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ROLE OF HIGH-RESOLUTION ULTRASOUND IN ASSESSING PERIPHERAL NERVE INVOLVEMENT IN LEPROUS NEUROPATHY: CORRELATION WITH CLINICAL AND ELECTROPHYSIOLOGICAL FINDINGS

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ABSTRACT

Background: Leprous neuropathy is often silent in onset, and early diagnosis is therefore difficult, despite being very important to prevent deformities. Complementary functional and morphological assessments of peripheral nerve involvement in Hansen's disease are provided by nerve conduction studies (NCS) and high-resolution ultrasound (HRUS), respectively, while clinical evaluation has its limitations. The objective of the current study was to measure peripheral nerve involvement in patients with leprosy by HRUS and correlate the results with clinical and electrophysiological features.

Materials and Methods: This prospective observational study included 35 newly diagnosed patients in a tertiary care hospital. Patients with leprosy, as confirmed by skin biopsy, attending the dermatology outpatient clinic, were included in the study based on the specified inclusion criteria. Clinical assessment, NCS, and HRUS of ulnar, median, common peroneal, and posterior tibial nerves were performed. Cross-sectional area (CSA) of nerves was measured and correlated with clinical thickening and electrophysiological parameters.

Results: The mean age of the cohort was 32.5 ± 10.3 years, with a male predominance (71.4%, M: F ratio 2.5:1). All patients had at least one clinically involved nerve, with the ulnar nerve being most frequently affected, followed by the common peroneal nerve. Sensory deficits (42.8%) were more common than motor (12.5%) or mixed deficits (22%). Deformities were observed in 29% of patients, most commonly ulnar claw hand. Electrophysiologically, subclinical neuropathy was evident, with abnormalities in both clinically thickened and non-thickened nerves, showing electroclinical dissociation. HRUS revealed increased CSA in 34% of nerves, with ulnar and common peroneal nerves most commonly thickened. Mean CSA values were 10.5 ± 3.5 mm² (ulnar), 6.1 ± 1.66 mm² (median), 11.5 ± 1.1 mm² (common peroneal), and 8.2 ± 1.27 mm² (posterior tibial). Significant correlation was observed between clinical thickening and CSA for the median nerve ($p < 0.05$), while correlation with NCS findings was inconsistent.

Conclusion: Leprosy is one of the main treatable causes of peripheral neuropathy in developing countries. Early diagnosis is of paramount importance in preventing deformities. We found a positive correlation between the clinical and CSA of the median nerve. However, no correlation was found between CSA and the electrophysiological function of the median nerve. In addition, no correlation was observed between clinical and electrophysiological function and CSA in the ulnar, common peroneal, and post-tibial nerves.

Keywords: Leprosy, Peripheral Neuropathy, High-Resolution Ultrasound, Nerve Conduction Study, Hansen's Disease.

INTRODUCTION

Leprosy or Hansen's disease is a chronic infectious disease due to Mycobacterium leprae that mostly affects the skin and the peripheral nervous system. Although officially announced to have been eradicated as a health care issue in India in 2005, with a prevalence rate of fewer than 1 in 10000, the country continues to bear a large share of the global burden of leprosy. In 2015, India alone had about 60 percent of the global leprosy cases, and it has reported a prevalence rate of 0.81 per 10,000 population, meaning that leprosy is still a significant line of public health problem in endemic areas.[1] Leprosy clinically has a spectrum of disease manifestations, from the localized paucibacillary tuberculoid form (hypopigmented, anesthetic patches) (tuberculoid leprosy, TT) to the disseminated, multibacillary lepromatous leprosy (LL). In between these extremes, there are the borderline forms that have fluctuating immunological responses.[2] The characteristic feature of leprosy is its propensity to affect the peripheral nerve, which may develop at the initial stages of the disease and may stay asymptomatic over a long-term period. Leprous neuropathy is a significant cause of morbidity with impairment of limb functions, trophic ulcer, and permanent deformity, which is highly socially stigmatized.[3] Therefore, it is of the essence that nerve involvement be detected early on before it results in irreparable disability. The classical diagnosis of leprous neuropathy is significantly dependent on the clinical assessment of nerve palpation and evaluation of the sensory and motor functions. However, clinical evaluation is subjective and may fail to detect subclinical nerve involvement.[4] Electrophysiological testing, during which nerve conduction studies are performed, is more sensitive to identifying early neuropathy. They are capable of showing subclinical pathology like slowed conduction velocity, delayed latency, and diminished motor and sensory nerve amplitudes in cases where clinical evidence is not apparent.[5] However, electrophysiology mainly provides details of functional impairment without direct evaluation of nerve morphology.

