

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 a nonsteroidal antiinflammatory 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)

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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 to recreational in industrialized 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

involvement predicts poor clinical outcome: the case-fatality rate for persons with SPHS is >50% (4–6). Most US leptospirosis cases are reported from Hawaii, where the annual incidence is 1.63 cases/100,000 person-years (1). Leptospirosis is endemic to Hawaii; however, SPHS is uncommonly reported (7).

We treated a 21-year-old active-duty Navy sailor for SPHS after he had a 5-day port visit in Hawaii, during which he went cliff-diving in Maunawili Falls. Afterwards, he returned to California and 2 days later sought medical attention in an outpatient clinic. Pharyngitis was diagnosed and azithromycin prescribed.

Two days later, he was hospitalized with fever, chills, pharyngitis, dyspnea, nonproductive cough, headache, myalgias, hemoptysis, epistaxis, diarrhea, nausea, emesis, meningismus, and a lower-extremity rash. Vital signs included temperature 38.3°C, pulse 132 beats/min, blood pressure 128/72 mm Hg, and oxygen saturation 98% on room air. Physical examination noted conjunctival suffusion, epistaxis, posterior cervical and inguinal lymphadenopathy, bilaterally diminished breath sounds, rhonchi and crackles, bloody cough, tachycardia, hepatosplenomegaly, and a macular rash over the lower extremities. Laboratory studies were noteworthy for reference range leukocyte count, hemoglobin (11.8 g/dL), platelets ($102 \times 10^3/\text{mm}^3$), creatine phosphokinase (1,719 IU/L), sodium (128 mmol/L), bicarbonate (23 mmol/L), blood-urea-nitrogen (29 mg/dL), creatinine (2.2 mg/dL), aspartate aminotransferase (171 U/L), alanine aminotransferase (147 U/L), bilirubin (1.9 mg/dL), and urinalysis (7 erythrocytes and 9 leukocytes/high-power field). Chest radiography showed multilobar bilateral opacities, and cerebrospinal fluid (CSF) showed mild pleocytosis.

The patient received intravenous acyclovir, ceftriaxone, and vancomycin and continued azithromycin.

At hospital admission, the patient experienced respiratory decompensation requiring endotracheal intubation and mechanical ventilation. Results of blood, urine, and CSF cultures and CFS PCR (herpes simplex virus and enterovirus) remained negative at 48 hours, prompting discontinuation of vancomycin and acyclovir. Serologic test results for HIV, dengue fever virus, mycoplasma, and *Chlamydomytila* and *Rickettsia* species were negative. Nasopharyngeal influenza PCR, *Streptococcus pneumoniae* and *Legionella* spp. urinary antigen test results, and hepatitis panel results were negative. *Leptospira* spp. test results by culture, PCR, and serologic testing (ELISA and microscopic agglutination testing) were negative.

Given an elevated suspicion for leptospirosis, ceftriaxone and azithromycin were continued through hospital day 7. The patient rapidly improved, was extubated after 48 hours, and was discharged on hospital day 7 with a 7-day course of oral doxycycline. A convalescent-phase serum sample had a titer of 1,600 against *L. interrogans* serovar Copenhageni, as determined by microscopic agglutination testing.

SPHS is associated with infection with *L. interrogans* serovars Copenhageni and Icterohaemorrhagiae (8), and the syndrome has been identified in diverse settings, including the Andaman Islands. Recent outbreaks have occurred in Nicaragua and Brazil (4,5). SPHS pathogenesis remains poorly understood. In animal models and human autopsy studies, immunoglobulin and complement are deposited along alveolar septa without a clear cause-and-effect relationship (9). Bacterial virulence factors are postulated but unproven. Leptospire induce endothelial

activation and pulmonary endothelial and epithelial injury (possibly by immune-complex deposition and/or autoimmune mechanisms) (9). Pulmonary histopathology demonstrates a paucity of leptospire, and antigen levels do not correlate with injury severity (9). Steroids, intravenous immunoglobulin, and plasma exchange are of unproven benefit but have been reported to be useful (9). Genetically determined responses include associations with human leukocyte antigen-DQ6 and hyperactive Toll-like receptor 4-dependent immunity.

