Background/Purpose Anti-carbamylated protein antibodies (anti-CarP) were detected in a large cohort of patients with Systemic Lupus Erythematosus (SLE) in correlation with erosive arthritis but not with disease activity indexes. In animal models, T-cells may be activated by carbamylated epitopes playing a role in the development of arthritis. The imbalance between circulating regulatory (Treg) and CD28- effector T-cells was described in association with disease damage, independently of different T-cell subtypes and anti-CarP levels in SLE.

Methods Eight SLE patients with a median (10th-90th percentile) SLEDAI-2K=0 (0–4), anti-dsDNA levels=34.1 (15.6–427.4) UI/ml (nv<7), SDI=1 (0–1.3) were enrolled. Serum anti-CarP levels were evaluated using a home-made ELISA (nv<340 AU/ml) and peripheral blood T cell immunophenotyping was done using Flow Cytometry (Beckman Coulter). Treg were defined as CD4+CD127lowCD25(high) T-cells.

Results Enrolled patients showed levels of anti-CarP=189.38 (93.5–341.1) AU/ml, Treg=2.2 (% of CD4+), CD4+CD28-=7.7 (4.8–22.5) (% of CD4+) and CD8+CD28-=30.3 (17.2–35.6) (% of CD8+). Analyzing possible correlations among different T-cell subtypes and anti-CarP levels, a significant inverse correlation was found between these autoantibodies and CD4+CD28- T cells (r=-0.8, p<0.01; Spearman rank correlation). No correlations were found between autoantibodies and other T-cell subpopulations or disease activity/damage indexes.

Conclusions In a small cohort of patients with serologically active SLE, anti-CarP autoantibodies were found as negatively correlated to circulating CD4+CD28- T-cells, which were described in association with disease damage, independently of age, gender, disease duration and activity. This suggest a potential role of anti-CarP as marker of SLE with a minor extent of T-cell activation and, consequently, with a possible better prognosis.

Methods Whole blood, serum and clinical data were obtained from 140 SLE individuals. Gene expression was assessed by NanoString® technology, using a panel of 37 probes allowing the computation of six IFN-I, one PMN and one PB scores. Protein levels were measured by ELISA.

Results High IFN-I gene expression was found in 45 to 50% of SLE individuals, depending on the score used. All 6 IFN-I scores were significantly associated with active skin involvement and 2 of 6 with arthritis. Interferon-induced GTP-binding protein MX1 (MX1) correlated with IFN-I score (p<0.0001) and was associated with a similar clinical phenotype. High PMN gene expression was found in 25% of individuals in association with SLE fever, serositis, leukopenia and glucocorticoid use. PB gene expression was highly influenced by immunosuppressant agents with no association with SLE features. The combined IFN-I and PMN gene expression was significantly associated with high disease activity and outperformed anti-dsDNA, anti C1q and complement levels to predict SLE activity.

Conclusions The IFN-I and PMN gene scores segregate with distinct SLE clinical features and their joint expression identify high disease activity. MX1 protein levels perform similarly to IFN-I gene expression.

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