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Leprosy: A Neurologist's Perspective

ABSTRACT

Leprosy (Hansen's disease) is a chronic infectious disease caused by Mycobacterium leprae that primarily affects the skin and peripheral nerves. Despite availability of effective treatment, leprosy continues to be an important public health problem, particularly in Southeast Asia. India accounts for a significant proportion of the global disease burden. Transmission typically occurs through prolonged close contact with infected individuals, most commonly via respiratory droplets. The bacillus demonstrates a predilection for cooler areas of the body and preferentially infects Schwann cells, leading to progressive peripheral nerve damage through both direct bacterial invasion and immune-mediated mechanisms. Clinically, leprosy presents across a spectrum ranging from tuberculoid to lepromatous disease, depending on the host's cell-mediated immune response. Classical manifestations include hypopigmented hypoesthetic skin lesions, thickened peripheral nerves, sensory loss, and painless ulcers. Peripheral neuropathy is a hallmark of the disease and may manifest as mononeuritis multiplex, pure neuritic leprosy, or generalized polyneuropathy. Early involvement of small sensory fibers results in loss of temperature sensation, often preceding impairment of other sensory modalities. Diagnosis is primarily clinical and is supported by slit-skin smear examination for acid-fast bacilli, electrophysiological studies, imaging, and histopathological evaluation. Early recognition is essential to prevent irreversible nerve injury, deformities, and disability. Management involves multidrug therapy with dapsone, rifampicin, and clofazimine as recommended by the World Health Organization. Additional management includes treatment of lepra reactions, control of neuropathic pain, rehabilitation, and preventive strategies such as chemoprophylaxis and vaccination. A multidisciplinary approach and early diagnosis are crucial for reducing long-term complications and social stigma associated with the disease.

Key words: Leprosy; Mycobacterium leprae; multidrug therapy; peripheral neuropathy; pure neuritic leprosy

INTRODUCTION

Leprosy (Hansen's disease) is a chronic infectious disease that has affected humans for centuries and is believed to have originated in East Africa.[1] It is caused by infection with Mycobacterium leprae, a slow-growing intracellular bacillus. The disease primarily involves the skin and peripheral nerves but may also affect the nasal mucosa, other mucosal surfaces, and the eyes.[2] Despite advances in diagnosis and treatment, leprosy continues to carry a significant social stigma because of its contagious nature and the visible deformities associated with long-standing disease.1 Patients often seek medical attention for cutaneous manifestations such as hypopigmented patches, deformities, or burns resulting from sensory loss. Consequently, management is frequently initiated by dermatologists, and neurological evaluation may occur later during the course of the illness.[1,3] Neurological involvement in leprosy can be diverse and, sometimes the only manifestation of the illness.[1,3]

According to the World Health Organization (WHO), the global prevalence of leprosy in 2018 was approximately 0.2 per 10,000 population.[4] Leprosy remains one of the leading infectious causes of peripheral neuropathy, particularly in tropical and developing regions such as India, Brazil, and Southeast Asia.[1] The Southeast Asia Region continues to report the highest prevalence and new case detection rates worldwide. Notably, India accounted for more than half of the global leprosy burden in 2014.[5] More recently, Brazil, India, and Indonesia each reported over 10,000 new cases in 2019.[6]

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ETIOLOGY AND PATHOGENESIS

In 1873, the Norwegian physician Gerhard Armauer Hansen first identified Mycobacterium leprae as the causative organism of leprosy.[7] M. leprae is an obligate intracellular bacillus characterized by extremely slow growth and a preference for cooler areas of the body, particularly affecting dermal nerves and peripheral nerve trunks.[8] The incubation period of the disease is typically prolonged, averaging around five years, and the organism grows optimally at temperatures between 28°C and 32°C.[8] Transmission generally occurs through prolonged close contact with an infected individual, most commonly via respiratory secretions such as nasal

droplets.[2,8] The bacillus demonstrates a particular affinity for Schwann cells, mediated through interactions with laminin-2 and dystroglycan receptors. Nerve damage may result from direct bacterial invasion as well as immune-mediated inflammatory responses.[2]