High-resolution ultrasonography (HRUS) has become a useful tool in these cases, being a non-invasive imaging modality in the assessment of peripheral nerves. It can be used to measure nerve structure in real time and identify its changes, including nerve enlargement, a change in echotexture, fascicular defects, and perineural vascularity.[6] In contrast to clinical and electrophysiological tests, HRUS allows visualization of structural changes in peripheral nerves directly, which allows the identification of clinically and silently occurring neuropathy.[7] Moreover, with the help of HRUS, it is possible to evaluate the nerve along its anatomical path, providing complementary information to the electrophysiological results.[8] The use of HRUS in leprosy has also been shown to be useful, as there is a good correlation between changes observed on morphology as assessed by ultrasonography and abnormal electrophysiology.[9] HRUS has also been particularly effective in the early detection of nerve thickening, which is a cardinal result of leprous neuropathy and difficult to clinically detect in many cases.[10] Further, it is possible to assess the peripheral nerve involvement in HRUS in combination with electrophysiological studies, and a more comprehensive assessment of the nerve involvement by combining structural and functional approaches is presented. Such a multimodal technique improves the accuracy of the diagnostic process, helps in tracking the progression of the disease, and could even influence the prompt therapeutic response, which would lead to decreased risks of disability and deformity. Because of the continued burden of leprosy in our country, the importance of detecting early nerve involvement and exploring the role of HRUHS in association with clinical and electrophysiological findings is essential. The current study aimed to assess peripheral nerve involvement in leprosy using HRUS and correlate the imaging findings with clinical and electrophysiological parameters.

MATERIALS AND METHODS

This prospective observational study was conducted in the Department of Dermatology and Radiology, Raja Rajeswari Medical College and Hospital, Bengaluru, Karnataka. Ethical clearance for the study was obtained from the Institutional Ethical Committee before commencement of the study. Written consent was obtained from all the participants of the study after explaining the nature of the study in the vernacular language.

Inclusion Criteria

1. All newly diagnosed cases of Hansen's disease attending the department of dermatology at RRMCH, Bengaluru.
2. Males and females
3. Signed the written consent for voluntary participation

Exclusion Criteria

1. Patients with any evidence of neuropathy other than Hansen's disease (diabetes mellitus, hypothyroidism, HIV, Poliomyelitis, Vitamin B12 deficiency, and trauma-related peripheral nerve disease)
2. Patients with chronic alcohol intake.
3. Patient with cardiac pacemakers.
4. Indeterminate type of leprosy patients.

A total of n=35 patients with clinical signs and symptoms suggestive of leprosy attending the department of dermatology, RRMCH Bengaluru, were selected based on the inclusion and exclusion criteria. Detailed history along with demographic

details such as age, sex, occupation was obtained, and patients were evaluated by general and systemic examination, dermatological examination for skin patches, and peripheral nerve examination was assessed for thickening of nerves, sensory and motor impairment related to ulnar, median, lateral popliteal, and posterior tibial nerves.

All the major nerves were palpated bilaterally to record the enlargement and were graded as follows:

Grade 0: Nerve not thicker than the contralateral nerve and with normal sensation

Grade 1: affected nerve thicker than the contralateral nerve

Grade 2: thickening of the affected nerve, which feels rope-like.

Grade 3: thickened nerve which feels beaded or nodular.

Patients were subjected to investigations, namely Slit skin smears to look for AFB, and a Skin biopsy was done in all cases to confirm the diagnosis of leprosy. Other routine investigations like Complete Hemogram, Liver Function tests, Renal Function Tests, and other related blood tests were done to rule out Diabetes, Hypothyroidism, and HIV.

Electrophysiological assessment was performed in all the enrolled subjects in agreement with the American Association of Neuromuscular & Electrodiagnostic Medicine (AANEM) recommendations using the Central research laboratory at Raja Rajeswari Medical College, using a Digital Nikon Kohden Machine (model neuropack S1 MEB-9400, Japan). All the tests were done in the same room with skin temperature kept >33° C.

