DOI: 10.7860/JCDR/2026/90246.24252

Case Series

Radiology Section

The Role of High-resolution Nerve Ultrasonography in Atypical Presentations of Hansen's Disease: A Case Series

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ABSTRACT

Hansen disease is a chronic infectious disease with a predilection for peripheral nerves. Clinical examination may underestimate the extent of nerve involvement, particularly in patients with suspected mononeuropathy or atypical presentations. This case series describes five cases of Hansen disease in which high-frequency ultrasonography contributed to diagnosis and disease mapping. Patients were referred for ultrasound evaluation either with suspected involvement of a single peripheral nerve or with an alternative clinical diagnosis. High-Resolution Ultrasound (HRUS) was performed using a Philips EPIQ machine with an 18 MHz linear transducer, assessing the symptomatic nerve and additional accessible peripheral nerves bilaterally. Sonographic features including nerve thickening, altered fascicular pattern, hypoechogenicity, focal or diffuse enlargement were recorded. Assessment of the nerves was done on long and short axis imaging of each nerve and single measurements of Cross-sectional Area (CSA) were taken at the point of maximum nerve thickening by a single observer. Ultrasound revealed additional clinically unsuspected nerve thickening in three cases and redirected clinical suspicion toward Hansen disease in two cases initially thought to have other pathology. This series highlights high-frequency ultrasonography as a useful, non-invasive adjunct for mapping peripheral nerve involvement in Hansen disease and for raising diagnostic suspicion in atypical or clinically underestimated presentation. Although the World Health Organisation (WHO) has prescribed three diagnostic cardinal signs which include definitive loss of sensation in a pale or reddish skin patch, thickened or enlarged peripheral nerves with associated sensory or motor loss, and microscopic identification of Acid-Fast Bacilli (AFB) in a slit-skin smear, in tertiary and specialised medical settings, the absolute confirmation of Hansen's Disease (HD) relies on histopathological examination of a skin biopsy. A significant advantage of HRUS is its non-invasive nature. It is also useful in cases of Pure Neural Leprosy (PNL) where patients do not have skin lesions and negative skin smears, HRUS allows for diagnosis by documenting absolute nerve asymmetry and thickening especially in cases where an invasive nerve biopsy could cause permanent nerve damage. HRUS is also useful to identify early subclinical structural nerve abnormalities, asymmetrical cross-sectional enlargement, and internal fascicular changes before nerve damage translates into sensory or motor loss which can be identified during a routine clinical exam. Ultrasound also helps visualise the exact location of nerve thickening and can differentiate it from other aetiologies such as entrapment neuropathy.

Keywords: Hansen's neuritis, Nerve biopsy, Peripheral nerve ultrasound, Pure neuritic leprosy

INTRODUCTION

The HD, or leprosy, is a chronic infectious granulomatous disease of the skin and peripheral nerves caused by *Mycobacterium leprae* and *Mycobacterium lepromatosis*. Despite being curable with Multi-Drug Therapy (MDT), it remains a significant global health burden with around 200,000 new cases reported globally every year (last reported in 2024) according to the WHO [1]. In India, the prevalence rate is 0.57 per 10,000 population with a new case detection rate of 7.0 per 100,000 in 2024-25 [2]. It has a predilection for Schwann cells [3], leading to inflammatory nerve destruction and subsequent sensory and motor neuropathy.

The Ridley-Jopling classification, a spectral system categorises HD into five groups, based on clinical features, histopathology, bacterial load, and the host's cell-mediated immune response to *Mycobacterium leprae*. It ranges from high-immunity forms with localised symptoms to low-immunity forms with widespread disease. The five main groups, ordered from highest to lowest immunity, are Tuberculoid (TT), Borderline Tuberculoid (BT), Mid-Borderline (BB), Borderline Lepromatous (BL), and Lepromatous (LL) [4]. The WHO simplifies this classification for field treatment, dividing cases into Paucibacillary (TT and BT) and Multibacillary (MB), which corresponds to BB, BL, and LL [4].

The diagnosis of HD is based on clinical presentation, and the diagnosis is confirmed by skin or nerve biopsy and acid-fast staining [4]. Traditional diagnosis relies heavily on clinical palpation to detect nerve thickening; however, this is inherently subjective and often fails to identify early-stage intraneural changes [5]. HRUS has emerged as a vital diagnostic tool, allowing for detection of early neural changes, providing objective quantification of Cross-sectional Area (CSA), detailed assessment of internal fascicular architecture, mapping the extent of involvement and helping to choose an appropriate nerve to target for biopsy [6].