Experimental studies have also suggested that M. leprae–induced nerve injury involves activation of the ErbB2 receptor tyrosine kinase signalling pathway, leading to demyelination. Repeated cycles of nerve damage, regeneration, and fibrosis eventually produce the characteristic enlargement of peripheral nerves seen in affected individuals.[2] Host genetic susceptibility appears to play an important role in disease development, with associations reported for the PARK2/PACRG locus on chromosome 6q25–q27. In addition, polymorphisms in the NRAMP1 gene on chromosome 2q35 may influence the immune response to the bacillus and contribute to the variability in clinical manifestations.[9]

CLINICAL PRESENTATION

The classical clinical features of leprosy include hypopigmented hypoesthetic skin lesions, mononeuritis multiplex, thickened peripheral nerves, and painless non-healing ulcers.[1,8] (Figure 1A). Clinically, the disease exists along a spectrum ranging from tuberculoid leprosy, which is characterized by well-defined localized lesions and strong cell-mediated immunity, to lepromatous leprosy, which presents with numerous poorly defined lesions and widespread systemic involvement. Cutaneous manifestations commonly appear as hypoesthetic or anesthetic patches involving cooler areas of the body such as the buttocks, back, trunk, face, and earlobes (Figure 1B-1D).[3] Epidemiological studies have shown that leprosy occurs more frequently in men than in women, which may reflect differences in environmental exposure, occupational risk, or host immune responses.

Ridley-Jopling classification: The various types were originally classified based on this scheme into tuberculoid disease (TT), lepromatous leprosy (LL) and the borderline leprosy subtypes. Patients with TT have a good cell-mediated immune response, few lesions and no detectable mycobacteria. Patients with LL are anergic to M leprae and, have multiple lesions with mycobacteria present. In the borderline leprosy types, patients have some cell-mediated immune response, multiple lesions, and unstable immunity.[10]

WHO classification: A simplified classification was proposed by WHO. The disease is classified based on the number of skin lesions- paucibacillary (up to five lesions) or multibacillary (more than five skin lesions).[12]

Clinical Features of Leprous Neuropathy

Peripheral nerve involvement is a hallmark of leprosy. In the early stages of the disease, temperature sensation is typically affected first, reflecting involvement of small sensory fibers. Larger sensory fibers, motor fibers, and deep tendon reflexes are generally preserved until the later stages of the disease. A distinct form known as pure neuritic leprosy may present primarily with neurological manifestations in the absence of obvious skin lesions. This presentation frequently results in delayed diagnosis and treatment, which may ultimately lead to progressive neuropathy and significant disability.[3,8,11] Less common neurological manifestations include cranial neuropathies, ganglionopathies, and generalized polyneuropathy.[12-18] The different patterns of neuropathy associated with leprosy are summarized in Table 1.[6,12,15,20-23]

Figure 1: Clinical photographs of patients demonstrating the following findings: (A) Unnoticed burns (black arrow) and deformity (arrowhead) of the fingers in a young female (Case 1) due to loss of temperature sensation. (B) Hypopigmented patch over the cheek (white arrow) in the same patient (Case 1). (C) Shiny, dry skin in a middle-aged male with lepromatous leprosy presenting with polyneuropathy (Case 2). (D) Skin changes over the palms secondary to autonomic neuropathy in Case 2. (E) Mononeuritis multiplex with an old healed burn over the palm of the right hand and wasting of the left hand in a young male with borderline tuberculoid leprosy (Case 3). (F) Dorsal aspect of the hands in Case 3 showing wasting of the left hand without any hypopigmented skin lesions.

Common neuropathic manifestations include

- Predominantly negative sensory symptoms, such as numbness, anhidrosis, painless wounds, and hypoesthetic or anesthetic skin patches. Loss of sweating (anhidrosis) is often one of the earliest signs of skin involvement.[19]
- Loss of temperature sensation, followed by impairment of touch and pain perception. Proprioceptive and kinesthetic sensations are typically preserved until later stages.[1]

- Thickening of peripheral nerves, which represents one of the most important clinical signs in the diagnosis of leprosy.
- Frequently involved nerves include the greater auricular, ulnar, median, superficial radial, dorsal cutaneous branches of the ulnar nerve, common peroneal (lateral popliteal), and superficial peroneal nerves.[20]

