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Original Article

The Role of Nerve Conduction Studies and Clinical Examination in Predicting Nerve Involvement: A Comparative Analysis

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Nerve involvement in Hansen's disease (leprosy) is a key determinant of disease severity and disability. Clinical examination and nerve conduction studies (NCS) are essential for assessing peripheral neuropathy in leprosy. While clinical examination provides insights into neurological signs and symptoms, NCS offers objective measurements of nerve conduction, aiding in the identification of pathological processes. This study aims to correlate the clinical manifestations of Hansen's disease with NCS findings to enhance the understanding of nerve involvement in leprosy and improve patient outcomes. This descriptive observational study involved 40 individuals recently diagnosed with Hansen's disease at a tertiary care center. Neurological examination focused on the ulnar, medial, and radial nerves in the upper limbs, as well as the sural, common peroneal, and posterior tibial nerves in the lower limbs. Sensory and motor functions were assessed using standard methods, and deformities due to muscle wasting were documented. All patients underwent bacteriological and histological evaluations, followed by nerve conduction tests which evaluated latency, amplitude, and conduction velocity using the Neuropack 2 EMG system. Data analysis was conducted using IBM SPSS Statistics, with p-values less than 0.05 considered statistically significant. The mean age of participants was 40.55 ± 16.78 years, with a male-to-female ratio of 7:1. Numbness (60%) and tingling (55%) were the most common presenting complaints. The most frequent grade I thickened nerve was the ulnar nerve (75% of cases), followed by the common peroneal nerve (70%). Sensory involvement was most observed in the left ulnar nerve (30% of cases). The borderline tuberculoid- BT spectrum was most common clinical presentation in our study seen in 14 (35.0%) of the 40 cases followed by borderline lepromatous - BL and pure neuritic spectrum in 10 cases (25.0%) each. 10 (25.0%) showed an NCS pattern suggestive of mononeuropathy while 8 (20.0%). had mononeuritis multiplex and 6 (15.0%) symmetrical distal polyneuropathies. Statistical analysis revealed significant associations between nerve thickening and abnormal latency, amplitude, and conduction velocity in both sensory and motor nerves. It is concluded that clinical examination and NCS are complementary in assessing nerve involvement, with each modality offering unique contributions to diagnosis. Discrepancies

Introduction

Leprosy is one of the oldest diseases known to mankind and is caused by *Mycobacterium leprae* which primarily involves the skin and peripheral nerves (Roberts 2020, Romero-Navarrete et al 2022). Involvement of peripheral nerves in leprosy leads to development of leprosy neuropathy which can either be due to

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assessing nerve involvement, with each modality offering unique contributions to diagnosis. Discrepancies between clinical findings and NCS results highlight the need for an integrated approach to optimize diagnostic accuracy and patient management. This study underscores the importance of combining clinical examination and NCS in the evaluation of nerve involvement in Hansen's disease, potentially informing more precise diagnostic algorithms and therapeutic strategies.

Keywords: Hansen's Disease, Leprosy, Nerve Involvement, Clinical Examination, Nerve Conduction Studies, Peripheral Neuropathy, Neurological Assessment.

the ability of leprae bacilli to invade Schwann cells of nerve fibres or due to infected host's immune response against bacilli which causes intense inflammation and damage to nerve fibres or by fibrosis within nerve fibre leading to interstitial neuropathy (Kim et al 2019). Leprosy neuropathy occurs initially due to damage to dermal nerves which are either non-myelinated or small myelinated fibres leading loss of temperature, touch, pain and pressure sensation along with loss of sweating. This is followed by involvement of superficial portions of peripheral nerve trunks in areas which are relatively cooler causing sensory and motor impairment along that nerve segment. Hence sensory loss precedes weakness in all types of leprosy (Midroni & Bilbao 2015). Nerve involvement in leprosy also varies across the spectrum of leprosy with asymmetrical nerve thickening usually around skin lesion seen in tuberculoid pole to late onset bilaterally symmetrical distal polyneuropathy in lepromatous pole. Clinically, it may present as silent neuritis (where there is ongoing nerve damage but patient is completely asymptomatic) or as a clinically manifest sudden onset neuritis (as in lepra reactions) equally affecting all three components of peripheral nerve that is autonomic, sensory & motor with varying severity (Joshua & Misri 2022). But sensory impairment involving loss of temperature and pain in patchy distribution is among the earliest and the most severely affected parameter.