Pure Neuritic Leprosy (PNL), characterised by involvement of peripheral nerves in the absence of skin lesions, constitutes approximately 4-18% of cases in Indian research and presents significant diagnostic challenges [7]. According to Sreejith K et al., high-resolution nerve ultrasound detected nerve enlargement in 47% of cases, whereas clinical diagnosis identified nerve enlargement in 20% of cases [5].

CASE SERIES

Authors present five cases of HD characterised by atypical clinical presentations masquerading as localised neurological or traumatic conditions. In all cases, HRUS played a pivotal role in redirecting the diagnosis by identifying characteristic sonomorphological patterns of leprosy-associated neuritis and these were subsequently confirmed by biopsy.

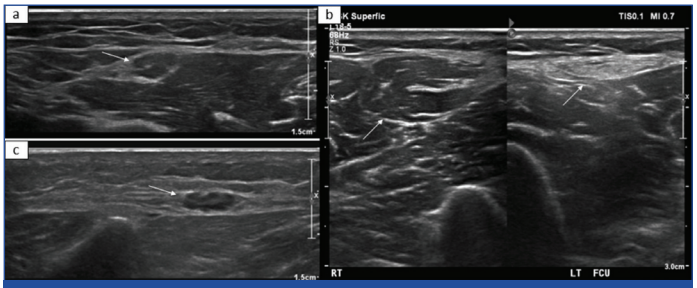
HRUS was performed using a Philips EPIQ machine with an 18 MHz linear transducer, assessing the symptomatic nerve and additional accessible peripheral nerves bilaterally.

These cases highlight the importance of considering HD in the differential diagnosis of peripheral neuropathies and nerve enlargements, even in the apparent absence of classic cutaneous findings or in the presence of confounding factors such as prior trauma.

Case 1

A 32-year-old male presented with a two-year history of chronic, progressive paraesthesia involving both the hands and feet, accompanied by progressive motor weakness of the left hand for the past six months. Clinical examination revealed a left ulnar claw hand and significant bilateral thickening of the ulnar nerves. No cutaneous lesions were present.

Nerve Conduction Study (NCS) showed sensory axonal polyneuropathy and asymmetric sensory motor neuropathy involving left upper limb. Electromyography (EMG) showed neurogenic changes in left ulnar innervated muscles. Slit skin smears were all negative for AFB. HRUS [Table/Fig-1] demonstrated diffuse thickening and hypoechogenicity with an increase in internal vascularity of the bilateral ulnar and median nerves, as well as the bilateral common peroneal and superficial peroneal nerves. No evidence of abscess. CSA values for thickened nerves were, right median- 13 mm^2, right common peroneal nerve- 15 mm^2, left superficial peroneal nerve- 13 mm^2, right superficial peroneal nerve- 11 mm^2 and left ulnar nerve- 15 mm^2.



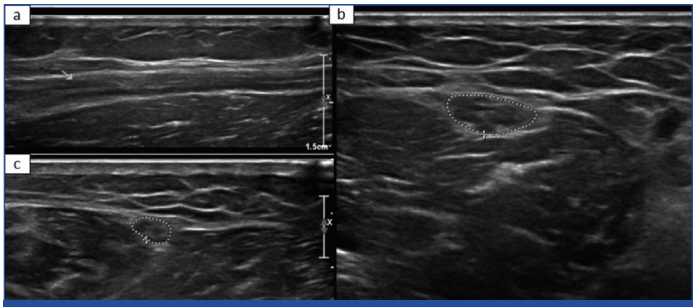
[Table/Fig-1]: High-resolution Ultrasound images in a 32-year-old gentleman with paraesthesia in both hands and weakness of the left hand
a. Left ulnar nerve in the forearm with hypoechoic thickening and loss of fascicular pattern.
b. Atrophy and fat replacement of the left flexor carpi ulnaris in comparison to the normal appearance on the right.
c. Hypoechoic thickening with maintained fascicular pattern of the right superficial peroneal nerve.

These findings were highly suggestive of HD. A biopsy of the cutaneous branch of the right ulnar nerve confirmed histopathological features of Hansen's neuritis with ill formed histiocytic granulomas with marked nerve fibre loss and absence of AFB on special stains-BT/TT in the Ridley Jopling classification. The absence of skin lesions confirmed a diagnosis of PNL. The patient was started on Multi-Bacillary MDT (MB-MDT) subsequently improved with MDT.

Case 2

A 31-year-old male presented with a hypopigmented patch on the back of his trunk, accompanied by sensory and motor deficits in the right hand for three years that had progressed to involve the left hand. Clinical examination revealed sensory loss and motor weakness along the ulnar distribution of both hands, with more pronounced involvement on the right side. Significant nerve enlargement was noted in the bilateral ulnar nerves and the left common peroneal nerve.

