Increasing incidence of leprosy and transmission from armadillos in Central Florida: A case series

Renee Domozych, BA,[a] Esther Kim, BA,[a] Sarah Hart, BA,[a] and Jeffrey Greenwald, MD, JD[a,b]
Orlando and Longwood, Florida

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INTRODUCTION

Leprosy, or Hansen's disease, is caused by the acid-fast bacillus *Mycobacterium leprae*. Leprosy is transmitted by human-to-human contact, although zoonotic transmission has been described, and contact with the nine-banded armadillo (*Dasypus novemcinctus*) is a risk factor for development of leprosy.[1-4] Cases 1 and 2 in this case series show zoonotic transmission from armadillos. An additional source of *M leprae* infection may be soil or land contaminated by leprosy-infected armadillos.[3,5,6] Cases 3 and 4 support this potential mode of transmission.

Florida recently experienced an increased incidence of leprosy, including 72 confirmed cases since 2010. We report 4 patients seen over 2 years in Central Florida, an area in which leprosy is not endemic.

Abbreviation used:

NHDP: National Hansen's Disease Program

CASE REPORTS

Case 1

An otherwise healthy 55-year-old white man from Central Florida presented to clinic in January 2014 with a 2-year history of enlarging, pink, anesthetic skin lesions on the left ankle and thigh. The patient denied a history of travel to areas where leprosy is endemic, and the patient had not previously come in contact with individuals with leprosy. He reported multiple exposures to armadillos over the last decade; the most significant exposure was when his vehicle struck an armadillo. Armadillo carcass transferred to the patient's shoes and skin on his left arm and ankle. He recalled smelling his left arm before wiping off the remains.

On physical examination, 8- to 15-cm hypopigmented, annular plaques with erythematous borders and central anesthesia to touch and pinprick were located on the left lower extremity (Fig 1). Two- to 5-cm annular, erythematous plaques were found on the scalp, face, trunk, and left upper extremity. Punch biopsies from ankle and leg lesions found mycobacterial infection with granulomatous inflammatory response suggestive of borderline tuberculoid leprosy (Fig 2). A punch biopsy of the upper midback was performed, and acid-fast bacilli were seen after application of a Fite stain (Fig 3). Biopsy specimens from the left lower extremity, which were sent to the National Hansen's Disease Program (NHDP), showed granulomatous infiltrates, and given the clinical findings, the patient had borderline tuberculoid leprosy diagnosed.

The patient received a combined daily regimen of dapsone (100 mg), minocycline (100 mg), and rifampin (600 mg) for 24 months. He responded well with no adverse events or immunologically mediated reactions. The smaller lesions disappeared within a week of treatment, with the larger lesions remaining as hyperpigmented anesthetic areas. There is no sign of relapse 12 months after completing treatment.

Case 2

An otherwise healthy 75-year-old white man from Central Florida presented to the clinic in November 2015 with a 5-year history of annular pink lesions on the trunk and thighs and a 3-year history of worsening paresthesias of the feet and distal fingers. He was being treated with gabapentin for peripheral neuropathy of unknown origin for more than 1 year.

From the University of Central Florida College of Medicine,[a] and the Dermatology Group PA.[b]

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Correspondence to: Jeffrey Greenwald, MD, JD, 515 West State Road 434, Suite 210, Longwood, FL 32750. E-mail: cinnamark@gmail.com.

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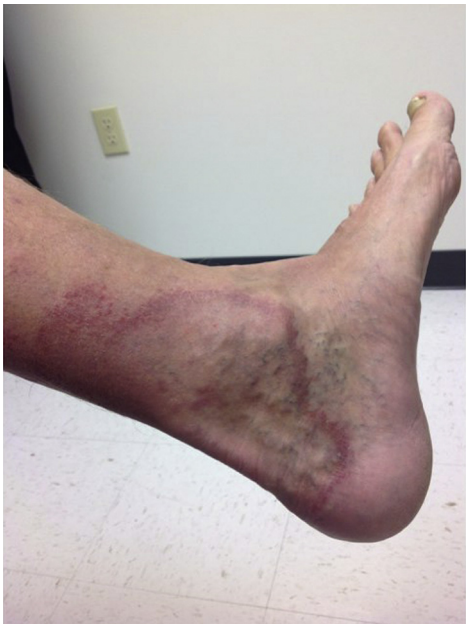

Fig 1. Borderline tuberculoid leprosy. Case 1: Annular, hypopigmented, anesthetic dermal plaques over the medial left ankle.