Recording Procedure: (a) Motor Nerve Conduction **Study (MNCS):** A Stimulator with water-soaked felt tips was placed on the skin overlying the nerve at proximal and distal sites. The recording and reference electrodes were placed using the belly tendon montage. The gain was set at 2–5 mV per division, and stimulation duration was in the range of 50–300 msec. The nerves were stimulated with a short burst of direct current, not exceeding 50 mA, since it was the upper limit available in the machine. The current was initially set to zero, then gradually increased with successive stimuli up to the point where the compound motor action potential (CMAP) no longer increased in size. Further, it was increased by another 20% to ensure the supra-maximal stimulation. For each stimulation site, CMAP, latency, amplitude, duration, and conduction velocity of the median, ulnar, radial, common peroneal, and posterior tibial nerves were recorded.

(b) Sensory nerve conduction study (SNCS): Ring and surface stimulating electrodes were used for stimulation, respectively. Electrodes were placed over a sensory portion of the nerves. Gain was set at 10–20 mV per division, and an electrical pulse of either 100 or 200 milliseconds in duration was used. Current was slowly increased from a baseline of 0 mA, usually by 3–5 mA at a time, until the supramaximal stimulation of the nerve was ensured. For each stimulation site, sensory nerve action potential (SNAP) amplitude, latency, duration, and conduction velocity were measured.

1. The normal values are taken from a previous study by Vashisht D et al,[11] CMAP's DL (mS), AMP (mV), CV (mS)
2. Median nerve >4 <4 <50
3. Ulnar nerve >3.5 <4 <50
4. Post tibial nerve >6 <4 <40
5. Common peroneal nerve >6 <2 <40

SNAPS AMP (mV) CV (mS)

1. Median nerve <10 <50
2. Ulnar <10 <50
3. Sural nerve <6 <40

High Resolution Ultrasonic (HRUS) imaging of the peripheral nerves for Cross-Section Area (CSA), texture, and vascularity was done. Bilateral peripheral nerves were imaged by an independent radiologist blinded to the clinical diagnosis using a GE HEALTHCARE ultrasound machine, Milwaukee, WI, USA, with a linear array broadband frequency of 10-15 MHz probe. Bilaterally, the ulnar nerve at the elbow and proximal to the medial epicondyle, the median nerve at the wrist, the Lateral Popliteal nerve at the fibular head, and Posterior tibial nerve at the ankle and proximal to the medial malleolus were examined, and the length of abnormality of the nerve was determined by the presence of abnormal size and echo reflectivity of the nerves. All the nerves were measured on transverse sections at a point where the nerve thickness is maximum in the visualized segment of the nerve. On transverse scans, the cross-sectional area of the nerve was determined from that area by one measurement within the hyperechoic rim surrounding the nerve. Normal values for cross-sectional area Jain S et al,[5] of the ulnar nerve, median nerve, common peroneal nerve, and posterior tibial nerve were taken from a previous study from South India, as our study population is ethnically similar. Cut-off values were as follows.

The echo texture of the nerves assessed on imaging was graded as follows:

1. Normal = norm echogenic (grade 0)
2. Mild =some hypo-reflectivity (grade 1)
3. Moderate = obvious hypo-reflectivity (grade 2)
4. Severe. = absence of any fascicular pattern (grade 3)

Statistical Analysis: All the available data were uploaded to an MS Excel spreadsheet and analyzed by SPSS version 26 in Windows format. Continuous variables (nerve cross-sectional area, conduction velocities) were expressed as mean ± SD or median (IQR), and categorical variables as frequencies and percentages. Group comparisons were performed using the Whitney U test for continuous data and the Chi-square test for categorical data. Correlation between HRUS parameters and electrophysiological findings was assessed using Spearman's correlation.

RESULTS

Demographic and Clinical Profile of the cohort is given in Table 1. The analysis of the table shows that out of the 35 cases of leprosy, 25 were males (71.4%) and 10 females (28.6%), with a mean age of 32.5 ± 10.3 years (range: 20–55 years). The most frequently involved age group was 20 – 29 years, with 45.7% of cases, followed by 30–39 years (31.4%) of cases. Distribution of cases based on Ridley–Jopling spectrum found that lepromatous leprosy (LL) was the most common presentation (28.6%), followed by borderline tuberculoid (BT, 25.7%), borderline lepromatous (BL, 20.0%), and tuberculoid (TT, 14.3%), while borderline (BB) accounted for 11.4%. Neurological assessment showed that 42.8% had isolated sensory deficits, 8.6% had isolated motor deficits, and 22.9% had combined sensorimotor involvement. Nearly three-fourths of patients (71.4%) had fewer than five clinically involved nerves.

