LETTERS

- Elfadil AA, Hasab-Allah KA, Dafa-Allah OM. Factors associated with Rift Valley fever in south-west Saudi Arabia. Rev Sci Tech. 2006;25:1137–45.
- Mellor PS, Hamblin C. African horse sickness. Vet Res. 2004;35:445–66. doi:10.1051/vetres:2004021
- Martín-Folgar R, Lorenzo G, Boshra H, Iglesias J, Mateos F, Borrego B, et al. Development and characterization of monoclonal antibodies against Rift Valley fever virus nucleocapsid protein generated by DNA immunization. MAbs. 2010;2:275– 84. doi:10.4161/mabs.2.3.11676
- Sotelo E, Llorente F, Rebollo B, Camuñas A, Venteo A, Gallardo C, et al. Development and evaluation of a new epitopeblocking ELISA for universal detection of antibodies to West Nile virus. J Virol Methods. 2011;174:35–41. doi:10.1016/j. jviromet.2011.03.015
- Scott GR, Coackley W, Roach RW, Cowdy NR. Rift Valley fever in camels. J Pathol Bacteriol. 1963;86:229–31. doi:10.1002/ path.1700860131
- Mariner JC, Morrill J, Ksiazek TG. Antibodies to hemorrhagic fever viruses in domestic livestock in Niger: Rift Valley fever and Crimean-Congo hemorrhagic fever. Am J Trop Med Hyg. 1995;53:217– 21.
- Eisa M. Preliminary survey of domestic animals of the Sudan for precipitating antibodies to Rift Valley fever virus. J Hyg (Lond). 1984;93:629–37. doi:10.1017/ S0022172400065207
- Olaleye OD, Tomori O, Schmitz H. Rift Valley fever in Nigeria: infections in domestic animals. Rev Sci Tech. 1996;15:937–46.
- Abd el-Rahim IH, Abd el-Hakim U, Hussein M. An epizootic of Rift Valley fever in Egypt in 1997. Rev Sci Tech. 1999;18:741–8.

Address for correspondence: Miguel Ángel Jiménez-Clavero, Centro del Investigación en Sanidad Animal (INIA), Ctra Algete- El Casar s/n, 28130, Valdeolmos, Spain; email: majimenez@inia.es



Brucellosis, Taiwan, 2011

To the Editor: Human brucellosis is the most common zoonosis worldwide (1-4). The disease is transmitted to humans through the consumption of infected meat and raw dairy products from domestic livestock or by direct or indirect contact with infected animals (1-3). The disease is multisystemic and shows wide clinical polymorphism (2-4).

A 54-year-old woman reported high fever, poor appetite, epigastralgia, mild dysuria, generalized myalgia, and mild left side pain for 6 days before she sought care at and was admitted to National Taiwan University Hospital, Taipei, Taiwan. She had a history of ovarian cancer (clear cell, stage Ic), which had been treated with surgery and chemotherapy 7 years earlier at our hospital. She had traveled to many countries, most recently to Algeria and Morocco 2 months before this admission. During her stay in North Africa, she had close contact with camels, ate cheese and yogurt, and drank milk, even in the desert. Fever occurred 1 month after she returned to Taiwan.

On physical examination, her body temperature was 39.9°C, blood pressure was 97/68 mm Hg, and pulse rate was 89 beats/min. There was mild tenderness on palpation in the epigastric area. Laboratory analysis of serum specimens showed elevated levels of alanine aminotransferase (534 U/L), aspartate aminotransferase (841 U/L), and alkaline phosphatase (337 U/L) but a total bilirubin level (0.48 mg/dL) within reference limits. Renal function was within reference ranges (blood urea nitrogen 9.5 mg/ dL, creatinine 0.6 mg/dL). C-reactive protein was elevated (4.59 mg/dL), but procalcitonin level was within reference range (0.13 ng/mL). The leukocyte count was 4,710 cells/mm³, and hemoglobin was 11.4 g/dL. Serologic tests for viral hepatitis were negative for hepatitis B virus, hepatitis A virus, cytomegalovirus, and Epstein-Barr virus infections. Abdominal ultrasound indicated mild splenomegaly and no evidence of vegetation. Abdominal and pelvic computed tomography showed focal splenic infarction with splenomegaly.

