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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Phuong L. Nguyen Thi,* Simon Yassibanda,† Awa Aidara,‡ Chantal Le Bouguenec,§ and Yves Germani*  
*Institut Pasteur de Bangui, Bangui, Central African Republic; †Hôpital de l’Amitié, Bangui, Bangui, Central African Republic; ‡Hôpital Pasteur de Dakar, Dakar, Sénégal; and §Institut Pasteur, Paris, France

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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 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 320. IgG antibody titer to phase 1 antigen was positive for C. burnetii 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, coccidiodomycosis, 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.

Pierre Tattevin,* Cédric Arvieux,* Mathieu Dupont,* Pascal Gugenbuhl,† Alexandre Lemeur,† and Christian Michelet†

* Hôpital Pontchaillou, Rennes, France; and †Hôpital Sud, Rennes, France

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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