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. Groups were compared using Pearson's Chi-Square for categorical variables and One-Way ANOVA or Kruskal-Wallis for numerical variables. COX regression analysis was used to investigate predictors of CKD (defined as estimated glomerular filtration rate [eGFR] lower than 60 mL/min/1.73m² for at least 3 months) and Kaplan-Meier curves were drawn.

Results: 236 patients were included. Median follow-up was 8 years (IQR 11; maximum 35 years). As seen in table 1, the level of proteinuria did not differ between groups; however, MLN patients presented with significantly lower serum creatinine. Levels of complement C3 and C4 were reduced in PLN but normal in MLN patients, and there were fewer patients with positive anti-dsDNA antibodies in the MLN group ($p<0.001$). On univariable COX regression, mixed histology was associated with progression to CKD (HR 26 [95% CI 3 - 255], $p=0.005$) (figure 1), however, it lost significance after adjusting for eGFR. In fact, $eGFR\leq 75$ at one year after the renal biopsy (HR 21 [95% CI 7 - 65], $p<0.001$) was the strongest predictor of CKD, even after adjusting for hypertension or histology.

Table 1. Comparative description of the Reuma.pt cohort of patients with proliferative, membranous and mixed LN

	PLN	MLN	Mixed	P
Total, N	186	42	8	
Females, N (%)	157 (85)	39 (95)	4 (50)	0.004
Ethnicity White European, N (%)	163 (90)	31 (78)	7 (88)	0.115
Other, N (%)	19 (10)	9 (23)	1 (13)	
Age LN diagnosis(y), median (IQR)	30 (20)	34 (16)	42 (25)	0.409
SLEDAI at LN diagnosis, median (IQR)	16 (9)	10 (10)	21 (17)	0.006*
uPCR at LN diagnosis, median (IQR)	1675 (2598)	1698 (2153)	2160 (3320)	0.629
Creatinine at LN diagnosis, median (IQR)	0.80 (0.32)	0.70 (0.20)	1.00 (0.95)	0.006*
eGFR at LN diagnosis, mean $\pm$ SD	98 $\pm$ 33	112 $\pm$ 17	82 $\pm$ 45	0.019*
Albumin at LN diagnosis, mean $\pm$ SD	34 $\pm$ 7	34 $\pm$ 7	30 $\pm$ 6	0.390
C3 at LN diagnosis, mean $\pm$ SD	0.65 $\pm$ 0.26	0.90 $\pm$ 0.35	0.53 $\pm$ 0.30	<0.001*
Positive anti-dsDNA LN diagnosis, N (%)	115 (91)	11 (48)	6 (86)	<0.001*
Use of antimalarials, N (%)	166 (94)	36 (92)	8 (100)	0.688
Use of immunosuppressants, N (%)	163 (94)	33 (87)	8 (100)	0.245
Use of corticosteroids, N (%)	145 (84)	33 (85)	7 (100)	0.511
CKD after LN diagnosis, N (%)	27 (15)	1 (3)	3 (38)	0.018*
ESRD, N (%)	7 (4)	1 (3)	2 (25)	0.016
Deaths, N (%)	14 (8)	2 (5)	0	0.610

uPCR: urinary protein-creatinine ratio, mg/g; y: years; Creatinine presented in mg/dL, eGFR in mL/min/1.73m²; albumin in g/L and C3 in g/L

Note: Baseline data (LN diagnosis) in grey; other data refer to the course of disease

*Significant difference between the proliferative and membranous groups

Figure 1. Kaplan-Meier curves showing cumulative survival free of CKD in patients with PLN, MLN and mixed LN

Conclusion: Our results support previous findings from single-centre studies suggesting that MLN has a different serological profile than PLN, possibly reflecting different pathogenesis. Renal function at one year predicts long-term outcome in LN.

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FRI0166

HIGH FREQUENCY OF KIDNEY STONES AND/OR NEPHROCALCINOSIS IN PRIMARY SJÖGREN'S SYNDROME MIGHT ACCELERATE CHRONIC RENAL DYSFUNCTION DUE TO TUBULOINTERSTITIAL DISEASE

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Background: The renal involvement of primary Sjögren's syndrome (pSS) is characterized by distal renal tubular acidosis (RTA), tubulointerstitial nephritis (TIN), and/or glomerulonephritis [1,2]. Kidney stones and nephrocalcinosis are presumably caused by subclinical distal tubular acidosis, but its clinical significance has not been clarified.

Objectives: This study aimed to clarify the frequency and clinical features of patients with pSS with kidney stones and/or nephrocalcinosis.

