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NERVE CONDUCTION STUDY IN LEPROSY PATIENTS: AN ELECTROPHYSIOLOGICAL EVALUATION OF MOTOR ULNAR AND MOTOR MEDIAN NERVES.

Physiology

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ABSTRACT

Background: In Leprosy disease involves peripheral nerves in the course of the disease leading to gross deformities and disabilities. By the time it becomes clinically apparent, the nerve damage is already quite advanced. If the preclinical damage is detected early in the course of disease, it can be prevented further deformities and disabilities.

Materials And Methods: This electrophysiological case-control study was conducted on 24(Cases;13, Controls:11) patients with clinically diagnosed leprosy, in the Dermatology Department of Acharya Vinoba Bhave Rural Hospital, Sawangi(M), Wardha. This study was done to assess the mean nerve conduction velocity, mean amplitude and mean latency of motor ulnar and median nerve.

Results And Conclusion: We found decreased mean amplitude in motor median and ulnar nerve, reduced mean conduction velocity in motor ulnar nerve besides no changes in mean latency in the median and ulnar nerves.

KEYWORDS

Electrophysiology, leprosy, nerve conduction study, amplitude, distal motor latency.

INTRODUCTION

Leprosy is one of the principal causes of nontraumatic neuropathy and is clinically manifested as lesions of the skin and peripheral nerves. [1] Functional derangement of nerves can be shown by nerve conduction studies before the appearance of clinical signs and symptoms of the disease. [2] Nerve damage in leprosy varies from involvement of an intradermal nerve in the cutaneous patch to a major lesion in the peripheral or the cranial nerve trunk. Neural involvement can manifest itself as enlargement of the superficial nerves such as great auricular, ulnar, median, radial cutaneous, superficial peroneal, sural, and posterior tibial which are clinically palpable against the corresponding bony prominences when thickened; associated with tenderness, in case of coexistent neuritis. Nerve damage in leprosy may present itself as silent neuropathy without overt signs and symptoms or as clinically manifest disease which may present as weakness, atrophy or contracture. Touch sensation is lost subsequently followed by that of pain. Patients may complain of anhidrosis if there is associated sympathetic nerve involvement. [4] The functional defect in the conduction velocity in the nerves always precedes clinically manifest nerve damage. A significant decline in motor nerve conduction velocities has also been reported in clinically normal nerves in leprosy. [5] The role of electrophysiological evaluation of nerve function in the diagnosis and assessment of various neuropathies has been studied. [6]

MATERIALS AND METHODS

The present case-control study was conducted on 24 patients which included already diagnosed cases of leporsy. Thirteen cases were of diagnosed leprosy while the rest eleven belonged to the normal group. The study was carried out in Central Electrophysiology Lab run under Physiology Department of Jawaharlal Nerhu Medical College, Sawangi(M), Wardha which spanned over a period of 2 years. Informed consent was taken from all the patients and after obtaining a brief history regarding the leprosy disease thorough clinical examination.

Inclusion Criteria: Diagnosed cases of leporsy disease.

Exclusion Criteria: The individuals having following clinical conditions were excluded -

- Known causes of neuropathy (Diabetic mellitus etc)
- History of limb injuries/trauma
- Ulcers
- Malignancy
- Neuromuscular transmission disorders, myopathy
- Alcoholism

Material

Instrument used was **Neuro Perfect 2 – Channel EMG NCV EP, Medicaid.**(Figure 1) This instrument was used to record parameters such as mean distal motor latency, mean CMAP amplitude, mean conduction velocity for motor nerve was recorded and analyzed.

Figure 1: Neuro Perfect 2- Channel EMG NCV EP, Medicaid

Method Of Data Collection

Nerve conduction study consisted of Motor nerve conduction of motor median and ulnar nerves.

Motor Nerve Conduction Study

Motor nerve conduction study involved stimulation of a motor nerve at two different sites with maximum stimulus, the distance was measured and automatically divided by conduction time between the two points (difference between proximal and distal motor latencies) which gave the conduction velocity.

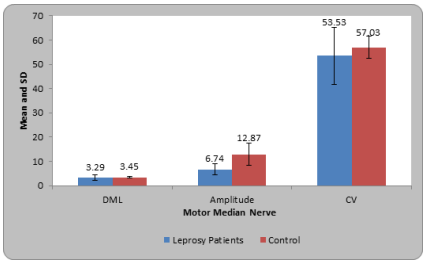
Ground electrode was placed between stimulating and recording electrodes. Surface disc electrode was placed on Abductor Pollicis Brevis muscle for median, on Abductor Digiti Minimi for ulnar nerve. Belly tendon montage was used with cathode and anode 3 cm apart. Nerve was stimulated at wrist and elbow for median and ulnar.