NCS showed asymmetric sensory motor axonal polyneuropathy in bilateral upper and lower limbs. Slit skin smears were all negative for AFB. HRUS assessment [Table/Fig-2] identified long-segment hypoechoic thickening and a loss of the normal fascicular pattern in the bilateral ulnar, superficial radial and superficial peroneal nerves. CSA values of the thickened nerves were, right ulnar - 14 mm^2, left ulnar - 15 mm^2, right superficial peroneal nerve - 6.1 mm^2 and left superficial peroneal nerve - 1.3 mm^2.



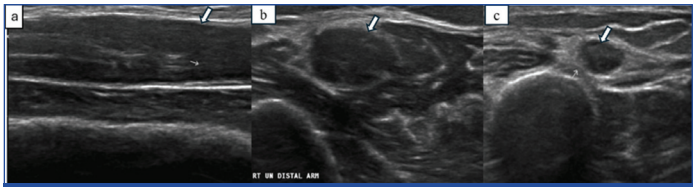
[Table/Fig-2]: High-resolution ultrasound images in 31-year-old gentleman with sensory and motor deficits in both hands
a. Right ulnar nerve in the arm with diffuse hypoechoic thickening.
b. Left ulnar nerve in the forearm with thickening and loss of fascicular pattern.
c. Hypoechoic thickening with maintained fascicular pattern of the right superficial peroneal nerve.

Additionally, fatty atrophy was observed in the right flexor carpi ulnaris muscle, indicating chronic denervation. A biopsy of a cutaneous branch of the right ulnar nerve confirmed features consistent with Hansen's neuritis with foamy histiocytes, ill-formed epithelioid cell granuloma and moderate lymphocytic infiltrates of the perineurium, special stains for AFB were negative - BB in the Ridley Jopling classification [4] Following the diagnosis, the patient was initiated on MB-MDT following which he had reduction in sensory symptoms and skin patches.

Case 3

A 62-year-old male presented with pain, swelling, and weakness of the right upper limb, three years following a fall from a bike. On examination he was found to have wasting and weakness of the muscles of the right hand with right claw hand, however tone was normal.

The NCS showed right ulnar neuropathy at wrist. Although tardy ulnar nerve palsy was initially suspected based on the clinical history of trauma, the patient was referred for a HRUS evaluation to further characterise the nerve involvement. The HRUS examination [Table/Fig-3] demonstrated fascicular thickening of the right ulnar nerve, without evidence of neuroma formation or nerve discontinuity. Notably, there was also thickening of the right superficial radial nerve. CSA of the right ulnar nerve in the arm was upto 47 mm^2 and right superficial radial nerve was 9 mm^2.



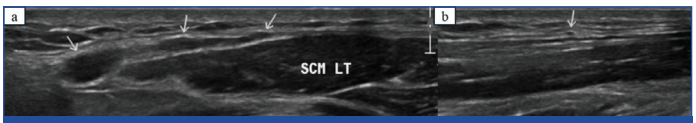
[Table/Fig-3]: High-resolution ultrasound findings in a 62-year-old man with pain and weakness of the right upper limb following a road traffic accident, clinically suspected to have tardy ulnar nerve palsy.
a. Longitudinal image showing diffuse hypoechoic thickening with loss of fascicular appearance of the right ulnar nerve in the distal arm.
b. Corresponding axial image of the right ulnar nerve showing hypoechoic thickening.
c. Axial image of the right superficial radial nerve in the mid forearm showing thickening with loss of the internal fascicular architecture, suggesting multifocal peripheral nerve involvement in favour of Hansen disease.

The identification of multifocal peripheral nerve thickening extending beyond the site of initial trauma raised a high clinical suspicion for HD. Upon retrospective physical examination, hypopigmented patch on the right hand which was seen but not previously attributed to HD was identified. Slit skin smears were all negative for AFB. Biopsy from the patch on the right hand showed dermal perivascular and perineural chronic inflammation.

The patient was subsequently initiated on MB-MDT therapy. Following the commencement of treatment, he showed significant clinical improvement, confirming the diagnosis of leprosy-associated neuritis masquerading as post-traumatic nerve palsy.

Case 4

A 54-year-old male presented with a three-year history of left lateral neck swelling and was referred for ultrasound evaluation. Initial imaging did not identify any focal lesions or fluid collections within the neck. HRUS demonstrated nodular thickening of the left greater auricular nerve [Table/Fig-4]. Subsequent clinical examination also revealed a skin patch on the patient's right forearm.



