



Weekly

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## Cat-Scratch Disease in Children --- Texas, September 2000--August 2001

Cat-scratch disease (CSD), a bacterial infection caused by *Bartonella henselae*, has emerged as a relatively common and occasionally serious zoonotic disease among children and adults. To illustrate the spectrum of clinical manifestations of CSD observed during a 1-year period, Texas Children's Hospital (TCH) in Houston reviewed the medical records of 32 children evaluated at TCH during September 2000--August 2001 whose antibody titers indicated recent *Bartonella* infection. This report summarizes the evaluations of these cases and highlights four manifestations of infection with this pathogen in children. The findings emphasize that although CSD is generally a mild, self-limited illness, the differential diagnosis often includes more serious conditions (e.g., lymphoma, carcinoma, mycobacterial or fungal infection, or neuroblastoma) that might result in protracted hospital stays and lengthy treatments before diagnosis. Timely assessment of CSD is important, particularly when invasive diagnostic measures are being considered.

### Case Reports

**Case 1.** In July 2000, a boy aged 5 years was admitted to a local hospital after having fever (with temperature reaching 104° F [40°C]) for 12 days and left upper quadrant pain for 8 days. Aspartate and alanine aminotransferase concentrations were normal; a blood culture grew a contaminant. The child was transferred to TCH for evaluation of unexplained fever. Except for fever and inflamed tympanic membranes, the physical examination was unremarkable. Peripheral white blood cell count was  $18.3 \times 10^3/\text{cu mm}$  (normal range:  $5\text{--}14.5 \times 10^3/\text{cu mm}$ ), erythrocyte sedimentation rate (ESR) was 97 mm/h (normal range: 0--20 mm/h), and IgG and IgM serologic test results for Epstein-Barr virus (EBV) were negative. A bone scan was unremarkable. Abdominal ultrasound revealed multiple small hypoechoic lesions in the spleen and retroperitoneal adenopathy. After 3 days of intravenous rifampin therapy, his temperature declined to <101° F (<38.3° C). The child had sustained a scratch from a kitten 2 months before onset of illness. His serologic titer for *B. henselae* obtained on day 14 of illness was 1:4096.

**Case 2.** In September 2000, a girl aged 10 years with a bicommissural aortic valve had persistent low-grade fever, myalgias, arthralgias, weight loss, splinter hemorrhages, and hematuria and was admitted to TCH for evaluation and surgical management of endocarditis. She had been evaluated during the previous 9 months at another medical center for culture negative endocarditis. A transesophageal echocardiogram showed aneurysmal dilatation of the ascending aorta and probable vegetations. She also had a pulsatile lesion on the right forearm. Endocarditis caused by *Chlamydia psittaci* was suspected on the basis of the patient's history of

bird contact. During surgery, a large pseudoaneurysm of the ascending aorta and thickened dysplastic aortic valves were replaced with an aortic valve homograft. Histology demonstrated microabscess formation at the mouth of the aneurysm, noncaseating granulomatous inflammation in the wall of the aneurysm, and numerous gram-negative bacilli within vegetations. She also had resection of a brachial artery aneurysm with reconstruction of the artery. All cultures of tissue were sterile. Serologic test results for *Coxiella burnetii*, *Brucella* spp., *Histoplasma capsulatum*, and *Coccidioides immitis* were negative. Because the child had exposure to kittens and birds, doxycycline was administered along with penicillin, cefotaxime, and gentamicin at the time of transfer back to the referring hospital. The *B. henselae* titer obtained on day 7 at TCH was 1:8192.

**Case 3.** In June 2001, a boy aged 4 years was admitted to TCH with a 4-day history of intermittent back pain and an inability to walk. He had no history of trauma or contact with cats. He had a temperature of 99° F (38.2° C), no tenderness over the vertebrae, normal reflexes, and a 2x3 cm right inguinal lymph node. ESR was 96 mm/h, and three blood cultures were negative. Plain radiographs of the back and a bone scan were normal. Magnetic resonance imagery (MRI) demonstrated a diffuse abnormal marrow signal in the L1 vertebral body without destruction or apparent collapse of adjacent disc spaces. A small amount of material elevating the subligamentous space was observed just posterior to the L1 vertebra. A CT-guided fine needle aspiration biopsy showed no pathologic abnormalities. During the next several weeks, the child's back pain resolved without specific therapy. A repeat MRI performed 2 months later was normal. His *B. henselae* titer obtained on day 8 of illness was 1:2048.