Diagnosis of leptospirosis may have been delayed for this patient because of early empiric azithromycin administration. Azithromycin is increasingly recognized as a potentially effective treatment that is comparable or superior to doxycycline (10) and thus warrants testing in human trials. Given the paucity of SPHS in leptospirosis case reports from Hawaii, potential sentinel cases may be harbingers of more virulent disease expression. A potential parallel is the emergence of SPHS in Salvador, Brazil, in 2003. No cases were identified before 2003, but 47 cases and a 75% case-fatality rate were identified during 2003–2005 (4,5). The entrenched active surveillance and physician awareness of SPHS in neighboring Brazilian cities suggests it is unlikely that this observation stemmed from prior underrecognition of disease; instead, it suggests de novo emergence.

Clinicians should consider leptospirosis (SPHS) in patients with acute fever accompanied by hemoptysis after travel to Hawaii, and leptospirosis should be suspected in any traveler with undifferentiated febrile illness, especially those reporting water exposures (2). Vigilant national surveillance is needed to determine further emergence of SPHS in Hawaii.

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**Christopher A. Duplessis,
Marvin J. Sklar, Ryan C. Maves,
Anne Spichler, Braden Hale,
Mark Johnson, Mary Bavaro,
and Joseph M. Vinetz**

Author affiliations: Naval Medical Center, San Diego, California, USA (C.A. Duplessis, M.J. Sklar, R.C. Maves, M. Johnson, M. Bavaro); University of California, La Jolla, California, USA (A. Spichler, J.M. Vinetz); and Naval Health Research Center, San Diego (B. Hale)

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Address for correspondence: Christopher A. Duplessis, Naval Medical Center San Diego, 34800 Bob Wilson Dr, Suite 5, San Diego, CA 92134-1005, USA; email: chris.duplessis@waldenu.edu

Salmonella enterica in Pinnipeds, Chile

To the Editor: Several wildlife-associated zoonotic agents have played a major role in the emergence of diseases in humans (1). However, diseases can also emerge in wildlife as a result of human activities, such as contamination of the marine environment and its fauna by the disposal of nontreated human sewage. *Salmonella enterica* is among the agents identified as causing infection in various marine birds and mammals, including pinnipeds, from different geographic regions (2–4).

The objective of our study was to determine whether *S. enterica* infection occurs in pinnipeds from the Chilean coast. During August–December 2010, we obtained samples from 13 South American sea lions

(*Otaria flavescens*) that the sanitary authority found malnourished and stranded at the northern Chilean beaches of Antofagasta (23°40'S, 70°24'W) and Los Vilos (31°54'S, 71°30'W) (Table). The pinnipeds showed no clinical signs or symptoms of disease; however, rectal swab samples were obtained during their stay for rehabilitation at the Buin Marino facilities (Santiago, Chile). After the animals recovered, they were released to their original habitat.

The swab samples were placed in Cary-Blair transport medium (COPAN, Murrieta, CA, USA) for shipment to the laboratory (Laboratory of Infectious Diseases, University of Chile, Santiago). To isolate bacteria, we placed the swab samples into 5 mL of buffered peptone water (Difco APT broth; Becton Dickinson, Franklin Lakes, NJ, USA), incubated them for 24 h at 42°C with agitation, and then aliquots of the suspension were transferred into the following media: modified semisolid Rappaport-Vassiliadis basal medium (Oxoid, São Paulo, Brazil) with novobiocin (20 µg/mL), selenite cysteine broth base (Oxoid), and xylose lysine desoxycholate agar (Difco XLD; Becton Dickinson). After the aliquots were incubated at 37°C for 24–48 h, we identified suspected colonies by using biochemical tests and *invA* gene detection by PCR (5). Results showed that 2 of the 13 animals were infected with *S. enterica* strains, which were serotyped as *S. enterica* serotype Newport and *S. enterica* serotype Havana (Table), according to the Kauffmann-White scheme (6). Testing showed that the strains were susceptible to the following antimicrobial drugs: ampicillin, chloramphenicol, tetracycline, amoxicillin/clavulanic acid, trimethoprim/sulfamethoxazole, cefotaxime, nalidixic acid, nitrofurantoin, ciprofloxacin, ceftazidime, and cefoxitin (7).

Our results confirm *S. enterica* infection in pinnipeds from Chile and,