dsDNA, C1q, chromatin, Smith, and ribosomal P (figure 1F). Autoantibody levels remained relatively stable in partial- and non-responder proliferative LN patients, as well as in patients with membranous LN.

Conclusions LN patients exhibit heterogeneous autoantibody profiles associated with ISN/RPS classification. Specifically, levels of autoantibodies against dsDNA, C1q, chromatin, and ribosomal P may serve as noninvasive biomarkers of proliferative LN. In patients with proliferative but not membranous LN, a decline in the titers of several autoantibodies, including many not routinely measured over time, such as anti-Sm, was associated with treatment response, suggesting a possible role in LN pathogenesis. In addition, these autoantibodies may serve as early biomarkers of treatment response.

LP-016 ANTI-HISTONE AND ANTI-NUCLEOSOME ANTIBODIES, RATHER THAN ANTI-DSDNA ANTIBODIES ARE ASSOCIATED WITH INTERFERON-INDUCED BIOMARKERS IN SUDANESE AND SWEDISH SLE PATIENTS

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Background In SLE, anti-dsDNA often exists together with autoantibodies against other chromatin components, like histones and nucleosomes. These antibodies can induce cytokines including interferon-alpha.

Methods We have measured ANA specificities and investigated their associations to inflammatory biomarkers. We included 93 Sudanese and 480 Swedish SLE patients. Serum levels of autoantibodies against dsDNA, Sm, the Sm/U1RNP complex, U1RNP, SSA/Ro52, SSA/Ro60, SSB/La, ribosomal P, PCNA and histones were quantified with a bead-based multiplex immunoassay. In the Swedish cohort also anti-nucleosome antibodies were investigated. Relative levels of 73 plasma biomarkers were determined with Proximity Extension Assay technique or ELISA. Adjusted p values were considered significant when <0.05.

Results Among Sudanese patients, levels of 5/73 biomarkers showed significant associations to ANA-associated antibodies. Anti-histone antibodies showed the strongest positive correlations with interferon-inducible factors MCP-1 and IP-10, and with MCP-3 and S100A12, and negative correlation with stem cell factor. Also anti-dsDNA antibodies associated with MCP-3, IP-10 and S100A12, but when combined in the same regression model, anti-dsDNA associations but not anti-histone lost significance.

Validation analyses among Swedish patients for MCP-1, IP-10, SA100A12 also demonstrated significantly stronger associations to anti-histone and anti-nucleosome antibodies respectively, compared to anti-dsDNA and other ANA specificities, and in combined regression models, anti-histone/nucleosome showed the strongest associations. When excluding anti-histone or anti-nucleosome positive patients, the associations between interferon-inducible factors MCP-1/IP-10 and anti-dsDNA and were lost. In contrary, when excluding anti-dsDNA positive patients, associations with anti-histone and anti-nucleosome respectively remained significant. SA100A12 associations with anti-dsDNA antibodies remained significant after exclusion of anti-histone positive patients but lost significance when excluding anti-nucleosome positive patients.

Conclusions Levels of mainly IFN-induced inflammatory biomarkers correlate stronger with anti-histone and anti-nucleosome antibodies compared to other ANA specificities including anti-dsDNA. Our results suggest that autoantibodies against DNA-complexes or DNA-associated proteins rather than anti-dsDNA induce the interferon signature in SLE.

LP-203 THE NEW MARKERS OF SYSTEMIC LUPUS ERYTHEMATOSUS ACTIVITY: FOCUS ON INTERLEUKIN (IL)-1B AND SOLUBLE IL-2 RECEPTOR

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Background Systemic lupus erythematosus (SLE) is a systemic autoimmune disease with unknown etiology, characterized by the hyperproduction of autoantibodies to various components of the cell nucleus and the resulting immune-inflammatory damage to tissues. Current trends of personalization therapy require the search for new serum markers, the production of which reflects aberrant activation of the immune system with further formation of autoimmunity. These are cytokines, chemokines and their receptors. Our aim was to determine the levels of interleukin (IL)-1b and soluble IL-2 receptor (sIL-2R) in patients with SLE, to evaluate their association with clinical and laboratory disease manifestations.

Methods The study included 26 patients (21 women, 5 men) with a diagnosis of SLE meeting the criteria of SLICC 2012 and EULAR/ACR 2019. The mean age of the patients was 33 ±11 years and the median disease duration was 14 [4;144] months. Examination of patients included standard laboratory and instrumental diagnostics. Disease activity was assessed using the SLEDAI-2K index. Serum levels of IL-1b and sIL-2R were determined by enzyme immunoassay (Invitrogen, Australia).

Results In the study cohort median IL-1b and sIL-2R levels were 3,3 [2,5;4,6] ng/mL and 0,0065 [0,005;0,008] pg/mL, respectively. Only negative correlation of IL-1b level with glomerular filtration rate was found (R=-0,48, p<0,01). sIL-2R level was associated with SLEDAI-2K (R=0,53, p<0,005), anti-dsDNA (R=0,55, p<0,003), C3 (R=-0,56, p<0,003) and ferritin level (R=0,47, p<0,05), CRP (R=0,45, p<0,002), urinary casts (R=0,46, p<0,01), leukocyturia (R=0,42, p<0,03). There were no statistically significant differences in the concentrations of both studied immunological markers between patients with lupus nephritis (LN) (n=18) and without LN (n=8).

Conclusions The concentration of sIL-2R correlates with laboratory indicators of SLE, SLEDAI-2K and urine sediments, suggesting its promising potential for SLE activity evaluation.