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 Tromsa 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) ≤5, 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 LLDAS, thus achieving LLDASS0. After correction for age and gender, LLDAS-50 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 true for female patients who spent 30% or more time in LLDAS, 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 0.42–1.29, 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


FRIO352

DAMAGE ACCURACY IN A LARGE MONOCENTRIC COHORT OF PRIMARY SJÖGREN’S SYNDROME PATIENTS: DETERMINANTS AND IMPACT ON PATIENT REPORTED OUTCOMES

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Background: Primary Sjögren’s Syndrome (gSS) is a chronic progressive disease potentially leading to irreversible organ damage. To date only a limited number of studies have analysed prevalence and factors associated with damage accrual in gSS.

Objectives: a) to characterise cumulative damage in gSS patients, b) to identify determinants associated with its presence and c) to evaluate the impact of damage on patient reported outcomes (PROs).

Methods: Data from a monocentric cohort of 466 gSS patients were analysed. Glandular and extra-glandular damage manifestations were assessed by the Sjögren’s Syndrome Disease Damage Index (SSDDI). Additional items of damage defined ‘a priori’ as being potentially related to treatment (i.e. osteoporosis, diabetes, infections) were analysed separately. The EULAR Sjögren’s Syndrome Disease Activity Index (ESSDAI) was used to measure disease activity at baseline and prospectively during the follow-up. The EULAR Sjögren’s Syndrome Patient Reported Index (ESSPRI), Oral Health Impact Profile (OHIP) and Ocular Surface Disease Index (OSDI) were used to record PROs. Patients’ comorbidities were assessed by the Charlson Comorbidity Index (CCI).

Results: A total of 466 gSS patients (446 F:20 M, median age (IQR): 59 years (48–69) were included in the study. The frequency of anti-Ro-SSA in the cohort was 69.5% (324/466). The median ESSDAI was 4 (IQR 1–8) at baseline and 2 (IQR 0–5) at the last evaluation, respectively. In addition to symptomatic agents, patients had been treated during the disease course with low-medium doses of glucocorticoids (GCs) (56%), hydroxychloroquine (HCQ) (62%) and DMARDs (16.6%). After a median follow-up of 5 years (IQR, 2–10), 208 patients (44.6%) had accrued some damage in either the oral damage items (33%), ocular damage items (20%) and/or systemic damage items (12%). In addition, 24/466 patients had developed a non-Hodgkin lymphoma and 2 patients a multiple myeloma. The SSDDI score ranged from 0 to 14. In the regression analysis; patients more likely to develop damage were those that were older, with a longer disease duration, higher baseline ESSDAI and who had been treated with DMARDs, whereas patients who had been ever treated with HCQ were less likely to develop disease-related damage. Similarly, treatment-related damage was independently associated with: disease duration, age of the patients, baseline ESSDAI, anti-Ro/SSA

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