Electrophysiological Study of Nerves in Type-II Reaction in Leprosy

Santosh Kumar Singh, Sunil Kumar Gupta\(^1\), R D Mukhija, A K Thacker\(^2\)

**Abstract**

**Background:** Leprosy is a chronic granulomatous infection primarily affecting the peripheral nervous system, skin and reticuloendothelial system. Cutaneous nerves are severely affected in lepra reaction and this leads to morbidity. **Objective:** To study electrophysiological pattern of different nerves involved in Type-II reactions in leprosy. **Method:** The present study was undertaken in 21 leprosy patients with Type-II reactions attending in and out-patient department of Dermatology & Venereology, B.R.D. Medical College, Gorakhpur from July 2005 to October 2006. This was a prospective case control study in which 20 healthy, age and sex matched people with no evidence of any disease (particularly nerve involvement) were included. **Limitation:** Lesser number of cases were studied. **Result:** The proximal motor conduction latency was significantly prolonged in both ulnar and common peroneal nerve and proximal motor conduction velocity was also significantly reduced. On examining the values beyond 2S.D. of the control value, distal latency was not affected and only proximal conduction was affected in ulnar nerve. **Conclusion:** In Type II lepra reaction the motor conduction abnormalities are not prominent. Abnormalities are relatively more marked in the proximal segment.

**Key Words:** Distal latency, erythema nodosum leprosum, leprosy, proximal motor conduction velocity, Type-II lepra reaction

**What was known?**

Leprosy primarily affects peripheral nervous system. Early change is in nerve conduction velocity.

**Introduction**

Leprosy is one of the oldest diseases of humankind. The first authentic description of leprosy is given in Sushruta Samhita written in India in 600 B. C. The chronic and placid course of leprosy is punctuated by episodes termed as “reactions.”\(^1\) The clinical diagnosis and treatment of these reactions are of immense importance as these determine the final functional outcome, especially with reference to the nerves.\(^1\) The determination of status of nerves is, therefore, of great importance in the study of the reactions.

**Objective**

The objective of this study was to find out electrophysiological pattern of different nerves involved in Type-II reactions in leprosy.

**Materials and Methods**

The present study was undertaken in 21 leprosy patients with Type-II reactions attending in- and out-patient department of Dermatology and Venereology, B. R. D. Medical College, Gorakhpur from July 2005 to October 2006. Electrophysiological studies were conducted.

**Neuro-electrophysiological studies**

A detailed neurological examination was carried out to assess the nerve damage. If the nerves were tender and had evidence of nerve damage in the form of motor weakness or sensory loss, then it was taken as clinically affected nerve, and if the nerves were nontender and did not show any motor or sensory dysfunction, it was taken as clinically unaffected nerve. A complete neurological examination would be carried out to assess the nerve damage during ENL. Motor nerve conduction velocities proximal as well as distal of all possible nerves would be measured using Neuro Perfect EMG 2000. Care was taken to ensure that one of the clinically affected and one of the clinically unaffected nerves were included in each case.

This is an open access article distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 3.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as the author is credited and the new creations are licensed under the identical terms.

For reprints contact: reprints@medknow.com

How to cite this article: Singh SK, Gupta SK, Mukhija RD, Thacker AK. Electrophysiological study of nerves in type-II reaction in leprosy. Indian J Dermatol 2017;62:644-8.

Received: January, 2017. Accepted: October, 2017.
Distal conduction studies

The technique of mean corpuscular volume (MCV) was as follows: a coaxial needle electrode was inserted in the most distal muscle of the nerve to be examined such as abductor digiti minimi for the ulnar nerve, abductor pollicis brevis for the median nerve, extensor digiti minimi for the lateral popliteal nerve, and abductor hallucis muscle for posterior tibial nerve. All the nerves were stimulated at two places proximally and distally. The ulnar nerve was stimulated proximally at just above the medial epicondyle posteriorly and distally at wrist along the course of nerve. The median nerve was stimulated proximally at anterior aspect of antecubital fossa and distally at wrist along the course of nerve. The lateral popliteal nerve was stimulated proximally at the neck of fibula and distally at ankle joint anteriorly. The posterior tibial nerve was stimulated proximally posterior to medial malleolus and distally along the course of nerve.