Methods: We examined 59 patients with anti SS-A/Ro positive pSS who underwent abdominal computed tomography and/or ultrasound between 1998 and 2019 at Kanazawa University Hospital. We identified 2 groups of patients with primary Sjögren's syndrome: 1) patients with kidney stones and/or nephrocalcinosis (group A: n=19) and 2) those without kidney stones and/or nephrocalcinosis (group B: n=40), and retrospectively analyzed their clinical features.

Results: Kidney stones and/or nephrocalcinosis were confirmed in 19 of 59 (32%) patients with pSS. The patients comprised 4 males and 55 females with an average age of 60 years (range, 30 to 83 years) and mean observation period of 96 months (range 1 to 336 months). Estimated glomerular filtration rate (eGFR) at the time of diagnostic imaging (group A vs group B: 71.5 mL/min/1.73 m² vs 82.8 mL/min/1.73 m²; $p=0.37$) and eGFR at last follow up (group A vs group B: 59.3 mL/min/1.73 m² vs 74.7 mL/min/1.73 m²; $p=0.03$) of group A were lower than those of group B and urinary β 2-microglobulin (group A vs group B: 7222 μ g/mL vs 437 μ g/mL; $p=0.01$) and urinary N-acetyl- β -D-glucosaminidase (group A vs group B: 5.8 U/L vs 3.9 U/L; $p=0.22$) of group A were higher than those of group B, while serum electrolytes (sodium, potassium, chloride, calcium, phosphorus), fractional excretion of calcium (group A vs group B: 1.2% vs 1.5%; $p=0.916$), ESS-DAI (group A vs group B: 7.6 vs 4.3; $p=0.069$), and eGFR decrease rate were not significantly different.

Conclusion: 32% patients with anti SS-A/Ro positive pSS had kidney stones and/or nephrocalcinosis in our cohort and their presence might accelerate chronic renal dysfunction due to tubulointerstitial disease (subclinical RTA or TIN).

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FRI0167

LONG TERM CLINICAL OUTCOME IN SYSTEMIC LUPUS ERYTHEMATOSUS PATIENTS FOLLOWED FOR MORE THAN 20 YEARS IN THREE ITALIAN TERTIARY REFERRAL CENTERS: THE MILAN SYSTEMIC LUPUS ERYTHEMATOSUS CONSORTIUM (SMILE) COHORT

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Background: the prognosis of Systemic lupus Erythematosus (SLE) patients has significantly improved over time, raising the need for more data about disease activity and damage accrual in the long term.

Objectives: to investigate the risk of long term disease activity and to identify viable prognostic markers for disease flares in SLE patients with long standing disease

Methods: data on SLE patients regularly followed at ASST PINI-CTO, Fondazione Ca' Granda Policlinico and Ospedale San Raffaele, Milan (Milan Systemic Lupus Erythematosus Consortium, SMILE, cohort) with disease duration ≥ 20 years, were retrospectively analyzed. Organ involvement as per the British Isles Lupus Assessment Group (BILAG) definitions was recorded along with achievement of clinical and complete remission (CR and CCR: clinical SLEDAI =0, PGA <0.5 and no prednisone or immunosuppression ± negative serology) and lupus low disease activity state (LLDAS) at 15 (T15) and 20 (T20) years of follow up. Damage accrual was estimated according to the SLE International Collaborating Clinics/American College of Rheumatology damage index (SDI).

Results: data from 168 patients (table 1) were available for analysis. Remission (CR+CCR) and LLDAS were achieved in 22% and 61% at T15 and 25% and 71% at T20. LLDAS was not associated with a history of involvement in any BILAG domain, but it was inversely associated with treatment with mycophenolate at any time (50 vs 23% treated vs not treated; p=0.02). SDI>0 was found in 49% patients at T15 and in 71% at T20. LLDAS at T15 was associated with lower flare rates in the following five years (HR= 0.395, 95%, CI=0.239-0.653; Figure, left panel; p<0.001). The risk of flaring for LLDAS was largely comparable to CCR and CR (Figure, middle panel). In the T15-T20 timeframe, 37% of patients had a flare. Patients with both low complement and anti-dsDNA positivity at T15 had an increased risk of flaring compared to serologically inactive patients (HR=2.86, 95%, CI=1.572-5.19; Figure right panel). Flaring patients were more likely to show an increase in SDI from T15 to T20 (37% vs 9% in patients with stable SDI; p<0.001)

Table 1. Demographic, laboratory and clinical characteristics of patients with SLE

