Leprosy in a Texan

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A 69-year-old man from Texas with an unremarkable past medical history presented with a 2-year history of a diffuse, spreading annular eruption involving most of his trunk. He noticed progressive numbness of his hands and feet but no other systemic symptoms. He had never traveled outside of the United States. Skin biopsy confirmed a diagnosis of leprosy, and he was initiated on appropriate therapy.

Leprosy, also known as Hansen's disease, is a chronic granulomatous disease of the peripheral nerves and superficial tissues. The diagnosis is challenging, as the incidence is relatively low in the United States. Only about 200 new cases are reported annually to the Centers for Disease Control and Prevention, with about 10% of those occurring in Texas. This report reminds practitioners of the clinical-pathological presentation of leprosy so that prompt treatment can be started.

CASE REPORT

A 69-year-old white man from Texas presented to our dermatology clinic for an annular eruption that had been spreading for the past 2 years. It began as an isolated lesion on his back and presented after he had been deer hunting and processed his own deer meat. It then spread to involve most of his trunk and arms. He denied any itching or burning associated with the eruption; however, he noted a worsening tingling and numbness sensation with intermittent pain in his hands and feet. Over 2 years, he was treated with antihistamines and intramuscular steroid injections. He did note some clearance with steroids, but the rash then quickly recurred. His daily medications were cetirizine, ranitidine, fexofenadine, and montelukast. No other family members were affected. He denied any travel outside of the United States or direct contact with armadillos.

Examination revealed diffuse erythematous, edematous annular plaques coalescing to cover most of his trunk and upper arms to the elbows (*Figure 1*). Islands of sparing, particularly in the axilla, were noted. He had no involvement of his face, lower extremities, or arms distal to the elbows. Punch biopsy from the edge of an indurated plaque demonstrated granulomatous inflammation with numerous acid-fast bacilli and Fite-positive organisms consistent with leprosy (*Figure 2*). Periodic acid Schiff stain was negative for fungal organisms. The patient was started

Figure 1. Diffuse erythematous slightly indurated plaques on chest, abdomen, and arms.

on dapsone 100 mg daily, rifampin 600 mg monthly, clofazimine 50 mg daily, and clofazimine 300 mg monthly for 12 months.

DISCUSSION

Leprosy is a chronic granulomatous disease of the peripheral nerves and superficial tissues that is caused by *Mycobacterium leprae*, an acid-fast bacillus (1). About 90% of patients with documented manifestations of infection with this organism reside in Africa, Asia, and South America, most commonly India, Brazil, Indonesia, and Nepal (2, 3). Most cases have occurred in India (2, 3).

M. leprae is an obligate intracellular parasite primarily multiplying within macrophages and Schwann cells (4). It is known that *M. leprae* grows at 30° to 33°C, which may be why it preferentially targets the lips, forehead, ears, and other acral areas (5). *M. leprae* causes a spectrum of illness with forms that are classified as either tuberculoid or lepromatous (6). In the tuberculoid

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Figure 2. (a) Granulomatous inflammation with prominent grenz zone (hematoxylin and eosin, original magnification 4×). (b) Granulomatous inflammation with perineural involvement (hematoxylin and eosin, original magnification 20×). (c) Fite stain highlighting *Mycobacterium leprae* (original magnification 40×).

form, lesions are granulomatous with few organisms, extensive epithelioid cells, giant cells, and lymphocytic infiltration (7). In the more disfiguring and widely recognized lepromatous form, lesions show dense infiltration with acid-fast bacilli (8). Bacilli often spread systemically and invade the peripheral sensory nerves which, when damaged, result in peripheral neuropathies that predispose patients to accidental trauma and subsequent nonhealing ulcers (9, 10). As exemplified by our patient, leprosy is characterized by a long and variable incubation period that may take up to 5 to 7 years from inoculation to presentation of clinical disease (11).

Leprosy may be diagnosed clinically and, in the case of the lepromatous form, confirmed by demonstrating the presence of acid-fast bacilli in stained biopsies or scrapings of infected tissue (12). Due to the paucity of organisms in the tuberculoid form, biopsies are often unable to localize any bacilli within the dermis (13). Treatment consists of multidrug therapy, often a combination of dapsone, rifampin, and clofazimine. Tuberculoid leprosy is treated with 6 months of dapsone and rifampin, whereas in cases of lepromatous leprosy, clofazimine in addition to both dapsone and rifampin is used for 12 months (14).

Tuberculoid leprosy is not highly contagious, and both tuberculoid and lepromatous forms are rare in the United States (15). Studies suggest that aerosolized droplets from untreated lepromatous leprosy patients, who are often undiagnosed for several years, are the main source of infection (16). Another natural host for *M. leprae* is the 9-banded armadillo (*Dasypus novemcinctus*) which is found in the central Gulf Coast area, particularly in Louisiana and Texas (17). The exact relation between armadillos and human infection with *M. leprae* is unclear, as many patients deny having ever had contact with armadillos (18).

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