After cleaning the skin, a supramaximal stimulus was applied to ensure activity in all functioning motor nerve fibers and was graded at one per second. It was applied through two electrodes place 18 mm apart along the nerve. The muscle action potential evoked by the stimulation was visually displayed on an oscilloscope, and the distal (DL) and proximal latencies (PL) were measured on the screen. The distance (d) between the stimulation points (proximal stimulation point to distal stimulation point) were measured with a measuring tape. The MCV was calculated using the formula $d/(DL-PL)$ in m/second for each nerve.

Proximal conduction studies by F-wave

F-wave studies were conducted in each patient in the same nerve, where the distal motor conduction was measured by the method described above. The nerve to be examined was stimulated by surface stimulation electrode dipped in normal saline. The recordings were made by needle electrodes placed at motor points of the relevant muscle. The ground electrode was placed between the stimulating and the recording electrodes. The stimulus frequency was one every second and the pulse was 0.1 ms. Supramaximal shocks were delivered to the nerve at the elbow for the upper limb and at the knee for lower limb respectively by adjusting the voltage to be 20% more than the required to produce a maximal direct motor response. Multiple F-wave at a sweep speed of 5 m s/division and sensitivity of 200 or 100 uv/division were recorded and the latency was measured individually. Latencies were uniformly measured to the beginning of the first deflection from the baseline. Ten F-responses were recorded, and the minimum latency of these (FL) was used to provide information regarding the fastest conducting motor nerve fiber, along the proximal segment. The proximal conduction velocity (FCV) was calculated using the formula $FCV = d × 2/(FL-PL)$; where the “$d$” is the distance between the proximal site of stimulation and C7 spinous process in upper limb and T12 spinous process in the lower limb.

Twenty healthy, age- and sex-matched persons with no evidence of any disease, involving the peripheral nerves with no history of any prolonged drug therapy were subjected to the motor nerve conduction studies to serve as controls. The values thus obtained were compared using the student’s t-test for statistical significance.

| Table 1: Comparative study of the duration between the start of reaction and electrophysiological study of nerve |
| Duration (days) | Number of patients (%) |
|-----------------|------------------------|
| 8-15            | 3 (14.28)              |
| 16-21           | 12 (57.15)             |
| >21             | 6 (28.57)              |
| Total           | 21 (100.0)             |

Above table shows that 12 (57.15%) cases have electrophysiological tests within 16-21 days after start of Type-II reaction, 6 cases (28.57%) after 21 days and 3 cases (14.28%) within 8-15 days after onset of Type-II reaction

| Table 2: Comparative study of distal latency (m/s) of control and patients |
|--------------------------|--------------------------|
| Nerves                   | Control group | Study group |
|--------------------------|---------------|-------------|
| Ulnar nerve              |               |             |
| Number of patients       | 20            | 21          |
| Mean±SD                  | 2.9±0.9       | 3.42±0.398  |
| $t$                      | -             | 2.412       |
| $P$                      |               | <0.05       |
| Median nerve             |               |             |
| Number of patients       | 20            | 21          |
| Mean±SD                  | 3.3±0.6       | 3.408±0.256 |
| $t$                      | -             | 0.756       |
| $P$                      |               | NS          |
| Common peroneal nerve    |               |             |
| Number of patients       | 20            | 21          |
| Mean±SD                  | 4.8±1.5       | 2.44±0.755  |
| $t$                      | -             | 6.410       |
| $P$                      |               | <0.001      |
| Posterior tibial nerve   |               |             |
| Number of patients       | 20            | 21          |
| Mean±SD                  | 3.9±0.9       | 4.41±0.657  |
| $t$                      | -             | 2.083       |
| $P$                      |               | >0.05       |

The significant prolongation of distal latency along the ulnar nerve, common peroneal nerve and posterior tibial nerve as compared to control group. SD: Standard deviation, NS: Not significant
Electrophysiological studies were carried out in 21 patients of Type-II reaction in leprosy. Twelve cases (57.15%) had electrophysiological tests within 16–21 days after start of Type-II reaction, 6 cases (28.57%) cases after 21 days, and 3 cases (14.28%) within 8–15 days after onset of Type-II reaction [Table 1]. In comparison to control values, distal latency showed a significant prolongation along the ulnar nerve, common peroneal nerve, and posterior tibial nerve [Table 2] while distal motor conduction velocity showed a significant reduction in ulnar nerve only [Table 3]. The proximal motor conduction latency was significantly prolonged in both ulnar nerve and common peroneal nerve and proximal motor conduction velocity was also significantly reduced in ulnar nerve and common peroneal nerve [Table 4]. On comparing these values, found to be beyond 2S. D. of the control value. It was again observed that a distal latency was not affected [Tables 5 and 6] and only proximal conduction was affected in three ulnar nerves [Tables 7 and 8].