Detailed neurological examination by the healthcare worker which involves palpation of superficial nerve trunks along with assessment of sensory, autonomic or motor impairment of the particular nerve is of paramount importance not only in diagnosing leprosy but also in early treatment and prevention of disabilities and deformities. It has been seen that even in the face of normal clinical testing, latent nerve damage exists: that is 30% of sensory nerve fibre is already damaged and the disease is well established in the nerve before there is clinical evidence of nerve function deficit (Shibasaki & Hallett 2022, Maher 2017). The role of electro diagnostic tests like nerve conduction velocity studies hence become important to pick nerve involvement even before the patient manifests clinically by measuring various parameters like latency, amplitude and conduction velocity in sensory and motor component of involved nerves. It also plays a role in assessing nature of nerve involvement and corroborate it with the neurological findings along with monitoring of patients of leprosy. It can also be beneficial in or in doubtful/ suspected cases that helps in timely treatment and prevention of deformities (Tavee 2019, O'Bryan & Kincaid 2021). This study was carried out to study and correlate the clinical manifestations of the Hansen 's disease with the nerve conduction velocity study findings and to make quality scientific observations to help in better understanding of nerve involvement in leprosy

Materials & Methods

This descriptive observational study involved 40 individuals who were recently diagnosed with Hansen's disease and sought treatment at the Dermatology department of a tertiary care centre. Prior to the commencement of the study, ethical approval was obtained from the institutional review board (IRB)/ethics committee of Command Hospital, Central Command, Lucknow, ensuring that the research adhered to the principles outlined in the Declaration of Helsinki.

The study encompassed all newly diagnosed patients of Hansen's disease who were 12 years of age or older, while all patients in the paediatric age range and individuals with any other aetiologies of nerve thickening or peripheral neuropathy, such as diabetes mellitus, thyroid problems, amyloidosis, or alcoholism were excluded. Demographic details, patient history was recorded. A detailed dermatological assessment was recorded, focusing on the classification of the primary lesion (categorised as patch, plaque, nodules, ulcers, or absence of skin lesion), the distribution of the lesion (whether it was symmetrical or asymmetrical), the total number of lesions, and any feelings experienced over the lesions. Neurological assessment was conducted and documented, following the method of Lehman et al (1997), focusing on palpating the nerves most affected in leprosy. This included examining the ulnar, medial, and radial nerves in the upper limbs, as well as the sural, common peroneal, and posterior tibial nerves in the lower limbs and simultaneously checked for tenderness. The hypoesthesia in the area supplied by the nerves under study was assessed for light touch using wisp of cotton wool gently stroked along the distribution of nerves under study (ulnar, median, radial, sural, common peroneal, and posterior tibial nerves). Patients were instructed to close their eyes and report the presence or absence of sensation, comparing bilateral sites and indicating hypoesthesia or anaesthesia clearly. Pain perception was assessed using a standardized disposable safety pin or a blunt needle, applying minimal pressure sufficient to elicit a response without skin injury. The sharp and dull ends of the instrument were alternately applied in random order, and patients were instructed to discriminate between sharp (painful) and dull (non-painful) stimuli. Responses were classified accordingly as normal, hypoaesthetic, or anaesthetic. Temperature discrimination was tested using warm (approximately 40°C) and cold (approximately 10°C) water-filled tubes. These tubes were gently placed on corresponding dermatomal areas of nerves tested. Patients, with eyes closed, indicated whether they perceived a sensation of warmth or cold. Impaired responses were recorded as abnormal temperature perception. The evaluation of muscular strength in the specific muscles of the hands and feet, which are supplied by the nerves being studied, was conducted using the voluntary muscle test (VMT), which is widely recognised as the standard method for assessing motor function in individuals with leprosy. Deformities arising due to complete muscle wasting was recorded. The subjects underwent bacteriological (Slit Skin Smear) and histological evaluation to verify the clinical diagnosis of Hansen's illness.

