

S-RI-1

ACR/SLICC Damage Scores in an Inception Cohort of Patients with Systemic Lupus Erythematosus (SLE) and their Relationship with Disease Flares and Various Clinical Variables

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Objectives: To study the damage scores in an inception cohort of SLE patients and their relationship with disease flares and various clinical variables. **Method:** 157 patients with SLE were recruited for an inception cohort study in 1997. Baseline clinical characteristics, profile of autoantibodies and the ACR/SLICC damage scores were obtained. These patients were followed prospectively and the damage scores were reviewed yearly. The number of severe and mild/moderate disease flares and new organ damage each year were also analyzed. Flares of SLE were defined by criteria modified from the SLEDAI-SELENA instrument. The damage scores at year 3 were compared with those at year 0 and the absolute increase in SLICC scores was correlated with various clinical variables at entry and the number of disease flares within this 3-year period. **Results:** 157 SLE patients (92% women) were followed. The mean age at entry was 35.9 ± 9.9 years and the mean duration of SLE was 84.7 ± 83 months. The major organ manifestations at entry were musculoskeletal (92%) mucocutaneous (83%), hematological (62%) and renal (43%) disease. At year 0, 62 (39.5%) patients had damage and this number increased to 79 (50.3%) at year 3. The contribution to damage by various systems at baseline, in decreasing order of frequency, was musculoskeletal (19%), neuropsychiatric (18%), renal (17%), ocular (11%), cardiovascular (16%) and gonadal (8%). The median SLICC score at year 0 and year 3 was 0 (IQR 0-1) and 1 (IQR 0-2), respectively. Compared with year 0, the mean damage scores of our patients at year 3 had increased significantly (0.85 ± 1.4 vs 1.27 ± 1.7 , $p < 0.001$, Wilcoxon's signed rank test) and the rate of increase was 0.14/patient-year. The increase in SLICC scores was mainly contributed by musculoskeletal, renal and gonadal damage, and was significantly higher in patients with damage at baseline than those without (0.17 vs 0.13 /patient-yr, M-W U, $p = 0.007$). The increment in SLICC scores correlated with the baseline scores (Spearman's rank; $p = 0.21$, $p = 0.01$) and the number of severe flares ($p = 0.31$, $p < 0.001$) but did not correlate with other clinical variables at entry. **Conclusions:** The damage scores of our cohort of SLE patients had increased significantly over a 3-year period of follow-up. Most new damage was contributed by renal disease, gonadal failure and musculoskeletal complications. Pre-existing damage and severe flares were associated with further damage.

S-RI-2

Exhaled and Serum Nitric Oxide Measurement in Patients with Systemic Sclerosis Complicated by Interstitial Lung Involvement

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Background: Interstitial lung disease (ILD) is a known complication of systemic sclerosis (SSc). Early fibrosing alveolitis (FA) is often asymptomatic and requires high resolution CT (HRCT) thorax, the gold standard for diagnosis. Nitric oxide (NO), which possesses dual pro- and anti-inflammatory actions, has been proposed to play a role in the pathogenesis of ILD in SSc. Measurement of exhaled and serum NO may help in diagnosis of early FA.

Objective: To measure exhaled and serum NO from patients with SSc and to correlate with different stages of development and severity of ILD.

Methodology: Exhaled and serum NO was measured in 40 patients with SSc and 40 healthy controls chemiluminescence and serum NO assay respectively. Lung function test, Doppler Echocardiogram and HRCT thorax were also performed and features of FA and honeycombing (HC) were graded according to severity.

Results: 40 SSc patients, with a mean disease duration of 9.5 ± 7.7 years, were recruited. Features of predominant FA and HC were detected in 30/40 (75%) and 10/40 (25%) patients respectively. Exhaled NO levels were not statistically different from patients with SSc and controls (23.2 vs 20.9 ppb, $p = 0.8$). Exhaled NO level, however, was found to correlate with the severity score of FA ($r^2 = 0.43$, $p = 0.006$) in SSc patients. Serum NO level in SSc patients was shown to be significantly higher than that in controls (74.1 vs 36.7 uM, $p < 0.001$). This, however, was not found to correlate with modified Rodnan skin score ($r = -0.05$, $p = 0.78$).

Conclusion: Nitric oxide may play an important role in the underlying pathogenesis of SSc.