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USE OF ANTI-RIBOSOMAL P ANTIBODY IN DIAGNOSIS OF CEREBRAL LUPUS: SUPERIORITY OF WESTERN BLOTTING OVER ELISA. KW Lee*, EYT Chan, OKH Ko, JWM Lawton, RWS Wong*, CS Lau*. *Department of Medicine and Department of Pathology, University of Hong Kong, Queen Mary Hospital.

Cerebral involvement in systemic lupus erythematosus (SLE) is associated with high rates of morbidity and mortality. However, the diagnosis of lupus cerebral events is difficult as most currently available blood markers are non-specific. Anti-ribosomal P antibody (anti-p) has been suggested to be useful. In this study, we have evaluated the usefulness of 2 anti-P assay systems in SLE.

Anti-P antibody was assayed by Western blotting and by ELISA in 32 SLE, 38 rheumatoid arthritis patients, 31 patients with other connective tissue disease and 40 normal individuals. By Western blotting, anti-P was specific for SLE (90% specificity) and was found in 58% of SLE patients. Most (13/18) patients had all 3 bands (P0, P1, P2) while the rest had only P1 and P2 bands. Detection of anti-P by Western blotting in SLE patients was significantly associated with major cerebral involvement ($p<0.01$), and was positive in 90% of those with cerebral complications and 41% of those without. For other connective tissue diseases, anti-P was only found in lupus-related diseases. Elevation of anti-P level measured by ELISA was also specific for SLE (99% specificity) but was present in only 44% of patients and was not associated with cerebral involvement. We conclude that anti-P is a specific but not sensitive marker of SLE. Measurement by Western blotting may also have a role in the diagnosis of cerebral complications in SLE patients.

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Dysregulation of the relative production of interleukin (IL) 2 and IL10 from peripheral blood mononuclear cells contributes to the in-vitro defective T cell response to tuberculin PPD in rheumatoid arthritis

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Introduction : Rheumatoid arthritis (RA) is a T cell-mediated chronic inflammatory disorder mainly involving the joint. Histologically, the rheumatoid synovium resembles that of a delayed hypersensitivity reaction with the infiltration of CD4+ve T cells, activated monocytes and B cells. Despite most of the CD4+ve T cells are activated / memory cells (CD45RO+ve), they proliferate very poorly to common recall antigens such as tuberculin PPD. The mechanism for this defect is unclear. We explored the possibility of an imbalance of TH1 and TH2 cytokine production from mononuclear cells as the cause of such a defect.

Methods : Fresh peripheral blood mononuclear cells (PBMC) isolated from RA patients and normal matched controls were cultured for 5 days in the presence and absence of PPD (15ug/ml). IL2 (5 I.U./ml) and a neutralising anti-IL10 monoclonal antibody (1ug/ml) were added at the beginning, either alone or in combination. Proliferation of T lymphocytes were measured by tritiated thymidine incorporation.

Results : IL2 supplementation did not specifically enhance T cell response to PPD in either normal controls or patients. Anti-IL10 increased the stimulation index (SI) of RA lymphocytes but this increment was not statistically significant (SI before and after anti-IL10 were 4.3±0.4 and 5.6±0.6 respectively). There was no effect of anti-IL10 on SI of normal lymphocytes. However, when IL2 and anti-IL10 were added together, a significant enhancement of SI (35.6%) was observed (SI before and after IL2 and anti-IL10 addition were 6.7±1.2 and 10.4±1.1 respectively, $p=0.02$). No effect of this combination could be demonstrated on normal lymphocytes.

Conclusion : The synergistic effect of IL2 and anti-IL10 on proliferation of lymphocytes to PPD supports the hypothesis that the defective T cell response to recall antigen is at least in part due to a dysregulation of the relative production of IL2 and IL10 from PBMC in RA