Following the confirmation of diagnosis, the individual cases were classified on the basis of IAL (1982) classification and WHO classification (PB/MB) as followed by National Leprosy Eradication Programme (NLEP 2013). Patients were diagnosed as a case of pure neuritic leprosy (PNL) based on diagnostic criteria described by Narang et al (2016). which relies on essential and auxiliary criteria. Essential criteria include

epidemiological factors such as residence in a leprosy-endemic area or history of contact with a known leprosy patient. Clinically, PNL is characterized by thickened peripheral nerves associated with definitive sensory impairment, which may be accompanied by motor impairment or functional loss, without any skin lesions (IAL 1982). Laboratory findings require negative slit-skin smears (SSS) from at least three sites, including the anaesthetic areas, and the absence of definitive histological evidence of leprosy in skin biopsies taken from sensory-impaired regions. Auxiliary criteria, supportive but not mandatory, include definitive or suggestive findings on nerve biopsy or fine needle aspiration cytology, such as acid-fast bacilli (AFB), caseous necrosis, perineural or endoneural infiltrates, and fibrosis indicative of leprous neuritis. Nerve conduction studies demonstrating reduced amplitude, diminished conduction velocities, or prolonged latency further substantiate the diagnosis of PNL. A nerve conduction test was conducted on all patients in the neurology outpatient department (OPD) utilising the Neuropack 2 EMG measuring device. The ambient temperature was consistently kept within the range of 29 to 32◦C. A separate nerve conduction examination was conducted for the sensory nerves (ulnar, medial, and sural) and the motor nerves (ulnar, medial, common peroneal, and tibial). The acquired data was compared to the standard values established in our facility. Nerve conduction studies (NCS) were deemed abnormal if any of the measured values for motor or sensory nerves fell beyond the established reference ranges. The statistical analysis was conducted utilising IBMSPSS Statistics software version 2015 and the p-values less than 0.05 was statistically significant.

Results

The mean age of study participants were 40.55 ± 16.78 years with a M:F ratio of 7:1. Skin symptoms ranges from complete absence of skin lesion to non-healing ulcers. Symptoms pertaining to peripheral nerve involvement included numbness, tingling, weakness of hands and feet and cord like swelling. 24 (60%) of the cases presented with numbness followed by tingling 22 (55%) involving distribution of one or more nerve.

The BT spectrum was most common clinical presentation in our study seen in 14 (35.0%) of the 40 cases followed by BL and pure neuritic types in 10 cases (25.0%) each.

In our study 14 presented with deformity with the most common deformities being ulnar claw hand and foot drop, each seen in 9 (22.5%) of the cases. All the deformities in our study were grade II deformities as per World Health Organization (1988) grading of disabilities. NCS was successfully performed in all enrolled patients, and none were excluded due to deformity or ulcers. Out of 40 cases studied, 21 cases presented with Type I reactions of which 13 (32.5%) presented with only neuritis and 3 (7.5%) had only cutaneous reaction while 5 (12.5%) presented with both cutaneous reaction and neuritis. Only 4 (10.0%) of the 40 cases presented with Type II Reaction.

The most common grade I thickened nerve was the ulnar nerve with Left and Right ulnar nerve involvement seen in 35 (87.5%) and 33 (82.5%) cases, followed by involvement of the left common peroneal nerve seen in 31 (77.5%) cases. The most common grade 2 thickened nerve was left ulnar nerve seen in 12.5% of the participants followed by right ulnar, right radial cutaneous and common peroneal nerve.

Out of the 40 cases studied, most common sensory involvement (30.0% cases) was seen in distribution of left ulnar nerve followed by right ulnar nerve (27.5% cases), right and left common peroneal (22.5% cases) and right sural nerve

Table 1 : Distribution of sensory involvement in the study group.

Sensory involvement (Hypoesthesia)	Frequency (%)
Right Ulnar Nerve	11(27.5%)
Left Ulnar Nerve	12 (30%)
Right Median Nerve	4 (10%)
Left Median Nerve	4 (10%)
Right Radial Nerve	2 (5%)
Left Radial Nerve	1 (2.5%)
Right Sural Nerve	9 (22.5%)
Left Sural Nerve	7 (17.5%)
Right Common Peroneal Nerve	9 (22.5%)
Left Common Peroneal Nerve	9 (22.5%)
Right Post Tibial Nerve	6 (15%)
Left Post Tibial Nerve	6 (15%)

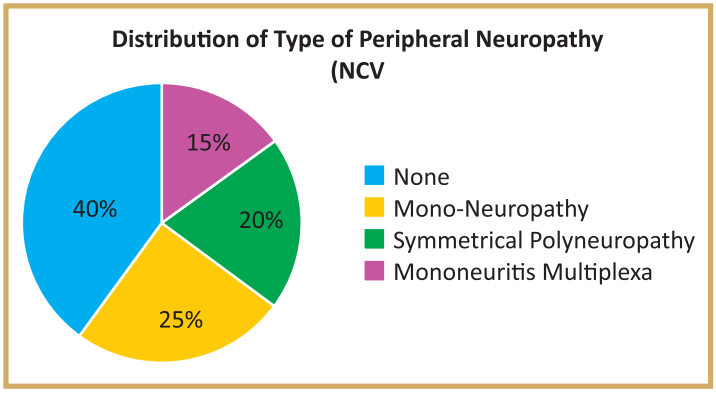

Fig. 1 : Distribution of pattern of nerve involvement.

