

CLINICAL VIGNETTE

An Unusual Cat-Provoked Infection: Superficial Cutaneous Nocardiosis

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Case Report

A 64-year-old man presented to the Emergency Department with left lower extremity swelling and pain for four days after being bitten by his own cat. Past medical history includes hypertension, obesity, and poorly controlled diabetes, with recent hemoglobin A1c of 9.7%. He initially presented to an urgent care center two days prior and was given oral cephalexin and trimethoprim/sulfamethoxazole (TMP-SMX). His symptoms continued to worsen despite oral antibiotics, including the development of fevers and chills. Although afebrile in the emergency room, his left leg distal to the knee was very erythematous, edematous, warm, and tender (Figure 1). Left groin lymphadenopathy was noted, and there were multiple punctate lesions on the left lateral foot, the largest with ulceration. His labs were remarkable for a white blood cell count of 12,000/k, C-reactive protein (CRP) greater than 8 mg/dL, negative blood cultures, and a negative nares colonization with methicillin-resistant *Staphylococcus aureus* (MRSA). X-rays revealed no soft tissue gas. The patient was started on intravenous ampicillin/sulbactam, and podiatry debrided the largest foot ulcer, noting gram-positive cocci. The patient's white count continued to decline, however, he developed fluctuant nodules. CT revealed a phlegmon at the left tibial plateau. Surgery was consulted and felt no intervention was required. On hospital day #6, wound cultures grew *Nocardia brasiliensis*. TMP-SMX was added to ampicillin/sulbactam and after clinical improvement the patient was discharged on TMP-SMX and amoxicillin/clavulanate with outpatient follow up with Infectious Disease and Podiatry.

Discussion

Microbiology of the animal bite wound in humans is usually polymicrobial and may include broad mixture of aerobic and anaerobic pathogens.¹ Pathogens often reflect the oral flora of the animal, the victim's own skin, the surrounding environment, or the animal's ingested food or prey. Only one-third of cultures from patients with cat bite wounds resulted in pure aerobic bacterial growth. The most common pathogens include *Pasteurella spp.*, *Streptococcus spp.*, *Staphylococcus spp.*, *Neisseria spp.*, *Moraxella spp.*, *Fusobacterium spp.*, *Porphyromonas spp.*, *Corynebacterium spp.* and *Bacteroides spp.* Cats can also harbor *Bartonella henselae*, the causative agent of cat scratch disease. In addition, exposure to infected cats may transmit *Francisella tularensis* resulting in tularemia, or *Yersinia pestis* resulting in the plague. Cats are also known sources of sporotrichosis and rabies.

Cat bites, compared with human or dog bites, get infected significantly more often. Wounds at risk for infection are those that are punctate; contaminated; penetrate into the synovial space, and those located on the hands, feet, face or peri-prosthetic joint area. Patients with impaired immune response or severe dermatitis that disrupts the skin are also at risk for severe infection after a bite.²

Nocardia is an exceedingly uncommon cause of cat-bite associated infection. *Nocardia* is a branching gram-positive aerobic actinomycete with the ability to cause localized or systemic suppurative disease in humans and animals. Its morphology is virtually indistinguishable from *Actinomyces spp.* on gram stain. Unlike an actinomycete, it grows in aerobic conditions, and is partially acid-fast due to presence of mycolic acid in its cell walls. *Nocardia* is a ubiquitous pathogen commonly found in soil, water, dust or decaying vegetation. It causes infection primarily following inhalation, and usually affects immunocompromised hosts, with only 1/3 of cases occurring in immunocompetent patients.³ Patients at risk include: transplant recipients, HIV patients with CD4 <100cells/mm³, cancer patients regardless of chemotherapy treatment, and those with diabetes mellitus.⁴ The incidence of nocardiosis in the United States is rising, probably due to increased availability of cultures, immunosuppressive therapies, and awareness of this organism.

The lung is the most frequently affected organ (39%) followed by generalized systemic infections (32%). Localized extrapulmonary infections constitute 12% of the infections with CNS and cutaneous/lymphocutaneous infections making up the majority (9% and 8% respectively).⁵ Smaller numbers of patients experience cutaneous nocardiosis due to direct inoculation into the skin or in the setting of trauma.⁶ Primary cutaneous nocardiosis can manifest as superficial skin infections (i.e. abscess, ulceration, cellulitis), lymphocutaneous variants or mycetomas. In general, most cases of primary cutaneous nocardiosis are caused by *N. brasiliensis*, like in our patient. Superficial cutaneous nocardiosis can mimic staphylococcal or streptococcal infection and is often misdiagnosed until formal cultures and gram stains of the wound are obtained. Growth of *Nocardia spp.* in culture is slow, and may take up to two weeks, further complicating patients' care.

Treatment of cutaneous nocardiosis is prolonged use of antibiotics and, if possible, surgical debridement and drainage.

TMP-SMX is the treatment of choice, but the infection will respond to cephalosporins, imipenem, minocycline, and clindamycin.

Our patient presented with classic infected cat bite wound, which did not respond to broad spectrum antibacterial therapy. Delayed growth of the *N. brasiliensis* on wound cultures further postponed initiation of appropriate therapy. After the final diagnosis of primary cutaneous nocardiosis was made, his antibiotics were adjusted, and his infection improved.

Conclusions

Primary cutaneous nocardiosis should be suspected in patients with cellulitis and poor response to broad spectrum antibacterial therapy. It is important to follow culture of the infected puncture wound for extended time to allow sufficient time for fastidious bacteria to grow.



Figure 1. Patient's erythematous and edematous left lower leg on presentation.

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