Characteristics	(n=168)
Demographic characteristics	
Female, n (%)	150 (89.3)
Age at diagnosis years, median (IQR)	24 (18-32)
Clinical and serological features during 15 years follow up, n (%)	
Musculoskeletal	140 (83)
Mucocutaneous	135 (80)
Constitutional	114 (68)
Haematological	103 (61)
Nephritis	81 (48)
Cardiopulmonary	49 (29)
NPSLE	31 (18)
Positive anti-dsDNA	136 (81)
Hypocomplementemia	136 (81)
Positive antiphospholipid	73 (44)
CR / CCR at T15	13 (8) / 23 (14)
CR / CCR at T20	17 (10) / 25 (15)

Conclusion: LLDAS is common in SLE patients with long disease duration although up to 37% of patients with 15-year disease duration may experience a flare during the following 5 years. The flare risk increases with failure to attain LLDAS at T15 and with active serology. Late flares associate with damage accrual.

References:

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Table 2. therapy during the 20 year follow up

Therapy (ever)	%
Prednisone	83
Hydroxychloroquine	91
Mycophenolate mofetil	33
Azathioprine	50
Methotrexate	23
Cyclophosphamide	36

Fig 1: risk of flare according to disease activity

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FRI0168 ASSOCIATION OF OVERWEIGHT/OBESITY WITH IMPAIRED HEALTH-RELATED QUALITY OF LIFE IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS

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Background: Patients with systemic lupus erythematosus (SLE) experience a considerably impaired health-related quality of life (HRQoL) compared with the general population. Previous literature has implied an association between high body mass index (BMI) and HRQoL diminutions. However, data are scarce and further exploration in large study populations and, importantly, with regard to the clinical significance of this association is needed.

Objectives: The aim of this study was to determine whether overweight and/or obesity were associated with impaired physical and/or mental HRQoL aspects in the SLE population of two large clinical trials.

Methods: We utilised pooled baseline data from the BLISS-52 (NCT00424476) and BLISS-76 (NCT00410384) clinical trials of belimumab (N=1684). Access to data was granted by GlaxoSmithKline. The patients were stratified into four groups based on their body mass index (BMI), according to WHO guidelines. We conducted comparisons between non-overweight versus overweight, and non-obese versus obese SLE patients. HRQoL was self-reported using the Medical Outcomes Study (MOS) short form 36 (SF-36) health survey, the functional assessment of chronic illness therapy (FACIT)-Fatigue scale and the three-level EuroQol- 5 Dimension (EQ-5D) questionnaire. We explored whether the differences in scores were clinically meaningful using previously determined thresholds for minimal clinically important differences (MCIDs). The non-parametric Mann-Whitney U test was used for comparisons between different BMI groups. Linear regression analysis was next applied to test independence in multivariable models, adjusting for age, sex, ethnicity, disease duration, disease activity, organ damage and standard of care treatment.

Results: Forty-four per cent (44%) of the patients had a BMI score over the normal range, and 18% were obese. The overweight group performed worse than the non-overweight with regard to FACIT-Fatigue scores (mean ± standard deviation: 27.7 ± 12.1 vs 32.0 ± 11.3; P<0.001), EQ-5D score (0.70 ± 0.19 vs 0.76 ± 0.18; P<0.001) and all SF-36 subscales and component summaries. The differences were greater than the MCIDs for physical component summary (PCS) scores (36.9 ± 9.3 vs 40.8 ± 9.6; P<0.001), physical functioning (53.3 ± 25.1 vs 63.6 ± 25.1; P<0.001), role physical (48.0 ± 27.1 vs 55.6 ± 26.9; P<0.001), bodily pain (43.8 ± 22.4 vs 52.5 ± 25.1; P<0.001), vitality (39.6 ± 21.7 vs 46.6 ± 21.3; P<0.001), and social functioning scores (55.8 ± 25.2 vs 62.6 ± 25.2; P<0.001). Likewise, obese patients reported worse FACIT-Fatigue scores (25.7 ± 11.9 vs 31.1 ± 11.6; P<0.001), EQ-5D scores (0.68 ± 0.20 vs 0.75 ± 0.18; P<0.001) and clinically important diminutions of HRQoL in all SF-36 items, except for the mental component summary (MCS), role emotional and mental health. In multivariable linear regression analysis, the overweight and obese group showed worse PCS scores (standardised coefficient: β=-0.09; P<0.001 and β=-0.13; P<0.001, respectively) and FACIT-Fatigue scores (β=-0.11; P<0.001 and β=-0.10; P<0.001, respectively), and overweight patients had significantly impaired MCS scores (β=-0.05; P=0.039), irrespective of other factors. High