Original Research Article

Effect of leprosy on median, ulnar, radial and facial nerve conduction

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Abstract

Background and aims: Leprosy causes a stage of functional blockade of conduction of nerve impulse in peripheral and cranial nerves. We failed to find studies focusing on this issue in leprosy patients of vidarbha region. Thus the present study aims to see difference between nerve conduction viz sensory and motor, parameters between newly diagnosed leprosy patients and age gender matched controls. **Methods:** This was a cross-sectional research carried out in randomly selected 30 newly diagnosed leprosy patients of age between 20 to 50 years and 30 age- gender matched controls. We studied distal motor latency, compound muscle action potential amplitude, motor nerve conduction velocity, F-minimum latency, sensory onset latency, sensory nerve action potential amplitude and sensory nerve conduction velocity in bilateral median, ulnar, radial and facial nerves using Aleron-RMS. **Results:** We found statistically significant reduction in CMAP, MNCV, SNAP and SNCV of bilateral median and ulnar nerves in cases as compared to controls. We also found statistically significant prolongation of F minimum latency in cases as compared to controls in above stated nerves. CMAPs of bilateral radial and facial nerves were reduced in cases compared to controls however they were not statistically significant. **Conclusions:** We found statistically significant difference between motor and sensory conduction of bilateral median and ulnar nerves in newly diagnosed leprosy patients and controls. Bilateral radial and facial motor nerve conduction also showed reduced amplitudes in leprosy patients however they were not statistically significant. Newly diagnosed leprosy patients showed mixed sensory motor axonal demyelinating pattern in upper limb nerve conduction.

Key words: compound muscle action potential, distal motor latency, mixed sensory motor axonal demyelinating polyneuropathy, neurophysiology, sensory nerve action potential,

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INTRODUCTION

Leprosy is the most common cause of treatable peripheral neuropathy in India. Peripheral neuropathy in leprosy varies from as small as, involvement of an intradermal nerve in the cutaneous patches to as large as major lesion in the peripheral or cranial nerve trunk.[1] This silent neuropathy starts with blockade of nerve impulse and then leads to visible pathological changes.[2] Cranial nerve involvement like facial, trigeminal or other nerves is also commonly seen in patients with leprosy. Early diagnosis and treatment of leprosy induced nerve damage with steroids is many times helpful in complete restoration of nerve function.[3] Prevalence of leprosy per 10,000 was found more in Vidarbha region, particularly tribal districts and areas, like ours.[4] Leprosy affects all physiological functions of peripheral nerves like sensory, motor and autonomic. Amongst these sensory functions are most severely affected. [5]Electrophysiological studies have now become recognized as early aids in assessing peripheral nerve dysfunction in leprosy even before appearance of clinical symptoms. Many studies have been conducted to relate a different patterns of nerve damage in different forms of leprosy. They found both axonal loss and demyelination nerve damage pattern on nerve conduction study.[5] However, we failed to find any such study focusing this issue in our local population. Thus present study aims to observe the difference between nerve conduction parameters viz motor and sensory in

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leprosy patients and controls. Our objective was to study median, ulnar, radial and facial nerve conduction in newly diagnosed leprosy patients from our local population.

METHODS

This was a cross-sectional research carried out in randomly selected 30 newly diagnosed leprosy patients of age between 20 to 50 years at our tertiary care teaching hospital, Skin outpatient department (OPD). We also enrolled, 30 age-gender matched, apparently healthy controls, who volunteered to participate in this research and were ready to give written informed consent. We carried out this study in neurophysiology OPD of our tertiary care teaching hospital, only after approval from institutional ethics committee. The mean age of cases and controls were 39.57 ± 2.032 yrs and 38.60 ± 1.880 yrs respectively. We excluded the patients suffering from Diabetes, Hypertension, Peripheral neuropathy, Rheumatoid arthritis and fracture at wrist joint which may affect our study parameters. Our study parameters were distal motor latency (DML), compound muscle action potential amplitude (CMAP), motor nerve conduction velocity (MNCV) and F-minimum latency in bilateral median and ulnar nerves to study the motor nerve conduction. Also we studied sensory latency, sensory nerve action potential amplitude (SNAP) and sensory nerve conduction velocity (SNCV) in bilateral median and ulnar nerves. In addition, we also recorded radial motor nerve conduction parameters like DML, CMAP and MNCV and facial nerve parameters like bilateral CMAP. We recorded symptoms/complaints of patients, anthropometric parameters like height and weight of each patient and controls, entered the data in pre-designed case record forms (CRF) as well as in computer attached with RMS-Aleron machine.

