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Role of immunological markers in latent TB infection

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Introduction: Tuberculosis (TB), primarily caused by *Mycobacterium tuberculosis* continues to be one of the most important infectious cause of mortality world-wide. A key challenge to greater progress in TB control is the pool of latent TB infection (LTBI) representing a huge long-lived reservoir of potential TB disease. Preventing active TB and tackling latent infection in addition to the Directly Observed Treatment, Short-Course (DOTS) strategy could improve TB control in high-burden settings, especially where there is a high prevalence of HIV co-infection. Based on the data analysis at our centre in the PPM (Public Private Mix) DOTS clinic, Mahavir Hospital & Research Center, Hyderabad, for the last 19 years we observed that, 15-20% of the patients who have been treated and cured, transmitting the disease to other household members. The risk of progression of infection to active disease was found to be highest during the first two years following infection. Hence there is a need to monitor the immunological and molecular responses in household contacts by following them for a period of two years that may help in identifying who are at risk.

Objectives: To determine the changes in cytokine production at different time intervals during the development of active disease from LTBI.

Methods: Our group is working on pulmonary tuberculosis patients, tuberculosis patients with diabetes mellitus and their household contacts with antigen 85A of *M. tb* Pro (IFN- γ, TNF- α, IL-1, IL-2, IL-12) and anti inflammatory cytokines (IL-4, IL-6, IL-10, TGF- β) were measured in culture supernatants and expression analysis was performed in the cultured cells. All the subjects were followed at different time points (0, 4, 6, 12, 18, 24 months). Based on the data analysis it has been observed that IFN-γ, IL-12 and TNF-α expression and production was low at the start of treatment and improved with treatment and the reverse was observed with IL-6, TGF-β and IL-10 cytokines. We are also working on the innate immunity by stimulation with ESAT-6 antigen and ligands PAM, LAM, LPS in TB patients and their contacts.

Results: In patients, the immune response was low with ESAT-6 and increased during the follow up, with LAM, PAM, LPS & MDP, a higher response was observed which decreased with treatment. Contrasting results were observed in the household contacts. The cytokines levels IL-1β were decreased during the follow up with ESAT-6 and with LAM and PAM increased with treatment. With LPS, patients had high levels and HHC had low levels at the time of diagnosis and did not vary with treatment. The levels of IL-12 were found to be increased with LAM, PAM, MDP and ESAT-6 during the follow up in patients as well as in house hold contacts.

Conclusion: SNPs were also carried out at different positions for the similar cytokines associated with TB. The genotypes of IL-2, IL-6, IL-18 cytokine genes were found to be susceptible towards the disease. Further studies with recombinant cytokines genes may confirm our results which may help to differentiate active TB from LTBI.

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