

PURE NEURITIC LEPROSY PRESENTING AS ISOLATED ULNAR NEUROPATHY: A CASE REPORT

Neurology

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KEYWORDS

INTRODUCTION

Leprosy, or Hansen disease, is a chronic infectious disease caused by *Mycobacterium leprae* that primarily affects the skin and peripheral nerves. Neurological involvement is an important cause of morbidity and disability, particularly because persistent nerve inflammation can result in sensory and motor deficits. [1] Pure neuritic leprosy (PNL) is a distinct clinical form characterized by peripheral nerve involvement in the absence of characteristic cutaneous lesions. In Indian studies, PNL accounts for approximately 4%–18% of leprosy cases and may present with paresthesia, neuropathic pain, sensory or motor deficits, and thickening or tenderness of affected peripheral nerves. [2]

The peripheral nerves of the upper limbs are commonly affected in PNL, with the ulnar nerve being among the most frequently involved. [2,3] PNL may present as focal or multifocal peripheral neuropathy, and diagnosis can be challenging in the absence of characteristic cutaneous manifestations. Slit-skin smears are typically negative in PNL, further complicating diagnosis. [3] Careful examination of peripheral nerves is therefore important when evaluating unexplained focal neuropathy.

We report a case of PNL presenting as isolated ulnar neuropathy with marked ulnar nerve enlargement and sensory symptoms in the absence of cutaneous manifestations, highlighting the importance of considering PNL in the evaluation of unexplained focal neuropathy.

Case Presentation

A 21-year-old man presented with a 2-month history of pain, paresthesia, and decreased sensation involving the right little finger and medial aspect of the ring finger. He reported intermittent episodes of tingling in the right little finger approximately 2 years earlier, particularly triggered by touch, which had become more persistent and painful over the preceding 2 months. There was no history of trauma.

Neurological examination revealed decreased sensation over the right little finger and medial aspect of the ring finger. The right ulnar nerve was markedly thickened and mildly tender on palpation at the elbow. There was no weakness or clawing, and the remainder of the neurological examination was unremarkable. No cutaneous lesions suggestive of leprosy were identified.

Ultrasonography of the right ulnar nerve demonstrated diffuse fusiform enlargement with thickened fascicles and altered echogenicity, extending across the cubital tunnel and involving the visualized portions of the arm and forearm. Slit-skin smear examination was negative.

Magnetic resonance imaging of the right elbow demonstrated extensive fusiform enlargement of the ulnar nerve extending from the lower third of the arm, across the cubital tunnel, into the upper third of the forearm. The maximum nerve thickness was approximately 8 mm, nearly twice that of the contralateral nerve. Increased intraneural signal, areas of altered fascicular architecture, and prominent perineural edema were noted (Figure 1).

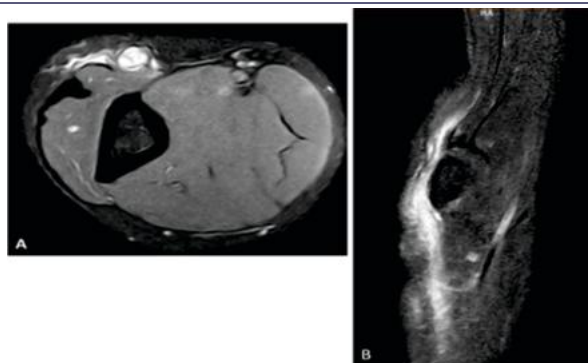


Figure 1. MRI Demonstrating Extensive Ulnar Nerve Involvement

Figure 1. MRI demonstrating extensive right ulnar nerve involvement. (A) Axial image demonstrating marked enlargement and abnormal signal of the ulnar nerve. (B) Longitudinal image demonstrating extensive fusiform enlargement and abnormal signal of the ulnar nerve with surrounding perineural edema.

post-contrast images demonstrated homogeneous enhancement of the nerve, without a definite loculated or intraneural abscess. The imaging findings favored an inflammatory neuritis/perineuritis process and were suggestive of Hansen disease (neuritic leprosy).

In view of the marked ulnar nerve thickening, corresponding sensory deficit, absence of cutaneous lesions, negative slit-skin smear, and supportive imaging findings, probable pure neuritic leprosy was diagnosed. The patient was subsequently initiated on multidrug therapy for leprosy along with corticosteroid therapy.

DISCUSSION

Pure neuritic leprosy presents a particular diagnostic challenge because its clinical manifestations are confined to peripheral nerves, and the diagnosis may be considered only after more common causes of focal neuropathy have been excluded. In this context, the pattern of nerve involvement can provide an important diagnostic clue. The ulnar nerve is among the most frequently affected nerves in PNL, and isolated mononeuropathy is a recognized presentation. In an Indian series of 65 patients, 40% presented with mononeuritis, while 60% had involvement of two or more nerves in the same extremity; the right ulnar nerve was the most frequently affected upper-limb nerve. [3]

An important feature of the present case was the disparity between the extent of structural nerve involvement and the degree of clinical disability. Despite marked enlargement of the ulnar nerve, the patient had sensory symptoms without clinically apparent weakness or clawing. This suggests that substantial neural pathology may precede the development of overt motor impairment. The history of intermittent neuropathic pain extending over approximately 2 years is

also relevant, as nerve pain in PNL may precede clinical diagnosis by months or years. [2] Recognition of such early or predominantly sensory manifestations may therefore be important for preventing progression to permanent neurological deficits.

The imaging findings complemented the clinical assessment by demonstrating the extent and character of the neural abnormality. Ultrasonography identified diffuse enlargement and fascicular abnormalities, while MRI further characterized the involved nerve and demonstrated associated inflammatory changes. In this case, the imaging abnormalities were considerably more extensive than might have been anticipated from the absence of motor deficit and provided additional support for significant neuritic involvement. Imaging therefore served primarily as an objective means of characterizing the extent of nerve involvement.

Histopathological examination of a peripheral nerve remains the diagnostic reference standard for PNL; however, nerve biopsy is invasive and carries a potential risk of neurological injury, particularly when a functioning major nerve is involved[2]. In the present case, biopsy of the functioning ulnar nerve was not pursued, and a diagnosis of probable PNL was made based on the clinical findings, characteristic nerve enlargement, negative slit-skin smear, and supportive ultrasonographic and MRI findings.

CONCLUSION

A markedly thickened peripheral nerve should not be overlooked in patients presenting with otherwise unexplained focal neuropathy. In an appropriate clinical setting, particularly in endemic regions, recognition of ulnar nerve enlargement and associated sensory dysfunction may provide an important opportunity to identify pure neuritic leprosy before progressive nerve damage results in permanent disability.

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