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Role of Surgical Decompression of Peripheral Nerves in Leprosy

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ABSTRACT

Leprosy is a particularly common cause of neuropathy in developing countries, although it is also seen in developed countries. In the United States, the prevalence of leprosy may increase with increasing immigration from regions in which the disease is endemic.[2] The disease is clinically characterized by one or more of the three cardinal signs: hypopigmented or erythematous skin patches with definite loss of sensation, thickened peripheral nerves, and acid-fast bacilli detected on skin smears or biopsy material. M. leprae primarily infects Schwann cells in the peripheral nerves leading to nerve damage and the development of disabilities. Despite reduced prevalence of M. leprae infection in the endemic countries following implementation of multidrug therapy (MDT) program by WHO to treat leprosy, new case detection rates are still high-indicating active transmission. The susceptibility to the mycobacteria and the clinical course of the disease are attributed to the host immune response, which heralds the review of immunopathology of this complex disease.

Peripheral nerve involvement results in deformities in leprosy.High doses (40-60 mg) of steroids along with anti-leprosy drugs is the preferred treatment, even though 70-75% cases still develop deformity. Early surgical decompression of nerves gives better chances of preventing deformity. We have analyzed the role of early surgical decompression in such cases.

KEYWORDS

Introduction

Leprosy is one of the most common primary causes of non-traumatic disease of the peripheral nerves worldwide.[1]Although its prevalence is steadily declining, leprosy remains an important cause of infectious neuropathy in tropical and subtropical countries.[2,3]The burden of leprosy and the public perception of the disease have dramatically changed in recent years. The use of multi-drug therapy (MDT) for the treatment of leprosy, as recommended by the World Health Organization (WHO), changed the natural history of this disease and dramatically decreased disabilities.[4]Concomitant attention to the follow-up of these patients translated into better identification of reactions and relapses.[5]Nonetheless, leprosy continues to be a disabling and stigmatizing disease.[6,7] Leprosy caused by Mycobacterium leprae, a microorganism that has a predilection for the skin and nerves. Though nonfatal, leprosy is one of the most common causes of nontraumatic peripheral neuropathy worldwide. The disease has been known to man since time immemorial[8] . Leprosy is a disease of nerves and known for its deformities. The peripheral nerve involvement in leprosy is common and results in damage leading to various deformities. The commonly involved nerves in the upper limb are the ulnar and median and in the lower limbs - posterior tibial and lateral poplitial nerve in that order. [9]

Nerves are known to get entrapped at various anatomical sites clinically manifesting in paresthesia and paresis even in nonleprotic conditions. It is also known that inflamed swollen nerves due to any cause are more prone to entrapment. [10–14]

Materials and Methods:

During 2006 to 2012, 80 ulnar nerves, 22 median nerves and 64 posterior tibial nerves were undertaken for decompression. These cases were on 40-60 mg of steroids for more than 12 weeks and did not show any improvement. The cases were paucibacillary, multibacillary and neuritic type. The paucibacillary cases were having less than five anesthetic patches along with nerve involvement while the multibacillary cases were having eight to fifteen anesthetic patches with skin infiltration and nerve involvement. The neuritic cases were not having any anesthetic patch over the skin; only thickened painful nerve with paresthesia was seen. detailed history was recorded from each patient. This included the duration of disease, duration of neural symptoms, history of treatment, mainly the anti-leprosy drugs and steroids with doses and duration of steroid intake.

Ulnar nerve:

Eighty cases were followed up for variable periods (2-4 years) following the decompression. Out of these, 51 cases were male and 29 were female. Detailed clinical examination was carried out. The ulnar nerve was palpated for thickening, tenderness and presence of abscess. The sensory function was examined with pinprick and feather touch/cotton wool. Complete charting of affected muscles and their motor power on MRC grading was done.

Median nerve:

Twenty two patients had nerve decompression (14 male and 8 female). All these had history of pain in lower forearm and wrist region and sensory loss over the palmar area of the thumb, index and middle fingers (not able to feel or differentiate pinprick, feather touch/cotton wool sensation).

Posterior tibial nerve:
Sixty-four patients having plantar ulcer (innervated by one or more of all three branches) for not more than six months duration were included in this study. fourty-one cases had history of recurrent nerve pain below the malleolus and the nerve was tender on mild touch. The ulcers were superficial. The common sites of ulcers were below the head of the first and second metatarsal (n = 39) and heel (n = 25). The size of ulcer varied from 1.5 to 3.0 cm. No bony involvement was detected on X-rays.

