Background: Disease activity in patients with Systemic Lupus Erythematosus (SLE) is an important contributor to organ damage and premature mortality. Current indices to capture disease activity are not well suited to reflect their contribution to long term outcome. Lupus Low Disease Activity State (LLDAS) has been developed as an alternative measure of long term disease activity.

Objectives: To determine whether 50% of time spent in Lupus Low Disease Activity State (LLDAS-50) impacts on mortality and damage accrual in SLE.

Methods: A retrospective analysis of prospectively collected data was conducted on 3650 clinic visits by 207 patients in the Troma Lupus Cohort. Lupus Low Disease Activity State –50 (LLDASS0) score was defined as at least 50% of follow-up time with SLE Disease Activity Index (SLEDAI) ≤4, no new disease activity, prednisone ≤7.5 mg/day and no escalation of maintenance immunosuppressant therapy. Cox regression analysis was used to evaluate the impact of LLDASS0 in terms of mortality and damage development (either new or severe) by Systemic Lupus Erythematosus Clinical Criteria (SLICC)/American College of Rheumatology (ACR) Damage Index (SDI). New damage was defined as a rise in SDI by 1 from baseline whereas severe damage was defined as a rise of 3 points or more from baseline.

Results: The median age at diagnosis of the cohort was 34 years with the majority (84%) being female. The median follow-up time was 125 months. A total of 69 patients (33.5%) spent at least half of their follow up time in LDAS, thus achieving LLDASS0. After correction for age and gender, LLDASS0 was associated with a significant reduction in risk of having any new damage (OR 0.65; 95% CI 0.44–0.96, p<0.01), severe damage (OR 0.46; 95% CI 0.25–0.83, p<0.01), and also a reduction in mortality risk (OR 0.42; 95% CI 0.21–0.82, p<0.01). These values were also found for female patients who spent 30% or more time in LDAS, and were also found to be significant for death (OR 0.46, 95% CI 0.26–0.83, p=0.05) but not for new damage (OR 0.92, 95% CI 0.62–1.35, p=0.67) or severe damage (OR 0.71, 95% CI 0.42–1.19, p=0.19).

Conclusions: The significant reduction in the risk of long term damage and mortality supports the use of LLDASS0 as a therapeutic goal.

Disclosure of Interest: None declared

positivity and use of GCs. Oral and ocular damage items significantly correlated with ESSPRI, CHOP and OSDI, whereas systemic damage items positively correlated with patients' health value.

Conclusions: This large pSS cohort confirmed that demographic and clinical characteristics as well as medication are independently associated with disease-related and treatment-related damage. In particular, this study shows a highly significant impact of baseline disease activity on the development of future damage and poor PROs in pSS patients.

Disclosure of Interest: None declared


FR0354 URINE METABOLIC FINGERPRINT AS DIAGNOSTIC BIOMARKER FOR LUPUS NEPHRITIS

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Background: Lupus nephritis (LN) represents the main prognostic factor for worsening in systemic lupus erythematosus (LES). The relevant classes of LN—due to the need of treatment—are the proliferative (III, IV, III/IV-V) and membranous (V).

Objectives: The aim of the study was to find a urinary metabolomic fingerprint to diagnose proliferative and/or membranous LN.

Methods: Cross-sectional study. Inclusion criteria: lupus patients with and without clinical significant lupus nephritis (classes III, IV, V and mixed classes).

Results: We included 29 lupus patients, 11 with LN. The median SLEDAI score in LN patients was of 13 vs. 3 in those without NL (p<0.0001). Class IV nephritis was present in 45%, mixed class in 36%, and class V in 18%. The median proteinuria of patients with NL was 1 g/L, (IQR 2.7).

Conclusions: We identified a urinary metabolomic fingerprint that involved several metabolic pathways; 2-nonanone and the ratio of 2-bromopropane/2-nonanone had the best diagnostic accuracy, (sensitivity of 0.87 and specificity of 0.93) of proliferative LN. Obtaining the ratio of 2-bromopropane/2-nonanone, the diagnostic accuracy improved, with a positive likelihood ratio (LR) of 14 and a negative LR of 0.1 (AUC 90%).

Metabolic pathways involved in LN were: methane, glycolysis, pyruvate and glycero-phospholipid pathways.