

TABLE 2. Comparison of Mitsuda reactions in 30 normal individuals using human and armadillo lepromin solutions administered one year apart.

Response <sup>a</sup>	No. of subjects
No change	10
Increased induration by 1 mm or less	8
Increased induration between 1 mm to 2 mm	2
Increased induration by more than 2 mm	10

<sup>a</sup> Responses to armadillo lepromin compared to responses to human lepromin one year later.

been done (<sup>2</sup>). Shepard and Saitz (<sup>3</sup>) found that sensitivity to tuberculin had little relationship to the lepromin reaction (Mitsuda). This present study confirms that relationship.

Twenty females and 10 males presented for the repeat lepromin test. Among the females, all positives remained positive at 21 days, while 5 cases previously negative had readings of 3 mm or greater. This gave a 90% positivity rate. Among the males, of the 5 subjects initially negative, 2 became positive and had readings of 5 mm and 10 mm. A further 5 subjects were initially positive, but on re-testing 2 were negative. Table 2 shows the comparison of the Mitsuda reaction using different lepromin solutions in those who were re-tested. The Mantoux reaction was greater than 10 mm in induration in those individuals who were negative to both human and armadillo lepromin.

Hansen's disease is still endemic in Sin-

gapore, although the number of new cases has been steadily decreasing.

The Mitsuda reaction is a consequence of an intact cell-mediated immune response to persistent antigenic material of *Mycobacterium leprae* as suggested by Wade (<sup>4</sup>). It is interesting to note the increase in positivity of those re-tested with armadillo lepromin 1 year after receiving human lepromin. That 2 males became negative after initially being positive is unusual. It is possible that technical factors could account for this. Of the 30 re-tested individuals, 20 showed an increased induration of 1 mm or more when re-challenged with armadillo lepromin (Table 2).

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## Bilateral Ulnar Nerve Abscess in Lepromatous Leprosy; A First Encounter

TO THE EDITOR:

We recently treated a patient with clinical and histopathologically confirmed polar lepromatous leprosy and skin manifestation of erythema nodosum leprosum (ENL) for right ulnar neuritis and subsequent ulnar nerve abscess, followed eight months later by a similar process involving the left ulnar nerve. Nerve abscesses in leprosy are rare

and are usually seen in tuberculoid patients; when seen in lepromatous leprosy, nerve abscesses are generally reported (<sup>1,2</sup>). The fact that our patient developed abscesses in both ulnar nerves, the evolving clinical picture, and the response to therapy make this case worth sharing with clinical colleagues who might encounter similar cases.

The patient, a 25-year-old Filipino male,

initially presented in August 1982 with previously untreated polar lepromatous leprosy. Detailed sensory and muscle strength evaluations were entirely normal and initial sensory and motor nerve conduction studies were noteworthy for minimal ulnar slowing bilaterally, 50 m/sec. Therapy was initiated with dapsone 100 mg and rifampin 600 mg daily. The patient responded well to therapy without complications until July 1984, when he experienced severe right ulnar pain and exquisite tenderness which resolved entirely on 60 mg of prednisone daily. In August 1984, after the prednisone had been reduced to 20 mg daily, the right ulnar nerve pain recurred and fusiform swelling and exquisite tenderness in the right ulnar nerve were noted. The right 1st dorsal interosseus muscle had lost 40% of its bulk and 90% of its strength. Early clawing with loss of sensation were noted in the right 4th and 5th fingers. The ulnar nerve pain, swelling, and tenderness resolved promptly upon increasing prednisone to 60 mg daily. In October 1984 the patient experienced, for the first time, skin lesions of ENL, fever (T. 38.3°C), and a swollen, exquisitely tender right ulnar nerve with adjacent skin erythema and induration. Therapy was initiated with prednisone 100 mg daily and thalidomide 300 mg nightly, an elbow sling, and splinting of the 4th and 5th fingers. Two days later the pain, tenderness, and swelling and signs of inflammation were markedly improved. One week later the right ulnar nerve was only mildly tender.