Empirical ceftriaxone (1 g every 12 h) and doxycycline (100 mg every 12 h) were administered, and fever subsided 5 days later. Two aerobic culture bottles (BacT/ALERT, bioMérieux Inc., La Balme les Grottes, France) from different sets of blood cultures on the day before admission yielded unidentified gramnegative tiny bacilli after 2 days of incubation. The organism was identified as Brucella melitensis by the Vitek 2 GN identification system (bioMérieux Inc.) (probability of identity 99%) and was confirmed by analysis of partial 16S rRNA gene sequencing. Two primers used: 8FPL (5'-AGAGT were TTGATCCTGGCTCAG-3') and 1492 RPL (5'-GGTTACCTTGTTACGAC TT-3'). We compared the partial sequences with published sequences in the GenBank database by using the BLASTN algorithm (www.ncbi.nlm. nih.gov/blast). The closest match was B. melitensis (GenBank accession no. CP001852.1; maximal identity 100%). MICs were determined by the Etest (AB Biodisk, Solna, Sweden) on Mueller-Hinton agar (BBL, Becton Dickinson, Sparks, MD, USA) supplemented with 5% sheep blood and were interpreted 2 days after incubation. The isolate was susceptible to doxycycline (MIC 0.25 $\mu g/mL$; susceptible MICs <2 $\mu g/mL$) but not susceptible to trimethoprim/ sulfamethoxazole (MIC 1/19 µg/ mL; susceptible MICs $<0.5 \mu g/mL$) (5,6). MIC values of tigecycline and gentamicin were 0.125 µg/mL and 2.0 µg/mL, respectively. A serum sample for examination of Brucella antibody by Rose Bengal test using B. abortus antigen (VLA Scientific, Winchester, UK) collected 12 days after fever onset was positive.

Antimicrobial drug treatment was changed to doxycycline and gentamicin. However, low-grade fever and low back pain developed 2 days after administration of gentamicin. The back pain was attributed to muscle pain and was almost completely relieved by 2-day treatment with nonsteroidal antiinflammatory а drug. Whole-body gallium scan and spine magnetic resonance imaging suggested osteomyelitis and epidural abscess over the third and fourth lumbar spines. The patient was treated with doxycycline for 6 weeks. A liver function test 2 weeks after admission showed values within reference limits.

Cases of human brucellosis and animal sources of Brucella spp. have been reported from Algeria and Morocco (7–10). The most common laboratory findings in patients with brucellosis are high C-reactive protein levels and anemia (3,4). The patient had high C-reactive protein levels but procalcitonin values within reference limits at admission. Hepatic involvement of brucellosis has been reported to range from 2% to 25% (3). The patient also had acute anicteric hepatitis, and serologic test results were negative for all hepatotropic viruses. The isolate from this patient exhibited high MICs for trimethoprim/ sulfamethoxazole, a finding rarely reported (5,6). The low MIC value of tigecycline suggests the potential role of this agent for the treatment of brucellosis.

This report confirms brucellosis in Taiwan. Brucellosis could become an emerging problem in this country, particularly given the frequency of travel between Taiwan and areas where brucellosis is endemic.