**Discussion**

The nerves in leprosy are chiefly involved in two zones, the dermal nerves at the periphery and the nerve trunks at certain specific sites. In most of these sites of predilection, the nerve trunks lie superficially over the bony structures. These observations are also reflected in most of the electrophysiological studies on patients with leprosy, where the proximal segments of the nerves are more severely affected than the distal segments. These changes are mainly noted along the clinically affected nerve in comparison to those nerves which are clinically unaffected, although some of the clinically unaffected nerves also reveal abnormal conduction. Reactions in leprosy lead to a generalized disturbance in the nerve functions and thus are of special importance in the final outcome of the disease. The effect of reaction is not only limited to the affected nerves, but it also involves the clinically unaffected nerves as well as the proximal segments of the nerves. The main alteration has been reported to occur in the motor nerve conduction velocities, especially in the affected nerves. The reason ascribed for this decrease in motor conduction velocities has been the inflammatory edema of the granuloma producing ischemia of the nerves associated with Schwann cell dysfunction. Since the ischemia of the nerves mainly involve the larger “a” fibers, the measurement of motor conduction velocity
Singh, et al.: Electrophysiological changes in nerves in ENL

Indian Journal of Dermatology | Volume 62 | Issue 6 | November - December 2017

Table 5: Number of patients with abnormal (beyond 2 standard deviation of control value) distal motor conduction velocity

| Nerves                          | Total number of cases | Abnormal Number of patients (%) |
|---------------------------------|-----------------------|----------------------------------|
| Ulnar nerve (mean-2SD=45.9)     | 21                    | -                               |
| Median nerve (mean-2SD=44.4)    | 21                    | -                               |
| Common peroneal nerve (mean-2SD=37.0) | 21                  | -                               |
| Posterior tibial nerve (mean-2SD=24.9) | 21                  | -                               |

There was not significant reduction in motor conduction velocity beyond 2SD of control values. SD: Standard deviation

Table 6: Number of patients with abnormal (beyond 2 standard deviation of control value) distal latency

| Nerves                          | Total number of cases | Abnormal Number of patients (%) |
|---------------------------------|-----------------------|----------------------------------|
| Ulnar nerve (mean-2SD=0.47)     | 21                    | -                               |
| Median nerve (mean-2SD=0.45)    | 21                    | -                               |
| Common peroneal nerve (mean-2SD=0.78) | 21                  | -                               |
| Posterior tibial nerve (mean-2SD=0.57) | 21                  | -                               |

There was not significant prolongation in distal latency beyond 2SD of the control value. SD: Standard deviation

Table 7: Number of patients with abnormal (beyond 2 standard deviation of control value) proximal motor conduction velocity (F-wave conduction velocity)

| Nerves                          | Total number of cases | Abnormal Number of patients (%) |
|---------------------------------|-----------------------|----------------------------------|
| Ulnar nerve (mean-2SD=44.5)     | 21                    | 1 (4.76)                         |
| Common peroneal nerve (mean-2SD=39.22) | 21                  | -                               |

There was significant reduction in proximal motor conduction velocity in 1 (4.76%) patient with ulnar nerve. SD: Standard deviation

Table 8: Number of patients with abnormal (beyond 2 standard deviation of control value) proximal motor conduction latency (F-latency)

| Nerves                          | Total number of cases | Abnormal Number of patients (%) |
|---------------------------------|-----------------------|----------------------------------|
| Ulnar nerve (mean-2SD=28.8)     | 21                    | 3 (14.28)                        |
| Common peroneal nerve (mean-2SD=47.5) | 21                  | -                               |

There was significant prolongation in proximal motor conduction latency in 3 (14.28%) patients with ulnar nerve. SD: Standard deviation

can be taken as an index of involvement of various nerves.[12,14,15]

Most of the electrophysiological studies conducted so far during reactions have been restricted to the distal segments of the nerves, and thus, they exclude the specific sites of predilection of the nerves involvement in leprosy. F waves by virtue of their passage along whole of the nerve segment are extremely useful in detecting abnormalities of motor nerve conduction along the proximal segments. There are studies where F waves are of use in detecting the proximal segment abnormalities in various neuropathies.[16-19] With this background, we have attempted to assess the electrophysiological alterations in the nerve functions of both the proximal as well as distal segment of nerves during Type-II reactions in leprosy.