Table 1: Types of neuropathies in leprosy

Type of neuropathy	Clinical features
Mononeuropathy and mononeuritis multiplex	Most common presentation Upper limb nerves are affected more than the lower limb nerves[21] (Figure 1E,1F) Can be associated with rheumatological manifestations like positive Rheumatoid factor, arthralgia, and rash.
Polyneuropathy	Sensory polyneuropathy (distal, symmetric small fiber type).[12] Deep tendon reflexes are preserved. Usually, no motor weakness. Preferentially involves Temperature>Touch>Pain.[1] Proprioception is rarely involved in multibacillary leprosy.[22] Higher association with ulceration and deformities.[8]
Autonomic Neuropathy	Seen in patients with multibacillary leprosy.[21] Anhidrosis causing dry and scaly skin with ulcerations. Widespread dysautonomia involving the cardiac and respiratory systems is well documented.[23]
Cranial neuropathies	Cranial nerves may be involved in up to 18% of cases.[6,21] The facial nerve is the most common cranial nerve affected in leprosy[15] The hallmark of cranial neuropathy in leprosy is its patchy involvement of the nerve.[20]
Acute neuritis	Seen during lepra reactions more commonly with type I lepra reaction.[1] Spontaneous nerve paraesthesia and pain followed by objective sensory-motor loss.
Ganglionitis	Dorsal root ganglion involvement causing severe proprioceptive impairment, pseudo-athetosis with areflexia in ataxic limbs. The extent and the severity of the process seem to correlate with the bacterial load.[22]

DIAGNOSIS

History and physical examination

The diagnosis of leprosy is primarily clinical, although several investigations may assist in confirming the diagnosis. These include nerve conduction studies, skin or nerve biopsy, nerve ultrasonography, and MR neurography. The WHO expert committee on leprosy defines a case of leprosy as: an individual with one of the following cardinal signs of leprosy, but who has not received a full course of multi-drug therapy (MDT) appropriate for the type of leprosy[6]

1. Definite loss of sensation in a pale (hypopigmented) or reddish skin patch
2. A thickened or enlarged peripheral nerve with a loss of sensation and/or weakness in the muscles supplied by the nerve
3. The presence of acid-fast bacilli (AFB) in slit skin smears.

Diagnostic Modalities

Electrophysiological studies often demonstrate reduced amplitudes of sensory nerve action potentials (SNAPs) with relatively preserved conduction velocities and latencies. Motor nerve conduction studies may remain normal during the early stages of the disease.[24] Sensory nerve conduction parameters, particularly reduced amplitudes and abnormal warm perception thresholds, are highly sensitive for detecting early nerve damage.[1,25] In patients with suspected leprosy, investigations like skin biopsy and nerve biopsy provide definitive diagnosis (Figure 2A,2B). The investigations that may aid diagnosis are discussed in Table 2.[1,3,8,26-31]

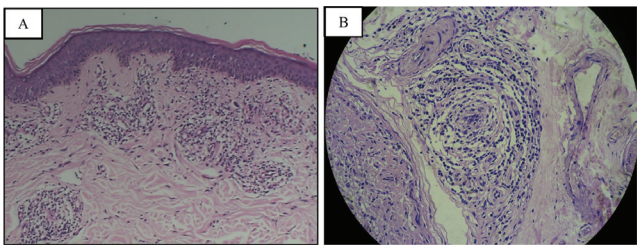

Figure 2: Histopathological findings: (A) Skin biopsy (Hematoxylin and eosin staining) showing granulomatous inflammation centered around the neurovascular bundles of the superficial and deep dermis, suggestive of borderline tuberculoid Hansen's disease (20×). (B) Nerve biopsy (Hematoxylin and eosin staining) of the medial plantar nerve showing inflammation and granuloma formation (20×). (Images Courtesy: Dr Uday Khopkar, Consultant Dermatologist, Mumbai)

Pitfalls in diagnosing leprosy neuropathy

Leprosy is the most common cause of a treatable neuropathy. Delayed and missed diagnosis of leprosy neuropathy leads to disability and deformities. Several atypical presentations may complicate diagnosis, including:

1. Pansensory involvement with kinaesthetic loss
2. Spontaneous sensory phenomena in the form of paresthesias.
3. Varied reflex patterns
4. Isolated cranial nerve involvement
5. Cooler and immune zones are present in concealed areas (axilla, groin, genitalia), hypoesthetic patches over these sites are often missed and under-reported.
6. Partially treated patients where the skin lesions have healed.
7. The common differential diagnoses are systemic connective tissue disorders, vasculitis and granulomatous disorders (as clinical and immunological features may be similar).

