Hepatosplenic cat scratch disease treated with corticosteroids

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A 3 year old girl with hepatosplenic cat scratch disease developed prolonged fever unresponsive to treatment with multiple antibiotics, including gentamicin, azithromycin, rifampin, and ciprofloxacin. Fever resolved with corticosteroid treatment.

Cat scratch disease (CSD), caused by infection with Bartonella henselae, usually presents as regional lymphadenitis that resolves without antimicrobial therapy. Less common manifestations of CSD include encephalitis, aseptic meningitis, osteomyelitis, pneumonia, and hepatosplenic abscesses. Results of antimicrobial therapy for these forms of CSD are inconsistent. We describe a child with hepatosplenic CSD who had fever for 90 days despite multiple antibiotic courses. The illness eventually resolved with corticosteroid therapy.

CASE REPORT

A 3 year old girl was referred with a 22 day history of fever of unknown origin. Her temperature reached 104°F daily but she had no other constitutional symptoms. On the 10th day of illness she experienced left flank pain and splenomegaly was noted; the pain resolved promptly and the spleen was not palpable on subsequent examinations. Other complaints included transient knee pain and a non-pruritic facial rash. Despite frequent contact with kittens and dogs in the home, the parents denied bites and scratches.

On the 15th day of fever, white blood cell count was 13.2 × 10⁹/l (69% neutrophils, 24% lymphocytes, and 6% monocytes) and Westergren sedimentation rate (WSR) was 42 mm/h. Epstein-Barr virus serology suggested past infection with the presence of IgG antibody to viral capsid and nuclear antigens. A chest radiograph and echocardiography were normal.

On referral, the child appeared healthy. Erythematous papules extended from the bridge of the nose to the left zygomatic arch. A non-tender lymph node measuring 0.5 cm was palpated in the right axilla but splenomegaly was absent. IgG antibody titre to B henselae was 160 but IgM was not detected (American Medical Laboratories, Chantilly, VA).

Abbreviations: CSD, cat scratch disease; CT, computed tomography; PCR, polymerase chain reaction; WSR, Westergren sedimentation rate

Figure 1 Graphic depiction of clinical course, therapy administered, and serial CT. GENT, gentamicin; AZITH, azithromycin; RIF, rifampin; CIP, ciprofloxacin; PDN, prednisone.
On the 29th day of fever, the IgG antibody titre to *B henselae* rose to $>1024$, consistent with acute *Bartonella* infection (IgM remained undetectable). Repeat WSR was 72 mm/h and computed tomography (CT) of the abdomen showed multiple hepatosplenic abscesses (fig 1). A 14 day course of intravenous gentamicin was initiated on the 33rd day. Persistent fever prompted a five day course of azithromycin on the 43rd day and then a 10 day course of rifampin on the 47th day of illness (antibiotic courses are depicted in the fig). By the 54th day, the *B henselae* IgG antibody titre fell to 128 but the WSR remained increased at 86 mm/h. No clinical response to antibiotics was evident.

On the 64th day of fever, abdominal CT showed renal abscesses with enlarging hepatosplenic lesions (fig 1). Biopsy of one hepatic lesion on the 66th day showed granulomatous inflammation with hepatocellular necrosis. Bacterial, fungal, and mycobacterial cultures were negative. Polymerase chain reaction (PCR) failed to detect *B henselae* sequences in the biopsied liver tissue (Molecular Diagnostics Laboratory, Vanderbilt University, Nashville, TN).

A seven day course of oral ciprofloxacin (250 mg twice daily) was initiated on the 74th day without response. Prednisone (2 mg/kg/day) was started on the 81st day and continued for 10 days (fig 1). Fever resolved after the eighth day of prednisone and did not recur. During 30 months of follow up, the patient was well, experienced normal weight gain and development, and no serious illnesses.

**DISCUSSION**

Prolonged fever with hepatosplenic CSD has been reported, but illness persisting for three months or more is rare. The present case is unusual because of the duration of fever despite treatment with multiple antimicrobial agents, and the apparent clinical response to prednisone. Additionally, this patient developed renal abscesses, a phenomenon not previously described.

Antibiotics with reported clinical efficacy in the treatment of CSD include gentamicin, rifampin, tetracycline, and ciprofloxacin, although only azithromycin has been studied in a prospective randomised trial. The patient reported here received four different antibiotics without change in clinical course. Prednisone is not currently recommended for treatment of CSD, although anecdotal experience suggests benefit in adults with severe systemic symptoms. In this case, the failure to detect *B henselae* sequences by PCR in granulomatous material obtained after 73 days of fever suggests a post-infectious, inflammatory process rather than ongoing infection, although the reported sensitivity of PCR is only 80% and detection is more difficult after 4–8 weeks of illness. The falling IgG antibody titre to *B henselae* despite persistent fever is consistent with a post-infectious process. Our experience suggests that prednisone may be useful as adjunctive therapy for hepatosplenic CSD in children with symptoms unresponsive to antibiotics.

**REFERENCES**