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Case Report

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A Case Report on Intraneural Ulnar Nerve Abscess in Lepromatous Hansen Disease

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ABSTRACT

Background: Leprosy (Hansen disease) primarily affects peripheral nerves and may present as neuritis, nerve thickening, and, rarely, abscess formation, leading to irreversible neuronal damage if untreated. Although nerve abscesses are more commonly seen in borderline tuberculoid leprosy, they can also occur in lepromatous leprosy, particularly in cases of inadequate multidrug therapy. This case highlights the diagnostic challenges and emphasizes the role of imaging in the early detection and management of intraneural ulnar nerve abscess.

Methods: A 34-year-old male with a history of inadequately treated lepromatous leprosy presented with pain, paresthesia, progressive numbness, and weakness along the medial aspect of the left forearm and hand. Clinical examination revealed a thickened, tender, nodular ulnar nerve with sensory loss and early clawing deformity. High-resolution ultrasonography demonstrated diffuse nerve enlargement, loss of fascicular architecture, intraneural hypoechoic foci, epineural thickening, and increased vascularity suggestive of active neuritis with abscess formation. MRI showed diffuse nerve thickening, T2 hyperintensity, fascicular effacement, and focal diffusion restriction consistent with intraneural purulent collection.

Results: Multidrug therapy was resumed, along with systemic corticosteroids and thalidomide, for a Type 2 reaction. Surgical decompression with epineurotomy and drainage of purulent material was performed. Imaging findings correlated with intraoperative confirmation of intraneural abscess.

Conclusion: Intraneural ulnar nerve abscess is a rare but significant complication of lepromatous leprosy. Early diagnosis through clinical examination and complementary use of ultrasonography and MRI, followed by prompt medical and surgical management, is essential to reduce morbidity and preserve nerve function.

Key-words: Leprosy, Ulnar nerve abscess, Ultrasonography, MRI, Nerve decompression

INTRODUCTION

Leprosy, or Hansen disease, is a granulomatous infection of the body that is not fatal, slowly progressive, and chronic, caused by *Mycobacterium leprae*, & mentioned in ancient Indian literature in the sixth century B.C. Preferential involvement of the skin, peripheral nervous system, upper respiratory tract, eyes, and testes is observed, as they are cooler than core body temperature. The immune-mediated reactions and neurotropism of the bacteria are very debilitating and mutilating and result in psychosocial destruction. Depending on the person's immune state, leprosy manifests as a continuum between polar tuberculoid and polar lepromatous leprosy. Abscesses of nerves, most commonly the ulnar nerve, may occur in patients with all

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types of leprosy, particularly in those with borderline tuberculoid (BT) leprosy [1,2].

Leprosy can be regarded as the most prevalent treatable neuropathy in the endemic areas; it is estimated that 600,000 new cases are detected each year, with 60% of the cases being detected in India. *M. leprae* is an obligatory intracellular bacterium as a result of reductive evolutionary loss of genes that encode metabolic and respiratory pathways. The basal lamina of Schwann cells is bound by a unique trisaccharide present in the wall of *M. leprae*. It allows invasion by Schwann cells and by endoneural and perineural macrophages. In leprosy, nerve disease is manifested across the spectrum of the disease and in lepra reactions. The neural lesion is usually a granuloma, but in rare cases, it can take the shape of an abscess, especially in BT leprosy patients [3].

In tuberculoid leprosy, the nerve is aggressively infiltrated by epithelioid and lymphoid cells, leading to thickening of the epineurium and perineurium and destruction of fascicles. The immune response of the lepromatous pole is less drastic, and the active multiplication of bacilli takes place, and the neural architecture is consequently better preserved. It has been conjectured that pressure-induced tissue anoxia, due to swollen neuronal fascicles caused by inflammation, will result in avascular necrosis and abscess development. The nerve swellings in leprosy are similar to those of entrapment neuropathies; although in leprosy, the nerve swelling is more complete [4].

The most common nerve to have an abscess is the ulnar nerve. In India, nerve abscesses occur in about 1.3% of leprosy patients, and some of these are calcified. There has been an increase in the incidence of nerve abscesses in leprosy, which has been credited to further application of multi-drug therapy programs with inadequate infrastructure to detect and treat early neuritis. Young children and teenagers are most affected [5,6].