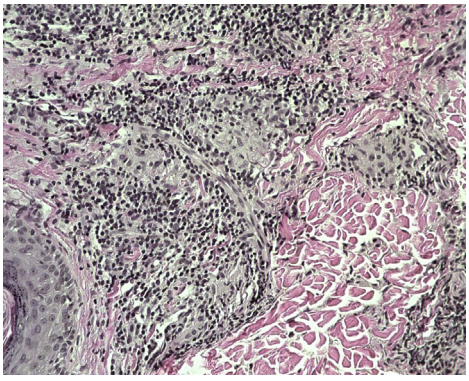

Fig 2. Borderline tuberculoid leprosy. Case 1: Granulomatous infiltrate. (Hematoxylin-eosin stain; original magnification: ×40.)

The patient denied a history of travel to areas where leprosy is endemic, and the patient had not come in contact with individuals with leprosy. He is a hunter and reported direct contact with armadillos and their carcasses over the last decade.

On physical examination, multiple asymmetric, erythematous, and annular plaques with central clearing were distributed on the trunk, thighs, and upper arms. The lesions, feet, and distal fingers were anesthetic to touch and pinprick. A punch biopsy found granulomatous dermatitis and epithelioid histiocytes surrounded by lymphocytes with perineural affinity. Histopathologic examination, after application of Fite stain, found the presence of multiple bacilli. The biopsy specimen was sent to the NHDP, and histopathology findings were diagnostic for leprosy. The patient has not begun treatment.

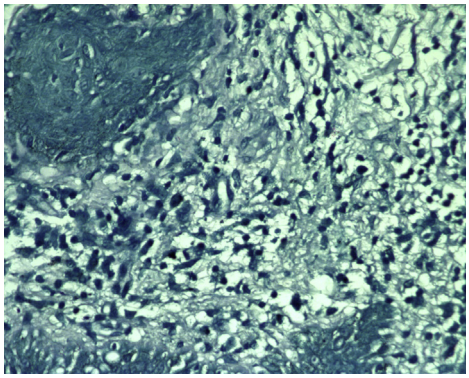

Fig 3. Borderline tuberculoid leprosy. Case 1: Punch biopsy from the upper midback shows acid-fast bacilli. (Fite stain; original magnification: ×100.)

Case 3

A healthy 70-year-old white man from Central Florida presented to clinic in January 2014 with concern for increasing number of lesions previously diagnosed as cutaneous sarcoidosis in 2012. The patient denied a history of travel to areas where leprosy is endemic, and the patient had not previously come in contact with individuals with leprosy. The patient denied direct contact with armadillos, but reported clearing an acre of overgrown wild land behind his house, which was known to be inhabited by armadillos for more than 20 years.

On physical examination, 8 annular, erythematous plaques, some with central clearing, were located on the trunk, back, and extremities. The plaques were anesthetic to touch and pinprick. Two punch biopsies from truncal lesions found granulomatous inflammatory infiltrate with scattered foci of necrosis (this histologic pattern is not typically seen with leprosy). Fite stain found rare acid-fast bacilli. The NHDP confirmed the presence of acid-fast bacilli and positive polymerase chain reaction for *M leprae* DNA. Tuberculoid Hansen's disease was diagnosed.

The patient received a combined daily regimen of rifampin (600 mg) and minocycline (100 mg) for 12 months. Prednisone was prescribed at the onset of treatment to suppress a Jarisch-Herxheimer reaction. The lesions resolved to the patient's native skin color. There is no sign of relapse 7 months after completing treatment.

Case 4

An otherwise healthy 73-year-old white woman from Central Florida presented to the clinic in