media thickness (cIMT) in patients with SLE in comparison with population controls is not clear.

Objectives: To examine in SLE and population controls (1) prevalence of risk factors overtime; (2) evolution of cIMT; (3) risk factors promoting cIMT evolution.

Methods: The study sample originated from the SLEVIC cohort (SLE vascular impact cohort study) which included consecutive patients with SLE and age and sex matched population controls. Seven years after inclusion all participants were asked to take part in the follow-up investigation. The standardized data collection and carotid ultrasound were performed at two assessments, 7 years apart. Effect of risk factors on cIMT overtime was examined with linear mixed models adjusted for age, sex and traditional CV risk factors.

Results: A total of 77 patients with SLE (68% of original cohort), 87% females, at inclusion had mean age 47 years, disease duration 11.4 years, SLEDAI 3.0, SLICC/ACR 1.1, and 74 controls (61% of original cohort), 89% females, mean age 51 years, completed 7-years follow-up. At inclusion, patients with SLE were younger and had lower levels of LDL than controls but were more likely to have hypertension and higher levels of triglycerides (TG). Between the assessments both groups were measured with an increase in blood pressure, levels of total cholesterol (TC), LDL and at follow-up the patients still had lower TC, LDL and HDL levels and higher TG than controls. At both assessments, patients used more frequent antihypertensive and aspirin than controls, p<0.001. The mean cIMT increased statistically significant in patients and controls, average absolute progression of 0.009 mm/year in patients and 0.011 mm/year in controls, p<0.001 for change in both groups, with no inter-group difference, p=0.867 age- and sex-adjusted.

In multivariate analysis, the patients showed a statistically significant association between mean cIMT overtime and higher systolic blood pressure, lower levels of HDL, higher TC/HDL- and LDL/HDL-ratio, higher tri-glycerides, dyslipidemia and detection with (any) carotid plaque at inclusion. In the control group, lower levels of HDL, history of dyslipidemia and finding with bilateral plaque at inclusion were associated with cIMT overtime. At follow-up, hypertension and blood lipid measures in patients (with exception for TG) and HDL in controls were still significantly associated with cIMT overtime. Of all, the strongest association was shown for HDL in both patients and controls. Effect of other cardiometabolic risk factors at inclusion and cumulative risks were not significantly after adjustment for age and sex.

In the patients, history of nephritis at inclusion and follow-up, and a higher average dose of prednisone used since diagnosis, but not other treatments, were associated with cIMT overtime.

Conclusion: We observed similar progression of cIMT over 7 years in patients with long-standing mild well-controlled SLE disease and population controls. Our findings suggest importance of recognition with dyslipidemia and hypertension, and support recommendation to limit use of corticosteroids in patients with SLE as a part of CV risk management. The factors protecting or promoting atherosclerotic progression should be further explored.

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SAT0183

LUPUS NEPHRITIS: A RETROSPECTIVE LONGITUDINAL STUDY LOOKING FOR CHRONIC RENAL DISEASE ASSOCIATED FACTORS, FROM THREE SOUTH-EUROPEAN COHORTS OF PATIENTS IN FOLLOW-UP SINCE 2000

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European Rheumatology departments. Variables: demographics, SLE related variables, including global activity (SLEDAI-2K), renal flares, therapies, ACR response criteria and CKD. Statistical analysis: descriptive, bivariate and multivariate analysis exploring factors associated to CKD.

Results: Seventy-six patients with biopsy-proven LN were included, 90.7% female; mean age: 33 years; mean disease: duration 14 years; mean follow-up time (since LN diagnosis): 8.5 years. LN class III, IV and V were present in 22%, 75% and 3% of the cases, respectively. At LN diagnosis 68 (89%) patients had a severe renal flare. Forty-one (56.1%) and 49 (64.4%) had HTA and nephrotic syndrome, respectively. The mean 24h proteinuria levels at LN diagnosis was 4.6g. Mean SLEDAI-2K at the time of flare was 20.3, with 69 (65.7%) patients having an extrarenal flare.

The treatments used to induce remission were: glucocorticoids (100%); pulses M-prednisolone in 49 (64%) and oral prednisone (mean starting dose): 43 mg/day (±20.6); intravenous cyclophosphamide in 42 (55%) patients; mycophenolate mofetil (MMF) in 21 (27%) patients; calcineurin inhibitors in 5 (11%) patients; rituximab in 4 (5.2%) patients; and oral cyclophosphamide in 4 (5.2%) patients. Forty-eight (63%) patients were receiving hydroxychloroquine. MMF was the immunosuppressant (IS) more frequently used (52%) as maintenance therapy.