Measurement of study parameters

For motor conduction, the gain was set at 2-5mV per division. The active electrode was placed on the centre of muscle belly and the reference electrode was placed distally on the tendon. Duration of pulse was set to 100μs and current 50-100mA for stimulation.[6] For sensory conduction, the gain was set at10-20 μV per division. A pair of ring electrodes was placed in line over the nerve at an inter-electrode distance of 3-4 cm. The active electrode placed closest to the stimulator. As sensory fibres have low threshold to stimulation current used was in the range of 5-30mA and duration for 100μs. We performed antidromic sensory conduction studies using ring electrodes.[6,7] Median motor nerve conduction parameters- Recording electrodes were placed over Abductor pollicis brevis muscle (lateral thenar eminence). Stimulation performed at two sites viz middle of the wrist and antecubital fossa. We recorded two waves at two sites of stimulation. But for our study we took only distal (wrist) stimulation wave parameters viz distal motor latency (DML) in milliseconds and compound muscle action potential amplitude (CMAP). We calculated motor nerve conduction velocity by entering distance between two stimulation points. F wave minimum latency (in milisec) recorded by stimulating at wrist and recording 8-10 waves on a rastered trace.

Median sensory nerve conduction parameters- A pair of ring electrodes were placed over second digit and stimulation was performed at middle of wrist with slowly increasing current from 0-50mA till we obtained a waveform. We recorded onset latency (milliseconds) and sensory nerve action potential amplitude (μV) (SNAP). We calculated sensory nerve conduction velocity by entering distance between active electrode and stimulator.[6,7]

Ulnar motor nerve conduction parameters- Recording electrodes were placed over abductor digiti minimi muscle (hypothenar eminence) and stimulation at medial wrist and below elbow. We calculated motor nerve conduction velocity of ulnar nerve by putting distance between elbow and wrist.

Ulnar sensory conduction parameters- Ring electrodes were placed over fifth digit and stimulation performed at medial wrist.

Radial motor nerve conduction parameters- Active electrode was placed after applying jelly over extensor indicis proprius. The reference surface electrode was put over styloid process of wrist. Ground was placed in between the two electrodes. The stimulation was given at two sites, first at the forearm and secondly at the spiral groove. MNCV was calculated from two stimulation points. Both hands were stimulated to obtain parameters.[6,7]

Facial motor nerve conduction – We placed active electrode on nasalis muscle for sharper take off of CMAP and reference electrode was put on the bridge of the nose. The ground electrode was placed on base of neck and stimulus was given just below the earlobe anterior to it. The gain was 1-2 mV/ division and sweep time was set at 2ms/division.[7]

Statistical analysis

Being a pilot study at our center, we chose a sample size of 30 newly diagnosed leprosy patients and 30 controls. We compared the means of all the study parameters for median, ulnar, radial and facial nerves by Mann-Whitney test using Graph pad prism software ver 5.01. We also compared F min latencies of both median and ulnar nerves with controls. 'p' value of less than 0.05 was considered as statistically significant.

RESULTS

The mean ages of leprosy cases and controls were 39.57 ± 2.032 years and 38.60 ± 1.880 years. The bilateral upper extremities of the participating subjects were evaluated electrophysiologically. Table 1 shows that, there was statistically significant reduction in CMAP, MNCV, SNAP and SNCV of right median nerve of leprosy cases as compared to controls. We also found statistically significant prolongation of F minimum latency. DML and SL were least affected.

