Conclusion: Cognitive impairment was common in both diseases but the cognitive domains affected were different. Rheumatologists should be aware of these differences when evaluating cognitive dysfunction in SLE and pSS patients.

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AB0411 COGNITIVE IMPAIRMENT IN PRIMARY SJÖGREN'S SYNDROME: A CASE-CONTROL STUDY

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Background: Neurological symptoms are common in primary Sjögren's syndrome (pSS) with a prevalence of 8.5 to 70%, focusing on cognitive impairment, information in pSS is scarce.

Many neuropsychological tests are used to diagnose cognitive impairment. The Montreal Cognitive Assessment (MoCA) is a validated, practical, and reliable instrument for screening mild cognitive impairment.

Objectives: To evaluate the prevalence of cognitive impairment with the MoCA test in pSS and compare it with controls.

Methods: Patients of a rheumatology clinic in Northeastern Mexico were recruited, who met the pSS AECG 2002 or ACR-EULAR 2016 classification criteria. Controls, matched by demographic characteristics were included for comparison. All subjects took the MoCA. The test has a range of 0-30 points, the highest score reflects better cognitive function, and explores 6 cognitive domains (Table 2).

Table 1. Demographic and clinical characteristics

Characteristics	pSS n=51	Control n=51
Age, Mean (SD)	56 (10.4)	54 (14)
Sex		
Female n (%)	47 (92.15)	48 (94)
Male n (%)	4 (7.85)	3 (7.3)
Disease duration (years), mean (SD)	6.38 (6.15)	
ESSPRI mean (SD)	4.94 (2.28)	
Years of education, median (q25-q75)	13 (10-17)	12 (10-15)
Employment, mean (%)	19 (37)	29 (56)

Table 2. MoCA subtest analysis by years of education in pSS and control group.

	Group	N	≤ 9 years of education			p-value	≥ 10 years of education			p-value
			mean, SD ¹	Min-	max		n	mean, SD ¹	Min-	max
MoCA total	pSS	17	25.65 (2.17)	20	29	0.248	46	26.67 (2.27)	20	30
	Control	14	24.36 (3.85)	17	30		36	27.22 (2.24)	21	30
Visuospatial	pSS	17	3.76 (0.9)	1	5	0.505	46	4.17 (1.03)	2	5
	Control	14	4.07 (1.59)	0	5		36	4.58 (0.87)	2	5
Naming	pSS	17	2.82 (0.39)	2	3	0.831	46	2.96 (0.2)	2	3
	Control	14	2.79 (0.57)	1	3		36	2.97 (0.16)	2	3
Delayed recall	pSS	17	3.06 (1.34)	1	5	0.251	46	3.48 (1.31)	0	5
	Control	14	2.43 (1.65)	0	5		36	3.44 (1.68)	0	5
Attention	pSS	17	5 (0.79)	3	6	0.041	46	5.37 (0.77)	4	6
	Control	14	4.29 (1.06)	3	6		36	5.53 (0.56)	4	6
Abstraction	pSS	17	1.71 (0.68)	0	2	0.464	46	1.89 (0.31)	1	2
	Control	14	1.86 (0.36)	1	2		36	2 (0.23)	1	3
Orientation	pSS	17	6 (0)	6	6	0.999	46	5.93 (0.25)	5	6
	Control	14	6 (0)	6	6		36	6 (0)	6	6
Language	pSS	17	2.41 (0.71)	1	3	0.741	46	2.61 (0.57)	1	3
	Control	14	2.5 (0.76)	1	3		36	2.58 (0.84)	0	3

¹SD: Standard deviation

We defined mild cognitive impairment as a score <26 and moderate-severe cognitive impairment as a score <24 as previously determined in Mexican population.

Results: Demographic and clinical characteristics are described in Table 1. Mild cognitive impairment was present in 13 (25.4%) in pSS group versus 14 (27%) in control group. Moderate-severe cognitive impairment was present in 9 (17%) of pSS group versus 8 (15%) in control ($p > 0.05$).

Results of the individual domains and comparison between groups are shown in Table 2. Attention was lower in the pSS group with ≤9 years of education compared to the control group ($p < 0.05$).

Conclusion: We did not find a difference in the prevalence of cognitive impairment, either mild or moderate-severe, in pSS subjects with low disease duration versus controls by MoCA. We found a lower attention score in the pSS group with less than 10 years of education.

The combination of neuropsychological examining and imaging techniques, such as SPECT or brain MRI, seem a more sensitive way to detect cognitive impairment in earlier stages.

