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Ulnar and median nerves in paucibacillary leprosy—a follow-up study of electrophysiological functions in patients before and after nerve trunk decompression

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Summary Electrophysiological functions of ulnar and median nerves in paucibacillary leprosy patients were studied. Patients who showed deterioration of sensory motor functions in spite of steroid therapy were offered nerve decompression together with oral steroids. On periodic follow-up of those who opted for surgery, it was observed, in general, that NCV and amplitude remained reduced even though clinical recovery occurred. Only 80% recovery of electrophysiological functions was seen (as compared to control levels), even in cases that showed good results. Motor function recovered better than sensory function. Complete electrophysiological recovery, if it occurs at all, takes much longer than clinical recovery.

Introduction

In leprosy, peripheral nerve involvement may vary from involvement of intradermal nerves in a cutaneous patch to a major lesion in the peripheral nerve trunk.[1,2] The nerve lesion may be insidious without any clinical manifestations, with mild clinical manifestations, or a sudden event, especially during reactions. Therapy may arrest the progress of neural damage and in several cases clinical recovery can occur.

Enhanced latencies and reduced sensory–motor conduction velocities and amplitudes have been observed, depending upon the extent of neural damage. If recovery occurs, these electrophysiological functions are expected to return to normal or near normal levels.

There are very few serial follow-up studies of electrophysiological functions in leprosy affected nerves.[3,4] The present study was undertaken to examine the electrophysiological behaviour of ulnar and median nerves trunks in paucibacillary (PB) leprosy before and after nerve trunk decompression.

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Materials and methods

PB leprosy patients attending the OPD at Central Jalma Institute for Leprosy, several of whom had some neurological symptoms related to ulnar and median nerve trunks, were included in the study. Most of the patients had been referred to the surgical unit by physicians, and only a few cases were self-referred. PB and multibacillary (MB) differentiation was performed as described previously.[5] Detailed physical and neurological evaluation was carried out using standard procedures to exclude cases having conditions other than leprosy as a cause of neuropathy. Palpation of nerve trunks was done by the same two persons independently to confirm the thickening of nerve trunks in early cases.

The nerves were clinically involved, thickened but non- tender. Fifty-nine cases had ulnar and 22 had median nerve involvement, and all were receiving MDT. Those with clinically manifest neural damage were also receiving oral corticosteroid therapy for varying periods at the time of their first electrophysiological evaluation. The age of the patients varied from 15 to 60 years (mean 35 years), the majority being males.

Since the study was retrospective, no specific study design could be made. Patients were classified, on the basis of clinical observations, as shown below. Twenty age- and sex-matched healthy controls for ulnar and 10 for median nerves were also included in the study.

GROUP 1: ULNAR NERVE

1. Group 1A. Disease duration 6 months or less; no neurological symptoms or clinical neurological deficit attributable to ulnar nerve trunk involvement (17 cases).
2. Group 1B. Neurological symptoms >6 months (up to 9 months); sensory loss in nerve trunk innervated area; muscle strength MRC grade 3–4 (20 cases).
3. Group 1C. Neurological symptoms of >9 months (up to 12 months); sensory loss in nerve trunk innervated area; Muscle strength MRC grade 1–2 (22 cases).

GROUP 2: MEDIAN NERVE

This group was further subdivided into groups 2A, 2B and 2C based on same criteria as for group 1.

ELECTROPHYSIOLOGICAL STUDIES

The recordings for sensory–motor nerve conduction velocities were done on a 4-channel EMG machine (Neuropack-2; Nihon Kohen Corporation, Japan). Standard procedures were used for stimulation and recording as described in the accompanying manual. During winter, the patients were acclimatized (room temperatures around 25°C) before recordings. Repeat examination was done every time under similar environmental conditions as far as possible.

Patients were informed regarding their nerve function status. The treatment modalities, including combined medical and surgical treatment available, were also explained to them. Some of those patients who opted for nerve decompression were operated on (by the same surgeon) and maintained postoperatively on daily doses of 10 mg prednisone, 300 mg aspirin and MDT. The operative procedure comprised complete de-roofing of the osseo-fascial cubital tunnel, along with epineurotomy. Since the treatment modalities are well known and have been in use over a long period, and the patients were free to choose their options, ethical

issues were not involved. The criteria for clinical improvement included symptomatic relief from pain and paraesthesia, improvement in sensory perception as tested with pin prick and tip of ball point pen, and improved muscle strength on MRC scale on manual testing.