Recent advances in high-frequency ultrasonography have improved the assessment of peripheral nerve damage in leprosy. Normal nerves have a typical fascicular structure: longitudinal scans reveal parallel hypoechoic fascicles separated by hyperechoic perineurium and epineurium, whereas transverse scans have a honeycomb structure. Sonography of the affected nerves may show focal thickening, especially near the medial epicondyle; hypoechoic granulomatous lesions; peripheral hyperechogenicity due to epineural fibrosis; abscesses; and augmented vascularity on color Doppler during lepra reactions. The electrophysiological examination of disturbed neuroanatomy can be a potent indicator of disease in the early stages, even in the absence of symptoms in contacts [5,6].

The MRI findings are also less specific, but they are generally diffuse nerve swelling and edema, which can be mixed with other hypertrophic neuropathies. But nodules, granulomas, or nerve sheath are favorable to leprosy. Nerve abscesses are hypointense on T1 and hyperintense on T2, with peripheral enhancement on post-contrast images. It is important to distinguish between differentiation and reversal reactions, as abscesses can respond to corticosteroids, whereas reversal reactions can be treated with surgical decompression. Early neuroarthropathic changes in the joints are identified using MRI [7].

CASE PRESENTATION

A 34-year-old man who is a manual worker reported to the outpatient section with complaints of intermittent pain, tingling, and progressive numbness along the medial side of his left forearm and hand that lasted two months. The symptoms were insidious, slowly progressive, and began with occasional paresthesia in the little finger and the ulnar half of the ring finger, followed by dull aching pain in the medial elbow. During the three weeks before, he complained that he was having more trouble with fine-motor activities, such as holding small objects between his fingers and pressing buttons on his clothing. No recent trauma history, no fever, no redness of the location, no constitutional symptoms, including weight loss or night sweats.

The patient was known to have a history of Hansen disease (Lepromatous leprosy) diagnosed two years previously, to which multidrug therapy (MDT) had been added. Nevertheless, he confessed that he stopped treatment within two months due to symptomatic response and socioeconomic reasons. He had no history of diabetes mellitus, tuberculosis, autoimmune disease, or any other similar neurological history. The family history of neuropathies and chronic infections was absent.

General physical examination revealed the patient to be afebrile and hemodynamically stable. The patient had no nodules on the cutaneous surface, hypopigmented areas, or trophic ulcers. No abnormalities were observed in the

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examination of the eyes, the nasal mucosa and the testes. Systemic analysis of cardiovascular, respiratory & abdominal was not outstanding. Neurological and musculoskeletal examination of the left upper limb showed obvious fullness on the medial side of the left arm just above the elbow. Palpation uncovered a thickened and cord-like ulnar nerve, which was tender and nodular, particularly immediately above the medial epicondylar region. There were several small hard swellings discernible along the nerve. No overlying skin erythema or discharge, local temperature not elevated.

Motor examination revealed ulnar intrinsic muscle weakness with partial clawing of the fourth and fifth digits. Grip strength was mildly reduced, and Froment's sign was weakly positive. Mild interossei atrophy was present, while thenar and hypothenar eminences were preserved.

There was reduced light touch, pinprick, and temperature sensation of the medial half of the palm, the back of the hand, and the little finger. There is intact proprioception. Deep tendon reflexes were normal. There was no deficit in the opposite limb. Since the patient had a history of leprosy and focal neurological weakness localized to the ulnar nerve, ulnar neuritis with potential inflammatory or infective etiology was a working diagnosis. Differential diagnoses were compressive neuropathy at the cubital tunnel, peripheral nerve sheath tumour, immune-mediated neuritis and intraneural abscess. Further characterization was therefore performed under imaging.

ULTRASONOGRAPHY

An 18-5MHz linear transducer was used to perform a high-resolution ultrasonography of the left ulnar nerve. On inspection of the distal arm and cubital tunnel, there was diffuse fusiform enlargement of the ulnar nerve above the medial epicondyle. The nerve proved to be extremely thick, with a cross-sectional area of about 33 mm^2, well above normal values. The fascicular honeycomb structure of the nerve common to the normal was completely lost.

The nerve was heterogeneously hypoechoic, with multiple scattered intraneural hypoechoic foci appearing as fluid collections on axial and longitudinal scans. The epineurium was thickened and irregular. Color Doppler interrogation revealed increased intraneural vascularity, consistent with active inflammatory neuritis. These characteristics were suspicious of the early abscess formation in the nerve substance. The imaging findings were consistent with diffuse thickening, loss of fascicular architecture, intraneural hypoechoic foci, epineural thickening, and increased vascularity. Since ultrasonography suggested intraneural collections, magnetic resonance imaging was performed to define the pathology better and assess the level of involvement. The diffuse enlargement of the ulnar nerve is confirmed in high-resolution ultrasonography of the distal arm and cubital tunnel on axial (A, B) and longitudinal (C) imaging. In axial images, the nerve looks very thickened and diffusely hypoechogenic and totally devoid of the normal fascicular honeycomb architecture. Several scattered intraneural hypoechoic foci are observed, which are in line with fascicular edema and inflammatory processes. The cross-sectional view is heterogeneous and nerve calibre is evidently enlarged as compared to what is anticipated of normal dimensions at the cubital tunnel level. Colour Doppler examination (D) shows increased intraneural vascularity, with internal blood flow visible, which is not normally seen in peripheral nerves (Fig. 1).