Conclusion

It is concluded that in Type-II reaction the motor conduction abnormalities are not prominent and the abnormalities are relatively more marked in proximal segment than the distal segment.

Acknowledgment

We would like to acknowledge Dr. Rashmi Gupta for continuous support on data collection.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

What is new?
Nerve conduction study in Type-II lepra reaction showed little abnormality and quite more in proximal segment than distal segment

References

1. Dharmendra. Acute exacerbations (reactions) in leprosy. In Dharmendra (Edit): Leprosy. 1st ed., Vol. 1. Bombay: Kothari Medical Publishing House; 1978. p. 108-39.
2. Bryceson A, Pfaltzgraff RE. Management of Reactions. In: Leprosy(Medicine in the tropics). 2nd ed. Churchill Livingstone, 1979;72-6.
3. Sehgal VN. Reactions in leprosy. In: Clinical Leprosy. Vol. XIII. Ghaziabad, UP, India: Vikas Publishing House Pvt. Ltd.; 1979. p. 52-8.
4. Job CK. Nerve damage in leprosy-XIII leprosy congress state of the art lectures. Int J Lepr 1989;57:532-9.
5. Pearson JM, Ross WF. Nerve involvement in leprosy – Pathology, differential diagnosis and principles of management. Lepr Rev 1975;46:199-212.
6. McLeod JG, Hargrave JC, Walsh JC, Booth GC, Gye RS, Barron SA. Nerve conduction studies in leprosy. Int J Lepr 1975;43:21-31.
7. Verghese M, Ittimani KV, Satyanarayan KR, Mathai R, Bhakthaviziam C. A study of the conduction velocity of the motor fibers of ulnar and median nerves in leprosy. Int J Lepr Other Mycobact Dis 1970;38:271-7.
8. Swift TR, Hackett ER, Shipley DE, Miner KM. The peroneal...
and tibial nerves in lepromatous leprosy clinical and electrophysiologic observations. Int J Lepr Other Mycobact Dis 1973;41:25-34.
9. Rao SP, Bharambe MS. ElectrophYSiological studies in early tuberculoid leprosy. Indian J Lepr 1993;65:181-7.
10. Sheskin J, Convit IJ. Results of a double blind study of the influence of thalidomide on lepra reaction. Lepr Rev 1969;37:135-46.
11. Magora A, Sheskin J, Sagher F, Goneny B. The condition of the peripheral nerve in leprosy under various forms of treatment. Conduction velocity studies in long-term follow-up. Int J Lepr Other Mycobact Dis 1970;38:149-63.
12. Sohi AS, Kandhari KC, Singh N. Motor nerve conduction studies in leprosy. Int J Dermatol 1971;10:151-5.
13. Naafs B, Pearson JM, Baar AJ. A follow up study of nerve lesion in leprosy during and after reaction using motor nerve conduction velocity. Int J Lepr 1976;44:188-97.
14. Garven HS, Gains FW, Smith G. The nerve fibre populations of the nerves of the leg in chronic occlusive arterial disease in man. Scott Med J 1962;7:250-65.
15. Miglietta O. Electrophysiologic studies in chronic occlusive peripheral vascular disease. Arch Phys Med Rehabil 1967;48:89-96.
16. Kimura J, Butzer JF. F-wave conduction velocity in Guillain-Barré syndrome. Assessment of nerve segment between axilla and spinal cord. Arch Neurol 1975;32:524-9.
17. David K, Peter A. Conduction velocity in the proximal segments of a motor nerve in the Guillain-Barre syndrome. J Neurol Neurosurg Neuropsychiatry 1976;39:538-44.
18. Lachman T, Shahani BT, Young RR. Late responses as an aide to diagnosis in peripheral neuropathy. J Neurol Neurosurg Neuropsychiatry 1980;43:156-62.
19. Driessens M, Saldien V, Dijs H, De Ridder A, Willems J, Mortier G, et al. F-wave latencies of the deep peroneal nerve in diabetic polyneuropathy. Electromyogr Clin Neurophysiol 1989;29:339-44.