

RI-05

**Cervical dysplasia in patients with systemic lupus erythematosus.**

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Background: SLE patients are prone to infection as a result of intrinsic immune defects as well as the use of immunosuppressive agents. The incidence and clinical course of malignancy, for instance, cervical intraepithelial neoplasia (CIN) in SLE patients are not certain.

Methodology: Consecutive female patients who fulfilled the ACR criteria for SLE were recruited from the rheumatology clinic. Demographic and clinical data including previous cyclophosphamide (CTX) use and smoking and sexual history were recorded. Cumulative dose of CTX was calculated. Gynaecological examination and Pap smear were performed on these patients.

Results: 331 SLE patients were studied. The mean  $\pm$  SD age at pap smear examination was 40.1 $\pm$ 8.9 (17-71) years. CIN lesion was found in 38/331 (11.5%) patients of various severity: Grade I 22/38 (57.9%), Grade II 6/38 (15.8%) and Grade III 9/38 (23.7%). Previous CTX use was found to be a strong risk factor for the development of CIN. CIN lesions was found in 20/38, (52.6%) and 95/293, (32.4%) patients who did or did not have previous use of CTX ( $p=0.02$ ). Patients with CIN lesions were also found to have consumed a higher cumulative dose of CTX (17521.6 $\pm$ 29436.7 mg) than those without CIN lesions (6279.1 $\pm$ 12498.4 mg). Smoking history and the number of sexual partners were not shown to relate to the development of CIN lesions in our cohort.

Conclusion: SLE patients are prone to develop CIN lesions. Previous CTX use is a strong risk factor and the risk is increased in those who had received higher cumulative dose of this drug. SLE patients, in particular the high risk subsets, should receive regular surveillance for CIN.

RI-06

**Influence of hyperlipidaemia on macrophage phagocytosis of apoptotic cells**

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**Introduction:** Failure of macrophages to remove apoptotic cells has been suggested to contribute to the development of systemic lupus erythematosus (SLE) and other autoimmune diseases. Though the mechanism of this defect is unclear, many serum factors are supposed to be involved. Since hyperlipidaemia is a common finding in SLE, we designed a pilot study to investigate its influence on macrophage phagocytosis function.

**Method:** High fat diet was given to healthy volunteers who had an overnight fast to induce transient hyperlipidaemia. Macrophage phagocytosis of apoptotic neutrophils was measured at 0, 4, 8, 12 and 24 hours post-prandial. The relationship between macrophage phagocytosis and hyperlipidaemia was analyzed.

**Results:** (1) After high fat diet, serum levels of triglyceride (TG) were significantly elevated at 8<sup>th</sup> hour (2.56 $\pm$ 2.13 mmol/l, n=7,  $p<0.05$ ) when compared to baseline (0.91 $\pm$ 0.35 mmol/l, n=7), (2) The phagocytosis index (PI) of macrophages cultured with post-prandial sera declined with time, with the significant time point at 8<sup>th</sup> hour (33.13 $\pm$ 18.50%, n=7;  $P=0.029$ ) when compared with baseline (42.91 $\pm$ 16.06%, n=7). (3) The PI of macrophages correlated negatively with the serum levels of TG ( $r=-0.38$ , n=42,  $p=0.013$ ).

**Conclusion:** Hyperlipidaemia, particularly hyper-TG, may influence the immune system through its negative influence on macrophages' ability to remove apoptotic cells.