MAGNETIC RESONANCE IMAGING

The left elbow MRI was done in a 1.5-Tesla machine with dedicated surface coils. Axial, coronal and sagittal T2-weighted images revealed a significantly thickened and edematous ulnar nerve, which stretched about 3-5 cm above the medial epicondyle. The T2-weighted images showed the nerve to be hyperintense, indicating edema and inflammatory changes. The normal fascicular pattern had been greatly destroyed and conglomerate hyperintense areas had replaced it.

Diffusion-weighted imaging (DWI) demonstrated the presence of focal areas of severe diffusion restriction in the swollen portion of the nerve itself, with the observed low apparent diffusion coefficient (ADC) values, which are typical of purulent collections. All these characteristics strongly indicated the presence of intraneural abscess formation rather than simple inflammatory neuritis. No muscular edema, bone marrow, or external-neural soft tissue abscesses were observed. The presence of a rather small, slightly swollen lymph node, i.e., 1.4 × 0.7 cm, was observed. Diffuse thickening, fascicular effacement, T2 hyperintensity, and diffusion restriction indicative of abscess were observed on MRI (Fig. 2).

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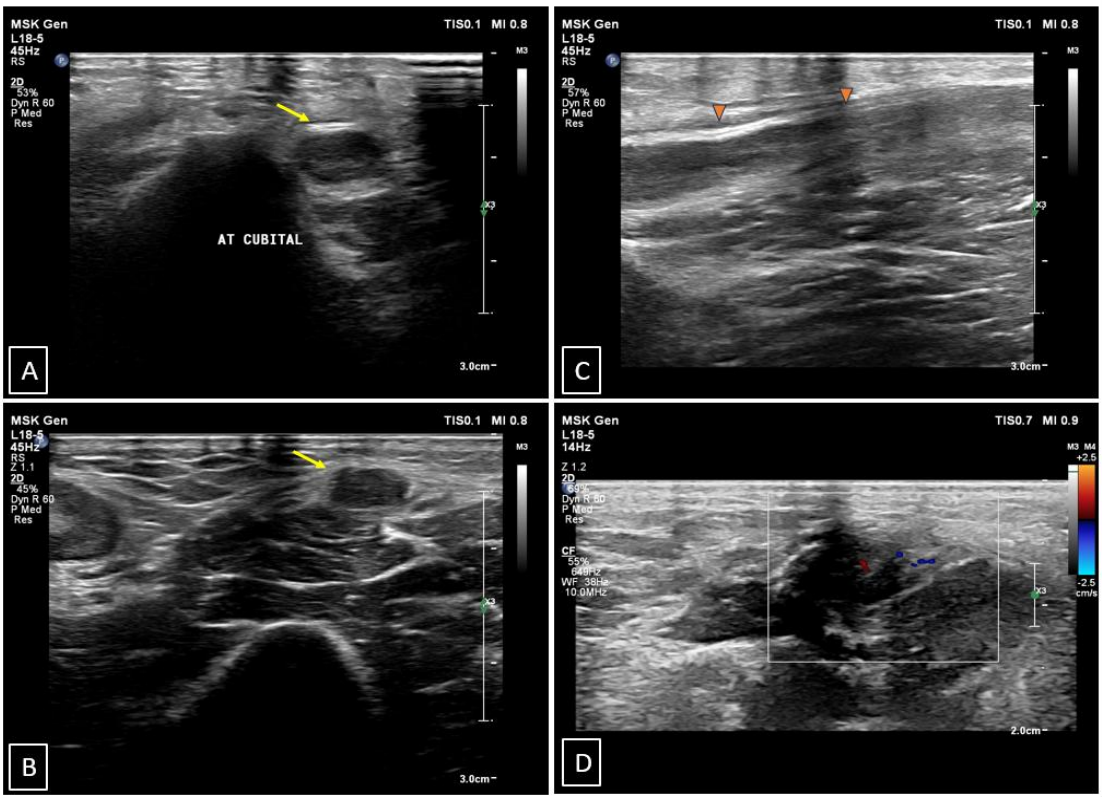

Fig. 1: Ultrasonography has revealed a significant degree of diffuse thickening of the ulnar nerve on axial (A, B) and longitudinal (C) images, and showing, on all of them, complete loss of normal fascicular structure and foci of intraneural hypoechogenesity. Epineural thickening is noticeable (arrowhead). Colour Doppler imaging (D) demonstrates increased intraneural vascularity, which is an active sign of neuritis.