(22.5% cases) (Table 1).

Among the 40 cases, 13 (32.5%) exhibited weakness in muscles innervated by the left ulnar nerve: 5 (12.5%) had MRC grade 2/5, 6 (15.0%) had 3/5, and 2 (5.0%) had 4/5. Similarly, 10 (25.0%) showed weakness in muscles supplied by the left common peroneal nerve: 2 (5.0%) had MRC grade 1/5, 2 (5.0%) had 2/5, 3 (7.5%) had 3/5, and 3 (7.5%) had 4/5.

Of the 40 cases 16 (40.0%) had normal NCS studies. Among the remaining 24 cases 10 (25.0%) showed an NCS pattern suggestive of mononeuropathy while 8 (20.0%). had mononeuritis multiplex and 6 (15.0%) symmetrical distal polyneuropathies (Fig. 1).

Among the 172 clinically thickened nerves evaluated, electrophysiological abnormalities were identified in 92 nerves (53.4%), while the remaining 80 nerves (46.6%) exhibited normal nerve conduction studies (NCS) despite clinical thickening. A comprehensive evaluation of 400 nerves (sensory and motor) was performed

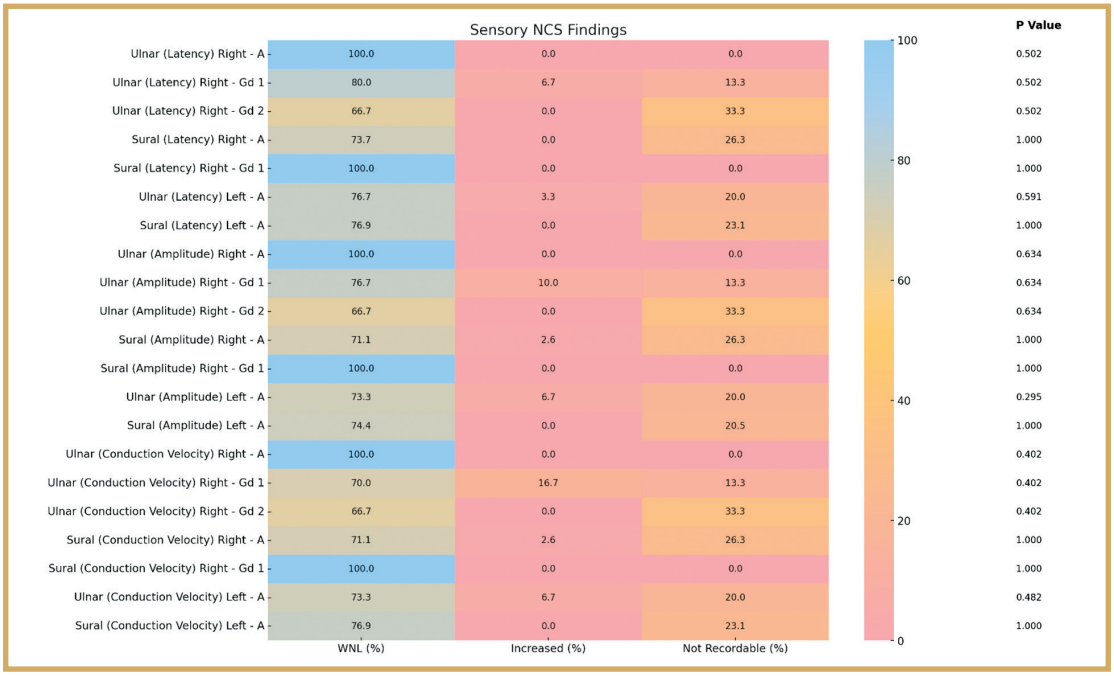

Fig. 2 : Association between thickened nerves and sensory NCS parameters.

