aim of our study is to investigate the value of B cells subsets as biomarkers in patients with active LN, in patients at the onset of renal manifestation or with renal flare, and finally in nephritic patients in relation to their clinical and laboratory characteristics at the baseline and during the course of the disease.

Methods 50 patients with active LN at disease onset or disease flare were enrolled and evaluated every tree months. Laboratory, immunological and disease activity data were collected at the baseline and at 6(T6), 12(T12), 24(T24), 36(T36) months and at the last follow-up(FU).Number of renal flares, time to renal remission and persistent proteinuria at the last FU were considered. B cell subsets were evaluated at baseline through cytofluorimetry and classified using C27/IgD classification. The characterisation of B cells subsets was realised in 50 LN patients and 37 healthy controls.

Results LN patients had a lower percentage of CD19 +cells than controls(9.2% vs 10.6%; p=0.01)as well as a lower percentage of memory unswitched cells CD27 +IgD+(10.7% vs 15.3%; p<0.001) while LN patients had an higher percentage of plasmablasts and double negative memory cells CD27-IgD- (respectively 5.9% vs 1%; p<0.001 and 10,9% vs 4,1%; p=001).

No significant differences regardless B cells subsets were found between early LN patients and long ones as well as between LN patients at the onset and LN patients during renal flare. We found a correlation between an higher disease activity (assessed with SLEDAI 2K) and lower percentage of memory B cells IgD-CD27+(p=0.02).Double negative B cells CD27-IgD- tended to be correlated with an higher disease activity. Of interest the correlation between persistent proteinuria detected during the follow-up and a lower percentage of plasmablasts at the baseline (p=0.015).

Conclusion The alteration of B cells subsets is an early event in LN without differences regardless the timing of renal involvement (nephritic onset or later LN development).The association between persistent proteinuria and a lower percentage of plasmablasts at the baseline could be a negative prognostic factor considering the correlation between persistent proteinuria and worse renal outcome.

PS1:10 EFFECTS OF BELIMUMAB TREATMENT ON B CELL HYPERACTIVITY AND TYPE-I INTERFERON EXPRESSION IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS

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Purpose Belimumab, a monoclonal anti-BAFF antibody, has been approved for patients with active systemic lupus erythematosus (SLE) despite standard of care immunosuppressive treatment (ST). However, the interference of belimumab with pathogenetic pathways of SLE is not fully understood. B cell hyperactivity and overexpression of type-I interferons (IFN) have been shown to be key elements in the pathogenesis of SLE. This study shows the effect of belimumab on biomarkers representing B cell hyperactivity and IFN expression in SLE patients.

Methods 20 SLE patients treated with belimumab (BT), 82 SLE patients with ST and 30 matched healthy controls (HC) were recruited. Siglec-1 expression on monocytes representing IFN signature, BCMA expression on different B cell subsets and the frequency of activated naive B cells (aNB) in PBMCs were analysed by FACS. Serum levels of BAFF plus soluble receptors sBCMA and sTACI were determined by ELISA.

Results Compared to ST, BCMA expression was reduced in BT on naive B (p<0.001) and memory B cells (p<0.05) but not on aNB, plasmablasts and plasma cells. In comparison to HC, BCMA expression was similar on all B cell subsets, except on aNB where it was higher in BT (p<0.001). The frequency of aNB among total B cells was reduced in BT compared to ST (p<0.001) and was comparable to HC. Siglec-1 expression on monocytes did not differ significantly between BT and ST; both groups showed a rise compared to HC (each p<0.001). There was no significant difference after belimumab treatment. Furthermore, serum BAFF levels in ST and BT were higher than in HC (each p<0.001), but did not differ significantly between BT and ST. Serum levels of sBCMA (p<0.05) and sTACI (p<0.001) were lower in BT compared to ST and also after belimumab treatment (each p<0.05). BT's sTACI levels were lower than in HC (p=0.01).

Conclusions This study provides deeper insights into the impact of belimumab on several pathogenetic pathways of SLE activity. Regarding the inhibition of B cell hyperactivity, one key pathogenetic element of SLE, belimumab treatment showed distinct advantages. Furthermore, these results suggested that belimumab treatment did not impair the type-I IFN pathway.

PS1:11 THE INTERFERON BIOMARKER SIGLEC1 REFLECTS DISEASE ACTIVITY IN PAEDIATRIC SYSTEMIC LUPUS ERYTHEMATOSUS

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Introduction SIGLEC1 (sialic acid-binding Ig-like lectin 1, CD169) is a monocytic adhesion molecule induced by interferon – α. In adult systemic lupus erythematosus (SLE), SIGLEC1 correlates cross-sectionally and longitudinally with disease activity. The aim of this work was to examine whether SIGLEC1 also reflects the disease activity in paediatric SLE.

Methods Over a period of 29 months the disease activity was clinically evaluated using SLEDAI (SLE-Disease Activity Index-2000). In 28 consecutive paediatric SLE patients (mean age 16 years, range 3–38 years, 86% female, 14% male), the number of SIGLEC1 molecules per CD14 +on blood monocyte was quantified using flow cytometry. At the same time, the level of anti-ds DNA-antibody titer (ELISA) and the concentration of complement factors C3 and C4 (nephelometry) were determined. The association between SIGLEC1, C3, C4 and ds DNA-antibody with SLEDAI was estimated using a mixed linear model to model the repeated measurement of parameters within a patient. The cut-off for the change in SIGLEC1 between two consecutive visits to predict minimal clinical improvement or worsening in SLEDAI was chosen on the maximum Youden Index.