8. Electrophysiologically, both demyelinating and axonal changes may be seen, without a strict correlation with the type of leprosy.

Table 2: Diagnostic tests for leprosy associated neuropathies

Electrophysiology[1,25]	Predominantly causes a secondary axonal neuropathy resulting in a reduction in amplitude of sensory nerve action potentials (SNAPs) and Compound muscle action potentials (CMAPs) Demyelinating features can be also seen, mainly in the ulnar nerve across the elbow. The blink reflex provides a quantitative assessment of cranial nerve involvement. To diagnose subclinical neural involvement in cutaneous leprosy. Impaired sudomotor function indicated by absent sympathetic skin response (SSR) Also useful to monitor toxicity of therapy e.g Dapsone, Thalidomide
Peripheral nerve ultrasound[27-29]	High-frequency ultrasound (12–18 MHz) is useful in the evaluation of thickened nerves, also in non-palpable sites and skin lesions in nerves in greater detail for thickening, nerve edema, micro-abscesses, and alteration of fascicular architecture.
MRI/ MR Neurography	Clinical and subclinical proximal nerve involvement may be detected MR Neurography characteristically shows diffusely enlarged nerves with increased signal intensity on T2-weighted images, and nodular enhancement[30] Especially useful in pure neuritic neuropathy with single nerve involvement[31] Associated brain and spinal cord have also been described using MRI[16]
Skin Biopsy[1,3] (Figure 2A)	Slit skin smears show rod-shaped bacilli Stains: Fite Faraco, acid-fast positive Specificity 100%, sensitivity 50% The features noted are: epithelioid granulomas and foamy macrophages IHC and culture on mouse foot pad help to improve the diagnostic yield
Nerve Biopsy [1,8] (Figure 2B)	Important in pure neuritic leprosy. The histological hallmark of leprosy is perineural and intra-neural inflammation. **Lepromatous leprosy:** macrophages and Schwann cells show plenty of bacilli **Tuberculoid leprosy:** caseating granuloma with few bacilli
Future tests[3,8]	**Serology:** No diagnostic antibody. Anti PGL-1 antibody (phenolic glycolipid), for detecting relapse and subclinical infection. **Molecular techniques**: PCR, RT PCR coupled with mutation detection analyses rapidly detect drug susceptibility and resistance patterns.

Differential Diagnosis

In regions where leprosy is endemic, the diagnosis is usually straightforward when patients present with multiple mononeuropathy or polyneuropathy associated with hypo-esthetic hypopigmented skin lesions. However, diagnosis becomes more challenging in cases of pure neuritic leprosy, where cutaneous manifestations are absent. In such situations, the differential diagnosis should be guided by the clinical pattern of neuropathy and may include other inflammatory, infectious, and systemic causes of peripheral neuropathy. (Figure 3)[33]

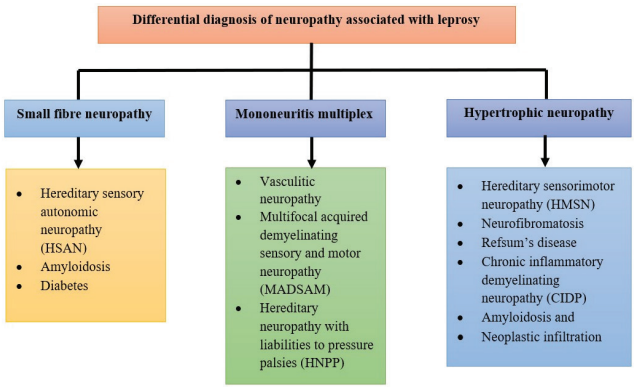

Figure 3: Flow-chart illustrating the differential diagnosis of leprosy. (Adapted from Neuromuscular disorders – a comprehensive approach with case illustrations: Khadilkar SV, et al)

NATURAL HISTORY AND PROGNOSIS

Leprosy is characterized by chronic granulomatous inflammation involving the skin and peripheral nerves. The clinical expression of the disease is largely determined by the host's cell-mediated immune response to M. leprae.[3,8] Peripheral nerve involvement may occur before, during, or even after completion of treatment. Progressive nerve damage can lead to long-term disability, deformities, and functional impairment.[3,34] The social stigma historically associated with leprosy is largely related to the chronic deformities resulting from untreated disease.[3] Delayed diagnosis increases the likelihood of irreversible nerve injury and disabling neuropathy.