Table 1: Comparison of study parameters in Right Median nerve

Study parameters	Cases(n=30) (mean±SEM)	95%CI	Controls (n=30) (mean±SEM)	95%CI	p value
DML(ms)	3.262±0.1847	2.885- 3.640	2.887±0.09154	2.699-3.074	0.1348
CMAP(mv)	10.19±0.9386	8.267- 12.11	15.72±0.9568	13.76-17.67	0.0004
MNCV(m/s)	47.37±1.522	44.26-50.48	53.48±0.9509	51.53-55.42	0.0023
F min L(ms)	28.32±0.7776	26.73- 29.91	24.76±0.8185	23.09- 26.43	0.0028
SL(ms)	2.504 ± 0.1177	2.264-2.745	2.348 ± 0.04657	2.252-2.443	0.4917
SNAP(µV)	20.65 ± 2.714	15.10- 26.20	39.51 ± 2.380	34.65-44.38	P<0.0001
SNCV(m/s)	40.82 ± 2.024	36.68-44.96	48.39 ± 1.094	46.15-50.63	0.0007

DML- distal motor latency; CMAP- compound muscle action potential amplitude; MNCV- motor nerve conduction velocity; SL- Sensory onset latency; SNAP- sensory nerve action potential amplitude; SNCV- sensory nerve conduction velocity. Similar to right median nerve, table 2 shows that, there was statistically significant difference in all study parameters of left median nerve except DML, SL and SNCV.

Table 2: Comparison of study parameters in Left Median nerve

Study parameters	Cases (n= 30) mean±SEM	95% CI	Controls (n= 30) mean±SEM	95%CI	p value
DML (ms)	3.090 ± 0.2047	2.671- 3.508	2.780 ± 0.06432	2.648- 2.911	0.1805
CMAP (mv)	9.854 ± 0.8877	8.038- 11.67	15.83 ± 0.8500	14.09-17.57	P<0.0001
MNCV (m/s)	45.76 ± 1.996	41.68-49.84	53.06 ±1.108	50.80-55.33	0.0023
F min L (ms)	27.58 ±0.6795	26.19- 28.97	25.55 ± 0.3006	24.94-26.16	0.0338
SL (ms)	2.562 ± 0.1144	2.328- 2.796	2.332 ±0.05937	2.211-2.453	0.2674
SNAP (µv)	20.16 ± 2.956	14.11- 26.20	40.42 ± 2.107	36.11-44.73	P<0.0001
SNCV (m/s)	43.19 ± 2.844	37.38-49.01	49.72 ±1.423	46.81-52.64	0.1413

DML- distal motor latency; CMAP- compound muscle action potential amplitude; MNCV- motor nerve conduction velocity; SL- Sensory onset latency; SNAP- sensory nerve action potential amplitude; SNCV- sensory nerve conduction velocity

There was statistically significant difference in all parameters of right ulnar nerve as shown in Table 3. DML, SL and F-min L were prolonged in leprosy cases, and CMAP, SNAP, SNCV and MNCV were decreased significantly.

Table 3: shows comparison of study parameters in right ulnar nerve

Study parameters	Cases (n=30) mean±SEM	95% CI	Controls (n=30) mean± SEM	95% CI	p value
DML (ms)	3.068 ±0.4041	2.242- 3.895	2.062 ± 0.1505	1.754-2.369	0.0162
CMAP (mv)	7.350 ±0.6972	5.924-8.776	12.69 ± 0.8210	11.01-14.37	P<0.0001
MNCV(m/s)	43.97 ±2.134	39.61-48.34	52.50 ±1.298	49.85-55.16	0.0014
F min L (ms)	29.67 ±1.013	27.60-31.74	26.27 ±0.3246	25.61-26.93	0.0112
SL (ms)	2.951 ±0.1695	2.604-3.297	2.168 ±0.05312	2.059-2.276	P<0.0001
SNAP(µV)	12.68 ±2.549	7.466-17.89	31.48 ±2.290	26.80-36.16	P<0.0001
SNCV(m/s)	33.17 ± 2.581	27.89-38.45	48.42 ±1.112	46.14-50.69	P<0.0001