**Case 4.** In August 2001, a girl aged 12 years was admitted to TCH after 3 weeks of intermittent fevers (101°--105.1° F [38.3°--40.6° C]), 2 days of right upper quadrant pain, and weight loss. Physical examination revealed enlarged and tender left and right inguinal lymph nodes. ESR was 93 mm/h. Two blood cultures and a urine culture were sterile. Stool cultures for various bacterial pathogens, including *Yersinia enterocolitica*, were negative. Several enlarged lymph nodes in the right lower quadrant were found on an ultrasound of the abdomen, but an abdominal CT was normal. Serologic test results for *Toxoplasma gondii*, cytomegalovirus, and EBV were negative. On day 7 of hospitalization, the patient underwent a colonoscopy, which was normal except for nodularity with mucosal edema in the terminal ileum. She had a recent history of dog and kitten scratches. Her *B. henselae* titer obtained during week 4 of illness was >1:8192.

Of the 32 patients, median age was 6 years (range: 2--15 years). Among the remaining 28 CSD cases observed at TCH during this 1-year period, clinical manifestations included fever and regional adenopathy (classic CSD)(20); prolonged fever without organ involvement (four); hepatosplenic granulotata (three); and encephalitis (one). Fourteen of the children were hospitalized.

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## Editorial Note:

CSD was first described as a clinical syndrome in 1931, but it was not until 1983 that a bacterial etiology was determined, and in 1992, the specific cause of CSD was identified. CSD is a feline-associated zoonotic disease, with an estimated annual incidence in the United States of 22,000 cases (1). Although CSD occurs in persons of all ages, the highest age-specific incidence

is among children aged <10 years (2). Infection with *B. henselae* is one of the most common causes of chronic lymphadenopathy among children, and in some case series up to 25% of the these infections result in severe systemic illness (3). Because TCH is a referral hospital, the frequency of severe manifestations seen in this series is probably disproportionately high relative to general practice. Other serious manifestations of CSD not included in this series are granulomatous conjunctivitis, neuroretinitis, and atypical pneumonia. In immunocompromised persons, *B. henselae* infections can cause other potentially life-threatening disease manifestations (e.g., bacillary angiomatosis and peliosis).

Serologic testing is the standard method of diagnosis (4,5) and should be considered for patients who present with adenopathy, fever, malaise, and history of feline contact. A single elevated indirect immunofluorescence assay titer or enzyme immunoassay value for IgG or IgM antibodies are generally sufficient to confirm CSD, because initiation of a humoral immune response generally precedes or is concurrent with symptom onset (4). IgG levels rise during the first 2 months after onset of illness, followed by a gradual decline (4). Other diagnostic assays, including polymerase chain reaction and bacterial culture, are available on a more limited basis at reference laboratories.

Treatment recommendations for *Bartonella*-associated diseases, including CSD, depend on the specific disease presentation. For most forms of CSD, assessing the efficacy of various antibiotics is difficult because symptoms are generally self-limiting over time, even in the absence of specific therapy. Recent experience with azithromycin suggests that this antibiotic hastens resolution of adenopathy of CSD (6). For patients with more severe disease, other antibiotic regimens have been successful, including azithromycin or doxycycline in combination with rifampin or rifampin alone (7); doxycycline or erythromycin are considered the drugs of choice for bacillary angiomatosis and peliosis (8).

CSD predominantly occurs in fall and winter because of either seasonal fluctuations in zoonotic transmission between felines or temporal changes in animal behavior and reproduction. Cat fleas (*Ctenocephalides felis*) are involved in the transmission of *B. henselae* among cats, but the role of fleas or other arthropods in the transmission of this pathogen to humans is not known. Scratches, licks, and bites from domestic cats, particularly kittens, are important risk factors for infection (9). Recommendations for prevention of CSD include vigilant elimination of fleas from feline pets and avoidance of traumatic injury from cats for persons who are immunocompromised (8) or who have heart-valve abnormalities (10). Cats rarely demonstrate overt signs of illness from infection, and no vaccines are commercially available to prevent *B. henselae* infection in animals.

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