

five of the patients with watery chronic diarrhea (one patient died), improved within 10 days of treatment.

In Dakar, during the study describing ordinary and opportunistic enteropathogens associated with diarrhea in adults (5), stool samples were collected from five HIV-infected adults with watery chronic diarrhea. In all cases, heavy *K. pneumoniae* growth was observed on the primary culture media, and no other known pathogens were recovered. These *K. pneumoniae* strains were subjected to the same phenotypic and genotypic tests as the strains isolated in Bangui. HEp-2-adherent *K. pneumoniae* was identified in four of these five samples. The condition of all the patients rapidly improved after treatment with ofloxacin. In Bangui and Dakar, repeated stool cultures were negative for *K. pneumoniae* by the end of treatment, providing further evidence that these *K. pneumoniae* were of etiologic importance, especially the HEp-2-adherent *K. pneumoniae* strains.

Only seven patients (four with mild, two with watery, and one with bloody chronic diarrhea) had not taken antibiotics during the 2 weeks before stool collection. The stool specimens from these seven patients yielded pure primary cultures of HEp-2-adherent *K. pneumoniae* and no other bacterial enteric pathogens. None of these seven participants was diagnosed with pseudomembranous colitis. The HEp-2-adherent *K. pneumoniae* strains isolated from the two participants with watery chronic diarrhea induced the accumulation of fluid in ligated rabbit ileal loops, and the HEp-2-adherent strains isolated from three of the participants with mild chronic diarrhea carried the *astA* gene, which is associated with pathogenic EAEC. Among the five patients with pseudomembranous colitis, all of whom had received antibiotics before the onset of illness, we found that the four isolates from the patients with bloody chronic diarrhea were cytopathogenic; the one isolate from the patient with watery chronic diarrhea

had the pathogenic marker for enteroaggregative *E. coli*. These findings suggest that not only is *K. pneumoniae* associated with chronic diarrhea in HIV-infected persons but also that infection with particular HEp-2-adherent *K. pneumoniae* subtypes may be associated with specific clinical illness.

Financial support was provided by Agence Nationale de Recherche sur le SIDA (contract 1227) and Groupe d'Etude des Infections Diarrhéiques (ACIP, Réseau International des Instituts Pasteur et Instituts Associés).

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Granulomatous Lymphadenitis as a Manifestation of Q Fever

To the Editor: Q fever is a worldwide zoonosis caused by the obligate intracellular pathogen *Coxiella burnetii* (1). Human infection is usually the result of exposure to infected cattle, sheep or goats. Acute Q fever may be asymptomatic or manifest as a self-limiting febrile illness, pneumonia, hepatitis, or meningoencephalitis. Most cases of acute Q fever will resolve without sequelae, but endocarditis, granulomatous hepatitis, osteomyelitis, and endovascular infections are well-documented manifestations of chronic *C. burnetii* infection (1). Recently, various atypical manifestations of acute (2), and chronic (3) Q fever have been reported as well as changing clinical presentation of Q fever endocarditis (4) and changing epidemiology of Q fever (5). Researchers have suggested that heightened awareness of Q fever among doctors, coupled with improved diagnostic methods, could increase the medical knowledge about this difficult-to-diagnose and difficult-to-treat infection (4). We report two cases of granulomatous lymphadenitis associated with *C. burnetii* infection.

A 70-year-old man was admitted to the hospital because of weight loss, night sweats, and a continuous high-

grade fever of 2 months' duration. His past medical history was unremarkable, except for pulmonary tuberculosis treated 55 years earlier and chronic glaucoma. He lived in a rural area and had rare contact with cattle. On admission, his body temperature was 39.5°C; his right laterocervical lymph nodes were enlarged (3 cm x 4 cm) and inflamed. Blood values were unremarkable except for an elevated C-reactive protein level of 150 mg/L (normal <6). A computed tomography scan of the chest showed hilar calcifications and enlarged mediastinal lymph nodes. A biopsy of cervical lymph nodes indicated granulomatous lymphadenitis with foci of necrosis. *C. burnetii* DNA was detected on the lymph nodes with a *C. burnetii*-specific pair of primers that amplified an *hpaAB*-associated repetitive element (6). Results of serologic testing by indirect immunofluorescence (IF) were positive for *C. burnetii* with immunoglobulin (Ig) G antibody titer to phase 1 and phase 2 antigen of 800 and 1,600, respectively, and IgM antibody titer to phase 2 antigen of 50.

A 44-year-old man was admitted to the hospital because of a continuous low-grade fever of 3 months' duration. He had worked as a farmer for 15 years and assisted in the birth of sheep and cattle. On admission, his body temperature was 38°C, and right inguinal lymph nodes were inflamed, measuring 4 x 4 cm. A lymph node biopsy showed granulomatous lymphadenitis with stellate abscesses surrounded by palisading epithelioid cells. Serologic testing by indirect IF was positive for *C. burnetii* with an IgG antibody titer to phase 1 antigen of 320.

For both patients, results of Ziehl staining and Lowenstein (Bio-Rad, Marne-La-Coquette, France) cultures of gastric aspirates (x 3) and lymph node specimens were negative for mycobacteria, as were the results of tuberculin skin tests. Other diseases were ruled out, including brucellosis, yersiniosis, bartonellosis, and chlamydial infections (by serologic testing)

and fungal infections (parasitologic studies on lymph node tissue). Antinuclear antibodies were absent, and angiotensin-converting-enzyme values were normal. Both patients received doxycycline, 200 mg once a day, and rifampin, 600 mg twice a day, for 1 year, and the symptoms resolved (follow-up at 18 months for patient 1 and 9 months for patient 2, respectively). For patient 1, serologic testing after 1 year of treatment showed an IgG antibody titer to phase 1 antigen of 320.

Granulomatous lymphadenitis has been described during mycobacterial infections, tularemia, cat scratch disease, yersiniosis, lymphogranuloma venereum, histoplasmosis, coccidioidomycosis, and chronic granulomatous diseases (7). One well-documented case of acute Q fever with necrotic cervical lymphadenitis has been recently reported (8); to our knowledge, granulomatous lymphadenitis has never been reported during Q fever. In both cases reported here, *C. burnetii* was the likely etiologic agent, given the results of polymerase chain reaction and serologic studies (patient 1) or the patient's occupation and results of the serologic testing (patient 2). Moreover, for both, no other potential cause could be identified, and the response to doxycycline-rifampin regimen was favorable. We suggest that granulomatous lymphadenitis be added to the list of atypical presentations of Q fever.

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Has *Coxiella burnetii* (Q fever) Been Introduced into New Zealand?

To the Editor: New Zealand has been an exception to the panglobal distribution of *Coxiella burnetii* (1), the causative organism of Q fever, as shown in a 1990-1991 study (2) of 12,556 sheepdogs and 2,181 aborting cattle, all seronegative for *C. burnetii*. In 1997, the *Rabbit hemorrhagic disease virus* (RHDV) was illegally imported from Australia into Central Otago, New Zealand, for the purpose of rabbit control. The unknown source and purity of RHDV, and the potential use of infected rabbits or their organs to transport it, meant that *C. burnetii* could have been coincidentally introduced along with the RHDV-infected rabbit material. To establish whether this occurred, we examined serum