At 3, 6 and 12th months, the mean proteinuria was 2.3g/24h, 1.53g/24h, 1.1g/24h, respectively (p< 0.001). Fifty-five (77.5%) of patients achieved complete response and 61 (84.7%) presented complete or partial response. Mean time to renal remission: 12.5 months (6.175). In 32 patients (42%) it was possible to discontinue IS. Sixteen (21.9%) patients developed CKD, 4 (5.3%) needing dialysis and 1 (0.76%) renal transplantation. Serious infection was noticed in 23 (34.8%) patients. Five (6.6%) patients died (2 cardiac vascular cause).

In the bivariate study, the following variables were significantly associated with CKD: male sex, hypertension, ACEI drugs, severe infection after LN, temporal dysaliasis, non ACR renal response, non use of hydroxychloroquine, time to achieve 10mg/day of prednisone, previous creatinine to LN, maximum creatinine at LN, hyperlipidemia at 3 months of LN, active proteinuria at 6 months was the only factor finally associated with CKD.

Conclusion: A considerable percentage of LN patients developed renal chronic failure (21.9%). A high percentage of ACR response was achieved using medium dose (40mg) of glucocorticoids for induction. A significative reduction of proteinuria was achieved at 3 months, but proteinuria at 6 and 12 months.

In the logistic regression model, using genetic algorithms, we found that proteinuria at 6 months was significantly associated with CKD (OR=2.95; 95%CI 1.19-9.29, p= 0.03). Hypertension and male sex were marginally associated (p=0.06, both).

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SAT0184

ASSOCIATION OF SERUM AND URINE LEVELS OF TWEAK, MCP-1 AND NGAL WITH DISEASE ACTIVITY IN SYSTEMIC LUPUS ERYTHEMATOSUS

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Background: TWEAK, MCP-1 and NGAL, mediators in pathogenesis of systemic lupus erythematosus (SLE), are proinflammatory cytokines/chemokines that are thought as potential biomarkers reflecting disease activity (1).

Objectives: In this study, we aimed to investigate the association of serum (s) and urine (u) levels of TWEAK, MCP-1 and NGAL with disease activity in both renal and non-renal SLE.

Methods: Thirty active patients with SLE (15 renal and 15 non-renal) were recruited. Thirty-one inactive patients with SLE (16 renal and 15 non-renal) were included as control group. SLEDAI-2K and SLEDAI-2K-s were used for evaluation disease activity. SLEDAI-2K-s calculates the frequency and severity of disease manifestations, while SLEDAI-2K evaluates only renal manifestations.

Results: A total of 45 patients with SLE were included in the study (25 renal and 20 non-renal). There was no statistical difference between the groups in the age, sex, race, education and duration of disease. The mean SLEDAI-2K-s was 0.9 and the mean SLEDAI-2K was 2.2 for the renal SLE group, while the mean SLEDAI-2K-s was 1.5 and the mean SLEDAI-2K was 3.5 for the non-renal SLE group.

Conclusion: Our study showed that serum TWEAK, MCP-1 and NGAL levels were significantly higher in active renal SLE patients compared to inactive renal SLE patients. Additionally, we found that serum TWEAK, MCP-1 and NGAL levels were significantly higher in active non-renal SLE patients compared to inactive non-renal SLE patients.

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non-renal), 14 patients with ANCA-associated vasculitis (AAV) all of whom had active renal involvement and 20 healthy volunteers were selected as control groups. Serum and urine levels of TWEAK, MCP-1 and NGAL were tested using ELISA.

Results: Sixty-one SLE patients, 51 (83.6%) of whom were female, with a median disease duration of 83 (23.5-135) months and a median age of 35 (27-47.5) were included in the study. Serum and urine levels of TWEAK and NGAL were significantly higher in the active SLE group compared to the control group (SLE: p<0.005; uTWEAK: p=0.026; sNGAL: p<0.001; uNGAL: p=0.002); whilst no significant differences regarding serum and urine MCP-1 levels were observed (p=0.189 and p=0.106). uTWEAK (p=0.237), sMCP-1 (p=0.141), uMCP-1 (p=0.206), sNGAL (p=0.419) and uNGAL (p=0.443) levels did not differ between patients with active LN and non-renal active SLE; yet levels of sTWEAK were higher in patients with active LN (p<0.006). There were no differences between active LN and renal active AAV. Levels of all biomarkers were correlated with SLEDAI (sTWEAK: p=0.001; uTWEAK: p=0.006; sMCP-1: p=0.049; uMCP-1: p=0.014; sNGAL: p=0.001; uNGAL: p=0.002).

Conclusion: sTWEAK, uTWEAK, sNGAL and uNGAL are significant biomarkers showing disease activity in SLE. However, our results implicate that these biomarkers may not be specific for SLE, and can be elevated in patients with active renal involvement of AAV. sTWEAK may be of use for discriminating active nephritis from non-renal active disease in SLE. Further studies are awaited to confirm these results (This study was funded by Istanbul University with the project number TTU-2017-24738 and Turkish Society for Rheumatology).

REFERENCE


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