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Common Peroneal Nerve Decompression in Leprosy

GN Malaviya, Sajid Husain

Plastic and Reconstructive Surgery Unit Central Jalma Institute for Leprosy, Tajganj, Agra (India)

Abstract

Common peroneal nerve decompression was performed in 21 leprosy cases of which 18 could be re-examined later. In 3 of them motor functions were normal whereas other cases had clinically manifest motor damage of 12 weeks or longer duration. Sensory improvement following neurolysis was seen in 3 cases. Of the 11 cases who had motor recovery, only five cases could get adequate foot lift during walking. Post-operatively several patients who did not get sensory recovery, complained of parasthesia in the common peroneal nerve innervated area of the leg and foot.

Key words: **Common peroneal nerve, Leprosy, Hensen's disease, sensory-motor deficit, multi drug therapy (MDT), paralysis, muscle power.**

Introduction

The involvement of nerve trunks in leprosy usually presents as parasthesiae and nerve pain and at times leads to sensory-motor deficits. Sensory loss generally preceeds the changes in motor function. In the last few decades reconstructive surgery has been able to restore function by means of tendon transfers. However, contradictory opinions have been expressed about the usefulness of nerve decompression procedures to restore the sensory - motor functions in the mixed nerve trunks affected with leprosy.

Damage to the mixed nerve trunks starts primarily as a result of internal compression caused by ongoing antigen - antibody reaction and resultant edema inside the nerve. External compression starts playing the role when turgid nerve becomes too thick to move and gets entrapped.

When leprosy lesions affect common peroneal nerve (CPN), the patient is at risk of paralysis of extensors of ankle and toes that gives static, dynamic and some times neurotrophic disorder of foot. Medical treatment alone is not found to be adequate in such situations and surgical intervention is called for. Muir (1948)[1] was of the opinion that a painful nerve swelling with pressure paralysis was suitable for decompression. Observations of several workers[2-6] suggest that in leprosy patients, after nerve decompression, pain and paresthesia disappear in nearly all the cases and cases with impending nerve paralysis tend to improve muscle power.

In literature there are many reports on surgical decompression of ulnar, median and posterior tibial nerves in leprosy but very few reports on common peroneal nerves. We have been performing nerve decompressions on mixed nerve trunks in leprosy for quite some time and report here our experiences with common peroneal nerve.

Materials and Methods

Twenty one patients, in 12-55 years age group, had decompression of CPN. Duration of disease varied from 3 months to 15 years. Fourteen of them had borderline tuberculoid leprosy, 3 neuritic, 2 borderline lepromatous and 1 borderline and 1 lepromatous leprosy each. Diabetes and spinal disease were ruled out in all the cases.

Correspondence: Dr. G.N. Malaviya, Post Box 25, GPO, Mall Road, Agra (India)-282001. Phone: 91-562-330618, Fax : 91-562-331755

Two cases did not turn up for follow-up examination and another had immediate tendon transfer for drop foot along with nerve decompression. These 3 cases were excluded from the series.

CPN involvement and/or damage varied from 3 months to 6 years (mean 13 months). Left side was affected in 15 and right in 6. Three cases had bilateral disease of which, 2 had recovery on the non-operated side under steroid therapy during the post-operative follow-up.

Average time between the onset of CPN symptoms/damage and operation was 3 months to 4 years (mean 9 months). Nerve swelling and disability were main presenting features. In 10 cases pre-operative steroids (up to 40 mg per day) were given for varying periods (8-16 weeks). The failure to respond to oral steroids had brought them for surgical relief.

CPN trunk was thickened and could be palpated well just below the head of fibula. There was no pain but tenderness on deep pressure could be elicited. Four cases had gross thickening of the nerve trunk.

Sensory loss was present in the CPN innervated area of the leg and foot in 17 cases. In 1 case it was confined to anterior tibial area (between the cleft of great toe and second toe). In one case there was no sensory loss. Motor damage was present in 15 cases and in 10 of them only anterior compartment muscles were affected. Three cases did not have any clinical motor damage. As a group only one patient did not have any manifest sensory-motor damage.

Nine patients in this series had complete posterior tibial nerve damage (bilateral in 2) and 5 had partial damage.

Surgical Procedure

Our approach aimed at extensive and wide exposure along with extraneural neurolysis. The nerve was identified close to biceps femoris tendon and was exposed in the popliteal fossa up to the neck of fibula and followed distally into fibular tunnel. Both superficial and deep parts of fibrous arch were released and its thick edges excised. The epineurium was carefully incised longitudinally, where the nerve was swollen, so as to release the tension avoiding injury to the blood vessels on the surface of the nerve. A curved hemostat forceps was passed and the passage was dilated in the downwards direction. The abscess, if present was evacuated and the wound was closed using skin sutures.