High-Resolution Ultrasound (HRUS) Findings recorded in the cases of study are depicted in Table 2. The cross-sectional area (CSA) assessment showed that significant nerve enlargement was present in clinically involved nerves. The common peroneal and ulnar nerves were commonly involved and markedly enlarged. The mean CSA value of 12.56 ± 2.11 mm^2 (left common peroneal) and 11.59 ± 3.14 mm^2 (left ulnar). Similarly, the posterior tibial nerves showed moderate thickening (mean CSA 8.88 ± 1.12 mm^2, left), while the median nerves were relatively less affected (mean CSA 6.45 ± 1.75 mm^2, left).

Table 3 shows the Echotexture Correlation with Clinical Spectrum in the cases of the study. Analysis of the table reveals that abnormal echotexture was significantly associated with multibacillary forms of leprosy. Although in the left median nerve, all TT and BT patients demonstrated normal echotexture, whereas abnormalities were more frequent in BL (14.3%), BB (50%), and LL (40%) cases (p=0.002). Similarly, for the right common peroneal nerve, echotextural abnormalities were detected in 75% of BB and 60% of LL patients, compared with none in BT and only 14.3% in BL (p=0.022).

Electrophysiological Findings are presented in Table 4. Electrophysiological abnormalities were pronounced in thickened nerves. Distal latencies were significantly increased in thickened compared with non-thickened nerves. In the right median motor nerve, distal latency was 8.28 ± 0.5 ms in thickened versus 4.11 ± 1.21 ms in non-thickened ($p<0.001$). Similar differences were found in the left median (6.96 ± 2.23 vs. 4.02 ± 1.25 ms, and $p<0.001$) and left posterior tibial nerves (8.73 ± 2.16 vs. 6.37 ± 2.5 ms, and p=0.039). Conduction velocities were found to be slower in the thickened nerves, but differences were not statistically significant.

Correlation of Clinical, HRUS, and Electrophysiological Parameters is given in Table 5. In the median nerve, the correlation between clinical thickness and HRUS was significant. HRUS-CSA was strongly associated with clinical thickening for the right median ($\rho = 0.507$, $\rho = 0.002$) and left median ($\rho = 0.571$, $p<0.001$). However, electrophysiological parameters, including conduction velocity, were not significantly correlated with HRUS or clinical findings ($p>0.05$). No significant correlation was found for ulnar, peroneal, or tibial nerves.

Table 1: Demographic and Clinical Characteristics of the Study Population (n=35)

Characteristic	Category	Number (%)
Sex	Male	25 (71.4)
	Female	10 (28.6)
Age (Years)	20-29	16 (45.7)
	30-39	11 (31.4)
	40-49	6 (17.1)
	≥ 50	2 (5.7)
	Mean ± SD	32.5 ± 10.3
Clinical Spectrum (Ridley-Jopling)	Lepromatous (LL)	10 (28.6)
	Borderline Lepromatous (BL)	7 (20.0)
	Borderline Tuberculoid (BT)	9 (25.7)
	Tuberculoid (TT)	5 (14.3)
	Borderline (BB)	40(1.4)
Neurological Findings	Sensory Deficits Only	15 (42.8)
	Motor Deficits Only	3 (8.6)
	Both Sensorimotor Deficits	8 (22.9)
	No Sensorimotor Deficits	9 (25.7)
	Nerves Involved (Clinical): <5	25 (71.4)
	Nerves Involved (Clinical): ≥ 5	10 (28.6)

Table 2: High-Resolution Ultrasound (HRUS) Findings of Peripheral Nerves

Nerve	Side	Cross-Sectional Area, Mean ± SD (mm^2)	Minimum	Maximum
Ulnar	Right	10.98 ± 3.33	7.30	18.90
	Left	11.59 ± 3.14	8.32	19.87
Common Peroneal	Right	11.66 ± 3.14	8.00	15.40
	Left	12.56 ± 2.11	8.70	15.80
Posterior Tibial	Right	8.44 ± 1.27	6.00	11.20
	Left	8.88 ± 1.12	6.80	11.00
Median	Right	6.17 ± 1.66	4.20	9.40
	Left	6.45 ± 1.75	4.30	10.20

