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AB0205 IL-22 SERUM LEVELS AS A BIOMARKER FOR EROSIVE **DISEASE IN RHEUMATOID ARTHRITIS**

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Background: Consistent with models of experimental arthritis implicating IL-22 in the development of joint destruction we could previously demonstrate that elevated IL-22 serum levels were associated with the development of erosive disease in patients with early RA.

Objectives: To study value of IL-22 as a marker for erosive disease in established RA, to assess whether IL-22 is increased in other joint destructive rheumatic diseases, and to evaluate the influence of comorbidities on IL-22 serum levels.

Methods: We measured serum IL-22 levels by Enzyme-Linked Immunosorbant Assay (ELISA) and analyzed their correlation to erosive disease (assessed by radiographs of hand and feet) and clinical parameters in patients with established RA (n=142), psoriatic arthritis (n=15), gout (n=15), age-matched patients with hypertension (n=10), diabetes (n=10), coronary heart disease (n=10), and healthy individuals as controls.

Results: 81 of 142 patients with established RA demonstrated elevated IL-22 levels compared with the range of healthy controls. A significant greater percentage of these "IL-22 high" patients (59%) demonstrated erosive disease compared the "IL-22 normal" patients (37%, p<0.05). In the "IL-22 high" compared to "IL-22 normal" group the fractions of patients positive for RF (70% vs 83%) and ACPA (74% vs 64%) were slightly higher, however not statistically significant different. Similar, measures of disease activity including DAS28 (2.3 vs 2.8) and CRP (0.5 vs 0.3 mg/dl) only tended to be higher in the "IL-22 high" than the "IL-22 low" group. Irrespective of erosive joint disease, patients with psoriatic arthritis and gout demonstrated lower IL-22 levels compared to established RA and only slightly higher than healthy individuals. Patients with hypertension, diabetes and coronary heart disease had IL-22 serum levels comparable to healthy controls.

Conclusions: High IL-22 levels are associated with erosive disease also in established RA, potentially in parts independent of serology, and might serve as a marker for joint destruction also in this cohort.

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