In addition, immune-mediated lepra reactions (Type 1 and Type 2) occur in approximately 30% of patients with multibacillary disease, particularly during or after initiation of multidrug therapy.[35] With improved treatment strategies, global new-case detection rates have gradually declined. In India, the National Leprosy Elimination Programme (NLEP) has played an important role in controlling the disease through active surveillance and early treatment.[36]

MANAGEMENT APPROACH

Management of leprosy requires a multidisciplinary approach that includes pharmacological treatment, prevention of disability, and rehabilitation.[1,3,8] Patient education and counseling are also essential to reduce disease transmission and prevent complications. The various lepra reactions should be discussed with the patient before initiating pharmacotherapy.[1]

1. **Medical Treatment of Leprosy:** According to WHO recommendations, patients meeting diagnostic criteria

should be treated with multidrug therapy (MDT) consisting of dapsone, rifampicin, and clofazimine, regardless of bacillary load.[6,8,36] (Table 3)

- **Paucibacillary leprosy:** 6 months of treatment
- **Multibacillary leprosy:** 12 months of treatment

Although uncommon, drug-resistant leprosy has been reported. In such cases, second-line agents including ofloxacin, minocycline, clarithromycin, and clofazimine may be used.

2. **Management of Neuropathic Pain:** While sensory loss is the predominant symptom in leprosy, neuropathic pain may occur during lepra reactions. Corticosteroids are the mainstay of treatment for inflammatory nerve damage and may also alleviate neuropathic pain.[1,3]

   Symptomatic treatment may include:

   - **Gabapentin:** 100–300 mg two to three times daily
   - **Pregabalin:** 75–150 mg one to three times daily
   - **Duloxetine:** 20–60 mg daily

   Adjunctive medications such as amitriptyline, nortriptyline, and clonazepam may also be considered.[3]
3. **Lepra reactions:** Lepra reactions are inflammatory episodes characterized by cutaneous and systemic manifestations due to dynamic immunological changes. They usually occur after starting the treatment, but they may be seen before or during the treatment as well. Two types of Lepra reactions are described (Table 4).

Type I lepra reaction: It is a type of "delayed type of hypersensitivity reaction". The major risk factors include: age, positive slit skin smear, multibacillary disease, and Anti phenolic glycolipid I seropositivity. It presents as increased inflammation of pre-existing skin lesions in which the skin lesions look more oedematous, tender, and erythematous while neurotic lesions produce more pain and paraesthesia. These are best treated with prednisolone initially starting at doses of 40-60mg/day and gradual taper after the inflammation subsides. Steroids must be continued at least for 3-6 months.[36,37]

Type II lepra reaction: Also known as "erythema nodosum leprosum". It is a type of Type III Hypersensitivity reaction with antibody and immune complex deposition in tissues. A high bacterial index is one of the major risk factors for this reaction. The clinical features include eruption of new erythematous skin lesions and tender nodules on the face, extremities, and trunk. Fever, generalized lymphadenopathy, orchitis, iridocyclitis, and severe neuritis may present as a systemic manifestation of the drug reaction. Mild type II reactions can be managed by antipyretics alone. Severe cases require systemic steroids for 1-2 weeks. Thalidomide (100-300 mg at night) can be used in recurring and persisting cases. Clofazimine in high doses (300 mg) has shown some efficacy in refractory cases.[36,37]

Table 3: Treatment regimens for various types of leprosy (WHO regimen)

