cells and relative mRNA expression of Nrf2 positively correlated with SLEDAI score.

Conclusion Our study clearly elucidates that intracellular oxidative stress was elevated in the subtypes of T-cells and their was alteration in Keap1/Nrf2/HMOX-1 and proportions, found to be associated with disease activity score(SLEDAI).

PO.2.29 SERUM LEVELS OF B-CELL RELATED FACTORS BELONGING TO THE TNF/TNFR SUPERFAMILY ARE LOWER IN ANTIPHOSPHOLIPID-RELATED SYNDROMES THAN IN SYSTEMIC LUPUS ERYTHEMATOSUS

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Purpose B-cell tolerance checkpoint defects are part of the pathomechanism in humoral autoimmune disorders, such as systemic lupus erythematosus (SLE) and antiphospholipid syndrome (APS).[1] In SLE, less stringent selection of autoreactive B cells at the transitional stage are potentially propagated by dysregulated BAFF (B-lymphocyte stimulator) homeostasis.[2] In APS, slight changes in circulating B-cell subsets were described, although with restricted autoantibody repertoire.[3] A recent study demonstrated that differences in the naïve B-cell repertoire could explain the higher number and variety of autoantibodies in SLE in comparison to APS, especially in those with obstetric complications.[4]

Increased levels of circulating BAFF were described in SLE and the efficacy of B-cell targeted therapies, such as belimumab and rituximab, was well demonstrated in clinical trials and/or observational studies.[5] In APS, despite the strong evidence of antibody mediated pathogenesis, specific B cell phenotype abnormalities, and by the development of the disease in patients with inborn errors of immunity involving B cell ontogeny, data on the use of therapies directed toward B cells are still lacking and anticoagulation remains the corner stone of management.[6]

The aim of this study was to measure serum levels of TNF/TNFR superfamily factors which are involved in B-cell homeostasis, looking for differences among diseases.

Methods Seventy-one patients [20 SLE, 10 SLE+aPL, 18 SLE +APS and 23 primary APS (PAPS)] were enrolled. The dosage was performed by high-sensitivity ELISA. Data are expressed by mean (in pg/ml, ng/ml for sBCMA). P values≤0.05 were considered statistically significant (*).

Results SLE patients had BAFF serum levels=1925 vs 1415 (in SLE+aPL), 965* (in SLE+APS), 1291 (PAPS);APRIL=8017 vs 3362*, 2502*, 2416*;sBCMA=14126 vs 10113, 8713*, 9145*;sTACI=8187 vs 2987, 1545*, 1570*;sCD40L=3965 vs 2943, 1663*, 2098*;TWEAK=11331 vs 4971, 1890, 1573.

The statistical differences in factor levels among the clinical conditions were maintained after analysis by mixed linear model with multiple imputation, controlling for several clinical parameters (sex, race, BMI, blood pressure, hormonal substitution, age, disease duration).

Conclusions Lower serum levels of B-cell related TNF superfamily factors in the aPL-related conditions compared with SLE suggest that selection of autoreactive B cells in the transitional stage, which is central in SLE, might not be the prominent mechanism for inducing aPL antibodies.

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PO.2.30 IGG/IGM AUTOANTIBODY RATIOS DO NOT RELATE TO PROGRESSION FROM INCOMPLETE SYSTEMIC LUPUS ERYTHEMATOSUS TO SYSTEMIC LUPUS ERYTHEMATOSUS

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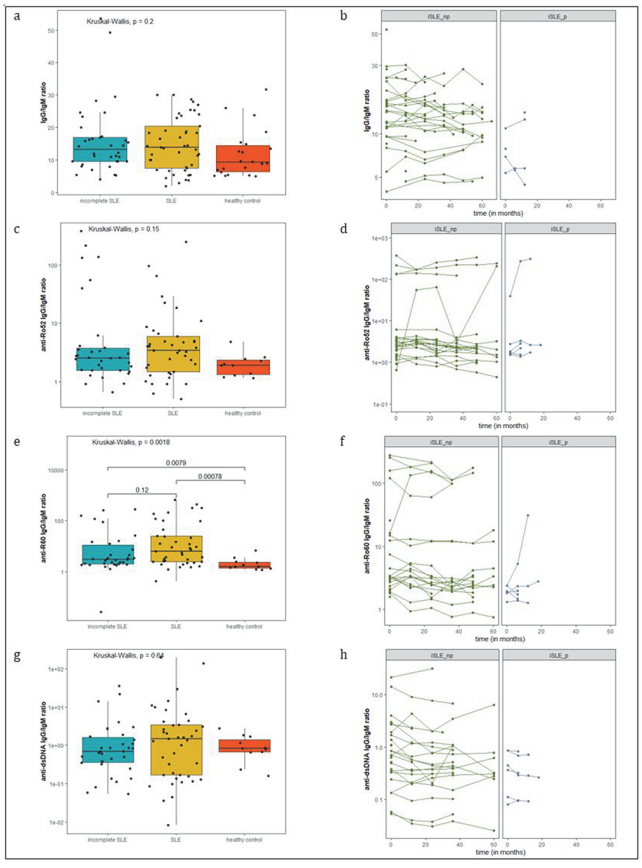
Purpose To identify whether IgG/IgM autoantibody ratios differ between patients with incomplete systemic lupus erythematosus (iSLE), patients with SLE and healthy controls (HC) and



Abstract PO.2.30 Figure 1 Median and interquartile ranges are depicted for a, c, e and g. Total IgG/IgM ratios (a), anti-Ro52 IgG/IgM ratios (c) and anti-dsDNA IgG/IgM ratios (g) were not significantly different between groups. Anti-Ro60 IgG/IgM ratios were significantly elevated in iSLE and SLE patients compared to healthy controls. Longitudinal line graphs of total IgG/IgM ratio (b), anti-Ro52 IgG/IgM ratio (d), anti-Ro60 IgG/IgM ratio (f) and anti-dsDNA IgG/IgM ratio (h) are shown for iSLE patients that did not progress to SLE (iSLE_np) and for iSLE patients that did progress to SLE (iSLE_p)