

Decompression and Anterior Transposition of Ulnar Nerve in Prevention and Progression of Claw Hand Deformity in Leprosy.

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ABSTRACT

Background: Leprosy is very common disease in India. It is known for its deformities and social problem associated with it. Our study was aimed to follow that surgical decompression and its anterior transposition of ulnar nerve prevents the progress of claw hand in leprosy. During last 20 years study was conducted at centers mentioned and statistic collected. It was found that all cases which did not responded to drugs or had deformity beforehand did responded to surgery and were made patient comfortable. **Methods:** ?. **Results:** ?. **Conclusion:** ?.

Keywords: Leprosy Ulnar Nerve Decompression Anterior Transposition and Deformity.

INTRODUCTION

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 nerve in the upper limb are Ulnar and Median nerve and in lower limb posterior Tibial and lateral Popliteal nerve in that order.^[1]

Nerves are known to get entrapped at various anatomical sites, clinically manifesting as paresthesia and paresis even in non leprotic condition. It is also known that inflamed and swollen nerve due to any cause are more prone to entrapment.^[2-6]

In multibacillary Leprosy nerve thickening occur following invasion of bacilli in nerve tissue or following Lepra reactions in the nerve. While in paucibacillary Leprosy hypersensitive reaction leads to sudden inflammation. Swollen edematous nerve passing through tunnel like structures suffer by getting compressed (entrapment neuropathy). Sensory loss is the most commonly reported symptom followed by motor loss. The autonomic

function loss leads to dryness, fissures ect. Motor loss manifests in form of paresis or paralysis in different parts (claw hand, foot drop, lagophthalmos). Sensory loss manifests in form of burn, blister, trophic ulcer. In India 25 – 30 % of leprosy cases develop deformities.^[7]

Several structures can compress the nerve and cause ischemia, venous obstruction, capillary stasis, intrafunicular hypoxia, edema and increased funicular tension. The net result of entrapment / ischemia is initial slowing of conduction velocity, later full conduction block and ultimately paralysis. The diseased and thickened Ulnar nerve can get further damaged due to physical trauma related to above mentioned anatomical factors.

Median nerve involvement is infrequently seen in leprosy but when ever it gets damaged, it results in functional loss affecting the pinch and grasp functions. Clinically, the median nerve involvement commonly presents as carpal tunnel syndrome, the wrist being unusual site for its involvement in leprosy. Munir was of opinion that the painful nerve causing progressive paralysis is suitable for decompression accomplished by splitting of nerve sheath that will conserve its function and its transposition which will prevent its future entrapment.^[4,6,8]

Ulceration of the foot is due to sensory loss, involvement and damage to the posterior Tibial nerve. The inflamed nerve is usually entrapped and

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compressed in the tarsal tunnel behind the medial malleolus. Steroid therapy to treat the inflammation is insufficient and entrapment need surgical decompression.^[2,9,10]

Early detection and diagnosis of nerve damage can be done by regular and periodic nerve function assessment (sensory by pin prick, and feather touch by cotton wool; motor status by Medical Research Council MCR muscle power grading) of all susceptible nerves on each visit. If the patient is developing nerve damage, steroid must be shared in adequate doses, 60 mg daily for first fortnight followed by tapering of 5 mg every two weeks is the protocol of choice. If there is no improvement in 8-12 weeks as evident by decrease in pain and tenderness over the nerve, reduced paresthesia or one observes worsening / deterioration in motor power, then such cases should be considered for decompression and anterior transposition.^[5,6]

Surgical intervention in form of epineurotomy by multiple longitudinal incision and external decompression to relieve internal pressure throughout the involved segment and anterior transposition was done in this study after steroids failure.

The present study is based on the research hypothesis that early surgical decompression of ulnar nerve with transposition in steroids failure cases specifically oriented for Ulnar nerve produces better functional results.

In the human anatomy, the Ulnar nerve is a nerve that runs near the Ulna bone. The Ulnar collateral ligament of elbow joint is in relation with the Ulnar nerve. The nerve is largest unprotected nerve in human body (meaning unprotected by muscle or bone) so injury is common. The nerve is directly connected to little finger, and the adjacent half of the ring finger, innervating the palmer side of these fingers, including both front and back of the tips, perhaps as far back as the finger nail beds.

The nerve can cause an electric shock like sensation by striking the medial epicondyl of the Humerus from posteriorly, or inferiorly with the elbow fixed. The Ulnar nerve is trapped between the bone and the overlying skin at this point. This is commonly referred as “funny bone “. The name is thought to be a pin, based on the sound resemblance between the name of the bone of upper arm, the “Humerus “and the word “Humorous”.