[Table/Fig-4]: High-resolution ultrasound findings in a 54-year-old patient presenting with left lateral neck swelling no focal neck lesion, mass or collections was identified on ultrasound.
a. Nodularity and hypoechoic thickening of the greater auricular nerve (white arrows), on the left aspect of the neck beneath the platysma where it emerges from behind the sternocleidomastoid (SCM).
b. Right greater auricular nerve (white arrow) is imaged for comparison, shows no abnormal thickening or nodularity.

The EMG in bilateral upper and lower limbs was normal. These findings, particularly the localised nerve enlargement in conjunction with a cutaneous lesion, raised a strong suspicion of HD.

Slit skin smears were negative for AFB, however in view of strong compelling clinical and ultrasound findings, the patient was classified as Multibacillary under the WHO classification [1] and was treated with MB-MDT for one year. Biopsy of the greater auricular nerve was not done. On subsequent follow-up, there was no pain in the region of the left greater auricular nerve and the nerve was not clinically palpable.

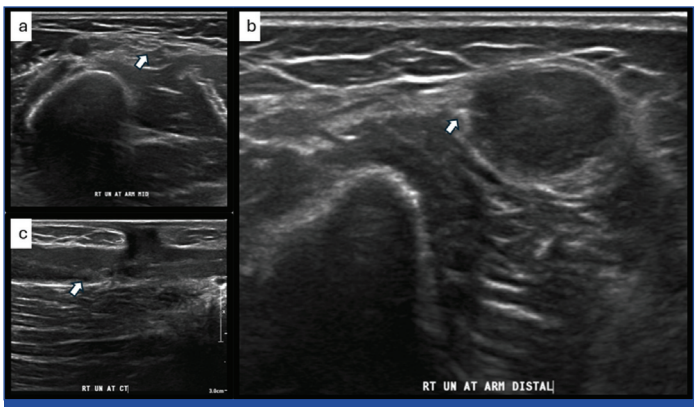
Case 5

A seven-year-old patient living in a children's home presented with a 6-month history of dry patches and impaired sensation on the medial side of the right hand, along with deformity of the right hand for three months. Clinical examination showed clawing of the right ring and little fingers, stiffness of the Proximal Interphalangeal (PIP) joint of the little finger, a small ulcer on the dorsum of the little finger, and thickening of the ulnar nerve. Mild erythema and oedema were also observed over the right palm. NCS was not performed, and slit skin smears tested negative for AFB.

The HRUS assessment [Table/Fig-5] revealed significant thickening of the ulnar nerve, with a complete loss of its normal fascicular structure from distal arm to distal forearm. The CSA of the right ulnar nerve was 53 mm^2. There were focal collections with associated sinus tracts in both the distal arm and distal forearm. These findings strongly indicate extensive ulnar nerve involvement, including nerve abscesses and sinus tract formation.

A skin biopsy from the ulnar side of the wrist revealed dermal necrotising granulomatous inflammation with deep scarring, and special stains were negative for AFB. Based on these clinical, bacteriological, and histopathological findings, the patient was classified as Borderline tuberculoid according to the Ridley Jopling system [4] and as a paucibacillary type based on the WHO classification [1]. The patient also experienced a Type 1 lepra reaction.

She was initiated on MBMDT monthly pulse and daily medications. For her right ulnar claw, she consulted Hand and Leprosy Reconstructive Surgery (HLRS), and physiotherapy exercises were taught and were given knuckle bender splint. The patient was counselled regarding necessity and the possible side-effects of treatment. Screening of the children in home was advised to her guardian and contact tracing was ordered to the social worker from the institution.



[Table/Fig-5]: High-resolution ultrasound images of a 7-year-old patient history of impaired sensation and deformity of the right hand.
a. Diffusely thickened ulnar nerve (white arrow) with loss of fascicular appearance in the mid arm.
b. Grossly thickened ulnar nerve (white arrow) with loss of fascicular appearance in the distal arm.
c. Focal hypoechoic tract (white arrow) extending from the skin in the region of the volar aspect of the carpal tunnel and hypoechoic abscesses within the thickened nerve.

DISCUSSION

The HRUS findings in this case series are comparable to those described in previous reports of HD-associated neuritis**.** Jain S et al., showed that HRUS can objectively identify nerve enlargement, altered echotexture, and increased vascularity, and HRUS often shows more extensive nerve involvement than clinical examination. In a case report of primary neuritic leprosy, Jain S et al., described marked ulnar nerve enlargement above the elbow, altered echotexture, increased epineural blood flow, and additional involvement of cutaneous nerves, with biopsy findings concordant with the ultrasound findings [6]. Elias J et al., similarly reported focal ulnar nerve thickening, hypoechoic areas, and loss of the normal fascicular pattern in patients with leprosy neuropathy [8], while Bathala LN et al., found that ulnar nerve enlargement in Hansen's neuropathy was commonly maximal a few centimeters proximal to the medial epicondyle and was associated with hypoechoic change, altered fascicular morphology, and doppler vascularity [9].