DML- distal motor latency; CMAP- compound muscle action potential amplitude; MNCV- motor nerve conduction velocity; SL- Sensory onset latency; SNAP- sensory nerve action potential amplitude; SNCV- sensory nerve conduction velocity

In the study parameters of Left ulnar nerve, there were statistically significant prolongation of distal motor latencies, F minimum latencies and sensory onset latencies. (Table 4) We also found statistically significant reduction in both motor and sensory amplitudes. Motor as well as sensory nerve conduction velocities were diminished markedly in cases as compared to controls.

Table 4: shows comparison of study parameters in left ulnar nerve

Study Parameters	Cases (n=30) mean±SEM	95% CI	Controls (n=30) mean±SEM	95% CI	p value
DML (ms)	3.208 ± 0.3802	2.431-3.986	2.014 ± 0.1485	1.710- 2.317	0.0082
CMAP (mv)	7.723 ± 0.5925	6.512 - 8.935	13.08 ± 0.8076	11.43- 14.73	P<0.0001
MNCV (m/s)	46.21 ±2.121	41.87- 50.55	52.22 ±1.163	49.84- 54.60	0.0219
F min L (ms)	29.30 ±0.9128	27.43 - 31.17	25.91 ±0.3198	25.25- 26.56	0.002
SL(ms)	2.610 ±0.1435	2.317 - 2.904	2.187 ±0.08949	2.004 - 2.370	0.0344
SNAP (µv)	17.77 ±2.988	11.66 - 23.88	30.98 ± 2.185	26.51- 35.45	0.0038
SNCV (m/s)	37.81 ± 3.032	31.61 - 44.01	49.65 ±1.409	46.77 - 52.54	0.0156

DML- distal motor latency; CMAP- compound muscle action potential amplitude; MNCV- motor nerve conduction velocity; SNAP- sensory nerve action potential amplitude; SNCV- sensory nerve conduction velocity

There was statistically significant difference in motor amplitude and motor nerve conduction velocity except DML. (Table 5)

Table 5: Comparison of study parameters in Right radial nerve

Study parameters	Cases (n= 30) mean±SEM	95% CI	Controls (n=30) mean± SEM	95% CI	p value
DML (ms)	2.022 ±0.1194	1.778 - 2.266	1.890 ±0.1255	1.633 - 2.147	0.5575
CMAP (mv)	4.717 ± 0.2620	4.181 - 5.253	6.180 ±0.3186	5.528 - 6.832	0.0007
MNCV(m/s)	49.51 ±1.743	45.94 - 53.07	58.18 ±1.900	54.30 - 62.07	0.0014

DML- distal motor latency; CMAP- compound muscle action potential amplitude; MNCV- motor nerve conduction velocity.

DML in left radial nerve in leprosy patients was comparatively increased and MNCV was decreased, however the differences were not statistically significant. (Table 6)

Table 6: Comparison of study parameters in left radial nerve

Study parameters	Cases (n=30) mean±SEM	95% CI	Controls (n=30) mean±SEM	95% CI	p value
DML (ms)	2.150 ±0.1405	1.863 - 2.438	1.804 ±0.1141	1.571 - 2.038	0.1085
CMAP (mv)	5.384 ±0.6384	4.079 - 6.690	5.297 ± 0.2734	4.738 - 5.856	0.3214
MNCV (m/s)	48.67 ±1.579	45.44 - 51.90	52.08 ±1.287	49.45 - 54.71	0.3254

DML- distal motor latency; CMAP- compound muscle action potential amplitude; MNCV- motor nerve conduction velocity.

