Results We identified 15 patients currently receiving BEL therapy. Of these, 9 were not analysed further because they had no history of LN or NPSLE.

One 48-y/o female patient after renal transplantation with background therapy consisting of prednisone (GC), hydroxychloroquine (HCQ), leflunomide (LEF) and tacrolimus (TAC) had a stable disease but no additional benefit (BEL stopped after 5 months). Three female patients with GC, HCQ and mycophenolate mofetil (MMF) had an improvement of proteinuria, steroid dosage and overall quality of life. One female patient is receiving BEL or placebo (PBO) during a clinical trial (BLISS-LN), she has markedly improved proteinuria with GC, HCQ, MMF and BEL/PBO. One 73-y/o male patient with NPSLE who failed or could not tolerate various standard and additional therapies (including Rituximab and Cyclophosphamide) had a persistent clinical improvement of cutaneous lupus and neuropsychiatric symptoms (dysarthria, concentration, ataxia) after the second BEL infusion. Overall, there was one upper respiratory tract infection but no other adverse

Conclusions In the six patients analysed, 3 had improved proteinuria, 1 had stable disease after renal transplantation, 1 improved regarding NPSLE symptoms and 1 had improved proteinuria, but in the last case, it is not yet clear whether the effect is due to BEL. Overall, while the results of the BLISS-LN trial are awaited, we experienced improved Lupus nephritis with BEL in addition to standard therapy and observed one case of improved NPSLE. While BEL has not been approved for these severe organ manifestations, it still might be effective in well-selected patients.

PS8:168 PROTEINURIA IN RELATION TO CLASS OF LUPUS NEPHRITIS – A RETROSPECTIVE SINGLE-CENTRE STUDY

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Purpose Lupus nephritis (LN) is one of the most severe organ complications of Systemic lupus erythematosus (SLE) affecting up to 60% throughout the course of their disease. Currently, LN is classified according to the ISN/RPS classification. Classes III/IV require aggressive immunosuppressive treatment to avoid end-stage renal disease. However, there are no clinical or serological parameters to predict the type of renal disease and overall renal prognosis.

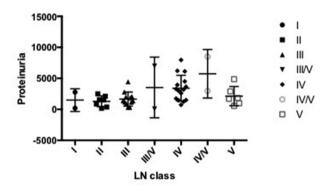
Methods We performed a single-centre study at our institution of all patients who underwent a renal biopsy between 2001 and 2017. Proteinuria, creatinine and other clinical/serological data were collected. Median values were analysed with ANOVA and Bonferroni's correction for multiple comparisons. Results 49 patients were analysed in our study. 3 patients were excluded because of incomplete data. The remaining 46 patients were stratified according to the histopathological class of Lupus nephritis. 2 patients had class I, 7 patients had class II, 12 patients had class III, 2 patients had class IV/V and 6 patients had pure class V.

Median proteinuria at or around the nearest time point to renal biopsy were 1487 mg/g creatinine (Cr) (class I), 1515 mg/g Cr (class II), 1373 mg/g Cr (class III), 3528 mg/g

Cr (class III/V), 3190 mg/g Cr (class IV), 5741 mg/g Cr (class IV/V) and 1773 mg/g Cr (class V).

While LN classes III/V, IV and IV/V showed the highest median proteinuria, there was no statistical difference between groups.

Conclusions Although often presumed, proteinuria is not a reliable marker for the various types of Lupus nephritis. There was a higher median proteinuria with class V (pure or combined) membranous nephropathy, however, even proteinuria in this group was not significantly different compared with the other groups. Lack of reliable clinical markers challenges the current lupus nephritis classification system, a combination of clinical, serological and histopathological findings might more appropriately predict the overall prognosis in LN.



Abstract PS8:168 Figure 1

PS8:169 PREVALENCE AND IMPACT OF DYSLIPIDEMIA IN LUPUS NEPHRITIS PATIENTS

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Dyslipidemia is a well-established atherosclerotic risk factor. It is also believed to affect the outcome of SLE, especially in lupus nephritis patients (LN). The aim of this study was to assess the prevalence and impact of dyslipidemia in our LN patients.

Methods We performed a retrospective clinical study, 140 patients with biopsy-proven LN from were analysed. The renal activity and classification were evaluated according to renal pathology. SLE disease activity was scored using the SLE Disease Activity Index (SLEDAI). Adverse outcome was defined by the occurrence of ESRD or death. The correlations between dyslipidemia and both ESRD and mortality were assessed.

Results Mean age of our patients was 34.63±12,7 years old, 83% were females. Class III, IV and V lupus nephritis accounted for 21%, 58.7% and 11.2% The prevalence of dyslipidemia with elevations in total cholesterol (TC), low-density lipoprotein (LDL), triglyceride (TG) were noted in in our LN patients, ranging from 41% at diagnosis to 59.7% or even higher after 24 months, and statitns were administered in 23% of the patients

After a mean follow-up of 22 months, ESRD occured in 24%, and death in 13% of cases. Moreover, dyslipidemia was significantly associated to both ESRD (p<0.02) and death (p<0.003).