Results

Of the cases included in the study, 59 had ulnar and 22 median nerve involvement. The observations made on ulnar nerves are shown in Table 1.

In group 1A (no clinical neurological deficit), 10 (of 17) nerves initially showed more or less normal sensory–motor nerve conduction velocities, latencies and amplitudes. On subsequent follow-up, three patients complained of pain in the cubital region and paraesthesia in the distribution of ulnar nerve and were put on 20 mg of steroids, which was gradually reduced to 10 mg over 3 months. Since their muscle strength deteriorated to MRC grade 3 and they developed complete sensory loss, cubital tunnel decompression and epineurotomy was performed. Two of these three cases improved in motor power and their electrophysiological functions returned to near normal (Table 1). One patient gradually deteriorated. His muscle strength fell to zero and nerve conduction velocities (NCV) were also reduced to a great extent.

Seven cases (of 17) in this asymptomatic group showed marked electrophysiological changes on first evaluation, with sensory conduction velocities (SCNV) much more reduced than motor conduction velocities (MNCV) and also in comparison to the controls. Five of these had nerve pain and deteriorated clinically (palmar anaesthesia along with deterioration of motor power to grade 3) at about 3 months and underwent surgery. Postoperatively pain subsided in all cases, but only three patients regained muscle power of grade 4+ and sensations; the other two deteriorated further to MRC grade 0. The SCNV and MCNV improved over its initial values, but did not return to normal (Table 1). The remaining two cases, who remained asymptomatic, showed no change in nerve function on subsequent follow-ups.

In group 1B (muscle power grade 3–4 and sensory loss), the electrophysiological functions showed marked changes. Of the 20 cases in this group, 17 cases underwent surgery. Of the operated cases, six regained near normal muscle strength and sensations over 12–15 months, their MNCV returned to normal and SNCV recovered to 90% of control values. The motor and sensory amplitudes not only recovered, but also gained 15% over the controls. Sensory–motor latencies increased to reach 90% of control (Table 2). Five cases did not gain any muscle strength but did not deteriorate either, and there was subjective improvement in sensory functions. The remaining six cases deteriorated to MRC grade 0–1 with complete sensory loss, and deterioration of electrophysiological functions.

Of the 22 cases in group 1C (with more or less complete sensory-motor loss), 15 opted for surgery and only four regained MRC grade 3. Sensory–motor latencies, amplitudes and NCV improved, but could not be restored to normal levels. Eleven cases remained at the preoperative levels of strength (MRC 1–2), but even in these patients, NCV were not zero (Table 2).

The median nerve involvement in different groups (in carpal tunnel) showed similar patterns of recordings preoperatively (Table 3).

Of the three cases in group 2A who showed electrophysiological changes at the time of first examination, one was not available for follow-up. The other two, who later complained

Table 1. Electrophysiological functions of ulnar nerve in clinically asymptomatic group before and after nerve trunk decompression. Postoperative observations only in those cases that improved on nerve decompression

