, Fusobacterium*, and *Clostridium* species (2, 3, 10). *C. bifermentans* has not been previously reported to have been associated with hepatic abscesses.

*C. bifermentans* has been found in soil and sewage and has been isolated from a variety of human infections, including soft tissue infections and bloodstream invasion (5, 8). It is similar to *Clostridium sordellii*, which also usually produces acid from glucose, fructose, and maltose and is both lecithinase and indole positive. Urease production by *C. sordellii* is the distinguishing characteristic (9).

Treatment for these frequently polymicrobial liver abscesses must include early antimicrobial coverage in addition to surgical drainage and debridement (1, 7). Cefoxitin alone will not adequately cover all anaerobes since about 35% of *Clostridium* species are reported to be cefoxitin resistant (6). Penicillin alone is insufficient for the treatment of *Bacteroides fragilis*. Although there is no consensus as to the best antimicrobial regimen for liver abscesses for which the etiology is unknown, prolonged antibiotic therapy including either a penicillin with chloramphenicol, erythromycin with chloramphenicol, or clindamycin and an aminoglycoside (12, 13) is needed in treatment of pyogenic anaerobic or mixed liver abscesses.

**LITERATURE CITED**