Post-operatively the patients were prescribed 10 mg prednisolone (betamethasone equivalent) and 300 mg aspirin daily for 6 months or more depending upon the response. Anti-leprosy drugs were continued till the disease became inactive. Wound dehiscence was seen in 3 cases.

Post-operatively only 14 cases received steroids. Five cases did not get steroids for reasons like plantar ulcers, wounds etc. Of these 5 cases one did not have any motor damage and other 2 had partial motor recovery. In all, 7 cases received steroids both pre and post-operatively.

Results

The patients were seen one month after operation, then at 3 monthly intervals upto the time they kept visiting us. This period varied from 4 months to 8 years (mean 32 months). Most of the cases were seen at least upto 12 months post surgery.

Follow-up was available in 18 cases. Fourteen cases came regularly and completed MDT schedules. Four cases were irregular and of these 2 did not recover. All the regular cases had recovery of some sensory and/or motor functions. In none of the patients apparent disability worsened even though muscles grade in toe extensors fell down to grade zero from grade 3 (2 cases). Post-operatively all except one had residual thickening of CPN near the neck of fibula.

Recovery of sensory and/or motor functions was seen as early as 3 months after surgery (range 3-16 months, mean 7 months). Sensory recovery was noted only in 3 cases of which one did not have any pre-operative loss of motor functions. Of the remaining 2, one did not get back the muscle strength and other had recovery of sensory and motor functions both. Of the patients who did not have sensory recovery, 9 complained of paresthesia in CPN innervated area of leg and foot.

Outcome about motor functions has been shown in Figure 1. Three cases had no pre-operative motor damage. Of the remaining 15 cases 11 showed some motor recovery. Even though some of the muscles gained normal strength, overall recovery was partial. Useful muscle strength to have dorsiflexion and adequate foot lift while walking was seen in 5 cases only. Peronei gained near normal strength in 5 out of 8 cases where they were paralysed. The order of muscle recovery was peronei > tibialis anterior > extensor hallucis > extensor digtorum longus in that order.

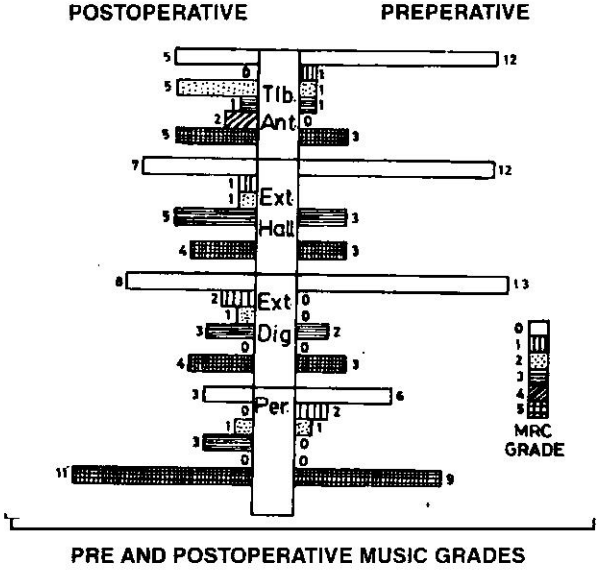

Figure: 1. Strength of CPN innervated musles before and after Nerve Decompression.

Three cases had tendon transfers for correction of drop foot deformity including one having drop foot for 12 years who opted for immediate surgery along with nerve trunk decompression.

Detailed analysis of cases who had motor recovery showed that in 9 of them, nerve damage was about 6 months old and they had received corticosteriods post-operatively. Seven of the 11 cases who showed motor recovery, had paralysis of both anterior and lateral compartment.

Nerve abscess was found, on exploration, in 5 cases. Of these, 3 did not have any clinical motor damage. Of the remaining, 2 one had only sensory recovery and another only motor recovery.

Discussion

The CPN is closely related to the head and neck of fibula and superior tibio-fibular joint. After leaving the popliteal fossa the nerve passes laterally behind and below the fibular head and over the upper fibres of soleus to reach the peroneus longus (PL) which is attached there by its two heads. The superficial head is attached to head of fibula and adjacent part of tibial condyle. The deep head is composed of tendinous tissue which extends upwards from the tibial shaft on to the neck of fibula. The nerve passes between the two heads of PL and here the nerve is in the form of thin flattened band with its constituent branches arranged as collateral bundles well separated from each other exposing the nutrient vessels which lie unprotected between them. It then curves round the neck of fibula and finally between the two heads of muscle and divides into superficial and deep branches. Just before the nerve enters PL it is held applied to bone and muscle by the attachment of deep fascia. Nerve therefore, is fixed as well as angulated at this site.

