P144 POTENTIAL REVERSIBLE CAUSES FOR FATIGUE IN SLE PATIENTS – DIFFERENCES BETWEEN MILD AND SEVERE FATIGUE

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Background/Purpose Fatigue is the most common symptom in SLE patients with a strong impact on patients' reported quality of life. Previous studies identified possible associations with fatigue with some contradictory results. The aim of this study was to identify additional potentially reversible associations with fatigue to generate targets for future interventions.

Methods Our study population consisted of 234 consecutively recruited SLE (1997 ACR criteria) outpatients from our university hospital based lupus reference centre. We analyzed clinical and demographic data from routine visits, laboratory variables, as well as sleeping disorders, disease perception, coping, social activities and health locus of control using validated questionnaires. We captured fatigue using the Fatigue Severity Scale (FSS). A score of $\geq$ 4 points is considered pathological. To assess depression, we used the German CES-D depression scale (values $\geq$ 23/60 points are considered pathological).

Results Our predominantly Caucasian cohort (99.1%) was mostly female (87.6%), with a mean age of 45.3 years (±13.4 [SD]) and a mean disease duration of 16.2 years (±9.5). In our cohort, 50.9% of patients reached a pathological result in the FSS. Depression was significantly associated with fatigue ($p<0.001$). Patients with poor health conditions (including sleeping disorders, pain, disease activity and damage) showed significantly more fatigue ($p<0.01$). Overall, in 71.4% of our SLE patients, we could identify at least one potentially reversible association for fatigue (shown in table 1).

Abstract P144 Table 1 Potential reversible associations in our cohort

		No Fatigue	Mild Fatigue	Severe Fatigue
Variable	N (%)	FSS <4 points	FSS 4.0–5.5 points	FSS =5.6–7.0 points
Obesity (BMI >30 kg/m^2)	27/234 (11.5)	11/115 (9.6)	5/48 (10.4)	11/71 (15.5)
Signs for depression (CES-D $\geq$ 23 points)	59/209 (28.2)	18/99 (18.2)	14/48 (29.2)	27/71 (38.0)
Vitamin D deficiency (25-hydroxyvitamin D <30 ng/ml)	73/213 (34.3)	39/106 (34.0)	14/48 (29.2)	20/71 (28.2)
Anemia (Hb <11.9 g/dl)	41/234 (17.5)	17/115 (14.8)	13/48 (27.1)	11/71 (15.5)
Hypothyroidism (TSH >4,2 µUI/ml)	6/225 (2.6)	2/112 (1.7)	2/48 (4.2)	2/71 (2.8)

Conclusion We observed a high prevalence of depressive disorders in our cohort and a significant correlation of depressive status with fatigue. Therefore, we suggest that psychological wellbeing is assessed in everyday clinical practice and treating physicians should react to patients' needs accordingly. Additionally, obesity, anaemia, hypothyroidism and vitamin D deficiency can easily be assessed. Optimizing these factors represent possible targets in order to improve fatigue in SLE patients.

P145 MEMBRANOUS AND PROLIFERATIVE LUPUS NEPHRITIS – ANALYSIS OF A NATIONWIDE MULTICENTRE COHORT

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Background Lupus Nephritis (LN) is one of the most severe manifestations of Systemic Lupus Erythematosus (SLE). We aimed to compare proliferative (PLN), membranous (MLN) and mixed LN regarding clinical and laboratory presentation, and serologic profiles. Previous work suggested that these groups differ in autoantibody profile and complement levels, but those reports mainly originate from single-centres.

Methods Multicentre observational study, with retrospective analysis of a prospective cohort, using data from the Portuguese registry of rheumatic diseases–Reuma.pt. Patients with biopsy-proven PLN, MLN and mixed LN were included. The first renal biopsy showing one of these classes was considered, for each patient. Groups were compared using Pearson's Chi-Square for categorical variables and One-Way ANOVA or Kruskal-Wallis for numerical variables.

Results 232 patients were included (87% females; 88.5% White Europeans). Median follow-up was 7 years (IQR 10.75; maximum 35 years). As seen in table1, the level of proteinuria did not differ between groups; however, MLN patients presented with significantly lower creatinine. Levels of complement were reduced in PLN and mixed LN but were normal in MLN patients, and this difference was statistically significant. Groups also differed regarding the proportion of positivity for anti-dsDNA (higher in PLN) and anti-RNP antibodies (higher in MLN). There was a lower SLEDAI in MLN, probably linked with the lower prevalence of anti-dsDNA antibodies and complement consumption in this group.

Conclusions Our results support previous findings from single-centre studies suggesting that MLN has a different serological profile than PLN, possibly reflecting different pathogenesis.

