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Perspective

Pathogenesis and Diagnosis of Leprosy

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INTRODUCTION

Leprosy is a chronic granulomatous ailment triggered by the bacillus *Mycobacterium leprae*. It mainly affects the skin and peripheral nerves and is still endemic in several regions of the world. Clinical presentation depends on the patient's immune status at the time of infection and during the course of the ailment. Leprosy is accompanying with disability and marginalization.

The portal of entry for *M. leprae* is extensively believed to be the nose, although skin-skin transmission has not been excluded. The initial lesions in the nasal mucosa cause mild, non-specific signs and are not biopsied, so the histopathologically features of this lesion are not known. Recognized nasal lesions are sometimes biopsied. Nasal lesions are normally lepromatous, with abundant bacilli. Tuberculosis granulomas may arise but probably cause such minor signs that they are usually not biopsied. Hematogenous dissemination is the likely mechanism of the spread of bacilli. In lepromatous patients, *M. leprae* may be found in buffy-coat preparations during hematogenous spread of the infection, although it is not typically accompanied by fever or other systemic symptoms. Although *M. leprae* prefers cooler temperatures, it can infect and survive for at least some time in deep tissues. *M. leprae* are, therefore, occasionally encountered in biopsies of the lymph node, liver, or bone marrow.

DESCRIPTION

PGL-I (Phenolic Glycolipid I) emerged in the early 1980's on the one hand as part of intensive efforts to describe the typing antigens of a host of *Mycobacterium* spp. and also from characterization of the lipids of skin biopsies from highly bacillary positive lepromatous leprosy patients. PGL-I, despite its extreme lipophilicity due to its intrinsic phthiocerol dimycocerosyl element, is highly antigenic inducing high titer IgM antibodies in lepromatous leprosy patients, attributable largely to the unique 3,6-di-O-methyl-β-D-glucosyl entity at the non-reducing terminus of its disaccharide. PGL-I itself or in the form of semisynthetic neoglycoproteins comprising the synthetic terminal disaccharide or the whole disaccharide chemically conjugated to such as bovine or human serum albumin, has found its greatest utility in the serological diagnosis, confirmation and management of lepromatous leprosy. PGL-I has also been involved in the tropism of *M. leprae* for Schwann cells, through particular binding to laminin, and to play a vital role in down regulation of the inflammatory immune response and inhibition of dendritic cell maturation and activation, thereby facilitating the persistence of *M. leprae*/leprosy.

Diagnosis is clinical and is made when the individual has at least 1 of the following cardinal signs stated by the World Health Organization: hypo pigmented or erythematous macules with sensory loss; thickened peripheral nerves; or positive acid-fast skin smear or skin biopsy with loss of adnexa at affected sites. Leprosy is treated with a multidrug combination of rifampicin, clofazimine, and dapsone. The World Health Organization advocates 2 leprosy treatment procedures on the basis of disease classification (as multibacillary or paucibacillary) by skin lesion count. This technique, which, in the Philippines, results in a high prevalence (78%) of patients with multibacillary leprosy, was directly equated with classification using standard. Histopathologically and microbiological criteria in 264 currently untreated patients with leprosy. Of those whose leprosy was classified as paucibacillary, 38%-51% of individuals had multibacillary leprosy according to classic criteria and were thus at risk of under treatment according to World Health Organization recommendations.

Over recent years, many significant advances have been made in evolving molecular diagnostics, in recognizing highly effective drugs and designing multidrug regimens for treatment, and in unraveling the genomic structure and functions of the leprosy bacillus. Using the new information about particular sequences of *M. leprae*, several gene probes and gene magnification systems for confirming diagnosis and monitoring treatment have been developed. Among these, polymerase chain reaction (PCR) based approaches have been useful in confirming the diagnosis in paucibacillary leprosy (where few bacilli are present). RNA-targeting systems for monitoring the improvement of treatment, in situ hybridization methods for analyzing specimens with nonspecific histological features, and molecular methods for direct detection of rifampicin/daps one resistance are other major technological advances with immense applied value.

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CONCLUSION

Several effective procedures for the treatment of leprosy have been advanced, which include rifampicin, clofazimine and daps one as core drugs. Although these regimens are generally satisfactory, limitations in terms of persevering activity and late reactions/relapses in paucibacillary leprosy, and persistence of dead and/or live organisms in multibacillary forms of the ailment, have been observed.