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**LETTERS**

**Ritual Slaughter as Overlooked Risk Factor for Brucellosis**

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To the Editor: The current rates of animal and human brucellosis in southern Israel are unacceptably high (1). Unsupervised livestock rearing, smuggling of herds, and the dissolution of Israel’s “test, slaughter, and compensate” program for small ruminants in 1997 have generated a large, uncontrolled animal reservoir in the region. The Bedouin Arab inhabitants who live in close proximity to herds and consume unpasteurized dairy products are disproportionately affected. Outbreaks from parts of Israel that were previously free of brucellosis are reported with increasing frequency. Moreover, 2 decades after supposed elimination of bovine brucellosis, a cattle herd adjacent to Bedouin grazing areas was found to be highly infected with \textit{Brucella melitensis} (2).

We report 5 cases of severe brucellosis in patients from a community whose members typically do not raise herds and do not consume unpasteurized dairy products. The patients were Ethiopian-born Jews who exhibited fever and either respiratory signs or radiologic evidence of new pulmonary findings (Table). The true nature of the infection only became evident when \textit{B. melitensis} was identified from blood cultures. Directed questioning revealed that all patients participated in ceremonial slaughter of sheep that were purchased from Bedouin owners in southern Israel.

The diagnostic pitfalls encountered by the medical staff are exemplified in the case of patient 1 (Table). In May 2014, this 68-year-old man was admitted to a hospital in southern Israel, with a 14-day history of fever, cough, and night sweats. His medical history was notable for asthma and previously treated pulmonary tuberculosis (TB). Computed tomography scan of his chest showed a new 1.5 cm\(^3\) apical lung lesion with irregular borders. Laboratory evaluations were negative for rickettsiae, \textit{Coxiella burnetii}, HIV, \textit{Plasmodium spp.}, and \textit{Mycobacterium tuberculosis}. Four days after admission, gram-negative cocccobacilli grew from his blood culture. \textit{Brucella} IgM titer was 1:1,920. The patient received appropriate treatment for 6 weeks (Table) and recovered. \textit{Brucella} IgM titers dropped to 1:40. Four months later, he returned to the infectious diseases clinic exhibiting fever, chills, and clinical and sonographic evidence of epididymo-orchitis. \textit{Brucella} IgM titers rose to 1:1,240. Treatment was resumed for 3 months. In addition, a thoracoscopic biopsy of the lung lesion was performed to rule out malignancy. Pathologic examination of the biopsy specimen revealed a focus of fibrosis, with giant cells surrounded by lymphocytes (online Technical Appendix Figure, http://wwwnc.cdc.gov/EID/article/22/4/15-1192-Techapp1.pdf). After treatment, the patient’s symptoms resolved and titers returned to low levels.

The long symptom-to-diagnosis interval (range 21–97 days) for patients in this report is alarming (Table). Treatment delays are associated with increased focal complications and relapse rates (3). Further, high case-fatality rates, allegedly due to low physician awareness, were reported in a largely immigrant cohort of brucellosis patients in Germany (4).

Several circumstances might have led to the failure to include brucellosis in the initial differential diagnosis for these patients, even in a disease-endemic region. First, we can assume that physicians are unfamiliar with the ceremonial slaughter central to the celebrations of Ethiopian Jews.
The tradition includes slaughtering, skinning, and eviscerating a sheep, followed by mincing of the sheep meat. This venerated ritual is performed by trained members of the Ethiopian community and supervised by the spiritual leader, the Kes (5). Second, patients were consistently reluctant to disclose their participation in ceremonial slaughter to medical staff. Third, the managing physicians considered differential diagnoses for febrile respiratory illness in line with the patients’ Ethiopian origins: reactivation of TB or suspected osteomyelitis C6; increased uptake bone scan.

All patients in our study had clinical or radiologic evidence of lung involvement. Causality between exposure to Brucella-infected aerosols and pulmonary manifestations of brucellosis has been demonstrated in animal models. After an aerosol challenge with B. melitensis, animal lungs have shown perivascular inflammation as well as microgranulomas (7). In a study of hunters infected with B. suis, in which 38% had respiratory symptoms, aerosol spread or conjunctival inoculation was considered the most likely route of infection (3).

Aerosol exposure during slaughter could be linked to the pulmonary manifestations of brucellosis observed in these patients. The granulomatous changes in the lung biopsy specimens of patient 1 are typical of lung involvement in brucellosis (8). The patients in this report did not use protective gear during contact with animal parts, which inevitably increased their risk for infection through direct or aerosol contact (9).