The nerve originates from the C 8 T 1 nerve roots which forms the part of medial cord of the brachial plexus and descends on the posteromedial aspect of Humerus passing behind the medial epicondyle (in cubital tunnel) at the elbow where it is exposed for several centimetres. It enters the anterior compartment of the forearm between the humeral and ulner head of flexor carpi ulnaris (FCU), lying under the aponeurosis of FCU alongside the ulna. There it supplies FCU and middle half of flexor

degetorum profundus (FDP), and courses with Ulnar artery .It travels down the Ulna and enter the palm of hand by Guyon’s canal.Ulnar nerve and artery passes superficial to flexor retinaculum via Ulnar canal.

IT innervates

- 1- Flexor Carpi Ulnaris.
- 2- Flexor Degetorum profundus medial half.
- 3 – Lumbricals muscle.
- 4 – Opponens Digity minimi
- 5 – Flexor Degetorum Minimi.
- 6 – Introssei.
- 7 – Adductor Pollicis

At elbow motor deficit causes

1. Weakness in flexion of the hand at wrist, loss of flexion of 4th and 5th digits loss of ability to cross the digits of the hand.
2. Presence of claw hand deformity when the hand is at rest, is due to hyperextension of 4 and 5 digit at metacarpophelengeal joint and flexion at interphalangeal joints.
3. Weakness of adduction of thumb, which may be assessed by presence of Froments sign.
4. Sensory deficit is loss of sensation or paresthesia in ulnar half of the palm and dorsum of hand, and medial 1 and ½ digits on both palmer and dorsal aspect of the hand.



Figure 1: claw hand dorsal view.



Figure 2: Claw hand lateral view.

MATERIALS AND METHODS

During 1997 to 2017, 226 Ulnar nerves were undertaken for decompression at Shyamal trauma and child care, Saket mod, Ansari road Muzaffarnagar U.P., Muzaffarnagar Medical college (2008 On wards) Muzaffarnagar U.P. and District Hospital Janjgir Chhatisgarh. These cases were on 40 -60 mg of steroids for more than 12 weeks and did not show any improvement. These cases were Paucibacillary, Multibacillary and Neuritic type. The Paucibacillary cases were having less than 5 anaesthetic patches alongwith nerve involvement while multibacillary cases were having 8 – 15 anaesthetic patches with skin infiltration and nerve involvement. The Neuritic cases were not having any anaesthetic patches over the skin only thickened painful nerve with paresthesia was seen.

All cases were of less than 6 month duration after diagnosis and on antileprotic treatment. A detail history was recorded from each patient, this included the duration of disease, duration of neural symptoms, history of treatment, mainly the antileprotic drugs and steroids with doses and duration of steroid intake.

226 patients were followed up for 5 to 20 years following decompression and anterior transposition. 200 patients were male and 26 were female. Youngest patient was of 16 years and oldest was of 60 years .with mean age around 40 years, and majority being adults. Of 226 cases, 118 cases were Paucibacillary, 82 cases were Multibacillary and 26 were Neuritic type of Leprosy. About 180 patients complained of severe pain at elbow which woke them at night.

Detail clinical examination was carried out. The Ulnar nerve was palpated for thickening, nodule, tenderness and presence of abscess. The sensory function was examined with cotton wool. Complete charting of affected muscles and on their motor power MRC grading was done.

All the 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 antileprotic treatment with steroids 40 – 60 mg / day for more than 12 weeks. There was no improvement in the nerve pain and paresthesia was increasing. At this stage case was taken for ulnar nerve decompression and anterior transposition. Cases were also taken to prevent deformity and there the criterion was clinical assessment of patient.

The ulnar nerve decompression were carried out either in brachial block or local infiltration of Xylocart 2 % with or without adrenaline (1 in 100000 concentration). The whole surgical procedure was over in 30 minutes and tournique was not used.

The Ulnar nerve at the elbow was exposed through a longitudinal incision about 6-8 cm. above and 4-6 cm below the epicondyle. The deep fascia of the anteromedial compartment of the upper arm was exposed and derroofing of fibrous tunnel was done by cutting the overlying fibrous tissue. The fibrous arch between the two heads of Flexor Carpi Ulnaris was cut and the distal end of tunnel was widened. The entire segment of the exposed nerve was cleared from the surrounding adhesions .This resulted in complete external release of ulnar nerve. The intraneural decompression was done by medial longitudinal epineurotomy without damaging the vascular network.^[1]

After the external and internal decompression nerve was mobilised without injuring its branches and a tunnel is made in common flexor origin, nerve is transported over medial condyl (i.e. removed from the groove behind the medial condyl) and buried into this muscular tunnel. Wound is closed in layers.

All Ulnar nerve were thickened and edematous. If abscess or caseation was found than was cleaned thoroughly with saline. Nerve had good vascularity overall. The adhesions were seen at more exposed tract of nerve.



Figure 3: Pre-operative draped elbow showing thickened (visible bulge) ulnar nerve



Figure 4: Ulnar nerve exposed and fibrous tunnel at elbow released nerve seen bulging after release



Figure 5: Nerve neurolysis done caeseous material seen protuding out and tunnel being made in common flexor origin for anterior transposition.