Yu-Chung Chuang, Szu-Chi Chen, Jung-Jung Mu, Hsiu-Ying Lin, Chih-Hsin Chang, Wei-Shiung Yang, and Po-Ren Hsueh Author affiliations: National Taiwan University Hospital, Taipei, Taiwan (Y.-C. Chuang, S.-C. Chen, H.-Y. Lin, C.-H. Chang, W.-S. Yang, P.-R. Hsueh); and Centers for Disease Control, Taipei (J.-J. Mu)

DOI: http://dx.doi.org/10.3201/eid1712.110739

References

- Pappas G, Papadimitriou P, Akritidis N, Christou L, Tsianos EV. The new global map of human brucellosis. Lancet Infect Dis. 2006;6:91–9. doi:10.1016/S1473-3099(06)70382-6
- Aggad H, Boukraa L. Prevalence of bovine and human brucellosis in western Algeria: comparison of screening tests. East Mediterr Health J. 2006;12:119–28.
- Gwida M, Al Dahouk S, Melzer F, Rösler U, Neubauer H, Tomaso H. Brucellosis—regionally emerging zoonotic disease? Croat Med J. 2010;51:289–95. doi:10.3325/cmj.2010.51.289
- Guenifi W, Rais M, Gasmi A, Ouyahia A, Boukhrissa H, Mechakra S, et al. Neurobrucellosis: description of 5 cases in Setif Hospital, Algeria [in French]. Med Trop (Mars). 2010;70:309–10.
- Ennibi K, Rabhi M, Chemsi M, Elouennass M, Chaari J, Toloune F. Nodular liver lesions with fever in a Moroccan man: hepatic brucelloma [in French]. Med Trop (Mars). 2009;69:509–11.
- Memish Z, Mah MW, Al Mahmoud S, Al Shaalan M, Khan MY. *Brucella* bacteraemia: clinical and laboratory observations in 160 patients. J Infect. 2000;40:59–63. doi:10.1053/jinf.1999.0586
- Buzgan T, Karahocagil MK, Irmak H, Baran AI, Karsen H, Evirgen O, et al. Clinical manifestations and complications in 1028 cases of brucellosis: a retrospective evaluation and review of the literature. Int J Infect Dis. 2010;14:e469–78. doi:10.1016/j. ijid.2009.06.031
- Marianelli C, Graziani C, Santangelo C, Xibilia MT, Imbriani A, Amato R, et al. Molecular epidemiological and antibiotic susceptibility characterization of *Brucella* isolates from humans in Sicily, Italy. J Clin Microbiol. 2007;45:2923–8. doi:10.1128/ JCM.00822-07
- Maves RC, Castillo R, Guillen A, Espinosa B, Meza R, Espinoza N, et al. Antimicrobial susceptibility of *Brucella melitensis* isolates in Peru. Antimicrob Agents Chemother. 2011;55:1279–81. doi:10.1128/AAC.00979-10
- Franco MP, Mulder M, Gilman RH, Smits HL. Human brucellosis. Lancet Infect Dis. 2007;7:775–86. doi:10.1016/S1473-3099(07)70286-4

Address for correspondence: Po-Ren Hsueh, Departments of Laboratory Medicine and Internal Medicine, National Taiwan University, No 7, Chung-Shan South Rd, 100 Taipei, Taiwan; email: hsporen@ntu.edu.tw

Hemoptysis Associated with Leptospirosis Acquired in Hawaii, USA

To the Editor: Severe pulmonary hemorrhagic syndrome (SPHS) is a serious complication of Leptospira infection, a globally widespread bacterial zoonosis that is increasing in incidence in tropical and subtropical regions. Despite decreasing endemicity of leptospirosis in industrialized regions, the disease is reemerging in travelers and recreationalists. Leptospirosis is an appreciable attributable cause of travel-related infections (typically associated with waterborne activities), and the incidence of travel-related leptospirosis is proportionally higher than that for endemic leptospirosis. Disease risk epidemiology has shifted concomitantly from occupational recreational in industrialized to countries (1-3). Risk factors include urbanization, climatic changes, and agricultural practices (1-3).

Clinical features of leptospirosis range from asymptomatic infections and undifferentiated febrile syndromes to multiorgan dysfunction and death. Weil syndrome (i.e., severe leptospirosis) is characterized by renal and hepatic dysfunction, hyperbilirubinemia (disproportionate to transaminase elevation), and hemorrhage (pulmonary, gastrointestinal, or intracranial). Pulmonary