One month later, in November 1984, while taking prednisone 60 mg daily and thalidomide 300 mg every other night, the patient again experienced many ENL skin lesions, fever (T. 37.8°C), and right ulnar nerve pain. An examination of the right ulnar nerve revealed findings similar to those of October 1984, including, to a lesser extent, the adjacent skin inflammation. Pain, tenderness, and signs of inflammation again resolved promptly upon increasing the prednisone to 100 mg daily and the thalidomide to 300 mg nightly. However, a week later in December 1984, right ulnar nerve pain and saccular swelling with adjacent skin inflammation recurred. The ulnar nerve was found to contain a very tender mass of 2½" × 3". The first dorsal interosseus function was now entirely gone, ad-

duction of the fingers of the right hand was no longer possible, and shotty tender right axillary nodes were noted. At surgical exposure, the right ulnar nerve was massively distended (3 cm) throughout the distal two thirds of the brachium. The ulnar nerve was severely compressed and necrotic in the cubital tunnel. Numerous intrafascicular abscesses were noted. Epineurolysis, intrafascicular neurolysis, and anterior transposition of the nerve were performed. A pathologic examination of multiple tissue fragments showed an extensive, acute inflammatory infiltrate with multiple microabscesses, extensive nerve destruction, a polymorphonuclear vasculitis, and acid-fast bacilli often in groups. There has been no postoperative recurrence of the right ulnar inflammatory symptoms, and there has been some slight improvement of the profound ulnar neuropathy.

In April 1985, without spontaneous pain, the left ulnar nerve was noted to be mildly tender; the patient was treated with prednisone 15 mg daily. Six weeks later in May 1985, the patient experienced severe left ulnar pain, the nerve being moderately swollen and very tender. Weakening (without clawing) of the left hand intrinsic muscles was noted one week later. On prednisone 80 mg daily, there was no spontaneous pain, and the ulnar nerve swelling was significantly decreased but exquisite tenderness remained which resolved promptly upon increasing the prednisone to 120 mg daily. One month later in June 1985, the patient had a few ENL skin lesions, and the left ulnar nerve was noted to be only mildly tender. Two weeks later in July 1985, while on prednisone 100 mg and thalidomide 150 mg daily, the left ulnar nerve became painful with a saccular swelling and overlying cellulitis. Further atrophy and decrease in intrinsic muscle strength in the left hand was noted. At surgery the left ulnar nerve was noted to be enormously enlarged with an especially thickened capsule from the mid-upper arm to the medial epicondyle. On opening the nerve sheath, a large amount of yellowish, edematous "casseous" material was identified and removed, and the ulnar nerve was transposed anteriorly. Microscopic examination revealed acute and chronic inflammation of the fibrous neural sheath with areas of necrosis and vast num-

bers of beaded acid-fast bacilli. Subsequently prednisone was tapered to 20 mg a day.

Six weeks later in August 1985, severe left ulnar pain recurred. Examination revealed a fluctuant 8 × 4 cm mass in the left ulnar nerve approximately 3 cm above the elbow. Early clawing of the 4th and 5th fingers with increasing sensory loss in the left ulnar distribution were found. At surgery there was considerable dense scar involving the full circumference of the left ulnar nerve from 6 cm above the elbow to the medial epicondyle; at its greatest diameter, the nerve was 2 cm. The ulnar nerve had displaced medially from its previous mooring. There was no frank pus. Scar tissue was extensively removed with further neurolysis, and the anterior transposition was revised. Surgical specimens contained muscle, fat, and nerve tissue with some necrosis. The inflammatory infiltrate consisted mostly of lymphocytes and neutrophils, but with a few plasma cells and histocytes—at times containing a few beaded acid-fast bacilli and giant cells. Steroids were slowly discontinued but, despite the severe recurrence of skin lesions of ENL, there has been no recurrence of the left ulnar pain or tenderness.

Long-term sequelae have been the loss of protective sensation in both 5th fingers, severe bilateral interosseus wasting, minimal bilateral 4th and 5th finger clawing, and complete bilateral absence of sensory and motor responses in the ulnar nerves following galvanic stimulation.

Although believed to be most commonly secondary to ENL, the etiology of nerve abscesses in lepromatous leprosy has not been firmly established. In our case, the fever and recurrent skin manifestations of ENL with the identification of ulnar intraneural poly-

morphonuclear vasculitis suggests such a relationship.

There are at least three lessons to be learned here. First, the marked ulnar nerve swelling and also the overlying signs of skin inflammation should alert one to the likelihood of a nerve abscess. Second, when a nerve abscess is suspected in lepromatous leprosy, prompt surgical intervention is indicated (<sup>1</sup>). In first the right and then the left ulnar nerves, typical "ENL neuritis" appeared to precede actual abscess formation; however, although corticosteroids and thalidomide ameliorated the signs and symptoms of ulnar nerve abscesses after they were suspected, anti-inflammatory therapy did not mitigate against the need for eventual operative intervention. In the interim, deterioration of ulnar nerve function occurred, and this may have been prevented by more rapid surgical decompression. Finally, aggressive and complete neurolysis is necessary to prevent recurrence. We hope that by sharing this unusual clinical experience others similarly confronted will be encouraged to proceed more effectively.

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