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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