All the 166 patients were free from diabetes and any other neurological problems and were not able to feel the pinprick/ feather touch sensation. These patients were on anti-leprosy treatment along with 40-60 mg/day steroid therapy for more than 12 weeks. There was no improvement in nerve pain and paresthesia was increasing. At this stage these cases were undertaken for nerve decompression.

Clinical features:
Signs and symptoms vary, depending on the type of leprosy. Symptoms of leprous neuropathy usually include the following:
- Anesthetic, painless, nonitchy skin patches
- Deformities due to weakness and wasting of muscles innervated by the affected peripheral nerves (eg, claw hand or foot drop secondary to muscle weakness)
- Sensory symptoms, such as diminished to complete loss of sensation, paresthesias in the distribution of affected nerves, and neuralgic pain when the nerve is struck or stretched
- Spontaneous blisters and trophic ulcers consequent to sensory loss. [15]

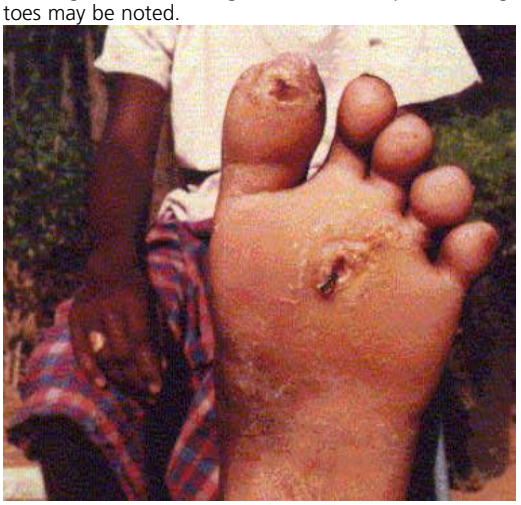
Examination Findings:
Peripheral nerve hypertrophy:
Nerves with a predilection for thickening include the following:
- Great auricular nerves
- Supraclavicular nerves as they cross the clavicle
- Ulnar nerves just above the elbow (the ulnar nerve is the one most commonly thickened)
- Dorsal cutaneous branches of the ulnar nerve at the wrist
- Median and superficial radial nerves
- Femoral cutaneous and lateral popliteal (common peroneal) nerves as they wind around the neck of the fibula
- Superficial peroneal nerves in front of the ankles
- Posterior tibial nerves immediately below the internal malleoli
- Sural nerves

Sensory and motor abnormalities:
With respect to sensory modalities, thermal sensation is affected first, followed by pain and touch. Proprioception and vibration modalities are often preserved. Topographical distribution of sensory loss is variable. Deep tendon reflexes generally preserved because the muscle spindles and large-fiber nerves are not involved.

Extremities, deformities, and trophic changes:
Claw-hand deformity (usually indicating ulnar nerve involvement) is most common, though it is a nonspecific manifestation of leprosy (see the images below).

Claw-hand deformities of both hands in a patient with neural leprosy.

Trophic ulcers, a common, nonspecific complication of pain sensation loss, occur on the sole of the foot and on the hands and fingers (see the images below). Absorption of fingers and toes may be noted.



Plantar trophic ulcers in a patient with leprous neuropathy.

Diagnosis:
Nerve Conduction Studies:
Abnormalities on nerve conduction studies.[16] include the following:

- Segmental slowing of conduction at common sites of entrapment (eg, elbow segment of the ulnar nerve)
- Prolonged distal latencies
- Reduced (sensory or motor) nerve conduction velocities
- Reduced amplitude of compound muscle action potentials
- Absent or low-amplitude sensory nerve action potentials
- Pattern of abnormalities suggesting mononeuropathy, mononeuropathy multiplex, entrapment neuropathy, or generalized polyneuropathy

Nerve Biopsy & Histology :
Nerve biopsy occasionally reveals abnormalities even in contacts of patients with leprosy. The results may rule out other diseases such as polyarteritis nodosa, hereditary neuropathies, or chronic inflammatory demyelinating polyradiculoneuropathy. Recognizing that not all people with thickened nerves, even those of in regions of endemic disease, have leprosy is important

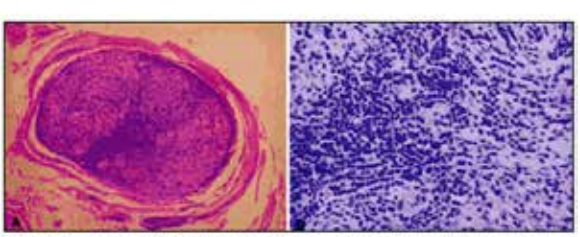


Fig 1. Sural nerve biopsy: A) Intense mononuclear inflammatory infiltrate in the endoneurium, perineurium and perineurium (H&E X 100). B) Other microscopic field shows an infiltrate in higher magnification (HE X 400).