			Group 1A									
			Ten ulnar nerves in PB cases < 6 months disease without any neurological symptoms and no EMG changes; 3 operated due to clinical deterioration of which two improved and one deteriorated					Seven ulnar nerves in PB cases < 6 months disease with out any neurological symptoms ; have EMG changes; 5 operated due to clinical deterioration of which three improved and 2 deteriorated				
					Improved cases $n = 2$					Improved cases $n = 3$		
					Postoperative					Postoperative		
Electrophysiological functions		Control	Initial	At time of operation	4–6 months	7–9 months	12–15 months	Initial	At time of operation	4–6 months	7–9 months	12–15 months
Motor	Mean NCV1 m/s	60·65	56·23	38·15	40·37	46·5	48·77	50·65	32·5	40·5	46·25	50·70
	% Change	100	92·71	62·90	66·56	76·67	80·41	83·51	53·59	66·78	76·26	83·59
	Mean AMP mV	6·6	6·77	4·14	4·58	4·81	5·45	4·67	3·60	4·25	4·90	5·60
	% Change	100	102·58	62·73	69·39	72·88	82·58	70·76	54·55	64·39	74·24	84·85
	Mean LAT ms	9·15	8·33	12·5	10·80	10·25	9·80	10·62	12·85	10·85	10·35	9·95
	% Change	100	91·04	136·61	118·03	112·02	107·10	116·07	140·44	118·58	113·12	108·74
Sensory	Mean NCV2 m/s	63·5	60·26	30·6	35·47	46·48	47·25	48·5	28·5	35·97	46·98	54·25
	% Change	100	94·90	48·19	55·86	73·20	74·41	76·38	44·88	56·65	73·98	85·43
	Mean AMP mV	28·95	27·3	20·36	22·36	24·65	25·35	21·5	18·25	20·36	23·65	23·75
	% Change	100	94·30	70·33	77·24	85·15	87·57	73·27	63·04	70·33	81·69	82·04
	Mean LAT ms	6·35	7·15	11·6	10·25	8·95	8·25	8·25	12·5	12·10	10·55	9·15
	% Change	100	112·6	182·68	161·42	140·95	129·92	129·92	196·85	190·55	166·14	144·10

Table 2. Electrophysiological functions of ulnar nerve in clinically symptomatic group before and after nerve trunk decompression. Postoperative observations only in those cases that improved on nerve decompression

			Group 1B				Group 1C			
			Twenty ulnar nerves in PB cases with motor power nerves 3+ and anaesthesia; 17 cases were operated, 6 improved, 5 remained status quo, 6 deteriorated				Twenty-two ulnar nerves in PB cases with motor power 1+ to 2+ and anaesthesia; 15 cases operated, 4 improved to 3+, 11 remained status quo			
				Improved cases $n = 6$				Improved cases $n = 4$		
				Postoperative				Postoperative		
Electrophysiological functions		Control	At time of operation	4–6 months	7–9 months	12–15 months	At time of operation	4–6 months	7–9 months	12–15 months
Motor	Mean NCV1 m/s	60·65	30·15	34·18	41·3	63·85	20·15	24·12	29·52	33·5
	% Change	100	49·71	56·36	68·10	105·28	33·22	39·77	48·67	55·24
	Mean AMP mV	6·6	2·90	3·80	5·20	6·15	2·10	2·93	3·66	3·89
	% Change	100	43·94	57·58	78·79	93·18	31·82	44·39	55·46	58·94
	Mean LAT ms	9·15	12·85	11·15	10·85	10·5	17·25	15·48	13·48	11·10
	% Change	100	140·44	121·86	118·58	114·75	188·53	169·18	147·32	121·31
Sensory	Mean NCV2 m/s	63·5	31·75	36·70	43·95	56·25	13·25	22·5	28·35	35·75
	% Change	100	50·0	57·80	69·21	88·58	20·87	35·43	44·65	56·30
	Mean AMP mV	28·95	20·65	22·5	25·15	26·3	10·25	14·20	16·25	18·60
	% Change	100	71·33	77·72	86·87	90·85	35·41	49·05	56·13	64·25
	Mean LAT ms	6·35	11·65	11·15	9·25	8·25	19·15	16·70	15·15	12·75
	% Change	100	183·46	175·59	145·67	129·92	301·58	262·99	238·58	200·79

Table 3. Electrophysiological functions of median nerve in clinically symptomatic group before and after nerve trunk decompression. Postoperative observations only in those cases that improved on nerve decompression

