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A CLINICAL STUDY OF NERVE FUNCTION IMPAIRMENT IN LEPROSY: AN ELECTROPHYSIOLOGICAL EVALUATION OF ULNER AND MEDIAN NERVES IN PATIENTS WITH CLINICAL NEURAL DEFICIT.

Clinical Research

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ABSTRACT

Background: Leprosy is a chronic granulomatous infectious disease caused by Mycobacterium leprae, which usually affects the peripheral nerves and the skin. The present study was conducted to assess nerve function impairment in leprosy. **Material & methods:** The present study was conducted on 40 patients which included already diagnosed cases of pauci- and multibacillary leprosy as well as the freshly diagnosed ones over a period of two months. This study was done to assess the nerve conduction velocity, amplitude and latency of ulnar and median nerves. **Results:** When examined for sensory impairment of touch, pain, and temperature, 32 of our patients complained of impaired sensations over the corresponding neural distribution, while the rest eight had glove and stocking pattern of sensory impairment. In case of ulner nerve, inpaucibacillary leprosy, amplitude and velocity was reduced in all cases and latency was prolonged in only 11 cases. In multibacillary leprosy, amplitude was reduced in all 18 cases, velocity was reduced in only 9 cases whereas latency was prolonged in 9 cases. In case of median nerve, in paucibacillary leprosy, amplitude was reduced in all cases, elocity in only 17 cases and latency was prolonged in only 11 cases. In multibacillary leprosy, amplitude was reduced in all 18 cases, velocity was reduced in only 4 cases and latency was prolonged in only 11 cases. **Conclusion:** The present study concluded that changes in sensory nerve conduction were more evident. Motor latencies and amplitude changes were more severe.

KEYWORDS

amplitude, sensory, motor, nerve conduction

INTRODUCTION

Leprosy is a contagious illness that continues to be endemic in many parts of the world, despite the fact that Brazil, India, and Indonesia account for over 80% of new cases worldwide. Mycobacterium leprae and (less commonly) Mycobacterium lepromatosis produce a persistentinfectious condition that affects infected people's skin and peripheral nerves.[1] According to the WHO 8th expert committee, leprosy is diagnosed in the presence of at least one of the following cardinal signs: Definite loss of sensation in a hypopigmented or erythematous skin lesion, A thickened or enlarged peripheral nerve, with loss of sensation with/without weakness of muscles supplied by that nerve, Presence of acid fast bacilli in a slit skin smear.[2] Peripheral nerve involvement in leprosy may vary from involvement of an intradermal nerve in the cutaneous patches to a major lesion in the peripheral nerve trunk. A stage of functional blockade of conduction of nerve impulse almost always precedes visible pathological changes in the nerve.[3] The role of electrophysiological evaluation of nerve function in the diagnosis and assessment of different neuropathies is well established.[4] Leprosy produces a spectrum of disease that Ridley and Jopling classified into five types based on clinical, bacteriological, histological, and immunological features. M. leprae has a predilection for peripheral nerves and skin, and peripheral nerves are implicated across the spectrum of the disease.[5,6] Around 10% of new leprosy cases registered every year have signs of sensory, motor or autonomic neuropathy at diagnosis.[7] Sensory loss is the earliest and most frequently affected modality but a predominantly motor loss can also occur. The nerves most likely to be involved are the mixed nerve trunks of the upper and lower extremity, especially in those segments where the nerve is subcutaneous and has a cooler temperature. These include great auricular nerves in the neck, the supraclavicular nerves as they cross the clavicles, the ulnar nerves just the elbows, the radial nerve at the spiral groove, the median nerves at the wrist, the radial cutaneous nerve in the lower forearm, the lateral popliteal nerves as they wind round the necks offibulae, the posterior tibial nerves behind the medial malleoli and the superficial peroneal nerves in front of the ankles and on the dorsa of the feet.[8] The present study was conducted to assess nerve function impairment in leprosy.

MATERIAL & METHODS

The present study was conducted on 40 patients which included already diagnosed cases of pauci- and multibacillary leprosy as well as the freshly diagnosed ones over a period of two months. Patients of cervical trauma, neurological disease, cardiovascular disease, diabetics, patients with pace makers, and chronic alcoholics were excluded from the study. Before the initiation of study, informed consent was taken from all the patients and after obtaining a brief history regarding the onset of their symptoms and treatment taken, if any; they were subjected to a thorough clinical examination. After a thorough cutaneous and neural assessment, routine hematological investigations such as complete blood counts, blood sugar, and erythrocyte sedimentation rate were done followed by skin slit smear for acid-fast bacilli. Skin biopsy for histopathological confirmation was taken from those patients who had a well-defined cutaneous patch or a well-defined area of sensory deficit. Patients who had clinically neural leprosy without a cutaneous patch was subject to nerve biopsy from sural nerve. The electrophysiological nerve conduction assessment was done for all the patients using RMS-EMG EP Mark 2 machine. Filters were set at 2-5 Hz and sweep speed was 5 ms per division for motor study and the corresponding settings for sensory study were 20-3 KHz and 2 ms per division. The duration for both the recordings was taken to be 100 μsec. The room temperature for the study was set at 30°C. The parameters studied for motor nerves were distal motor latency, compound muscle action potential, and conduction velocity while for sensory nerves sensory nerve action potential (SNAP), onset latency, and conduction velocity were recorded. The sites for stimulation for median and ulnar nerves were the wrist and the elbow, and the recording sites were motor point of abductor pollicisbrevis and abductor digiti minimi, respectively. Reference electrode was placed at 4 cm distally over first metacarpopharyngeal joint for median nerve and over fifth metacarpopharyngeal joint for ulnar nerve. Belly-tendon montage was used with cathode and anode set 3 cm apart. Antidromic study was done for sensory nerves by placing the electrodes at index and little finger for median and unar nerves, respectively. SNAP amplitude was taken from peak to base and the ground electrode was placed between stimulation and recording electrode. ELISA for human immunodeficiency virus (HIV) infection was performed on all the patients to rule out immunocompromised state.