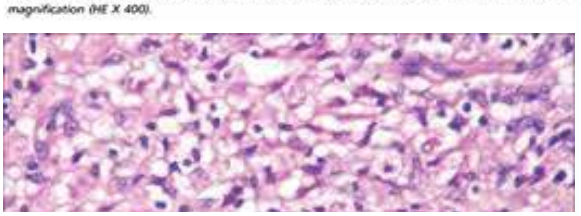


Fig 2: Under the microscope large histiocytes with abundant eosinophilic to foamy cytoplasm & vesicular nucleus are seen.

Surgical Treatment :
All the nerve decompressions were carried out under local anesthesia without any tourniquet. Lignocaine 2% with adrenaline (1 in 100,000) was used for infiltration anesthesia. The whole surgical procedure was over in 30 minutes.

The ulnar nerve at the elbow was exposed through a longitudinal incision about 7-10 cm above and 3-4 cm below the epicondyle. The deep fascia of the anterior-medial compartment of the upper arm was exposed and de-roofing of the fibro-osseous tunnel was done by cutting the over lying fibrous tissue. The fibrous arch between the two heads of the flexor carpi ulnaris was cut and the distal end of the tunnel was widened. The entire segment of the exposed nerve was cleared from the surrounding adhesions without lifting the nerve from its bed. This resulted in the complete external release of the ulnar nerve.

The median nerve was exposed 2 cm above the proximal wrist crease and the adhesions were separated from the surrounding tissues. The flexor retinaculum was cut to its free edge up to the origin of the abductor pollcis brevis fibers. The adhesions inside the carpal tunnel if any, were surgically released. An epineurotomy was done along the length of the nerve taking care not to injure the blood vessels.

The posterior tibial nerve was exposed by an "L"-shaped incision just behind the medial malleolus extending 5 to 6 cm above and below it to reach up to the lower border of the calcaneum. The flexor retinaculum was incised and the neurovascular bundle was identified. The nerve was carefully separated from the posterior tibial vessels. [10,17]

All the nerves were thickened, edematous and had good vascularity. The adhesions were seen along the whole exposed tract of the nerve.

Results:
All the 80 ulnar nerves decompressed and followed up at 2 to 4 years, had no pain in the ulnar nerve at elbow. The patients allowed touch or pressure on the nerve. Sensory recovery was noted in about 50% of the cases . In 22 patients the ability to feel the touch (subjective sensory improvement) was noticed as early as four weeks, though the usual recovery to pinprick and feather touch too started on an average in about 24 weeks. The improvement gradually progressed to complete recovery and maximum benefits were observed at the end of the first year after nerve decompression.

In median nerve, full sensory recovery for pinprick and feather touch was seen in 08 cases. While the other 14 cases were able to feel pinprick sensation but cotton wool and feather touch sensation was poor. All these cases had full functional hand.

For posterior tibial nerve the results of decompression were very promising. The subjective sensory recovery (the patient perception or feel for touch) took place two to three weeks postoperatively while recovery of pinprick and cotton wool/ feather touch sensations took place after six to eight weeks in all cases.

Discussion:
The follow-up period varied from 2 to 4 Years. Pain was the first symptom to disappear. [11,18] The sensory improvement was noticed in some cases as early as four weeks, though the actual recovery took place in about 20 weeks postoperatively. The improvement gradually progressed to complete recovery and the maximum benefit was noticed in about a year after nerve decompression.[11] Forty-eight per cent cases showed complete sensory recovery while others had improved sensations as compared to preoperative state. Fifty per cent cases retained their motor power at Grade 5 while 38% cases were able to maintain motor power up to Grade 3. The improvement in motor function was slow to occur and was seen after 24 weeks. It was more gradual and in some cases it took about two years to obtain the maximum motor recovery.

Full sensory recovery was seen in 55% of median nerve cases while the rest of the cases showed partial sensory improvement which helps the patients to be safe from secondary problems like burns, injuries, etc. [12,13,14] Thirty-five per cent cases improved to motor power Grade 5 and had normal functional hand while the another 35% were able to maintain motor power Grade 3, with a reasonably good functional hand.

Posterior tibial nerve decompression results were very promising. Forty-four per cent cases obtained full sensory recovery in the sole while 66% cases recovered the sensations partially. The healing of ulcers was also 100% in our series.

Conclusion:
The overall observation suggests that along with basic care of hands and feet, the cases not responding to steroid therapy of 12 weeks, who had nerve decompression showed better functional hands and feet which would not have been possible without timely surgical intervention.

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Conflict Of Interest : None Declared

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