Cat-Scratch Disease Mimicking Pancreatic Malignancy: Case Report

* Bartonella henselae* is a small gram-negative rod that causes cat-scratch disease (CSD) [1, 2]. Many cases of atypical CSD have been reported, including cases of CSD that presented as abdominal visceral granulomas [3, 4]. A case of CSD that presented as severe abdominal pain and biliary tract obstruction in an HIV-infected patient has also been reported [5]. We describe a patient with CSD that mimicked pancreatic cancer.

An immunocompetent, previously healthy 56-year-old male physician was admitted to our hospital with a 5-month history of abdominal distention and discomfort that was associated with progressive fatigue. He had lost 5 kg. One month before admission, he had treated himself for 1 week with metronidazole, tetracycline, and clarithromycin. If symptoms occur, they should abate after the drug is discontinued.

Physical examination revealed no abnormalities other than jaundice. Laboratory tests revealed the following values: plasma y-glutamyltransferase, 401 U/L; alkaline phosphatase, 699 U/L; alanine aminotransferase, 120 U/L; and aspartate aminotransferase, 59 U/L. Unfortunately, the plasma bilirubin level was not measured. A CT scan and an ultrasonogram revealed a hypodense, 6-cm retroperitoneal mass in the area of the pancreatic head (figure 1) as well as near occlusion of the portal vein, with markedly decreased blood flow. The common bile duct (CBD) was dilated (diameter, 9 mm). Endoscopic retrograde cholangiopancreatography showed relative stenosis of the CBD (length, 4 cm) and no abnormalities of the pancreatic duct.

A stent was placed in the CBD. Examination of cells obtained by puncture of the mass was not diagnostic. Histological examination of a biopsy specimen obtained during laparotomy showed granulomatous necrotizing lymphadenopathy. Stains (hematoxylin-eosin, Grocott-Gomori methenamine silver nitrate, Ziehl-Neelsen, and Warthin-Starry silver stain) did not reveal any microorganisms. Titer of serum IgG antibody to *B. henselae*, measured with a whole-cell ELISA, were strongly positive (2,379 U/mL in a first sample obtained on admission, 6,044 U/mL at 4 weeks, and 412 U/mL at 11 months after admission [cutoff value for probable infection, >500 U/mL]), whereas titers of IgM antibody remained negative. A PCR assay for *B. henselae* with use of the primers p24E and p12B and appropriate negative and positive controls [1] was strongly positive.

On the basis of the positive results of serology and PCR, a diagnosis of CSD was made. The patient was treated with oral clarithromycin (500 mg twice daily) for 6 weeks as well as oral anticoagulants to prevent occlusion of the portal vein. After 3 weeks, the CBD stent was removed. A sonogram and a CT scan obtained 5 months after admission showed a marked decrease in the size of the retroperitoneal mass. Normal blood flow in the portal vein was restored, and there were no more signs of CBD compression.
Our patient’s CSD presented as painless jaundice that was due to a retroperitoneal mass; this lesion almost completely disappeared after 5 months of follow-up. Although the primers used for the PCR assay in this case are not able to distinguish *B. henselae* from *Bartonella quintana* and the amplified fragment was not sequenced, we still believe that the diagnosis of CSD was justified. First, histopathological analysis of a specimen from this patient showed necrotizing lymphadenitis, which is a typical feature of CSD in immunocompetent hosts but not of *B. quintana* infection [6]. Second, our patient remembered no contact with lice, the only known vector of *B. quintana*.

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Enhanced Amphotericin B Nephrotoxicity in Intensive Care Patients with Elevated Levels of Low-Density Lipoprotein Cholesterol

The clinical use of the antifungal agent amphotericin B has been limited by dose-dependent renal toxicity, which can result in a twofold decrease in the glomerular filtration rate and renal plasma flow that leads to renal potassium and magnesium wasting [1-4]. In most cases the underlying electrolyte imbalances that are a consequence of amphotericin B therapy can be treated with electrolyte supplements [1-4]; however, the long-term consequences of amphotericin B–induced renal tissue damage remain unclear.

Recommendations pertaining to the dosage and administration of amphotericin B in the treatment of candidemia and cryptococcal meningitis have been standardized and are well accepted. A 1-mg test dose of amphotericin B is given over 30 minutes and, if tolerated, is followed by an infusion of 0.2 mg/kg on the first day. The dosage is then increased by 0.1–0.2 mg/(kg · d) until a therapeutic or maximum-tolerated dosage is reached, usually 0.5–1.0 mg/(kg · d) in adults and 1.0–1.5 mg/(kg · d) in children [3–6].