Table 3: Association of HRUS Echotexture with Clinical Spectrum of Leprosy

Nerve	Echo texture	TT (n=5)	BT (n=9)	BB (n=4)	BL (n=7)	BL (n=7)	LL (1=10)	p-value
Left Median	Normal	5 (100.0)	9 (100.0)	2 (50.0)	6 (85.7)	6 (85.7)	6 (60.0)	0.002*
	Abnormal	0 (0.0)	0 (0.0)	2 (50.0)	1 (14.3)	1 (14.3)	4 (40,0)	
Right Peroneal	Normal	3 (60.0)	9 (100.0)	1 (25.0)	6 (85.7)	6 (85.7)	4 (40,0)	0.022*
	Abnormal	2 (40.0)	0 (0.0)	3 (75.0)	1 (14.3)	1 (14.3)	6 (60.0)	
*Significant								

Table 4: Electrophysiological Parameters in Thickened vs. Non-Thickened Nerves

Nerve & Parameter	Thickened Nerves (Mean ± SD)	Non-Thickened Nerves (Mean ± SD)	p-value
Right Median Motor			
Distal Latency (ms)	8.28 ± 0.5	4.11 ± 1.21	<0.001*
Conduction Velocity (m/s)	27.45 ± 19.77	31.24 ± 19.88	0.722
Left Median Motor			
Distal Latency (ms)	6.96 ± 2.23	4.02 ± 1.25	<0.001*
Conduction Velocity (m/s)	21.80 ± 11.27	46.74 ± 71.68	0.449
Left Posterior Tibial Motor			
Distal Latency (ms)	8.73 ± 2.16	6.37 ± 2.5	0.039*
Conduction Velocity (m/s)	42.15 ± 6.03	40.66 ± 6.09	0.589
*Statistically significant ($p < 0.05$). Amplitude differences were not significant for these nerves.			

Table 5: Correlation between Clinical Nerve Thickening, HRUS, and Electrophysiology

Nerve	Parameter	Spearman's ρ	p-value
Right Median	HRUS Cross-Sectional Area	0.507	0.002*
	Motor Conduction Velocity	-0.04	0.819
Left Median	HRUS Cross-Sectional Area	0.571	<0.001*
	Motor Conduction Velocity	-0.239	0.168
Statistically significant ($p < 0.01$). No significant correlations were found for the ulnar, peroneal, or tibial nerves.			

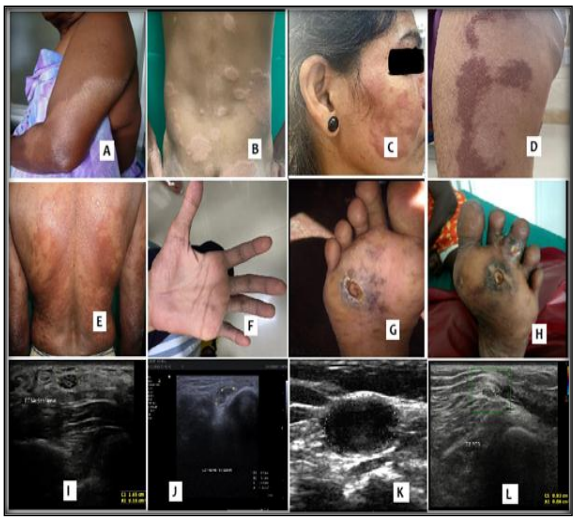

Figure 1: A: Tuberculoid leprosy. An ill-defined, large hypopigmented patch over the arm. B: Borderline Tuberculoid leprosy. Well-defined hypopigmented to erythematous plaque with raised edges and satellite lesions over the back. C: Mid Borderline Leprosy. to an ill-defined erythematous plaque with an inner edge well demarcated, outer edge sloping towards normal skin (Swiss cheese pattern) over the right cheek. D: Mid Borderline Leprosy. to an ill-defined erythematous plaque with areas of normal skin within the plaque over the lateral aspect of the left thigh. E: Lepromatous leprosy. Ill-defined large erythematous patches and nodules over the back of the trunk and arms. F: Claw hand deformity. G & H: trophic ulcer over the plantar aspect of the foot in patients with leprosy. I: High-resolution ultrasound of the normal median nerve at the wrist. (green dots). J: Thickened left ulnar nerve of patient with leprosy (yellow Dotted ellipse). K: Loss of fascicular pattern seen on the ulnar nerve of a patient with leprosy (Dotted ellipse). L: Thickened right peroneal nerve of patient with leprosy (yellow Dotted ellipse).