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Conclusions Dyslipidemia is a significant comorbidity of LN that severely affects its renal and overall outcome. Its treatment represents a modifiable risk factor; adequate management can decrease its complications in LN patients and therefore improve their overall morbidity and mortality.

PS8:170

LUPUS NEPHRITIS IN A MULTI- ETHNIC COHORT OF PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS FROM BERKSHIRE, UK

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Background Previous studies suggest that ethnicity influences the clinical phenotype in systemic lupus erythematosus (SLE), with lupus nephritis (LN) being more frequent in non-Caucasians. However, there are scarce data regarding prevalence of LN in South-Asian population (Indians, Pakistani, Sri Lanka, or Bangladeshi).

Objectives This study aimed to compare the prevalence of LN between lupus patients of different ethnicities, and to compare demographics and disease characteristics between LN patients. Methods This is a retrospective chart review study of 100 lupus patients followed from 2013 to 2017 at Wexham Park Hospital, a large district hospital in Southern England. The patients were categorised into four ethnic groups Caucasians, South-Asians, Blacks and Others (mixed race, Orientals, Arabs). LN prevalence, demographic and clinical data were compared using Fisher/Chi-Square tests for categorical variables and Wilcoxon test for continuous variables.

Results Of 100 patients in the study sample, 51% were Caucasians, 31% were South-Asians, 11% were Blacks and 7% had other ethnicities. Mean age was 48 yo and 90% were females. Prevalence of LN was 26% in the full study sample and 24%, 16%, 64% and 57%, respectively, among Caucasians, South-Asians, Blacks and Others. LN prevalence was significantly lower in Caucasians vs Blacks (p=0.01), South-Asians vs Blacks (<0.01) and South-Asians vs Other (p=0.02). Among patients with LN, mean age was lower in South-Asians and Blacks, than Caucasians and Others (44 and 45 yo vs 52 and 51 yo). Blacks appear to include more males (43% vs <25% in the other groups). Renal biopsy, available for 22/26 LN patients, suggested Class II predominance in Caucasians (44% vs 33% in the other groups). Proliferative LN (Class III and IV) was confirmed in 7 patients, without significant predominance in any ethnic group. Specific lupus autoantibodies (anti-dsDNA Ab and/or antiSm Ab) were found in 64% Caucasians, 75% south-Asians, 86% Blacks and 75% others with LN.

Conclusion In our cohort, prevalence of LN in South-Asians was lower than in Blacks, but not statistically different comparing with Caucasians. However, South-Asians and Blacks with LN were younger than Caucasians. These results should be re-examined in larger similar multi-ethnic cohorts.

PS8:171

ENDOTHELIAL DYSFUNCTION AND VASCULAR RISK FACTORS IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS

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Objectives Lupus systemic erythematosus is characterised by an increasing risk of premature cardiovascular disease (CVD). CVD is one of the most common causes of death in SLE. Subclinical atherosclerosis in comparison to general population is also more prevalent, especially the presence of plaques at the carotid level, as well as thickening of the carotid intima.

The aetiology of atherosclerotic disease is completely unknown. It involves: traditional risk factors (age, male gender, smoking, diabetes, hypertension, dyslipidemia, obesity) as well as risk factors related to the disease itself and the treatments used

Methods A cross-sectional study was carried out from March to November 2015 in 119 patients. Patients were recruited from consultation at the Systemic Autoimmune Diseases Unit for a routine medical check. Clinical data on the disease (from diagnosis to the time of inclusion in the study) were obtained by reviewing the medical history.

The population was divided into two groups: patients with lupus and endothelial dysfunction and patients with lupus without endothelial dysfunction. The existence of endothelial dysfunction was explained by the presence of plaques at the carotid and/or intimate mean thickness >0.8 in a doppler ultrasonography.

Results There is no association with taking antimalarials, immunosuppressants, corticosteroids prior to high doses.

As for the classification criteria there is no relation with the presence of malar rash, Photosensitivity, Oral ulcers, Arthritis, Serositis, Nephropathy, Cytopenias and DNA.

No significant differences were detected in the determination of antibodies or complement levels.

No differences were found with SLEDAI. Since lupus is a disease that occurs in outbreaks, finding no differences may be due to the fact that at the time of inclusion patients had a low activity.

The presence of hypertension and dyslipidemia favours the existence of endothelial dysfunction. Hypertensive patients have a five-fold increased risk of developing endothelial dysfunction (5,593, 95% CI: 2340 to 14,015) as well as patients with dyslipidemia with a nearly 3-fold increased risk (2,976 CI: 1191 to 7,591).

Conclusions Hypertension and dyslipidemia remain the classic risk factors associated with increased endothelial dysfunction. Strict control of them is imperative.

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