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 the pathogenic marker for entero-aggregative *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.

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References

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 hiliar 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 htpAB-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 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 (normal<6). A computed tomography scan of the chest showed hiliar 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 htpAB-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 antigen of 800 and 1,600, respectively, and IgM antibody titer to phase 2 antigen of 50.

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