and CSF of an immunocompetent patient with an acute encephalitic syndrome.

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References

Prevalence of Bartonella henselae Antibodies Among Human Immunodeficiency Virus–Infected Patients from Bahrain

Bartonella (formerly Rochalimaea) henselae is the most frequent etiologic agent of cat-scratch disease. In the HIV-infected or otherwise immunocompromised host, B. henselae may cause bacillary angiomatosis, parenchymal bacillary peliosis, bacteremia, weight loss, and prolonged fever [1, 2].

Despite the proliferating literature about B. henselae, little is known about the seroprevalence of this infection in different geographical areas. This study aims to determine the frequency of B. henselae antibodies in the serum of HIV-infected patients in Bahrain, a small island nation located in the Arabian (Persian) Gulf.

The B. henselae reference strain was provided by Jean Creek from the Office of the San Diego County Veterinarian. The strain was recultured on tryptic soy agar with 5% sheep blood and harvested on the 10th day in sterile PBS (pH, 7.4); autoagglutination was not observed. This suspension was fixed in 3.7% formalin for 1 hour and washed 3 times in PBS. After the final wash, the pellet was resuspended in coating buffer (0.05 M carbonate; pH, 9.6) and adjusted to $1.2 \times 10^7$ bacteria/mL with use of the McFarland opacity standard.

During performance of the indirect fluorescent antibody assay, we compared the antigen prepared in our laboratory with the killed antigen provided by the Division of Viral and Rickettsial Diseases at the Centers for Disease Control and Prevention (CDC) in Atlanta using the basic method described by Slater et al. [4]. Convalescent human sera (provided by the CDC) of patients with cat-scratch disease were used as positive controls; titers of $\geq 1:128$ were considered positive. All the immunofluorescent antibody assays were done in parallel, once with the antigen prepared in our laboratory and another time with the antigen provided by the CDC. The HIV-infected patient sera were obtained from HIV-infected patients identified in Bahrain by blood donations. The control sera were from age-matched HIV-seronegative blood donors in Bahrain.

Nine (16%) of 56 HIV-seropositive patients had antibodies to B. henselae, although only two (3.5%) of 56 HIV-seronegative individuals had detectable titers (estimated relative risk, 4.5; $P = .026$ by Mantel-Haenszel). Both patients who were not infected with HIV who had detectable antibodies had titers of $1:32$. In the HIV-seropositive group, two (22%) of nine had a titer of $1:32$, one (11%) of nine had a titer of $1:64$, and the others (six of nine [66%]) had high titers ($\geq 1:128$). When the B. henselae antigen prepared in our laboratory was used, the results were the same as those obtained when the antigen provided by the CDC was used.

Given the broad array of illnesses caused by B. henselae in patients who are infected with HIV, it is surprising how little is known about the geographic epidemiology of bartonella infections. Within the United States, regional differences in the incidence of cat-scratch disease have been documented, with lower rates in the western states [5]. In the current study, we found that 16% of HIV-infected subjects in Bahrain were seropositive for B. henselae as compared with only 3% of healthy blood donor controls. The 3% seropositivity among the HIV-negative subjects is similar to published seropositivity rates for healthy controls from the United States [3].

It is unclear why HIV-infected subjects were more frequently seropositive for B. henselae. Perhaps HIV-infected patients have more persistent B. henselae infections than the HIV-negative controls, who would most likely have had self-limited cat-scratch disease as their only B. henselae illness. Another plausible explanation is that there is a higher rate of cat ownership among HIV-

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infected patients. It is not surprising that *B. henselae* would be a common infection in Bahrain as cats are the most common household pets in this country; cats are risk factors for both cat-scratch disease and the more aggressive forms of *B. henselae* infection in immunocompromised hosts [2, 3].

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Peritonitis Due to a Ruptured Splenic Abscess

Splenic abscess is a rare entity of which the incidence at autopsy is <0.7% [1]. However, recently it has been reported with much greater frequency, probably because of improved diagnostic techniques and an increase in the number of immunocompromised patients. Rupture into the peritoneal cavity is the most severe complication and is associated with mortality rates ranging from 50% to 100% [1, 2]. We describe a case of generalized peritonitis due to a ruptured splenic abscess.

An 87-year-old woman was admitted to our facility because of a 2-day history of fever and abdominal pain. The most remarkable aspects of her medical history were diabetes mellitus, cholecystectomy, and left hemicolectomy for neoplasm 3 years previously.

Physical examination revealed a temperature of 38°C and mild left abdominal pain; other findings were unremarkable. Laboratory studies showed leukocytosis (leukocytes, 32.5 X 10⁹/L), with 95% polymorphonuclear cells; the hematocrit was 35%, and the hemoglobin level was 10.5 g/L. Urinalysis showed numerous WBCs, and roentgenography of the chest revealed elevation of the left side of the diaphragm. Abdominal ultrasonography was negative.

The patient was treated with antibiotics for possible urinary sepsis. Her abdominal symptoms were attributed to the same condition. After 2 days the abdominal pain worsened, and the abdomen was rigid, silent, and tender. The hematocrit decreased to 23%, and a CT scan showed splenomegaly with a large single collection inside, as well as perisplenic and pelvic fluid (figure 1).

Laparotomy showed diffuse peritonitis caused by a freely ruptured splenic abscess, and a splenectomy was performed. Cultures of blood and of the peritoneal fluid yielded *Bacteroides fragilis*, which was susceptible to metronidazole. Histologic examination revealed normal parenchyma surrounding the abscess. Postoperative evolution was uncomplicated, and the patient was discharged on the fifteenth postoperative day.

Hematogenous bacterial seeding from a septic focus is the most frequent cause of splenic infection (75% of cases) [1, 3], and bacterial endocarditis is the most frequent source (10% to 20% of cases), followed by urinary tract infection [1, 4]. Splenic abscesses occurring after septic abortion, appendicitis, diverticulitis, pneumonia, and other conditions have also been reported [1] and are more frequent in immunocompromised patients, such as those with AIDS, with chronic illness, or undergoing chemotherapy. Recently, splenic abscesses have been recognized with greater frequency in IV drug abusers [2, 4, 5].

Abnormalities in splenic tissue—such as hematomas following trauma or infarcts that occur in certain hematologic diseases—may favor bacterial growth [1]. Contiguous spread of infection is a less frequent cause (10%) and usually is of pancreatic, colonic, or gastric origin [1, 5]. In our case, the cause of the abscess was the hematogenous spread of *B. fragilis*, but the septic focus remained unclear.

The type of organism isolated suggested a colonic origin, but laparotomy did not show lesions in the large bowel.

For many years, aerobic gram-positive organisms (e.g., *Streptococcus* and *Staphylococcus* species) have been the commonest...