Mycobacterium tuberculosis Beijing Strain, Bamako, Mali

To the Editor: *Mycobacterium tuberculosis* has ≥36 identified genotype families (*1*). Four genotypes cause 35% of documented cases of active tuberculosis (TB): Beijing (10%–11%), Latin American–Mediterranean (9.3%), Haarlem (7.5%), and the X clade (7%) (*1*,*2*) The Beijing clade strains, reported in 1995 from the People's Republic of China, are widely recognized as highly pathogenic with a possible predilection for multidrug resistance (*3*). Predominant in Asia, these strains have been documented in other parts of the world (*1*,*4*,*5*). The virulence, propensity to become resistant, and distinct geographic distribution of the Beijing clade suggest it may have some adaptive advantage in producing disease in humans. Limited data suggest that its presence in Africa is low (*2*,*4*,*5***)**.

In Bamako, Mali, 2 patients with active pulmonary TB came to the research clinic at Point G Hospital, affiliated with the University of Bamako Medical School, for recruitment under a US National Institute of Allergy and Infectious Diseases' institutional review board–approved protocol. The first patient, a previously healthy 34-year-old man, sought treatment in March 2008. He had a 3-month history of fever, cough, shortness of breath, and left-sided chest pain; respiratory rate of 24/min; temperature of 36.8ºC; and pulse rate of 68/min. He weighed 60 kg. His leukocyte count was 8,700 cells/μL, and he was positive for HIV-1 with a CD4+ T-cell count of 468 cells/μL. He reported contact with persons from other countries in Africa, China, and other parts of Asia.

Chest radiograph showed a cavitary lesion on the left upper lobe and opacities throughout the left lung. Three sputum samples collected 3 days apart were digested and decontaminated with N-acetyl-L-cysteine, 4% NaOH; concentrated by high-speed centrifugation; stained with auramine-rhodamine; and evaluated by using fluorescent microscopy. The many acid-fast bacilli (AFB) seen were identified by using nucleic acid probes (AccuProbe, Gen-Probe, San Diego CA, USA). Antimycobacterial drug susceptibility was determined by using a manual indirect susceptibility test (mycobacterial growth indicator tube, AST SIRE System; BBL, Becton Dickinson, Franklin Lakes, NJ, USA) showed the isolate sensitive to isoniazid (0.1 μg/mL), rifampin (1.0 μg/mL), and ethambutol (3.5 μg/mL) but resistant to streptomycin (0.8 μg/mL). Spoligotyping using a commercially available kit (Spoligotyping Isogen Life Science, De Meern, the Netherlands) showed characteristics of the Beijing clade (online Appendix Figure, panel A, www.cdc.gov/EID/content/16/2/362-appF.htm) (*6*).

The patient began treatment with the standard first-line regimen of isoniazid, rifampin, pyrazinamide, and ethambutol fixed-dose combination (Svizera Laboratory, Mumbai, India) according to Malian National Guidelines. Follow-up sputum samples at 13 and 18 weeks of treatment were smear- and culture-negative for AFB.

The second patient, a 28-year-old woman, sought treatment in July 2008. For 1 year, she had received first-line and retreatment regimens that failed to clear her sputum of AFB. She had begun second-line treatment for multidrug-resistant disease 2 days earlier. She had a history of fever, cough, and weight loss; temperature of 37.1ºC; heart rate of 104 beats/min; respiratory rate of 24/min; and blood pressure of 90/60 Hg mm. She weighed 49 kg. Leukocyte count was 9,400 cells/μL. Serologic results for HIV-1 and -2 were negative. Chest radiograph showed a right apical cavitary lesion and a fibrotic lesion in the right middle lung field. She did not recall any exposure to TB. She worked as an assistant at a local telephone center.

Two sputum samples, processed as described above, were positive for, and Gen-Probe testing confirmed, *M. tuberculosis*. According to antimycobacterial susceptibility testing, the strain was resistant to isoniazid (0.1 μg/mL), rifampin (1.0 μg/mL), ethambutol (3.5 μg/mL), and streptomycin (0.8 μg/mL). Spoligotyping confirmed the strain as Beijing clade, and restriction fragment length polymorphism (7) confirmed that it differed from that of patient 1 (online Appendix Figure, panel B).

The relevance of different genotypes, such as the Beijing clade, to disease progression is being studied. Evidence indicates the genotype may factor in transmission or pathogenesis. In a study in Cape Town, South Africa, disease produced by the Beijing clade increased exponentially over time, suggesting a possible pathogenic advantage; although most cases were drug susceptible, the likelihood of unsuccessful treatment was greater than for non-Beijing variants (*8*). Although the Beijing clade does not appear to have greater propensity than non-Beijing genotypes for acquiring resistance, certain variants within the group that become multidrug resistant may be more likely to acquire such resistance. Beijing strains particularly may tend to acquire resistance more easily than others under conditions of suboptimal treatment (*9*). In Cape Town during 2000–2003, the Beijing clade as a cause of disease in children increased from 13% to 33%, suggesting a selective advantage in transmissibility and disease production (*10*).

These cases highlight the need to diagnose disease and resistance early and to begin appropriate treatment in TB-endemic countries. Knowledge of circulating strains and their resistance patterns is essential to developing

effective programs to curtail the spread of TB within the country and the region; in this era of globalization, it is required for the successful control of TB worldwide.

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Bassirou Diarra,[1] Sophia Siddiqui,[1] Dramane Sogoba, Brehima Traore, Mamoudou Maiga, Janice Washington, Anatole Tounkara, and Michael A. Polis

Author affiliations: Project SEREFO-NIAID/University of Bamako Research Collaboration on HIV/TB, Bamako, Mali (B. Diarra, D. Sogoba, B. Traore, M. Maiga, A. Tounkara); National Institute of Allergy and Infectious Diseases, Bethesda, Maryland, USA (S. Siddiqui, M.A. Polis); and US Food and Drug Administration, Silver Spring, Maryland, USA (J. Washington)

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[1]These authors contributed equally to this article.

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Address for correspondence: Sophia Siddiqui, Collaborative Clinical Research Branch, National Institute of Allergy and Infectious Diseases, National Institutes of Health, 6700A Rockledge Dr, Rm 1105, Bethesda, MD 20892, USA; email: ssiddiqui@niaid.nih.gov

Hemorrhagic Fever with Renal Syndrome, Vietnam

To the Editor: Hantaviruses are primarily rodent borne and can cause hemorrhagic fever with renal syndrome (HFRS) in persons who inhale aerosolized excreta from infected rodents. The clinical characteristics of HFRS are fever, hemorrhage, and varying degrees of renal and hepatic dysfunction. Although HFRS is endemic primarily to Eurasian regions, there is serologic evidence of hantavirus infections in rodents and humans worldwide (*1*). Little is known about the occurrence of hantavirus infection in rodents or humans in Vietnam. One study found 5.4% prevalence of antibodies against Hantaan 76–118 and Puumala strains among residents of the Hanoi Metropolitan (*2*), whereas another study in southern Vietnam did not find evidence of hantavirus infection in humans (*3*). We describe autochthonous HFRS from Vietnam, possible reservoir hosts, and the follow-up investigation, which implies the presence of a strain of Seoul virus (SEOV).

The case-patient was a previously healthy 25-year-old nurse working in a referral hospital and residing in a semiurban district of Ho Chi Minh City. On September 23, 2008, she was admitted to the referral hospital with a history of high fever, chills, myalgia, nausea, vomiting, hematuria, and abdominal and lower back pain for 3 days. Physical examination showed a body