Electrophysiological functions		Control	Group 2B: Eight cases of median nerves in PB cases; 3+ motor power with anesthesia; 6 cases operated 3 improved to 5; 1 status quo and 2 deteriorated				Group 2C: Five cases of median nerves in PB cases with motor power 1+ to 2+ with anesthesia; 3 cases operated, 1 improved to 3+ and 2 remained status quo			
				Improved cases $n = 3$				Improved cases $n = 1$		
				Postoperative				Postoperative		
			At time of operation	4–6 months	7–9 months	12–15 months	At time of operation	4–6 months	7–9 months	12–15 months
Motor	Mean NCV1 m/s	59·6	26·5	33·78	40·80	44·5	12·5	18·5	23·25	25·75
	% Change	100	44·46	56·68	68·46	74·66	20·97	31·04	39·01	43·21
	Mean AMP mV	7·76	3·87	5·23	6·15	6·75	2·42	2·85	4·15	3·80
	% Change	100	49·87	67·40	79·25	86·99	31·19	36·73	53·48	48·97
	Mean LAT ms	4·76	6·65	5·85	5·31	5·25	16·80	14·90	10·85	9·5
	% Change	100	139·71	122·90	111·56	110·29	352·94	313·03	227·94	199·58
Sensory	Mean NCV2 m/s	59·43	19·4	33·58	43·60	47·12	10·25	17·5	22·25	30·50
	% Change	100	32·64	56·50	73·36	79·29	17·25	29·50	37·44	51·32
	Mean AMP mV	42·26	20·5	26·22	30·75	35·15	8·80	12·50	16·75	22·75
	% Change	100	48·51	62·05	72·76	83·18	20·82	29·58	39·64	53·83
	Mean LAT ms	3·5	5·75	5·10	4·70	4·20	11·60	10·25	8·70	7·25
	% Change	100	164·29	145·71	134·29	120·0	331·43	292·86	248·57	207·14

of pain and swelling of the median nerve at the wrist, were put on 20 mg of steroids and observed for 3 months. The swelling increased in size, pain also increased and thumb and index finger became anaesthetic. These two cases underwent surgery and a nerve abscess was evacuated. They were subsequently put on 10 mg prednisolone along with isoniazid 300 mg and rifampicin 450 mg daily for 6 months. The pain subsided, sensory loss and conduction velocities improved.

Six of the eight cases in group 2B opted for nerve decompression, of which three regained normal muscle strength and improved sensations. Sensory–motor NCV improved to 80% and amplitudes to about 85% of the control values. Latencies also improved but remained enhanced by 10–20% (Table 3). Two cases deteriorated to MRC grade 1+ to 0, and one did not show any change. In group 2C, three cases opted for surgery but only one was able to gain muscle strength of 3+ and sensations, gaining only 50% in electrophysiological parameters (Table 3). Two cases remained at the preoperative levels of strength and one was not available for follow-up.

Discussion

Analysis of three basic parameters, NCV, latency and amplitude of the evoked response, helps in interpretation of observations made on nerve conduction studies.

Studies on leprosy patients have shown that in a significant proportion of cases, SNCV is at the lower limit of normal or slightly delayed while amplitude and duration of action potential is with in the normal range.[6,7] This suggests that the disease results in diffuse neuropathy even at an early stage where it cannot be detected by routine methods of clinical testing.

In another study, it was observed that, even though clinically normal, 16% of ulnar and 20% of median nerves were electrically abnormal. Since damage to C and A-delta fibres precedes involvement of A-alpha fibres in leprosy, increasing damage to C and A-delta fibres is accompanied by increasing A-alpha fibre involvement.[6,9] This means that a reduction of NCV indicates severe damage to A-delta and C fibres in leprosy, and as segmental demyelination progresses, a greater number of fibres are affected, resulting in reduced NCV due to distorted conduction along small segments of demyelination in the majority of fibres. It is therefore evident that for clinical impairment to occur, damage to the nerve has to be quite extensive.

Other groups of workers[2,3,8] have observed that normal sensory–motor NCV can be found in the diseased nerves, which could be explained by involvement of certain fascicles of the affected nerve with little or insignificant involvement of others. In early cases where damage is more in slow conducting fibres (average velocity fibres), change in NCV may not be marked.[8] Since NCV is calculated on the basis of fast conducting fibres, it may be normal if slow conducting fibres are predominantly damaged. The reduced NCV in cases with clinically normal nerve functions probably represents the preclinical stage of damage. The damage becomes manifest only when certain defined quanta of nerve fibres become non-functional.

