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LETTER TO THE EDITOR

First case report of *Nocardia brasiliensis* infection causing necrotizing fasciitis in an immunocompetent patient

To the Editor,

We have read with interest the article by Lai et al[1] in the *Journal of Microbiology, Immunology, and Infection*, in which the authors reported on fetal empyema thoracis caused by imipenem-resistant *Nocardia abscessus*. We here describe another serious infection by *Nocardia brasiliensis* that initially presented with cellulitis but progressed to necrotizing fasciitis requiring surgical intervention to cure.

A 64-year-old previously healthy woman without systemic diseases sought medical attention because of a chronic nonhealing wound on her left lower leg. Two weeks earlier, she met with a traffic accident whereby she incurred the abrasive wound on her left leg. The initial wound did not heal but instead progressed to an ulcerative drainage lesion. Physical examination disclosed a tender nodular lesion measuring 2.0 cm in diameter, with shallow ulceration and pustules in the center (Figure 1A). Laboratory findings were as follows: white blood cells, 5530/mm^3 (56.3% segmented, 34.4% lymphocyte, 6.0% monocyte, 3.1% eosinophil, and 0.2% basophil). The blood chemistry profile of the patient was as follows: aspartate aminotransferase, 27 IU/L; alanine aminotransferase, 21 IU/L; blood urea nitrogen, 16.5 mg/dL; creatinine, 0.6 mg/dL; and C-reactive protein, 2.4 mg/dL. She was admitted for cellulitis, which was empirically treated with intravenous cefazolin initially. A wound biopsy specimen was obtained for culture. After 2 days of incubation, the culture yielded pure aerobic bacteria, and further 16S ribosomal RNA (16S rRNA) gene sequence analysis confirmed the organism as *N. brasiliensis*. The antibiotics were then shifted to trimethoprim–sulfamethoxazole. Four days after admission, the

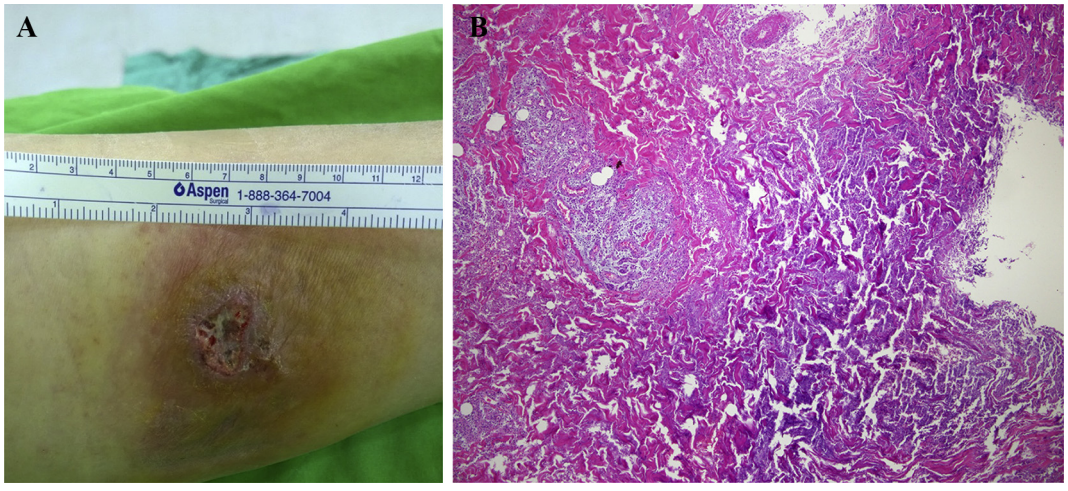

Figure 1. (A) Erythematous nodular ulcerative lesion on the left leg. (B) Representative pathological finding of inflammatory cells infiltrating the necrotic tissue and fragmented fascia.

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patient developed progressive erythema and swelling over her left lower limb with intense pain. Surgical debridement was then performed under the impression of necrotizing fasciitis. The intraoperative findings, including fascia necrosis and edema of the fascial layer, were suggestive of necrotizing fasciitis, which was further confirmed by histological examination of surgical specimens (Figure 1B). After combined medical and surgical treatment, the patient was discharged under a relatively stable condition. Prolonged use of trimethoprim—sulfamethoxazole (for a total of 3 months) was performed until wound healing, and no relapse of the infection was observed on follow-up evaluations.

Cutaneous nocardiosis manifests as either primary or secondary cutaneous nocardiosis (secondary dissemination from the disease of the internal organs).[2] Primary cutaneous nocardiosis can be further divided into three major subtypes, namely, actinomycotic mycetoma, superficial cutaneous nocardiosis, and lymphocutaneous nocardiosis.[2,3] In contrast to previously reported primary cutaneous nocardia cases that manifested limited superficial skin infection,[1] our nonimmunocompromised case with initial superficial skin lesions progressed to necrotizing fasciitis warranting curative surgical debridement. We found only one reported case of necrotizing skin infection by *Nocardia* species do date after literature review.[4] The case described by Ricci et al[4] was that of an elderly patient with long-term corticosteroid treatment of antineutrophil cytoplasmic autoantibodies-associated small vessel vasculitis that developed a necrotizing soft tissue infection of the upper extremity caused by *Nocardia asteroides* successfully cured with surgical debridement and antibiotic use.[1,2] Despite its rarity, the previously reported case and the present case should alert clinicians that cutaneous nocardiosis has a potential risk to cause serious complications.

Conflicts of interest

None declared.

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