Figure 6: Nerve being transposed in tunnel made in common flexor origin over medial condyl.



Figure 7: Wound stitched.

RESULTS

All 226 ulnar nerve were decompressed and anteriorly transposed then followed up for 5 to 20 years had no pain in the ulnar nerve course. The patient allowed touch or pressure over the transposed nerve.

Sensory recovery was noted in nearly 60 % of the cases [Table 1]. IN 80 patients the ability to feel the touch (subjective sensory improvement) was noticed as early as 4 weeks, though the usual recovery to pin prick and feather touch too started recovering in about 20 weeks. The improvement gradually progressed to complete recovery and maximum benefits were observed at the end of first year after nerve decompression and anterior transposition.

Table 1: Sensory Results of Procedure

Disease	No of cases	Sensory recovery
Pausibacillary	118	60 (50.84%)
Multibacillary	82	52 (63.41 %)
Neuritic	26	20 (76.92 %)
Total	226	132 (58.40 %)

We noted that the ulnar nerve supplied muscle retain their functional ability to prevent the deformity up to MRC grade 3. Hence we grouped the muscle strength in to 3 category – improved, remain same with the usual muscle power and deteriorated for our post-operative evaluation.

Table 2: Results of Motor Functions of the Ulnar Nerve

No of cases	Motor power		No of cases
	Preoperative	Post-operative	
82	Grade 1	Grade 3	76
		Grade 2	6
106	Grade 1-3	Grade 5	90
		Grade 4	16
38	Grade 3-5	Grade 5	31
		Grade 4	7

It was observed that after decompression and anterior transposition ulnar claw hand significantly improved and patient were so satisfied they did not opted for tendon transfer procedure if suggested subsequently. The results were evaluated on the basis of subjective improvement and objective findings related to both sensory and motor modalities and periodic comparison were made with previous assessments. Full sensory recovery (perception to pin prick and feather / cotton wool touch) was 58.4%, relief from pain (I.e. patient did not complain of any pain at elbow or wrist) and patient allowed to touch nerve in its course was 100 %after decompression and anterior transposition. Motor recovery was seen to grade 5 in 121 cases. However no case had any progression to claw hand again for a mean period of follow up of 10.5 years.

One more important behaviour pattern was observed .All patient tried to move out of hospital as soon as possible so that their disease does not become known in society as it still carry social stigma though they were so satisfied next time they visited they usually brought one more patient with them and escorted them safely to us.

DISCUSSION

Leprosy (Hansens Disease) is an ancient disease that continues to impose significant societal burden and is still relevant to peripheral nerve.

Paul Brand (1914 to 2003) observed that the Hensens bacilli, Mycobacterium Lepreae, preferentially targeted peripheral nerve. He saw abnormal “nerve swelling “occurred at specific locations where the nerve lay close to skin surface like Tebial nerve behind ankle, Paroneal nerve behind knee, Ulnar nerve behind elbow and Median and Ulnar nerve at wrist.^[12]

At the turn of century Neg et al,^[13] demonstrated that Phenolic Glycolipid 1 of M. Leprae binds specifically to Lamanin 2 around C terminal of Lamanin G – like domain 4 and 5 of the Lamin alpha 2 chain with in the basal lamina of Schawnn cell – Axon unit, promoting bacterial invasion and even after bacterial cell death , damage to Schawnn cell and nerve. Around the same time Scollard observed that M. Leprae extensively colonise epi and endoneural blood and lymphatic vasculature as well.^[14]

In 2010 TEbes et. al.^[15] found that M . Leprae can also bind Mannose receptors on Schwann cell via Lipoarabino Mannan which as Bahia El Idrissi et. al.^[16] recently demonstrated, can also activate compliment and promote inflammation. Molecular studies in 2007 reveled that M. Leprae has a defective heat stress response that restricts the bacteria to superficial and cooler area of the body such as peripheral nerve.^[17] Taken together the evidence seems to support that such effected nerve is more likely to suffer ischemia from inflammation, trauma, and mechanical stress (sucn nerve compression at tunnel of near joint),^[10] contributing to development of neuropathy.^[18,19]

The follow up period varied from 5 – 20 years. Pain was the first symptom to disappear. Sensory improvement was noticed in some cases as early as 4 weeks, though the actual recovery took place in about 20 weeks postoperatively. The improvement gradually progressed to complete recovery and maximum benefit was noted in about a year in nearly 60 % of patients. Eventually all cases showed improvement to some extent. About 53.5 % of cases retained their motor power at grade 5 while nearly 41.5 % had more than grade 3. The improvement in motor function was slow to occur i.e. seen after 24 weeks and improved up to 2 year. It was tried that all patient take anti Leprotic medicine after surgery.

CONCLUSION

The over all observation suggested that along with basic care of hand and feet , the cases not responding to steroid therapy for 12 weeks who had nerve decompression and anterior transposition

showed better functional hand which would not have been possible with out timely surgical intervention.

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