The sensory fibres, mainly non-myelinated, are more prone to damage and therefore show a much wider spectrum of changes in NCV as compared to motor nerve fibres in the early stages of damage. However, amplitude changes are much more marked for motor nerve fibres. Slowing of MNCV has also been observed in patients without having any clinical abnormality.[3]

A significant reduction of NCV occurs in all forms of leprosy at a certain stage of disease. Since sensory potentials are obtained from afferent nerves supplying anaesthetic areas, a loss of sensation in leprosy does not necessarily mean destruction of all nerve fibres; probably the cutaneous afferents are much more affected than muscle afferents.[11]

The absence of detectable potentials from area showing complete loss of sensation may mean absence of myelinated fibres, failure of surviving fibres to be stimulated due to increased thresholds or a complete dispersion of waves. Even in the absence of sensations, action potentials could be recorded from nerve fibres supplying the area concerned. Therefore it is likely that sufficient fibres in the myelinated group survive to account for the presence of these potentials.

It is interesting to note that NCV never became zero, i.e. some conduction continued to occur even in cases showing no response on clinical testing for sensory–motor functions. This does not appear to be explained by volume conduction alone. Several workers have demonstrated healthy nerve fibres within anaesthetic areas.[10] This suggests that some nerve fibres continue to function and will probably be helpful in useful sensory recovery (protective sensations), provided an environment conducive to growth and function of existing nerve fibres can be supplied.

The significance of MNCV or SNCV as a prognostic tool can be demonstrated by the degree of improvement on treatment, which always precedes any clinical recovery.[4] Electrophysiological normality may not be restored (or probably takes much longer, even in cases with complete clinical recovery). It has been observed, in general, that even though clinical recovery occurs, NCV and amplitude remained reduced and best results showed recovery only up to 80% of control values. Motor latencies recovered better and reached 90% of control values, whereas sensory latencies reached about 80%. Since our follow-up was limited, it is possible that further improvement may occur in these functions with time.

Of the asymptomatic cases who had no electrophysiological impairment (10), three subsequently had nerve pain and showed deterioration. The other seven, asymptomatic but having electrophysiological changes, became symptomatic after about 3 months and manifested sensory–motor deficit. In group 1B, the symptomatic group, 17 of the 20 cases underwent surgery, following which one-third improved to normal, while another one-third deteriorated in spite of surgery and steroid therapy. In the remaining third, the process of damage was arrested.

Let us consider that the above two sets of situations exist, i.e. normal neurological function with clinically thickened nerves and thickened nerves with clinically manifest partial nerve functional impairment. The second group comprises cases that deteriorated under supervised treatment, probably due to multiple factors during the course of illness.

Since steroids take about 1 month to be really effective as an antiinflammatory agent, two options can be exercised:

1. Add steroids in low doses (about 10 mg prednisone equivalent) along with MDT to all PB cases for 6 months, or the total duration of MDT. Periodic monitoring of the immune status of patient against *Mycobacterium leprae* antigens and *M. leprae* modified neural antigens can be done if found practical, since a spurt in immunological function may be a sudden event.
2. Load these patients with heavy doses of steroids along with other effective quick acting immunosuppressants, so that immunosuppression is almost immediate if not instant. The

second option requires that patient has to be hospitalized. By the time he is transported from the field, the damage is likely already to have occurred.

Little can be done when complete palsy has developed. Only 20% cases improved with steroid therapy complimented with nerve decompression and could gain only some useful strength. Combined treatment can be tried if facilities are available.

It is difficult to decide whether or not a clinically enlarged nerve in a patient is necessarily abnormal and at risk. If the nerve is found to be enlarged but conduction studies are normal, a further period of observation is indicated.[8] This may not hold good in endemic situations where subclinical disease is known to exist. Such patients need monitoring, and to be put on appropriate treatment if required.

In patients with known leprosy having a normal clinical picture, abnormally slow conduction at the elbow may be an early sign of nerve involvement, thus being of some prognostic value. For these cases with neural inflammation, appropriate therapy can be started before the patient becomes disabled, which at times is sudden in onset. Such patients will be very few and can easily be monitored.

Even though the suggestion has often been made to conduct 'proper controlled studies', in practice this seems very difficult. To have identical groups is a distant dream, the situation being further complicated because of the possibility of a 'spontaneous recovery'. If facilities are available, high-risk group among asymptomatics can be identified by evaluating electrophysiological function, because fairly extensive large fibre damage has to occur for NCV changes to become manifest. Studies on refractory periods and differential evaluation of small and large fibres can be of help in this context, but because the procedure requires considerable technical expertise, it is beyond the purview of most field programmes, at least for the time being.

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