DISCUSSION

Leprous neuropathy is frequently clinically silent during the early course of the disease; therefore, it complicates diagnosis in early stages and increases the possibility of irreversible disability and deformity. Detection of nerve involvement early is therefore vital for preventing irreversible damage. In this study, we assessed peripheral nerve pathology of biopsy-proven Hansen's disease using nerve conduction studies (NCS) and high-resolution ultrasound (HRUS) and correlated them with clinical features. HRUS therefore provides a non-invasive evaluation of nerve morphology that complements electrophysiological studies that show functional integrity.[4,6,8] Our study population size was (n=35), mostly male (71.4 %), with a mean age of 32.5 years. These results are comparable with earlier Indian studies where they observed increased prevalence of Hansen's disease in young adult males.[7,9,10] The male preponderance has been explained by health-seeking behaviors, sociocultural impediments, and occupational exposures.[12] The mean number of months of symptoms was 12.4, which is less than that

of Jain et al. (4), where they found a mean prevalence of symptoms for 24.7 months. Since our mean duration of symptoms was shorter, it could be because of earlier referral in our cohort. Clinically, all patients had at least one affected nerve, with 71.4% having less than five nerves. The ulnar nerve was most commonly affected, followed by the common peroneal nerve, a finding in keeping with their superficial position and lower temperature, which promotes M. leprae invasion.[13,14] The majority was sensory deficit (42.8%), which conforms with the known susceptibility of sensory fibres in leprosy.[15] In contrast, some series have described motor symptoms as the first presentation.[7,5] The multibacillary spectrum was the predominant patient category, of which lepromatous leprosy (28.6%) was the most frequent subtype. Even in clinically normal nerves, subclinical neuropathy was evident from abnormalities detected in electrophysiological testing. Distal latency was significantly increased in thickened nerves, especially the median nerve and posterior tibial nerve; however, the conduction velocity difference was not statistically significant. These findings are in accordance with earlier studies in this field, where they found the existence of axonal neuropathy with mixed demyelinating characteristics. [16,17] Mononeuritis multiplex was the most frequent pattern, in accordance with previous studies. [18,19] However, electrophysiology lacks the spatial information, reinforcing the importance of HRUS.

On sonographic assessment, CSA of the nerves was significantly increased, mainly in the ulnar and common peroneal nerves. Echotextural abnormalities were more common in multibacillary forms and especially in lepromatous leprosy, and were statistically significant. Our findings are similar to those of Jain et al,[4] and Bathala et al,[9] who reported HRUS identification of clinically apparent and subclinical nerve involvement. Importantly, HRUS abnormalities were noted in some nerves without clinical thickening, affirming its greater sensitivity. Correlational analysis showed a significant correlation between HRUS CSA and clinical thickening of the median nerves, but no significant correlation with electrophysiological parameters. Bathala et al,[9] showed a correlation of ulnar nerve CSA with conduction abnormality, and Park et al,[20] reported results similar to ours. Discrepancies may be attributed to sample size heterogeneity, disease spectrum, or technical limits of lower limb nerve imaging. Our results corroborate the complementary nature of clinical evaluation, NCS, and HRUS. Clinical examination still is the cornerstone, but HRUS supplies objective morphologic data and NCS detects functional abnormality many times before clinical changes. Together, these modalities help increase diagnostic accuracy and potentially enable early intervention to prevent disability. Limitations of this study are that there was no control group, and normative CSA values were adopted from previous studies carried out in South India. HRUS interpretation can be affected by other conditions, such as edema, especially in the lower limb nerves. Therefore, future studies with larger cohorts and multicenter studies with standardized CSA threshold and long-term follow-up can provide essential data for the validity of HRUS as a reliable tool for surveillance of leprous neuropathy.

CONCLUSION

Leprosy is one of the main treatable causes of peripheral neuropathy in developing countries. Early diagnosis is of paramount importance in preventing deformities. We found a positive correlation between the clinical and CSA of the median nerve. However, no correlation was found between CSA and the electrophysiological function of the median nerve. In addition, no correlation was observed between clinical and electrophysiological function and CSA in the ulnar, common peroneal, and post-tibial nerves. From the above findings, we can conclude that HRUS alone is not an effective tool to determine early involvement of leprous neuropathy, and the aid of other tools, such as clinical examination and electrophysiological studies, is required for early detection of leprous neuropathy, since all three parameters have not shown significant correlation. Hence, a comprehensive approach, including clinical, electrophysiological, and HRUS assessments, is essential for the assessment of the morphology and function of nerves involved in leprosy for better outcomes.

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