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Review

Boar hunting and brucellosis caused by *Brucella suis*

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ABSTRACT

Brucellosis remains as a significant public health concern in many areas where the infection persists in domestic hosts (i.e. goats, cattle, and domestic swine) with subsequent risk of transmission to human populations. Brucellosis caused *by B. suis* remains an important threat to human populations in many countries exposed to domestic and feral swine. In the U.S., swine brucellosis has been under control for many years. Meanwhile, it is a widespread infection among feral swine, particularly in the Southeastern United States; and exposure to infected animals pose a growing threat to humans. We present the case of a 31-year male hunter who six weeks after a knife injury to his hand while field dressing a wild boar, developed a febrile illness associated with hematologic abnormalities and splenic abscesses caused by *Brucella suis* infection.

1. Introduction

Brucellosis is a zoonosis that has affected human populations for millennia and its cycle of transmission was further amplified after the onset of animal domestication practices [1]. Despite some improvement in controlling the disease in many countries, there are many regions where brucellosis continues to impose an important disease burden in terms of cause morbidity and clinical sequelae [2–6]. In addition, this infection is associated with a high risk of relapse imposing substantial healthcare costs and utilization [8–10]. Worldwide, most cases of brucellosis are caused *by B. melitensis*, *B. abortus,* followed by *B. suis, and B. canis* [11,12].

In the U.S., control efforts instituted after World War II reduced human cases by the use of a live attenuated vaccine in domestic animals and by the widespread pasteurization of milk. In spite of these efforts, many cases continued to occur among those handling animals including abattoir workers, shepherds, veterinarians, dairy-industry professionals; and via aerosolized bacteria among microbiology laboratory personnel [13]. In the last few decades, a final push has reduced cases of brucellosis due to *B. melitensis* and *B. abortus* in the U.S. to those occurring only among travelers and immigrants from highly endemic areas [14].

Historically, human brucellosis caused by *Brucella suis* was responsible for a large number of human cases of brucellosis in the U.S., particularly when swine production intensified in the second part of the twentieth Century. Due to rigorous control interventions, swine brucellosis has nearly been eliminated in domestic herds [14]. Nonetheless, free-living swine remain an important sylvatic reservoir [15]; and with the geographic spread of feral pig among many States, there is an increasing threat of occupational and recreational exposure to brucellosis [16,17]. Herein, we present a case of *Brucella suis* infection in a wild boar hunter. This is the second confirmed case of *Brucella suis* infection seen at our institution in Southwest Georgia within the last 4 years. In both cases, the risk factor for acquiring this infection was a skin injury involving a cutting instrument used during field dressing of wild boars (also called feral pigs, feral hogs, or wild pigs). In addition to these confirmed clinical cases, we have recently participated in providing post-exposure prophylaxis to veterinarians, dog-owners, and kennels personnel exposed to *B. suis*-infected hunting dogs [18].

1.1. Case presentation

A 31-year old male previously healthy presented with a 2-week history of fever, night sweats, malaise, and decreased appetite. He is

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married and works as a bus driver and lives in Sylvester, GA (Southwest GA, United States). He had no recent history of travel outside the State of Georgia, no recent exposure to ticks, but reports recreational hunting of wild boars. Approximately, six weeks prior to his onset of symptoms reported suffering a small cut with a razor blade used during the field dressing of a wild boar. At that time, he was not wearing gloves or any other protective equipment. On admission to the hospital, his vital signs demonstrated a temperature of 101.8 °F, pulse rate of 90, respiratory rate of 17, and blood pressure of 130/73 mm/Hg. His physical examination revealed splenomegaly. His laboratories showed a White Blood Cell count of 3.4 K/mm^3, Hb 8.9 g/dL, hematocrit of 26.5%, and a platelet count of 98,000 K/mm^3. His liver enzymes showed an AST of 115 U/L, ALT of 83 U/L, and alkaline phosphatase of 74 U/L. HIV testing was reported negative. Computed tomography of the abdomen demonstrated multiple small hypodensities in the spleen consistent with small abscesses and 2 larger subcapsular ones (Fig. 1). Two sets of blood cultures performed at the time of admission revealed the presence of small gram-negative cocobacilli (Fig. 2). Bacterial isolates were submitted to the Zoonoses and Select Agent Laboratory, Centers for Disease Control and Prevention, Atlanta, GA. *Brucella suis* was identified by performing the following tests: gel formation negative, positive hydrogen sulfide production, AMOS PCR, urease production with positive <5 min (Christensens Method), negative lysis by Phage Tbilisi RTD but positive lysis by Phage Tbilisi RTD x 10^4. Brucella LRN was positive by PCR BRU 1 25, BRU2 23, and BRU 3 25. Serology for *Brucella* (IgG) was also reported positive at a titer of >1:1280.