Several types of entrapment of CPN is described viz. postural, dynamic and idiopathic.[7] Peroneal nerve entrapment usually is attributed to an excessively thick, overhanging edge of the fibrous arch formed by the superficial head of PL.

The arch comprises of two bands - superficial and deep, which must be released in order to completely decompress the nerve. Beyond this, the nerve is compressed between the two heads of the muscle and narrowing of the fibular tunnel through which it passes. Well developed muscle mass adds to the compression.

In this confined space there is slight sliding movement of the nerve during leg and foot movements particularly those of inversion and plantar flexion. A friction constrictive fibrosis developing at this site may impair nerve function. The fibrous arch therefore also contributes to dynamic entrapment during activities like running, jogging, fast walking and to postural entrapment during activities like squatting, kneeling or cross-legged sitting. Superficial peroneal nerve can also get entrapped at the place where it pierces the fascia of lateral compartment to emerge on the dorsum of foot near ankle. It is possible that a double compression can exist in some cases thereby preventing the recovery of sensory functions on the dorsum of foot.

Chronic irritation of leprosy-affected inflammed entrapped nerve both from inside and outside leads to intraneural and extraneural edema which further restricts mobility of the nerve in its sheath during flexion-extension of the knee leading to formation of adhesions, further aggravating the problem. Repeated flexion- extension of the knee causing movement of the nerve within the tight arch can precipitate the damage in already inflammed nerve.

The relative fixation of nerve makes it more susceptible to traction injuries. Proximal part of the nerve trunk suffers more in the process. The traction forces are strong enough to disrupt the nutrient capillaries leaving the perineurium intact. There are two reasons for comparative lack of resistance of CPN to ischemia - (i) Its exposed nutrient vessels and scarcity of the interfascicular connective and fatty tissue (ii) The main intrinsic vessels run loose in the epineurium in 88% cases. There are few large funicular bundles is the nerve as it leaves the popliteal fossa and a relative lack of areolar connective tissue which make them more prone to compression.[8]

Carayon and Huet[9] reported a series of 9 cases of early leprous neuritis of CPN (about 6 months old) of which 6 had complete and one had partial recovery after nerve decompression. In another series [10] 12 of the 32 (37%) operated cases of CPN neuritis had recovery of muscle power. Good results have been reported in a series of non-leprosy cases also.[7]

We have reported very good results of ulnar and median nerve decompressions[6,11-12] with recovery rates varying from 50 to 60%. However, with common peroneal decompression results are not that impressive. In our series of 15 leprosy cases only 5 cases (33%) improved to have useful function in contrast to what we observe with ulnar and median nerves where many cases with complete palsy also improve.

Appearance and persistence of parasthesiae in cases who did not have sensory recovery probably indicates the irritability of regenerating neurons attempting to reach their final destination. The patients who had sensory recovery did not get parasthesiae.

Poor success rate with common peroneal neurolysis even though done at the "appropriate time" needs explanation. Post-operative splinting of the foot may not have been done for adequate period. Keeping foot off the ground during period of recovery which may be as long as 6 months, is not practical. Even if done, it needs supervised mobilisation as there is risk of pathological fractures in foot bones due to increased osteoporosis because of steroids and immobilisation, defeating the very purpose for which the surgery has been performed. The blood supply of the nerve somehow got compromised, may be due to disease process, thereby making any recovery impossible. The patients are probably

presenting too late for surgery as evident by failure of sensory recovery.

Non-operative treatment is generally prescribed because spontaneous recovery is known to occur atleast in leprosy. Some workers have advocated waiting for spontaneous recovery to take place. The spontaneous recovery takes very long time and what percentage of these cases fail to recover is not documented. Many a times, even though steroids are prescribed, proper splinting and rest is not practical as it interferes with the mobility of the patient. Such splinting, to be effective needs at least 6-12 weeks and by this time enough osteoporosis and disuse atrophy of proximal muscles occurs. Stretched leg muscles are slow to gain strength and at times recovery is so poor to be of any functional value.

There are many leprologists who still believe that steriod alone will do good may be at higher doses given for a longer time. This apparently speaks their ignorance about the anatomical peculiarities of the region, pathomechanics of nerve damage and strong belief that inflammed nerve returns to its "normal" thickness once the inflammation has subsided. There is nothing like surgery versus steroids as interpreted by many people. A combined approach appears more logical and in larger interests of the patients.

It seems better to decompress the nerve if there is no response to oral steroids within 3 months of therapy. Operative decompression is safe and time taken for recovery is usually reduced. We feel convinced that operation should be performed relatively early in the course of disease as an adjunct to the steriod therapy in interest of the patients. Correction of drop foot is more troublesome than the nerve decompression. Limited waiting period of 8-10 weeks is justified so as not to interfere with a rapid spontaneous recovery which is always a possibility.

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