Abstract P145 Table 1 Comparative description of patients with proliferative, membranous and mixed LN enrolled in Renuma.pt – the Portuguese registry of rheumatic disease covering over 20 centres throughout Portugal

	Class III and IV	Class V	III+V or IV+V	P
Total, N	184	41	7	
Females, N (%)	157 (85)	39 (95)	5 (71)	0.121
Ethnicity **White European**, N (%)	159 (91)	28 (78)	6 (86)	0.079
Other, N (%)	16 (9)	8 (22)	1 (14)	
Age LN diagnosis(y), median (IQR)	30 (20)	34 (16)	42 (27)	0.932
Time SLE-LN(y), median (IQR)	1.5 (6.8)	1 (6)	0.5 (2.3)	0.477
SLEDAI at LN diagnosis, median (IQR)	**16 (10)**	**10 (10)**	**21 (17)**	**0.005**
uPCR at LN diagnosis, median (IQR)	1650 (2580)	1600 (1934)	2580 (3619)	0.692
Creatinine at LN diagnosis, median (IQR)	**0.80 (0.32)**	**0.69 (0.21)**	**1.0 (0.95)**	**0.005**
Albumin at LN diagnosis, mean ± SD	34 ± 7	34 ± 7	29 ± 6	0.294
C3 at LN diagnosis, mean ± SD	**0.65 ± 0.25**	**0.92 ± 0.36**	**0.55 ± 0.32**	**<0.001**
Ever low Complement, N (%)	**160 (90)**	**27 (71)**	**7 (100)**	**0.004**
Ever anti-dsDNA positive, N (%)	**174 (95)**	**31 (78)**	**6 (86)**	**0.001**
Ever anti-Sm positive, N (%)	37 (21)	12 (32)	3 (43)	0.155
Ever anti-Ro positive, N (%)	36 (34)	14 (58)	3 (50)	0.074
Ever anti-La positive, N (%)	18 (17)	5 (22)	2 (33)	0.562
Ever anti-RNP positive, N (%)	**32 (30)**	**15 (65)**	**2 (33)**	**0.007**
> 1 renal biopsy, N (%)	26 (14)	4 (10)	0	0.440
Different class in subseq. biopsy, N (%)	4 (15)	2 (50)	NA	0.169

SLE: Systemic Lupus Erythematosus; LN: Lupus Nephritis; uPCR: urinary protein-creatinine ratio, mg/g; y: years; Creatinine is presented in mg/dL, albumin in g/L and C3 in g/L.

P146 FRACTURE RISK IN SYSTEMIC LUPUS ERYTHEMATOSUS PATIENTS OVER 25 YEARS

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Background Osteoporosis and fractures are complications of glucocorticoid treatment. Current EULAR guidelines for the treatment of SLE recommend minimising long term glucocorticoid aiming at ≤ 7.5 mg/day. We examined the relationship of glucocorticoid dosing on fracture risk in patients with SLE.

Methods Retrospective data collection on SLE patient attending University College London Hospital clinic over a 35year period. The data included consecutive steroid dosing, Bone marrow density scans (BMD) and fragility fractures.

Results We reviewed 250 patients selected because we had a minimum of 10yrs follow up on them, 229 female (92%), 130 Caucasian (52%), 62 (25%) Afro-Caribbean and 45 (18%) South Asian. Mean age of SLE diagnosis was 27years and 27% were smokers. Fragility fractures were diagnosed in 28 patients (11%), mean age of the first fracture 51 years ± 16 years. Ten patients (36%) were diagnosed with osteoporosis prior to the fracture (p 0.006). The majority, 94% of patients were treated with glucocorticoids with an average daily dosing of 6.20 mg/day. Patients with fractures, had a lower average daily dosing – 5.36 mg/day (p 0.127), but had a higher median cumulative dose (25.19 g versus 20.96 g, p 0.229). The majority of patients received vitamin D and calcium supplementation (92% p 0.109 and 84% p 0.163 respectively). However, hyperparathyroidism (n =6) was significantly associated with fragility fractures (p value 0.020). The presence of end-chronic kidney disease or rheumatoid arthritis were not related to the development of fractures (p 0.381; p 0.139, respectively). Regarding treatment, 22 patients with fractures were treated with bisphosphonates (p <0.001), two had denosumab (p 0.034) and two had teriparatide (p 0.012).

Conclusions In our cohort, there was no statistically significant association between glucocorticoids treatment and fragility fractures. Hyperparathyroidism was significantly correlated with fragility fractures in our group. The majority of patients averaged daily dosing in accordance with EULAR recommendations.