Bartonella (Rochalimaea) henselae Hepatosplenic Infection Occurring Simultaneously in Two Siblings

Cat-scratch disease (CSD) is an infection caused by a gram-negative bacillus currently known as Bartonella henselae (formerly Rochalimaea henselae) [1]. Atypical presentations of CSD are seen in 5%-14% of cases and may be a cause of fever of unknown origin; hepatosplenic disease occurs in only 0.3%-0.7% of patients [2]. Reports of systemic disease occurring simultaneously in more than one family member are rare [3]. We report the cases of two siblings who had simultaneous hepatic and/or splenic CSD without regional lymphadenopathy and who were initially evaluated for fever of unknown origin; the conditions of both patients improved while they were receiving rifampin therapy.

A 5 1/2-year-old male (patient 1) was referred to Texas Children’s Hospital (Houston) for evaluation of a 1-month history of periumbilical abdominal pain and persistent fever (temperature to 104°F). His private physician treated him over a 3-week period for viral syndrome, otitis media, and arthralgias of his knees and ankles, but his condition did not improve. The erythrocyte sedimentation rate was 59 mm/hr; all other laboratory data were within normal limits. Because of the persistence of fever (temperature to 103°F–104°F) 1 week later, he was referred to the same hospital for further evaluation. He lived on a farm in Louisiana where he was frequently exposed to a variety of animals, including cats and dogs. He may have been scratched on his lip by a kitten 2 weeks before the onset of his symptoms.

On admission to the hospital the patient had a temperature of 100.1°F; physical examination was significant only for a liver edge that was palpable 3 cm below the right costal margin; there was some mild tenderness to palpation. An abdominal ultrasonogram revealed multiple hypoechoic lesions of the liver and spleen along with periportal and peripancreatic lymphadenopathy (figure 1). Serum was sent to the Centers for Disease Control and Prevention (CDC) for determination of B. henselae titers, and the patient was discharged to his home with instructions to complete a 2-week course of rifampin (15 mg/kg/d) q 12 h. A follow-up examination several days later revealed that the patient’s fever and abdominal pain had resolved completely. His titer of antibodies to B. henselae was >1:1,025.

The second patient was the 4-year-old brother of patient 1. He developed rhinorrhea, fever (temperature to 103.7°F) and a sore throat 2 weeks after his brother became ill. He was evaluated by his private physician, who thought he had a viral syndrome; however, over the next 12 days he continued to have fever (temperature to 106°F), and he was hospitalized for 4 days and treated for presumed bacterial sepsis. The erythrocyte sedimentation rate was 56 mm/hr, and the peripheral WBC count was 22,000/mm³ with a mild left shift. The rest of an extensive laboratory workup was negative or within normal limits. He was also referred to Texas Children’s Hospital for further evaluation. His medical history was noteworthy in that he had been exposed to some or all of the same animals that his brother had been exposed to.

On physical examination he did not appear ill. He had some mild pharyngeal erythema, and a liver edge was palpable 1–2 cm below the right costal margin. An abdominal ultrasonogram demonstrated an hypoechoic defect at the tip of his spleen, thickening of the gallbladder wall, and lymphadenopathy in the porta hepatitis. He was discharged to his home with instructions to complete a 2-week course of rifampin (15 mg/kg/d) q 12 h. A follow-up examination revealed that the patient’s symptoms had completely resolved within 1 week of starting therapy. His titer of antibodies to B. henselae was >1:1,025 (determined by the CDC).

Atypical presentations of CSD occur in up to 10% of patients and include multifocal hepatosplenic granulomas, Parinaud’s ocu-lodular syndrome, meningoencephalitis, hematologic abnormalities, and osteomyelitis [4]. The clinical diagnosis of atypical CSD is difficult to make, especially when the patient does not have palpable lymphadenopathy or a history of exposure to cats, and requires a high level of suspicion. These patients usually present with a lengthy history of fever and with variable systemic symptoms and are referred for evaluation of a fever of unknown origin.

The hepatosplenic form of CSD usually produces hepatic and splenic abscesses with multifocal granulomatous hepatitis [4–7]. These lesions may be demonstrated ultrasonographically in the liver and/or spleen as rounded, hypoechoic defects ranging in size from several millimeters to several centimeters in diameter. Lymphadenopathy in the paraaortic, periportal, and peripancreatic areas may also be present. CT shows fairly well defined areas of low attenuation whose margins may enhance following administration of intravenous contrast [6, 7]. B. henselae antibody titers may be used to support the diagnosis of CSD and have been reported to be very elevated in patients with the hepatosplenic form of the disease; in addition, histopathologic examination of biopsy specimens from liver lesions typically demonstrates necrotizing granulomas [5, 6, 8].

CSD is a benign, self-limited illness that usually resolves spontaneously. Antimicrobial therapy is typically reserved for prolonged, systemic, or severe illness; the ideal treatment regimen and duration have not been established. It is not uncommon for CSD to affect more than one family member simultaneously, but the simultaneous occurrence of hepatosplenic CSD (without adenopathy) in siblings has not been reported. The hepatosplenic presentation of CSD is being recognized more frequently as a cause of fever of unknown origin, and a trial of antimicrobial therapy should be given since this form of the disease seems to be responsive to treatment.

Tina Q. Tan, Milton L. Wagner, and Sheldon L. Kaplan
Departments of Pediatrics and Diagnostic Imaging, Baylor College of Medicine, Houston, and Texas Children’s Hospital, Houston, Texas; and Department of Pediatrics, Northwestern University Medical School, Children’s Memorial Hospital, Chicago, Illinois

Reprints or correspondence: Dr. Tina Q. Tan, Division of Infectious Diseases, Children’s Memorial Hospital, 2300 Children’s Plaza, Box 20, Chicago, Illinois 60614.

Clinical Infectious Diseases 1996;22:721–2
© 1996 by The University of Chicago. All rights reserved.
ISSN 1058-4838/96/2204-0025$02.00
References

Prosthetic Valve Endocarditis Due to Staphylococcus saccharolyticus

Anaerobic gram-positive cocci rarely cause endocarditis. Staphylococcus saccharolyticus, previously known as Peptococcus saccharolyticus, has been reported as a cause of native valve endocarditis (NVE) [1] but not, to our knowledge, as a cause of prosthetic valve endocarditis (PVE).

A 57-year-old woman with a prosthetic mitral valve that had been implanted several years previously was found to have fever, a systolic murmur, tender hepatomegaly, anemia, and leukocytosis. A transthoracic echocardiogram revealed a large mass that partially obstructed the tilting-disc-type prosthetic valve, as well as several mobile masses in the mitral outflow tract. Blood cultures yielded S. saccharolyticus from all bottles (Bactec NR 730 System; Becton Dickinson, Cockeysville, MD) under anaerobic conditions at 24 hours and under aerobic conditions after 11 days of incubation. The organism was identified as S. saccharolyticus by two anaerobic biochemical profiles (Rapido ANA II System, Innovative Diagnostic Systems [Atlanta] and An-Ident, Analytab Products [Plainview, NY]). The organism was resistant to all β-lactam agents including oxacillin (MIC, >32 μg/mL) and all cephalosporins (MICs, >64 μg/mL) and to metronidazole and tetracycline (MIC of each drug, >16 μg/mL). It was susceptible to vancomycin (MIC, 2 μg/mL; MBC, 8 μg/mL), clindamycin (MIC, <0.5 μg/mL), and chloramphenicol (MIC, 8 μg/mL). Susceptibility tests were performed by means of breakpoint MIC determinations and by the Kirby-Bauer agar dilution method on Mueller-Hinton me-