CASE REPORTS

An Unusual Wound Infection Due to Acinetobacter junii on the Island of Oahu

A Case Report

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ABSTRACT

The genus Acinetobacter has long been associated with war wounds. Indeed, A baumannii was responsible for so many infected wounds during Operation Iraqi Freedom that it was given the nickname “Iraqibacter.” Therefore, it is important to monitor the occurrence and spread of Acinetobacter species in military populations and to identify new or unusual sources of infection. A junii is an infrequently reported human pathogen. Here, we report a case of a slow-healing wound infection with A junii in a woman on the island of Oahu. This case highlights the pathogenic potential of this organism and the need for proper wound care when dealing with slow-healing wounds of unknown etiology. It also underscores the need for identifying species of Acinetobacter that are not A baumannii to better understand the epidemiology of slow-healing wound infections.

KEYWORDS: Acinetobacter junii; emerging infection; Hawaii; Oahu; wound

Case Report

A 50-year-old woman was treated in the emergency department (ED) for a right shin injury sustained during a fall on a treadmill running deck. A preliminary medical history revealed the injury had occurred 5 days before the ED visit and that there was no obvious history of malignancy or other condition that would predispose the patient to wound infection. A 4-cm ulceration was noted during the physical examination and an empirical diagnosis of cellulitis was made. No culture or sensitivity studies were taken during the initial examination. The patient was treated with a broad-spectrum cephalosporin (cefalexin 1000mg twice daily) and a lincosamide for 14 days after the initiation of ciprofloxacin therapy.

The next examination occurred in the general surgery clinic 4 days after the ED visit. At this time, the wound was described as a 2.5cm × 3.5cm ulceration with a central eschar. It was treated with periodic dressing changes and debridement over the next 50 days until the patient was referred to the vascular limb salvage clinic (VLSC) for the treatment of a nonhealing wound. Observations undertaken at the VLSC revealed a 0.5m × 0.3cm ulceration and a rash extending approximately 5cm beyond the wound margin. There were palpable distal pulses and no evidence of edema. A radiologic examination showed no evidence of osteomyelitis or any acute osseous abnormality, although there was evidence for the presence of intra-articular bodies, which may explain some of the pain reported by the patient. Treatment consisted of 0.1% triamcinolone for contact dermatitis, medical-grade honey, and a 4 in. × 4 in. gauze stockinet. Furthermore, no additional adhesives were applied to the skin (to prevent exacerbation of the contact dermatitis). At a follow-up appointment in the VLSC 6 days later, it was noted that although the rash had resolved, there was continued pain and discomfort at the wound site, which was now producing a honey-colored exudate. Samples were collected for culture and sensitivity studies. Treatment with medical-grade honey was discontinued and a silver-coated dressing treatment was begun.

The initial Gram stain of the material submitted for culture and sensitivity analyses indicated the presence of polymorphonuclear lymphocytes, mononuclear cells, and gram-negative rods. All anaerobic cultures were negative; however, the aerobic culture demonstrated the presence of A junii and a coagulase-negative Staphylococcus spp. (an established skin commensal). Antimicrobial sensitivity studies of the A junii isolate indicated sensitivity to gentamicin, trimethoprim, sulfamethoxazole, ciprofloxacin, ampicillin, sulbactam, and cefepime. On the basis of these results, the patient was prescribed 500mg of ciprofloxacin for 10 days to be taken twice daily, and wound care was continued to include immobilization of the shin with a boot that controlled ankle movement. The wound resolved after 11 days after the initiation of ciprofloxacin therapy.

Discussion

The genus Acinetobacter consists of aerobic gram-negative bacilli characterized by the production of catalase, a lack of cytochrome oxidase, the inability to reduce nitrate to nitrite, and the inability to cleave indole from tryptophan.1 The first description of this genus was made in 1911 by Beijerinck, who isolated a representative strain from soil.2 The most common pathogenic species in this genus are A baumannii, A nosocomialis, A pittii, and A calcoaceticus.3 Although the mechanisms of Acinetobacter virulence have not been well described,
Acinetobacter species are common environmental contaminants. This is partially due to the ability of these organisms to survive for a long time on dry environmental surfaces. Significantly, Acinetobacter species are often found as inhabiting the outer surface of human skin.

Acinetobacter junii was first described as a distinct species in 1986. Because of biochemical similarities between A junii and the other members of the genus Acinetobacter, molecular methods are often necessary for accurate identification. Indeed, the results of molecular analyses in the late 1980s were used to determine that a previously described strain of Acinetobacter known as A grimontii was synonymous with A junii. Recently, mass spectrometry has been introduced as an alternative to the polymerase chain reaction and DNA sequencing for discriminating among phenotypically similar species of Acinetobacter and for the identification of A junii.

The pathogenic potential of A junii was demonstrated in 1997 when septicemia developed in six infants being treated in a neonatal unit. In those cases, a total of 12 isolates were recovered and identified as A junii by a combination of molecular and phenotypic techniques. All six infants were successfully treated with either ciprofloxacin alone or with a combination of ciprofloxacin and gentamicin. Although A junii is infrequently identified as a cause of human disease, it is typically found in immunocompromised patients or in patients with hematologic malignancies. Acinetobacter junii is very rarely isolated from wound infections. However, a case of cellulitis caused by a community-acquired A junii infection was reported in 2012 and a community-acquired strain was recovered from a corneal ulcer in 2000.

A closely related organism, A baumannii, gained notoriety during Operation Iraqi Freedom as a significant source of wound infection among troops injured during combat operations throughout the Middle East. Increases in wound infections tend to disrupt the normal operation of military medical centers and are an impediment to patient care. Indeed, an outbreak of A baumannii in the military health care system between 2003 and 2004 prompted a multisite investigation into the sources of infection. Such investigations consume human and material resources and illustrate the need for maintaining a high degree of vigilance in the clinical setting so new or unusual sources of infection can be identified and eliminated quickly.

In the present case, the infection was most likely acquired in the community at the time of injury. This case is significant because it represents, to our knowledge, the first case of a wound infection caused by A junii to be reported from the island of Oahu and because it demonstrates the importance of proper wound care combined with the rational application of antibiotic therapy in the treatment and resolution of nonhealing or slow-healing wounds of unusual etiology.

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The views expressed herein are those of the author and do not reflect the position of the US Military Academy, the Department of the Army, or the Department of Defense.

The authors have nothing to disclose.

Author Contributions
JG identified and treated the patient. JB and MW drafted the document and performed the literature review.

References