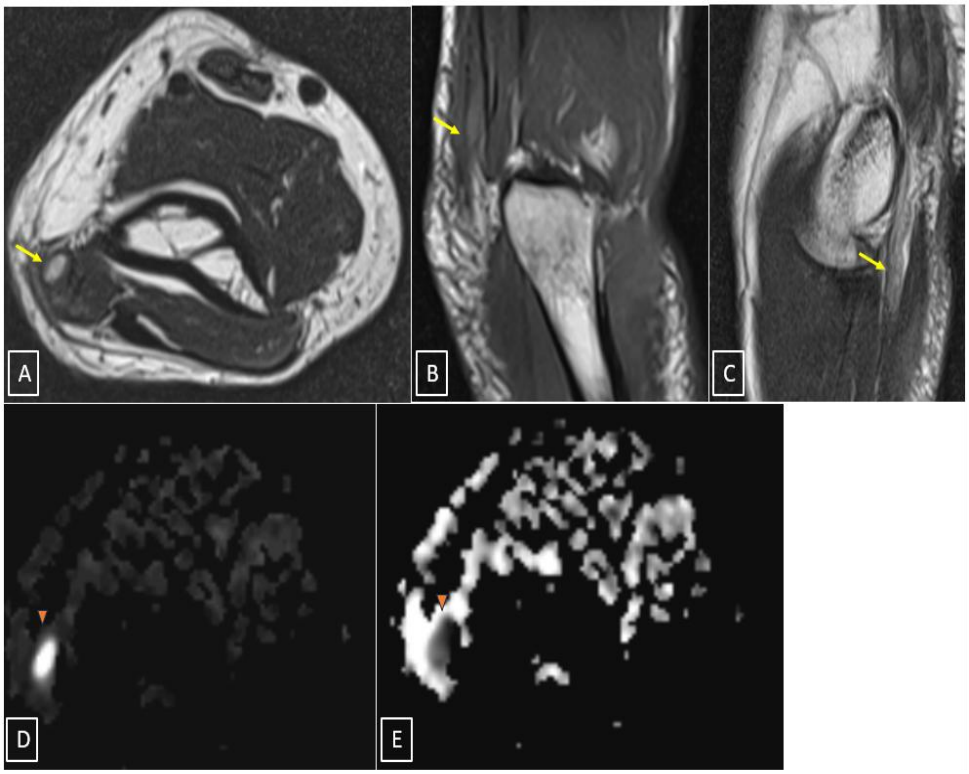

Fig. 2: The T2-weighted MRI images are of axial (A), coronal (B), and sagittal (C) images that show a diffusely thickened, edematous ulnar nerve with loss of the normal fascicular structure. Diffusion-weighted imaging (D, E) demonstrates significant diffusion restriction in the diseased segment, which is in line with the appearance of intraneural abscesses.

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MANAGEMENT

The patient was admitted to undergo additional management. Multidrug therapy for leprosy was resumed in accordance with national guidelines. Oral corticosteroids were also used to decrease neuritic inflammation and nerve edema. The patient was treated as an atypical presentation Type 2 reaction (bullous lesions) lepromatous leprosy treated by three drug multidrug therapy, cap thalidomide 100 mg thrice daily and oral prednisolone 1mg/kg initiated and continued. Consultation with surgery was given due to the presence of imaging evidence of an abscess and the risk of progressive neurological compromise. Regional anaesthesia of the ulnar nerve of the cubital tunnel was performed. During surgery, the nerve was observed to be thick, tense and attached to the adjacent tissues. Purulent material was found on the nerve sheath after careful epineurotomy and decompression, which indicated the existence of an intraneural abscess. The abscess cavity was debrided, drained, and microbiological and histopathological samples were sent. It created a decompression along the affected part of the nerve to decongest it and regain vascularity.

DISCUSSION

Leprosy is a skin and nerve disease; both peripheral and cutaneous nerves are usually involved. Schwann cells serve as a reservoir for *M. leprae*. In tuberculoid and lepromatous leprosy, the pathology differs. In TB leprosy, the cell-mediated immune response results in granuloma formation that, in other instances, can liquefy and degenerate into an abscess. In lepromatous leprosy, an abscess may be a consequence of an antigen-antibody response, e.g., an ENL response, exacerbation of a lepromatous lesion, necrosis of a lepromatous granuloma, or, possibly, an iatrogenic response as a complication of perineural or intraneural injection [8].