There was no statistically significant difference in motor amplitudes of facial nerve, bilaterally. (Table 7)

Table 7: Comparison of CMAPs in facial nerve

Study Parameters	Cases (n= 30) mean± SEM	95% CI	Controls (n=30) mean± SEM	95% CI	p value
Right CMAP (mv)	1.220 ± 0.07632	1.064 - 1.376	1.307 ±0.04888	1.207- 1.407	0.6915
Left CMAP (mv)	1.240 ±0.07529	1.086- 1.394	1.243 ± 0.03886	1.164- 1.323	0.8106

CMAP- compound muscle action potential amplitude.

Right ulnar nerve was the most affected among all upper extremity nerves. Both motor and sensory fibres were affected. Distal latencies were prolonged, amplitudes were significantly reduced and nerve conduction velocities were also decreased. Thus mixed sensory motor axonal-demyelinating type of neuropathy was observed.

DISCUSSION

We conducted nerve conduction studies on 30 newly diagnosed leprosy patients and 30 healthy controls. We found changes in almost all sensory and motor nerve conduction parameters in median and ulnar nerves in leprosy patients. Motor conduction parameters like DML showed prolongation, CMAP amplitudes were reduced and MNCV was decreased. Sensory conduction parameters like onset latencies were prolonged and SNAP amplitudes and SNCV were reduced in both median and

ulnar nerves in leprosy cases. Radial and facial motor nerve conduction showed minor changes which was not statistically significant. Peripheral nerves are involved early in leprosy than cranial nerves due to their superficial anatomical course. Cooler temperature facilitates multiplication of lepra bacilli at these sites. Antigens released by live or dead lepra bacilli cause nerve damage in leprosy.[2,8] Sensory and slow conducting fibres of mixed nerves are involved early as compared to motor fast conducting myelinated fibres. [9] This results in prolongation of latencies in sensory and motor nerves and subsequently leads to slowed conduction velocity.[10]Axonal loss, is shown in NCS as reduction in amplitudes, while demyelinating is represented as prolonged latencies and reduced conduction velocities. Present study showed marked changes in all nerve conduction parameters in median and ulnar nerves which are suggestive of mixed axonal-demyelinating type of neural damage of both sensory and motor fibres. Similar findings were observed by Kar *et al*, Maharatta *et al*, Chaurasia *et al*, Husain *et al*.[1,2,5,9,11] There are certain causes for the occurrence of multiple changes in NCS, even in newly diagnosed cases at our center, viz. prevalent illiteracy among study population and attached social stigma with the disease, making the presentation late. Otherwise Hussain *et al* states that, Axonal damage precedes, the demyelination in leprosy patients, however mixed type is the most common presentation.[2,5,9] Right radial nerve showed reduced amplitude and conduction velocities on motor nerve conduction. Van Brakel *et al* studied radial nerve conduction in leprosy patients. They found that index branch of the radial cutaneous nerve was also involved in leprosy cases and observed impaired sensory nerve conduction.[12]We also studied bilateral facial nerves of leprosy patients and found reduced CMAP as compared to controls but results were not statistically significant. Reichart PA *et al* stated that zygomatic branch of facial nerve was most frequently affected and it was involved late in the disease which had an average duration of 12.1 years.[13] However present research dealt with newly diagnosed cases, we could not find the similar observations.

Limitations of the study

Being a pilot research, the sample size was insufficient to achieve desired power, to find all appropriate observations. Our study was also limited to very small geographical area. Hence more such electrophysiological studies need to be conducted/repeated with larger sample size.

CONCLUSION

We observed that leprosy as a disease affects nerve conduction studies in many ways. Median and ulnar nerve motor conduction showed prolongation of distal motor latencies, reduced compound muscle action potential amplitudes and decreased conduction velocities. We found prolonged latencies, reduced sensory nerve action potential amplitudes and decreased conduction velocities on sensory conduction of both nerves. Radial and facial nerves also showed reduced compound muscle action potential amplitudes in leprosy patients which were not statistically significant. Leprosy causes mixed sensory-motor axonal demyelinating changes on nerve conduction study in bilateral median and ulnar nerves.

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