After the initiation of antimicrobial therapy with doxycycline (200 mg loading dose, followed by 100 mg PO BID) and rifampin (600 mg daily), his fever curve, hematologic parameters, and liver enzyme values returned to normal limits within 10 days. Our patient was discharged to complete a 60-day course of this antimicrobial drug-regimen. A repeat computed tomography of the abdomen pelvis performed at the time of completion of his antimicrobial therapy revealed complete resolution of the splenic abscesses.

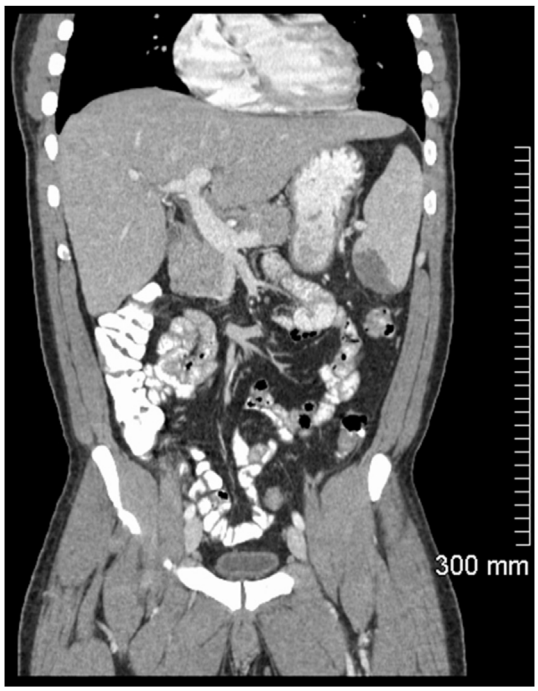

Fig. 1. Computed tomography of the abdomen demonstrating a large subcapsular splenic abscesses.

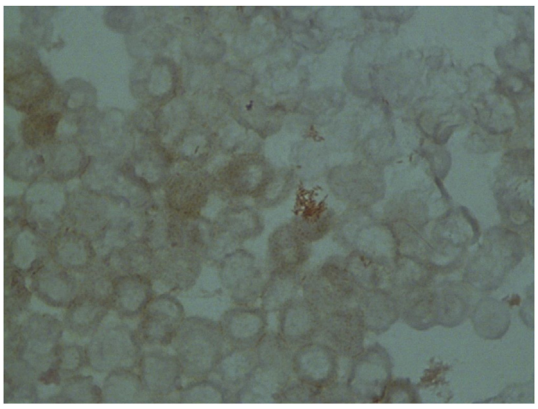

Fig. 2. Gram staining of peripheral blood demonstrating small gram negative coccobacilli.

Approximately, twelve weeks later, he presented with a new three-day episode of fever, night sweats, and malaise. *Brucella suis* was isolated once again in blood cultures, but this time he did not have any evidence of anemia, thrombocytopenia or splenic abscesses. Patient was successfully treated with gentamicin intravenously for 7 days and doxycycline for 45 days with no evidence of any further relapses (clinical improvement with resolution of fever, and two surveillance blood cultures reported with no growth obtained at one and three months after completion of antimicrobial therapy).

2. Discussion

2.1. Brucellosis is the leading bacterial zoonosis worldwide

Brucellosis caused by *Brucella melitensis, Brucella abortus, Brucella suis* remain as a significant public health concern in many areas where the infection persists in domestic hosts (i.e. goats, cattle, and domestic swine) [5–7]. Most of the burden of diseases currently takes place in the Mediterranean region, the Indian subcontinent, the Arabian Peninsula, Central and South America, and Mexico [5,7].

The *Brucella* genus contains a group of small gram-negative cocobacilli that belong to the alpha-2 subdivision of the Proteobacteria [7]. These are gram-negative facultative intracellular pathogens that are non-spore forming, non-motile that may infect domestic or wildlife through a cycle of transmission involving direct contact or by the sexual route [6,7,9–11]. Human infection occurs by: a) direct contact with infected animal parts allowing the entrance of the organism through skin abrasions or mucosal membranes; b) consumption of infected and unpasteurized animal-milk products; or c) inhalation of aerosolized bacteria. The agents of brucellosis produce acute and chronic granulomatous disease through their intracellular ability to survive inside macrophages and promote its replication through the creation of membrane-bound compartments that inhibits fusion with lysosomes [7].

Brucella spp are described by their epidemiology and predilection for specific domestic hosts (Table 1). Brucellosis caused by any of the pathogenic species manifests either as an acute febrile illness in approximately 50% of the cases (incubation period averaging two to six weeks) or with an insidious onset over a period of weeks or months from the initial infection [5]. Clinical manifestations include fever, night sweats, malaise, weight loss, arthralgia, headache, or back pain. In some patient, when brucellosis goes