

## 9. ABSTRACT

Leprosy is a chronic infectious disease caused by the *Mycobacterium leprae,* an obligated intracellular parasite, yet to be cultivated in artificial media. The disease may present with a wide clinical range that will correspond to the immunological response of the host to the M. *leprae.* In one pole is the resistant form, the tuberculoid leprosy (HT), in which the strong specific cellular immune response controls bacillary multiplication. The other pole is represented by lepromatous leprosy (HV), low resistant form in which the cellular immunity fails to eliminate bacilli from the infected host and infection disseminates. The borderline (HD) presents variable intermediate manifestations between HT and HV, depending on the grade of immunity against *M. leprae.* 

Considering that anti-PGL-I antibodies, neopterin and C reactive protein (CRP) were evaluated in a few studies, at diagnosis and during multidrugtherapy (MDT) treatment, the aims of the present study were:

A. Evaluate the immune and inflammatory responses in leprosy patients at diagnosis of the disease and 2, 4, 6 and 12 months of treatment with MDT, and at reactional episodes. This was accomplished by anti-PGL-I antibodies, neopterin and CRP serum levels determination.

B. Evaluate the bacilloscopic index (IB) of leprosy patients at diagnosis and correlate it with serum levels of anti-PGL-I antibodies, neopterin and CRP. Twenty-five non-treated patients attended at the Instituto Lauro de Souza Lima took part of this study. They were classified according to the criteria established at the 6° International Leprosy Congress, Madri. There were 09 HV, 06 HD and 10 HT. According to the World Health Organization orientation for treatment scheme, the 15 patients were classified as multibacillary (MB) and 10 as paucibacillary (PB). Among the 25 evaluated patients, 13 presented reactional episodes, 08 were type 1 and 05 type 2. Cutaneous lymph was collected at diagnosis for IB determination. Blood samples were drawn at five moments: at diagnosis, 2, 4, 6 and 12 months of treatment. Sera were used for anti-PGL-I antibodies, neopterin and CRP

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serum level determination. The ELISA kit developed by the Royal Tropical Institute of Amsterdam was used for anti-PGL1 antibody quantification. Neopterin levels were determined using a competitive ELISA test developed by IBL, Hamburg (Germany). The CRP levels were determined using a latex passive agglutination test. Results showed low levels of anti-PGL-I antibodies in HT patient, average levels in HD and increased levels in HV. Throughout treatment there was a significant decrease in the anti-PGL-I antibodies levels in MB patients. A positive correlation was obtained between anti-PGL-I levels and IB at the moment of diagnosis. Neopterin serum levels were increased in MB patients, especially on HV. There was a positive correlation between neopterin and IB. The results, however, showed little monitoring value for patients on MDT scheme. No significant results were obtained during treatment for CRP levels, but when comparing the groups of patients, HV showed increased CRP levels. No statistical difference was found for anti- PGL-I levels between patients who presented type 1 and type 2 reactions during the study. Neopterin levels were elevated in patients with type 2 reaction. The CRP levels were equally elevated in the type 2 reactional patients. We could conclude that: detection of anti-PGL-I antibodies may be used for follow-up of HV and HD patients on MDT; neopterin level is a good indicator for reactional episodes, mainly in patients with type 2 reaction; CRP was not efficient for follow-up of leprosy patients on treatment.