Setting for upper limb duration was set up at 100 µs, sweep speed was 5 ms/D, filter was between 2Hz to 5 KHz, and for lower limb duration was 200 µs, filter was between 2Hz to 10 KHz, sweep speed was kept the same as upper limb.

RESULTS

Table 1: Comparison Of Motor Median Nerve In Two Groups

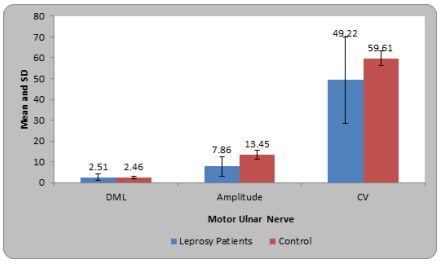
	Leprosy Patients	Control	t-value	p-value
DML	3.29±1.03	3.45±0.43	0.66	0.51,NS
Amplitude	6.74±2.37	12.87±4.58	4.45	0.0001,S
CV	53.53±11.81	57.03±4.51	1.25	0.21,NS



Graph 1: Comparison Of Motor Median Nerve In Two Groups

Table 2: Comparison Of Motor Ulnar Nerve In Two Groups

	Leprosy Patients	Control	t-value	p-value
DML	2.51±1.64	2.46±0.43	0.13	0.89,NS
Amplitude	7.86±4.72	13.45±2.19	4.77	0.0001,S
CV	49.22±20.83	59.61±3.56	2.30	0.028,S



Graph 2: Comparison Of Motor Ulnar Nerve In Two Groups

Table 1 reveals comparison of motor median nerve in leprosy patients and controls. Mean distal motor latency in leprosy patients was 3.29±1.03 and in control group it was 3.45±0.43. By using Student's unpaired t test statistically no significant differenece was found in motor median nerve in patients of both the groups(t=0.66, p=0.51).

Mean amplitude in leprosy patients was 6.74±2.37 and in Control group it was 12.87±4.58. By using Student's unpaired t test statistically significant differenece was found in motor median nerve in patients of both the groups(t=4.45, p=0.0001).

Mean conduction velocity in leprosy patients was 53.53±11.81 and in Control group it was 57.03±4.51. By using Student's unpaired t test statistically no significant differenece was found in moor median nerve in patients of both the groups(t=1.25, p=0.21).

Table 2 reveals comparison of motor ulnar nerve in leprosy patients and control group. Mean distal motor latency in leprosy patients was 2.51±1.64 and in Control group it was 2.46±0.43. By using Student's unpaired t test statistically no significant differenece was found in motor ulnar nerve in patients of both the groups(t=0.13, p=0.89).

Mean amplitude in leprosy patients was 7.86±4.72 and in Control group it was 13.45±2.19. By using Student's unpaired t test statistically significant differenece was found in motor ulnar nerve in patients of both the groups(t=4.77, p=0.0001).

Mean conduction velocity in leprosy patients was 49.22±20.83 and in Control group it was 59.61±3.56. By using Student's unpaired t test statistically significant differenece was found in motor ulnar nerve in patients of both the groups(t=2.30, p=0.028).

DISCUSSION

The destructive capability of granulomatous inflammation which is present in the leprosy is well known and has often been accepted as the basic explanation for nerve injury in leprosy patients.

The evaluation of electrophysiological study of nerve conduction is assessed by three criteria, i.e., conduction velocity, amplitude, and distal motor latency.

In the preclinical stage of the leprosy, where there are no signs and symptoms suggestive of nerve damage, slowing of motor nerve conduction velocity has been observed. This hidden stage of neural deficit escapes early and timely detection and later progress to manifested disease when certain defined quantum of nerve fibers becomes nonfunctional. [12] Since it is the fast conducting fibers that are taken into account while calculating nerve conduction velocities, and the results may differ if slow conducting fibers are predominantly damaged. [13] In the present study it was observed that the 13 patients had impaired mean nerve conduction velocities along with reduced mean amplitude and no changes in mean latencies in all the cases.

In yet another case–control study by BK Gupta and DK Kochar on leprosy patients in Bikaner, motor nerve conduction velocity was found to be reduced in more number of patients.

Thus, we conclude that nerve conduction studies are reliable diagnostic and prognostic indicators useful in leprosy especially in areas that are endemic for the disease like ours and we are conducting further research work in the field with the use of various electrophysiological studies in this regard.

CONCLUSION

From the current study we conclude that nerve conduction studies are reliable diagnostic and prognostic good indicators of diagnosis in leprosy especially in areas that are endemic for the disease and we are conducting further research work in the field with the use of various electrophysiological studies.

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