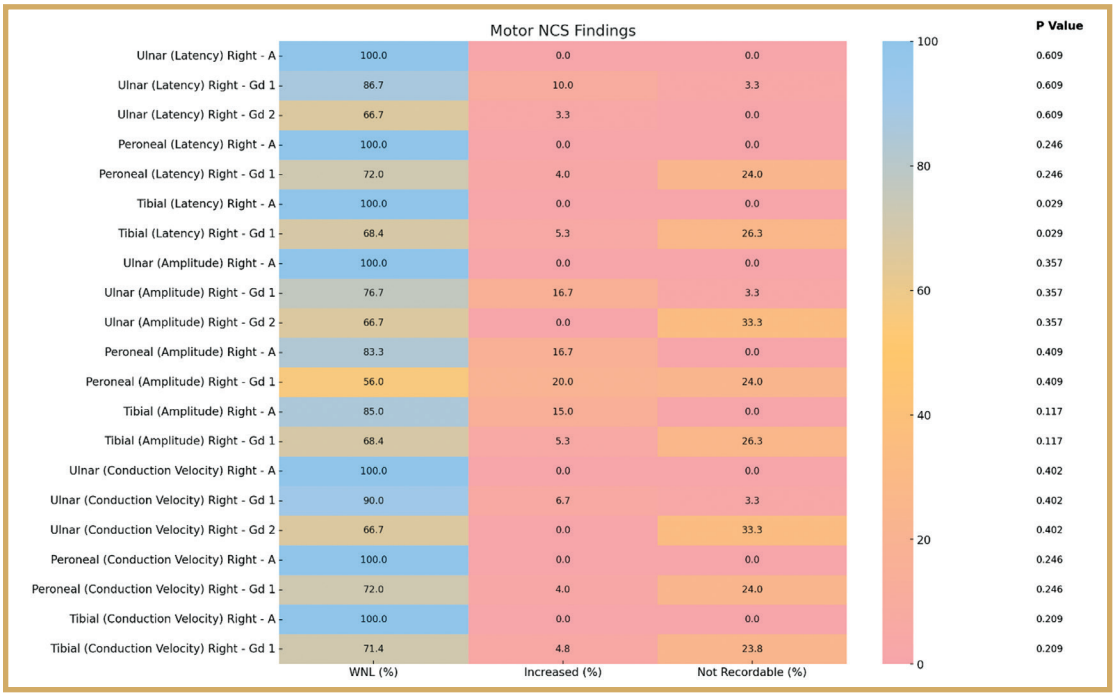

Fig. 3 : Association between thickened nerves and motor NCS parameters.