This report illustrates an unsuspected mode of brucellosis transmission in an area with soaring brucellosis.

<table>
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<th>Pt no.</th>
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<tr>
<td>1</td>
<td>68/M</td>
<td>Cervical neck pain; cough; night sweats; 38°C</td>
<td>Hb, 11.9 mg/dL; leukocytes, 16.3 x 10^9 μL; AST, 63 U/L; ALT, 118 U/L</td>
<td>CT: apical lung finding, new onset</td>
<td>Asthma exacerbation; lung malignancy</td>
<td>21</td>
<td>Focal lung lesion; relapse: epididymo-orchitis; suspected osteomyelitis C6; increased uptake bone scan</td>
<td>STR/2 wk, dox + cipro/6 wk; relapse: rising Brucella titer + epididymitis; same 3 drugs/12 wk; recovery</td>
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<td>1st admission: fever; productive cough</td>
<td>Hb, 14.9 mg/dL; leukocytes, 12 x 10^3 cells/μL; platelets, 136 x 10^3 μL; AST, 61 U/L; ALT, 47 U/L</td>
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<td>Asthma exacerbation; bronchitis</td>
<td>NA</td>
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<td></td>
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<td>Fever; prolonged headache</td>
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<td>TB; cryptococcal meningitis</td>
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<td>49/F</td>
<td>Cough; fever</td>
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<td>TB; infective endocarditis caused by Actinobacillus ureae</td>
<td>90</td>
<td>Genta/2 wk; dox + rif/7 wk; recovery</td>
<td></td>
</tr>
</tbody>
</table>

*Pt, patient; lab, laboratory; Hb, hemoglobin; leukocyte: leukocytes; AST, aspartate aminotransferase; ALT, alanine aminotransferase; CT, computed tomographic scan; C6, cervical vertebra 6; STR, streptomycin; dox, doxycycline; cipro, ciprofloxacin; NA, not available; IV, intravenous; Na, sodium; RUL, right upper lobe; TB, tuberculosis; ESR, erythrocyte sedimentation rate; MRI, magnetic resonance imaging; genta, gentamicin; cotrim, cotrimoxazole; CRP, C-reactive protein; rx, prescription; rif, rifampin.*
rates: transmission from infected animals to persons clandestinely engaging in ritual slaughter; specifically, an Ethiopian Jewish community. Physicians in countries receiving immigrants should be aware of ceremonial practices that place patients at risk for zoonoses. The severe respiratory manifestations that ensued following aerosol exposure to animal blood or secretions suggest that brucellosis with pulmonary involvement after inhalation of Brucella-infected aerosols might be more common than previously documented.

References


Follow-up of Ebola Patient, 2014–2015

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To the Editor: The 2014–2015 epidemic of Ebola virus disease (EVD) in West Africa affected 23,666 persons and caused 14,603 deaths (1). The World Health Organization (WHO) declared the epidemic a public health emergency (2). Although Ebola virus is transmitted by unprotected physical contact with infected persons, published reports about which body fluids are infected or the risk for fomite transmission are few (3). For most cases, virus was detected by reverse transcription PCR (RT-PCR) of clinical (saliva, feces, semen, breast milk, tears, nasal blood, skin swab) and environmental specimens (4). Earlier reports of the follow-up of recovered patients stated that viral RNA was detected by RT-PCR for up to 33 days in vaginal, rectal, and conjunctival swab samples from 1 patient and up to 101 days in seminal fluid from 4 patients. Infectious virus was detected in 1 seminal fluid sample 82 days after disease onset (4,5).

Attendees at the Eighth Meeting of the WHO Advisory Group on the EVD Response (1) discussed potential risk factors, including hidden chains of transmission and sexual transmission, and determined the following criteria. A country can declare “interruption of transmission” when 42 days have elapsed since the last diagnosis of a case. A country can declare that the “outbreak has stopped” when test results from the last case are negative twice or after another 90-day interval. For determining a cutoff for finally declaring the strategy and criteria for elimination, extensive follow-up on infectivity of semen in Ebola survivors is needed.

We report follow-up of a man who recovered from EVD and was monitored for 165 days after he was declared Ebola-free. The 26-year-old man from India returned to New Delhi, India, from Liberia on November 10, 2014, with a certificate from the government of Liberia stating that he was “cured” of Ebola. Because EVD is considered an exotic disease in India, he was placed in isolation at the Airport Health Organization quarantine center at Indira Gandhi International Airport, New Delhi (6). Serum and semen samples were collected and sent to the National Centre for Disease Control (NCDC), New Delhi, and the National Institute of Virology (NIV), Pune, India. The serum was negative by

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