Type of leprosy	Drug used	Frequency of Administration	Dosage			Duration
			Adults	Children (10-14 yrs)	Children (<10 yrs)	
Multibacillary Leprosy	Rifampicin	Once monthly	600 mg	450 mg	300 mg	12 months
	Clofazimine	Once monthly	300 mg	150 mg	100mg	
	Dapsone	Daily Once	100 mg	50 mg	25 mg	
	Clofazimine	Daily for adults (every other day for children)	50 mg	50 mg (alternate day, not daily)	50 mg (weekly twice)	
Paucibacillary Leprosy	Rifampicin	Once monthly	600 mg	450 mg	300 mg	6 months
	Clofazimine	Once monthly	300 mg	150 mg	100mg	
	Dapsone	Daily Once	100 mg	50 mg	25 mg	
	Clofazimine	Daily for adults (every other day for children)	50 mg	50 mg (alternate day, not daily)	50 mg (weekly twice)	

Table 4: Characteristics of lepra reactions[1,3]

Associated features	Reversal reaction	Erythema nodosum leprosum
Leprosy subtype	BL, BB, BT	LL, BL
Type of reaction	Type 1 (Delayed type hypersensitivity)	Type 2 (Antibody and immune complex deposition in tissues)
Clinical features	Increased Erythema, induration of pre-existing lesions, gradual in onset	New erythematous lesions, tender nodules on the face, extremities, and trunk, sudden in onset
Systemic involvement	Absent	Present (includes arthritis, iritis, lymphadenopathy, orchitis, severe nerve involvement)
Prognosis	Good	Worse

4. **Role of Steroids:** Steroids are being used more and more, as anti-inflammatory agents in nerve involvement but specific dose and duration are not yet defined.[1,38] Oral prednisolone (given for 12 weeks) may be effective in neuritis with less than 6 months duration.[38-40]
5. **Prophylaxis:** The spread of disease in close contact should be prevented while taking care of patients. Prevention often avoids clinical and social consequences, especially social isolation. Single-dose rifampicin (SDR) was found to be effective in reducing the risk of leprosy over 5–6 years in leprosy contacts in a recent randomized control trial.[41,42] The dose of rifampicin suggested is as follows: Older contacts >15 years of age (600 mg single dose) and contacts with 10–14 years (450 mg single dose). The doses in children vary with age and weight: children (between 6 and 9 years and ≥20 kg)- (300 mg single dose), and children (<20 kg)- (10–15 mg/kg of body weight, single dose).[1] The Bacillus Calmette Guerin (BCG) vaccine given at birth is effective in preventing leprosy as well as tuberculosis. In highly endemic areas, compulsory BCG vaccine use is recommended. Besides, the vaccine also potentiates the protective effect of single-dose rifampicin in contacts.[3,43]
6. **Surgical management:** Neurolysis and decompression and acute nerve abscess drainage help to prevent further nerve damage.[8,44-46]
7. **Management of disabilities:** Supportive care focuses on preventing complications related to sensory loss and motor weakness.[1,6,8] Measures include:
    - Eye protection to prevent corneal xerosis and ulcers
    - Proper wound care for neuropathic ulcers
    - Use of orthotic devices or splints for deformities such as wrist drop or foot drop
    - Protective footwear to prevent trophic plantar ulcers
    - Regular skin care with moisturizers to prevent dryness and cracking
8. **Leprosy Vaccine:** As described earlier, BCG seems to have a potential prophylactic role for leprosy. Killed mycobacterial vaccines comprising Mycobacterium indicus pranii (MIP) also seem effective for immunoprophylaxis.[47-49] The vaccine LepVax was tested in Armadillos, an experimental animal model for lepromatous neuropathy. The results of the study show promise for both safety and effectiveness as post-exposure immunoprophylaxis in the neuropathy model.[50] There was a reduction in sensory nerve damage and delayed nerve damage in animals infected with high doses of M. leprae post LepVax administration. A phase 1, open-label clinical trial is soon to commence on the use of LepVax in healthy adult subjects.[51]

CONCLUSION

Leprosy remains an important and potentially treatable cause of peripheral neuropathy, particularly in endemic regions. Early recognition of its neurological manifestations is essential, as delayed diagnosis can lead to irreversible nerve damage, deformities, and long-term disability. Multidrug therapy recommended by the WHO has significantly improved treatment outcomes and reduced disease burden worldwide. In addition to pharmacological management, prompt treatment of lepra reactions, prevention of disability, rehabilitation, and patient education play vital roles in comprehensive care. Continued awareness among clinicians and strengthening of national control programs are essential to minimize complications, reduce transmission, and address the social stigma associated with leprosy.

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