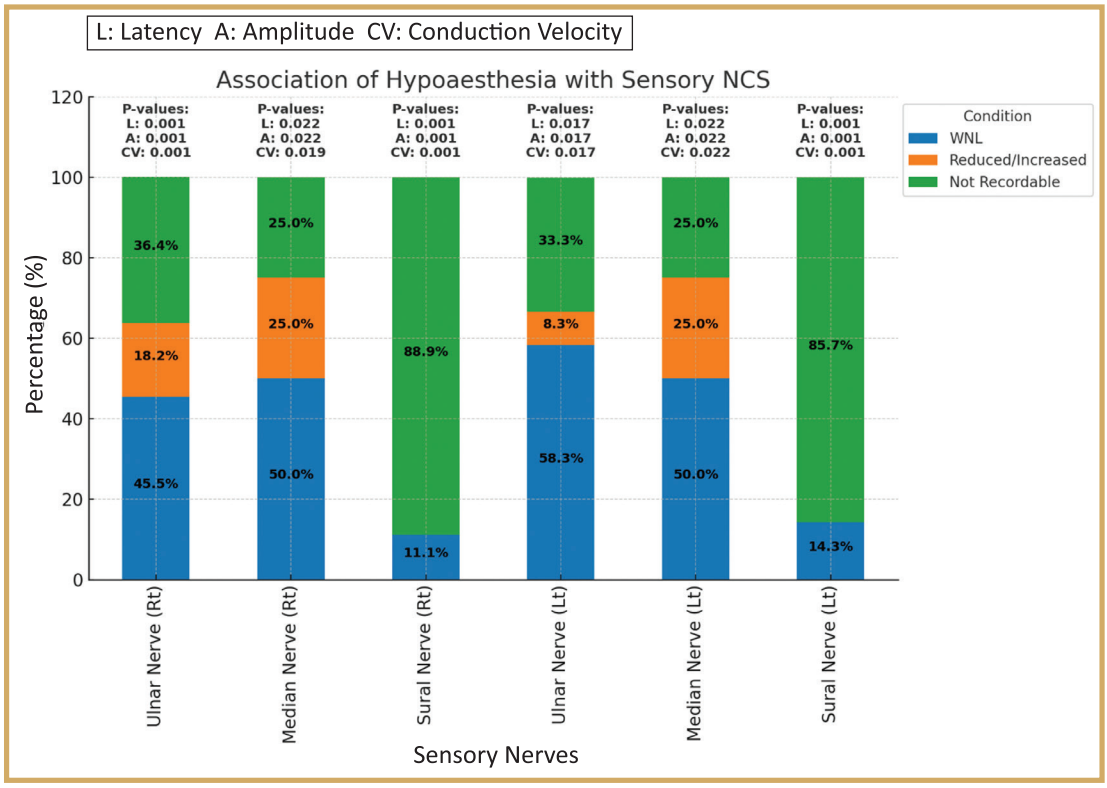

Fig. 4 : Association between hypoesthesia (sensory loss) and NCS parameters.

across the cohort of 40 patients. Of the 240 sensory nerves assessed (ulnar, median, and sural nerves), clinical sensory impairment (hypoesthesia) was documented in 47 nerves (19.5%), while electrophysiological abnormalities were identified in 46 sensory nerves (19.1%) and was normal in 194 (80.8%) sensory nerves. Motor function analysis involved assessment of 320 motor nerves (ulnar, median, tibial, and common peroneal). Clinical motor deficits (muscle weakness) were observed in 57 nerves (17.8%), whereas electrophysiological abnormalities were detected in a higher number of motor nerves, totaling 65 (20.30%) and was normal in 255 (79.6%) of motor nerves.

Association of nerve thickening with right and left latency sensory nerves, amplitude sensory nerve and conduction velocity sensory nerve is shown in Fig. 2.

Association of nerve thickening with right and left latency, amplitude and conduction velocity in right and left motor nerve is shown in Fig. 3.

Association of hypoesthesia with latency, amplitude and conduction velocity in right and left sensory nerve is presented in Fig. 4.

Association of loss of power with latency, amplitude and conduction velocity in right and left motor nerves revealed the following results: Increased latency correlated with muscle power loss for the right ulnar ($p = 0.020$), right median ($p = 0.001$), right common peroneal ($p = 0.005$), and right tibial nerves ($p < 0.001$), as well as the left ulnar ($p = 0.032$), left median ($p = 0.005$), left common peroneal ($p = 0.006$), and left

tibial nerves ($p < 0.001$). Reduced amplitude was significantly associated with weakness in muscles supplied by the right ulnar ($p = 0.001$), right common peroneal ($p = 0.003$), and right tibial nerves ($p < 0.001$), and similarly for the left ulnar ($p = 0.001$), left common peroneal ($p < 0.001$), and left tibial nerves ($p = 0.001$). No significant association was found for median nerves bilaterally ($p = 1.000$ for both right and left). Slower conduction velocities correlated with muscle weakness in the right ulnar ($p = 0.007$), right median ($p = 0.001$), right common peroneal ($p = 0.005$), and right tibial nerves ($p < 0.001$), as well as the left ulnar ($p = 0.009$), left median ($p = 0.005$), left common peroneal ($p = 0.005$), and left tibial nerves ($p < 0.001$).

Discussion

Demographic and Clinical Findings: The mean age of participants (40.55 ± 16.78 years) and male predominance observed in this study align with previous Indian studies, reflecting typical demographic patterns in leprosy-affected populations. Numbness (60%) and tingling (55%) were the most prevalent presenting symptoms, consistent with earlier observations by Jardim et al (2003). Clinically, ulnar nerve involvement was predominant (82.5%), corroborating findings by Chaudhary et al (2023).

Nerve Conduction Study Findings: Despite evident clinical nerve thickening, 53.4% of cases had normal nerve conduction study (NCS) results, suggesting clinical thickening alone is insufficient to reliably predict electrophysiological impairment. Conversely, 16.2% of clinically uninvolved nerves demonstrated abnormal NCS findings, highlighting the utility of NCS in detecting subclinical neuropathy, aligning with results reported by Khambati et al (2009).