RESULTS

In the present study 26 cases were fresh and 14 cases were known cases of leprosy. Out of 14 known cases of leprosy, 7 cases among them had already completed the multibacillary regimen of antileprosy treatment, while the other 7 were on treatment and the rest six were freshly diagnosed cases. Out of the 40 cases, 22 were paucibacillary and the rest 18 were multibacillary. When examined for sensory impairment of touch, pain, and temperature, 32 of our patients complained of impaired sensations over the corresponding neural distribution, while the rest eight had glove and stocking pattern of sensory impairment. In case of ulner nerve, in paucibacillaryleprosy,

amplitude and velocity was reduced in all cases and latency was prolonged in only 11 cases. In multibacillary leprosy, amplitude was reduced in all 18 cases, velocity was reduced in only 9 cases whereas latency was prolonged in 9 cases. In case of median nerve, in paucibacillary leprosy, amplitude was reduced in all cases, elocity in only 17 cases and latency was prolonged in only 11 cases. In multibacillary leprosy, amplitude was reduced in all 18 cases, velocity was reduced in only 4 cases and latency was prolonged in only 11 cases.

Table 1: Diagnosis and the treatment status of the patients

Diagnosis status	No.of cases	Casescompleted/under treatment
Fresh cases	26	0
Known cases	14	7/7

Table 2: Type of leprosy cases

Type of leprosy cases	No.of cases
Paucibacillary leprosy	22
Multibacillary leprosy	18

Table 3: Showing changes in amplitude, velocity and latency in ulnar nerve

Type of leprosy cases	Amplitude	Velocity	Latency
Paucibacillary leprosy	Reduced in all 22 cases	Reduced in Reduced in all 22 cases	Prolonged in only 11 cases
Multibacillary leprosy	Reduced in all 18 cases	Reduced in only 9 cases	Prolonged in only 9 cases

Table 4: Showing changes in amplitude, velocity and latency in median nerve

Type of leprosy cases	Amplitude	Velocity	Latency
Paucibacillary leprosy	Reduced in all 22 cases	Reduced in only 17 cases	Prolonged in only 11 cases
Multibacillary leprosy	Reduced in all 18 cases	Reduced in only 4 cases	Prolonged in only 11 cases

DISCUSSION

About 33-56% of newly registered leprosy patients already have clinically detectable nerve function impairment (NFI), often no longer amenable to MDT. Unfortunately, many patients are diagnosed late and are at greater risk of developing the reactions and neuritis. If these reactions are treated effectively, early nerve damage can be reversed and disability can still be prevented.[7]

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Nerve conduction studies have demonstrated predominantly axonal neuropathy in leprosy, i.e., reduction in sensory nerve action potential (SNAP) and compound muscle action potential (CMAP) amplitude. Sensory nerves are more commonly affected than motor, and SNAP amplitudes are altered more than CMAP. Nerve conduction was found to be affected most frequently, followed by warm detection thresholds (WDT) with QST. These two methods were able to detect abnormalities in the nerves up to 12 weeks before MFT becomes abnormal. Multibacillary leprosy patients had significantly more severe changes in nerve conduction parameters compared with paucibacillary leprosy. The lower extremity was more frequently and severely involved than the upper limbs in both groups of patients.[8-15]

The sensory fibers are damaged early in leprosy and therefore show more quantum of changes inconduction velocities as compared to motor nerve fibers in the early stages of damage. However, the amplitude changes are much more marked for motor nerve fibers.[16,17]

In a study conducted by J. Mc Knight, PG Nicholls, Das Loretta, K.V Desikan, et al. in leprosy patients in northern India, it was found that the commonest and the earliest impairment was reported in sensory nerve conduction of sural nerve.[18]

Kar S et al . found reduced conduction velocities besides changes in latency and amplitude in the affected nerves. Changes in sensory nerve conduction were more pronounced. Also, sensory latencies and amplitude changes were more severe than motor latencies and amplitude in those presenting with muscle palsies.[19]

CONCLUSION

The present study concluded that changes in sensory nerve conduction were more evident. Motor latencies and amplitude changes were more severe.

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