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Review Article

Leprosy: A Concise Review of Diagnosis, Treatment, and Elimination Strategies

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Leprosy, or Hansen's disease, stems from Mycobacterium leprae and M. lepromatosis, posing ongoing risks in low- and middle-income regions despite progress. This compact review details epidemiology, disease mechanisms, clinical signs, diagnostics, multidrug therapy (MDT), reactions, rehabilitation, and control efforts. It emphasizes WHO strategies, resistance challenges, and innovations like vaccines to end transmission..

Keywords: Leprosy, M. leprae, MDT, diagnostics, reactions, stigma, elimination.

INTRODUCTION

Leprosy has afflicted humans for millennia, targeting skin, peripheral nerves, eyes, and upper respiratory tract. Untreated cases lead to deformities, but WHO-led MDT has cut global prevalence. Still, ~200,000 new cases emerge yearly, mainly in India, Brazil, and Indonesia, urging sustained action.

Epidemiology and Risk Factors

Endemic in Asia, Africa, and Latin America, leprosy spreads via respiratory droplets from untreated patients; armadillos act as reservoirs in the Americas. Key risks include close contact with cases, genetic factors, malnutrition, and poor hygiene. In 2023, India reported the highest burden.

Pathogenesis and Classification

The bacterium invades Schwann cells, with outcomes tied to immunity: strong response yields tuberculoid leprosy (TT, few lesions); weak immunity causes lepromatous (LL, widespread). Ridley-Jopling classifies into TT, BT, BB, BL, LL based on histology and immunity. WHO uses paucibacillary (PB, ≤5 lesions) vs. multibacillary (MB).

Clinical Features

Symptoms span a spectrum:

Skin: Hypopigmented/erythematous patches with sensory loss, nodules, plaques.

Nerves: Thickening, anesthesia, weakness, paralysis.

Eyes: Lagophthalmos, corneal opacity, blindness.

Other: Nasal issues, testicular atrophy.

Early detection prevents disability.

Diagnosis

Clinical suspicion (numb patches, palpable nerves, contacts) prompts:

Slit-skin smears for acid-fast bacilli.

Biopsy showing granulomas/bacilli.

PCR for early/low-burden cases.

Serology (anti-PGL-1, limited utility).

Challenges: mimicry by other diseases, access gaps, stigma delays.

Treatment: WHO MDT

Standard regimens prevent resistance:

PB (6 months): Rifampicin 600 mg monthly (supervised); dapsone 100 mg daily.

MB (12 months): Rifampicin 600 mg monthly; dapsone 100 mg daily; clofazimine 300 mg monthly + 50 mg daily.

Resistant cases use alternatives; ROM (rifampicin-ofloxacin-minocycline) suits single lesions. U-MDT trials simplify care.

Leprosy Reactions

Type 1 (Reversal): Borderline cases; nerve swelling treated with prednisolone.

Type 2 (ENL): LL/BL; nodules, fever managed by steroids, clofazimine, thalidomide.

Prompt control averts damage.

Public Health Control

WHO aims for zero transmission via:

Early case detection/treatment.

Contact chemoprophylaxis.

Stigma reduction, integration into primary care.

Barriers: underdiagnosis, diagnostics access, resistance.

Future Directions

Promising: LepVax vaccine, rapid PCR tests, resistance surveillance, awareness drives. Coordinated efforts can eliminate leprosy.

CONCLUSION

Leprosy control hinges on MDT, diagnostics, and anti-stigma measures. Global commitment will halt transmission and support affected individuals. Despite remarkable strides through WHO-led MDT programs reducing prevalence by over 99% since 1985, leprosy endures as a preventable yet stigmatized threat, with ~200,000 annual cases signaling persistent transmission. Success demands integrated action: accelerating early detection via point-of-care PCR and training frontline health workers; fortifying supply chains for free MDT to counter emerging rifampicin/dapsone resistance; scaling rehabilitation to restore dignity and livelihoods for millions facing disabilities; and dismantling social barriers through community education and policy reforms.

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