Leprosy is a chronic infectious disease caused by *M. leprae*. Leprosy is known to vary in its clinical presentation due primarily to the microorganism's affinity for skin and peripheral nerve tissue, as well as patients' genetic susceptibility to *M. leprae*, which is genetically determined and variable among individuals. Nerve involvement was noted at both ends of the disease spectrum, including type 1 reaction. The combination of local bacillus infiltration and immunologically mediated reactions, including reversal reactions and acute neuritis, may involve the nerves. Neural lesions are usually granulomas, although in rare cases, especially in persons with tuberculoid or borderline tuberculoid leprosy, they may progress to abscesses and result in permanent nerve damage and abnormality. Nerve abscesses are present in 1.3% of patients with leprosy in India. T-cells damage Schwann cells and axons, besides destroying the perineurium of a peripheral nerve [7,8].

Even though the bacteria favour the Schwann cell habitat of the nerve, in such situations, it is usually the ensheathed axons that are affected. Occasionally, due to severe inflammatory changes, caseous necrosis and liquefaction of the granuloma may occur. A coalescing of caseating lesions in the nerve may result in a nerve abscess. Most affected nerves are the ulnar nerve and the peroneal nerve, the median nerve and other cutaneous nerves of the upper and lower limbs. In tuberculoid leprosy, a cell-mediated immune response leads to the development of a granuloma, which may liquify and form an abscess [9]. Conversely, an antigen-antibody complex, e.g., an ENL reaction, extension of a lepromatous lesion, necrosis of a lepromatous granuloma, or, perhaps, an iatrogenic reaction due to perineural or intraneural injection, may result in the occurrence of lepromatous leprosy abscesses. ENL is mediated by immune complexes and characterized by a Th2 response. Evidence of a Th2 response is the finding of high levels of IL-6, IL-8, and IL-10 in the lesions, but also of high levels of TNF-α and TGF-β [10].

Calcification may develop in caseation and necrotic tissue in tuberculoid and borderline tuberculoid leprosy due to the deposition of calcium in dying, dead, and chronically inflamed tissue. 3 cases of calcification in nerves were reported by Malaviya *et al*. [11]. It was reported that Ramanujam *et al*. [12], Jopling, and Ellis had some calcification in the nerves. The ulnar nerve was the most frequently affected in most of the reports. Exploration of the nerves revealed white chalky material [8].

Nerves in the area of leprosy on colour Doppler images reveal focal thickening, more prominent in proximity to the medial epicondyle, and epineural fibrosis, manifesting as peripheral hyperechogenicity and hypervascularity. Symptoms that cannot be resolved by steroid therapy, chronic need for high doses of steroid therapy, and worsening of sensory and/or motor loss are

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the primary reasons for surgery in the cases of nerve abscess [13].

With advances in high-frequency transducers, it is now possible to study nerves in detail. As the perineurium/epineurium is the area between the neuronal fascicles (several hypoechoic parallel linear areas), longitudinal scans of normal nerves have an appearance of fasciculations (hyperechoic bands). Transverse scan of nerves depicts a honeycomb structure with rounded hypoechoic neuronal fascicles on a hyperechoic background. Nerve damage due to leprosy may be observed on ultrasound with focal thickening, hypoechoic lesions containing a disorganised fascicular pattern and focal hyperechoic foci. On colour Doppler imaging, nerves affected by leprosy exhibit focal thickening, which is more prominent towards the medial epicondyle, epineural fibrosis, peripheral hyperechogenicity, and increased vascularity [14,15].

CONCLUSIONS

Ulnar nerve abscess is a rare but significant complication of Hansen disease that can lead to substantial neurological morbidity if not identified early. Although more commonly associated with tuberculoid and borderline forms, this case demonstrates that it can also occur in lepromatous leprosy, particularly in the setting of immune-mediated reactions such as ENL or irregular multidrug therapy. Clinically, the presentation may mimic compressive ulnar neuropathy, making careful evaluation essential in endemic regions. High-resolution ultrasonography serves as an effective primary imaging modality for detecting nerve enlargement, fascicular disruption, and intraneural collections. At the same time, MRI provides confirmatory evidence of abscess formation through T2 hyperintensity and diffusion restriction. Early multidisciplinary management, including multidrug therapy, corticosteroids, thalidomide, and timely surgical decompression with drainage, is crucial. Prompt diagnosis and intervention help relieve intraneural pressure, prevent further axonal damage, and preserve neurological function.

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