The ulnar nerve was the most frequently involved nerve in present case series with the following CSAs:

Case 1: left ulnar nerve -15 mm^2

Case 2: right ulnar nerve-14 mm^2, left ulnar nerve-15 mm^2

Case 3: right ulnar nerve upto 47 mm^2

Case 5: right ulnar nerve upto 53 mm^2

The ulnar nerve cut-off proposed by Elias J et al., is 9.8 mm^2 [8] and our CSAs were above the reported cut-offs in the literature. CSA of the right median nerve in case 1 is 13 mm^2 which exceeded the cut-off of 10.17 mm^2 proposed by Sreejith K et al., [5]. The presence of multifocal involvement of the ulnar, median, peroneal, superficial radial and greater auricular nerves in present case series supports the asymmetric, regional, non-uniform involvement of the nerves published in the previous studies [10].

Even though HRUS is helpful in the assessing nerve thickening in Hansen's neuritis, there are other close mimickers that can share similar features as Hansens neuritis such as Chronic Inflammatory Demyelinating Polyneuropathy (CIDP), Multifocal Motor Neuropathy (MMN), hereditary neuropathy with liability to pressure palsies (HNPP), or other granulomatous neuropathies [11]. In Hansen's neuritis, the HRUS finding commonly shows asymmetric, non-uniform segmental thickening of the nerves [10]. In CIDP, the nerve enlargement is more diffuse and also proximal [12]. In MMN, HRUS demonstrates focal fascicular enlargement of the nerve fascicle which corresponds to the conduction block on electrophysiology studies [13]. In HNPP, the nerve enlargement predominantly involves the entrapment sites [11].

The MR neurography would be the other imaging modality for peripheral nerve assessment. With its high resolution, it can provide valuable information of the deeper nerves difficult to screen on HRUS and also proximal most nerves and plexuses. It can demonstrate

nerve thickening, enhancement, presence of intraneural and micro-abscesses and associated muscle denervation [14,15].

This case series had several limitations that must be acknowledged. Firstly, the sample size is modest (n=5), which limits the generalisability of the findings and precludes the application of formal statistical analysis. Secondly, inter-observer variability was not formally evaluated because all examinations were conducted by a single operator. Thirdly, NCS data were unavailable for all cases at the initial assessment. Lastly, the series originates from a single tertiary referral centre, thereby introducing potential referral bias towards atypical and diagnostically challenging cases.

CONCLUSION(S)

HRUS is a valuable, non-invasive tool in diagnosing HD, capable of identifying subclinical nerve involvement, mapping multifocal disease, and guiding biopsies. Based on this series and supporting literature, HRUS should be considered in clinical scenarios such as: (1) suspected PNL with negative skin smear; (2) unexplained peripheral neuropathy or nerve enlargement in HD-endemic regions; (3) paediatric nerve cases in communal or institutional environments; and (4) isolated nerve enlargement that resembles soft-tissue masses. HRUS can detect nerve enlargement even before the classic clinical symptoms of leprosy at the stage of subclinical neuropathy.

The results of this series support integrating HRUS into the HD diagnostic process alongside clinical examination, Slit skin smear, and NCS, with nerve biopsy used for histopathological confirmation. However, due to the small size of this series, these findings need validation through larger, prospective studies. Future studies should include a large cohort of patients from different centres using standardised HRUS protocol and normative CSA measurements. Correlation with NCS is also needed to better define the role of HRUS in Hansen's neuritis.

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PLAGIARISM CHECKING METHODS: [Jain H et al.]
- Plagiarism X-checker: May 09, 2026
- Manual Googling: Jul 11, 2026
- iThenticate Software: Jul 13, 2026 (8%)

ETYMOLOGY: Author Origin

EMENDATIONS: 6

AUTHOR DECLARATION:
- Financial or Other Competing Interests: None
- Was informed consent obtained from the subjects involved in the study? No (Waived in view of retrospective case series)
- For any images presented appropriate consent has been obtained from the subjects. No (Waived in view of retrospective case series)

Date of Submission: **May 04, 2026**
Date of Peer Review: **May 28, 2026**
Date of Acceptance: **Jul 15, 2026**
Date of Publishing: **Sep 01, 2026**