CORRESPONDENCE

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An Unusual Case of Untreated Polar Lepromatous Leprosy Associated with Rare *M. leprae*

TO THE EDITOR:

Recently we were confronted with a unique case of untreated, clinically typical, nodular lepromatous leprosy with characteristic skin histopathology on hematoxylin and eosin (H&E) staining, but only exceedingly rare acid-fast bacilli (AFB). Because of the unusual nature of this combination of findings and because others might encounter similar cases, we report here the clinical, histological, and bacteriologic observations of this case.

The patient, a 78-year-old Filipino male, presented with a two-month history of a non-pruritic nodular rash which began on his legs but rapidly spread to involve his trunk, face, and upper extremities. Over the preceding few months, the patient noted progressive anesthesia of the extremities, particularly the feet. He reported having burned his left foot while bathing two weeks prior to medical presentation. On repeated questioning, he denied a previous diagnosis of, or therapy for, leprosy. He had no known contact with leprosy patients, had left the Philippines to reside in the United States 35 years prior to presentation, and had only returned to the Philippines once during the intervening years, four years previously, for a visit of one month's duration. The patient presented with multiple, symmetrical, mildly erythematous, 0.5–2 cm round nodules over his trunk, face, and extremities. These nodules were nearly confluent on the back. Bilaterally enlarged peroneal nerves were noted and significant loss of sensation was observed in the left foot, which demonstrated a small, healing, second-degree burn. On monofilament testing of the upper extremities, there was diminished light touch of the right hand on both the volar and dorsal aspects and diminished protective sensation of the right volar hypothenar eminence. The left hand showed diminished light touch on only the volar aspect in both the median and ulnar distributions.

Hemogram, fasting blood sugar, BUN, serum creatinine, liver function tests, chest X-ray, and EKG were unremarkable. Nerve conduction studies showed a number of significant abnormalities, including: a) inability to elicit a response from the left peroneal and tibial nerves, the left median and sural sensory, and the ulnar sensory bilaterally; b) slowing of conduction velocity of the right ulnar nerve from the axilla to below the elbow, and c) slowing of conduction of the right peroneal nerve across the fibular head. The amplitude of the right peroneal response was markedly reduced.

The nodules regressed significantly after a few months of dapsone therapy and later disappeared entirely.

Prior to the initiation of therapy a full-thickness, 10 mm punch biopsy of the skin was taken from one of the nodules of the left anterior thigh. H&E-stained sections of this biopsy were typical of lepromatous leprosy (LL), with an abundant amount of highly vacuolated foam cells noted. A thorough evaluation of the Fite-stained specimen, for which there was a good positive control section of lepromatous leprosy

stained concurrently, revealed no bacilli. Approximately two-thirds of the biopsy was processed by usual techniques ([2]) and initially found to contain less than 10^3 *Mycobacterium leprae* (no bacilli on 60 high-powered fields). More thorough examination, however, revealed a few definite AFB, morphologically compatible with *M. leprae*. From this biopsy no growth was noted on Löwenstein-Jensen media for up to four months. From this biopsy both hind foot pads of BALB/c mice were infected with no more than 8×10^2 *M. leprae,* which resulted 20 months later in a harvest (four feet, two mice) of 2.3×10^5 AFB per foot pad. Five $\times 10^3$ organisms from these mice harvested at 20 months were inoculated into both hind foot pads of mice and multiplied therein, yielding 5×10^5 bacilli at six months and 7×10^5 bacilli per foot pad at 11 months. Five $\times 10^3$ bacilli per foot pad from these six-month mouse harvests were again passaged to groups of mice so as to assess antimicrobial sensitivity. Untreated mice yielded 6×10^5 bacilli per foot pad by six months; isoniazid 0.01% w/w in the mouse diet did not prevent multiplication (AFB per foot pad at six months were 2×10^5). On the other hand, dapsone 0.01%, 0.001%, 0.0001%, clofazimine 0.001%, and rifampin 0.01% resulted in suspension of bacterial growth (AFB per foot pad less than 8×10^3 at eight months). Organisms from the untreated mice were plated on Löwenstein-Jensen media and again no growth was observed. A suspension containing AFB from the mouse foot pad homogenate of the untreated mice after six months in the last-described passage was sent to Dr. Patrick Brennan. Utilizing a modification of the dot ELISA technique described by Hawkes, *et al.* ([1]), he confirmed the presence of the *M. leprae*-specific phenolic glycolipid I (PGI).

In conclusion, this patient without a previous diagnosis of or therapy for leprosy presented with skin lesions, nerve enlargement, and peripheral neuropathy consistent with polar lepromatous leprosy. H&E-stained skin biopsy material was entirely compatible with that diagnosis, yet Fite-stained sections showed no AFB. Lack of growth on Löwenstein-Jensen media, characteristic growth in the mouse foot pad, antimicrobial susceptibility, the demonstration of PGI in the organism, and the clinical response to dapsone, all confirm that *M. leprae* was responsible for the disease in this patient. The mechanism of the unique host-parasite interaction demonstrated by this case is under investigation. Whatever the mechanism operative in this case, it is hoped that this report will serve to alert other clinicians to this unusual combination of features resulting from infection with *M. leprae.*

—Robert H. Gelber, M.D.

Medical Director
Hansen's Disease Program
Seton Medical Center
1900 Sullivan Avenue
Daly City, California 94015, U.S.A.

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