Patterns of Neuropathy: Axonal neuropathy (37.5%) emerged as the predominant electrophysiological pattern, consistent with Capadia et al (2010). In leprosy, nerve enlargement and entrapment in anatomic tunnels, along with inflammation, may lead to axonal loss sensory nerve fibers initially exhibited reduced conduction velocities followed by amplitude reductions, indicative of early demyelination progressing to axonal loss. In contrast, motor nerve fibers primarily showed initial amplitude impairments, confirming earlier observations by Husain and Malaviya (2007) about differential vulnerability between sensory and motor fibers.

Association of Nerve Thickening with NCS Findings: Approximately half of clinically thickened nerves demonstrated electrophysiological abnormalities, indicating that nerve enlargement alone does not uniformly predict functional impairment on NCS. Conversely, 16.2% of non-thickened nerves exhibited abnormal conduction, with a majority involving the sural nerve, suggesting its potential role as an early indicator of subclinical neuropathy. Similar proportions have been reported by Khambati et al (2009), who observed abnormal NCS in 61% of thickened nerves and notable subclinical changes in clinically normal sural nerves. Possible explanations for normal NCS in enlarged nerves include preserved functional fibers, focal or fascicular pathology beyond the tested segment, or ongoing regenerative processes. These observations underscore that clinical nerve thickening should trigger, but not replace, electrophysiological assessment; integrating both approaches enhances detection of overt and silent neuropathy.

Association of Clinical Sensory Loss and NCS Parameters: Clinical sensory loss (hypoesthesia) demonstrated significant correlation with NCS abnormalities, notably in the sural nerve (87.5%), underscoring its diagnostic importance as an early indicator of sensory neuropathy.

This finding supports earlier research indicating the value of electrophysiological studies in revealing neuropathy even before clinical detection. In some instances, nerves exhibiting clinical hypoesthesia demonstrated normal NCS findings. This discrepancy may arise because standard NCS assesses only a limited nerve segment and can fail to capture focal or proximal lesions. Additionally, fast-conducting fibers may remain intact despite pathology in slower fibers, yielding normal electrophysiological responses. Regenerating axons can also produce preserved conduction despite clinical sensory loss. Histopathological studies in leprosy have shown the presence of surviving functional fibers within apparently anaesthetic nerve regions, supporting this phenomenon. Khambati et al (2009). similarly reported that only 65% of nerves with clinical hypoesthesia exhibited abnormal NCS findings, underscoring the limitation of electrophysiological studies in detecting all forms of nerve pathology.

Association of Muscle Power and NCS Parameters: Clinical muscle weakness strongly correlated with motor nerve conduction abnormalities, particularly in the tibial and common peroneal nerves. However, correlations with ulnar nerve involvement were weaker, consistent with findings by van Brakel et al (2005), suggesting variability based on nerve segments tested and some fast-conducting fibers may remain unaffected producing a normal electrophysiological test.

Clinical Spectrum and NCS Parameters: Severity of electrophysiological abnormalities was significantly associated with the clinical spectrum of leprosy, with greater NCS abnormalities seen in multibacillary (BL/LL) forms. This aligns with Gupta et al findings, highlighting more pronounced nerve impairment in advanced leprosy types compared to paucibacillary forms.

Limitations

A significant limitation of our study is the lack of assessment of posterior column sensations, specifically vibration sense and proprioception, which are mediated predominantly by large, fast-conducting myelinated fibers. Although nerve conduction studies primarily evaluate large myelinated sensory and motor fibers, they do not directly reflect posterior column function. Consequently, our findings may underestimate or miss subtle neuropathic changes involving these sensory pathways. Due to practical constraints and logistical challenges, we were unable to recall patients to complete these additional sensory evaluations, which limits the comprehensiveness of our neuropathic assessment.

Conclusion

Leprosy neuropathy involves complex immune-mediated inflammatory processes causing asymmetric and progressive nerve damage. While thorough dermatological and neurological examinations, supported by bacteriological and histopathological assessments, remain the gold standard, nerve conduction studies (NCS) effectively identify subclinical nerve involvement. NCS is particularly valuable for early detection, precise assessment of nerve function, and guiding patient management. Our study highlights that electrophysiological tests can detect neuropathological changes missed during clinical examinations, emphasizing their complementary role. Incorporating NCS into diagnostic algorithms, especially in endemic areas and cases with inconclusive clinical findings, could significantly enhance early diagnosis, effective monitoring, and prevention of disability in Hansen's disease.

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