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AB0412 URINARY SOLUBLE VCAM-1 IS A USEFUL BIOMARKER OF DISEASE ACTIVITY AND TREATMENT RESPONSE IN LUPUS NEPHRITIS

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Background: The traditional lupus nephritis (LN) biomarkers are not sensitive nor specific enough for detecting ongoing disease activity and early relapse of nephritis and they do not reflect kidney damage nor have prognostic value¹. Urinary biomarkers are directly excreted by the kidney and are easily obtained. They can also differentiate the renal activity of the disease from other organic manifestations more accurately than the serum biomarkers². Vascular cell adhesion molecule-1 (VCAM-1) is involved in the progression of glomerular and tubulointerstitial injury in LN and its soluble form can be easily assessed in urine (uVCAM-1)³. Several studies correlated the uVCAM-1 levels with urine protein-creatinine ratio (UPC), with general disease activity and with active LN³.

Objectives: To assess uVCAM-1 as a biomarker of disease activity and treatment response in LN.

Methods: This prospective study enrolled patients with class III, IV or V LN diagnosed within the last three years and divided them in two groups: with and without active nephritis at the inclusion. The patients with active nephritis were included before they started a new immunosuppressive treatment. Active LN was defined as proteinuria (UPC ≥ 0.5) plus active urinary sediment (hematuria, leukocyturia or cellular hematic/granular casts). At each visit, a urine sample was collected for uVCAM-1 evaluation and the nephritis status was accessed.

Results: Median uVCAM-1 level was elevated in patients with active compared to inactive LN ($p < 0.001$). The ROC curve of uVCAM-1 demonstrated an AUC of 0.84 and a cutoff of 47.2 ng/mgCr yielded a good sensitivity (74.2%) and specificity (74.2%) for the diagnosis of active LN. A significant correlation was found between uVCAM-1 level and renal activity scores and traditional biomarkers of LN (table 1). The level of uVCAM-1 dropped in patients with active LN who went into remission ($p < 0.001$), increased in patients who went into activity ($p = 0.002$) and did not change in patients who remained inactive ($p = 0.797$) (figure 1). The level of uVCAM-1 peaked during the flare of LN ($p < 0.05$) (figure 2).

Table 1. Correlations between urinary soluble VCAM-1 and other LN biomarkers/disease scores

LN biomarkers/disease scores	VCAM-1
SLEDAI-2k	0.597***
Renal SLEDAI	0.569***
Renal SLAM-R	0.470***
Renal SLICC	0.620***
Anti-dsDNA	0.342**
C3	-0.344**
C4	-0.382**
UPC	0.654***

Spearman's correlation coefficients

*p value <0.05; **p value <0.01; ***p value <0.001

C, complement; LN, lupus nephritis; SLEDAI, Systemic Lupus Erythematosus Disease Activity Index; renal SLAM-R, The renal Systemic Lupus Activity Measure revised; renal SLICC, The Systemic Lupus International Collaborating Clinics renal activity/response exercise; UPC, urine protein-creatinine ratio.

Conclusion: The uVCAM-1 is a reliable biomarker that reflects renal disease activity and is useful for monitoring individual patients with lupus nephritis over time.

Figure 1. Urinary soluble VCAM-1 levels according to lupus nephritis status. Active LN was defined as proteinuria (UPC≥0.5) plus active urinary sediment (hematuria, leukocyturia or cellular hematic/granular casts).

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Figure 2. Urinary soluble VCAM-1 levels at different time points relative to a lupus nephritis flare. The levels of uVCAM-1 of seven lupus nephritis patients were evaluated 8 and 4 months before and after a flare, including at the time of the flare itself (time point 0). The number of patients who contributed at each moment was informed. Graph represents median and interquartile range. *p<0.05 compared with level at flare.

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AB0413

INVESTIGATION OF THE ASSOCIATION OF CARDIOVASCULAR EVENTS AND ANTI-SS-A ANTIBODIES AS RISK OF DEVELOPMENT IN PATIENTS WITH LUPUS NEPHRITIS FROM THE LUNA REGISTRY: A CROSS-SECTIONAL STUDY

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Background: Cardiovascular disease(CVD) has been identified as a major cause of morbidity and mortality in patients with lupus nephritis(LN)^{1,2}. There is a clear causal relationship between the onset of neonatal lupus (cardiac complications) and SS-A antibodies^{3,4}, but no association has been reported in adults. In recent years, there have been reports from overseas that suggest the association between CVD and anti-SS-A antibody in adult systemic lupus erythematosus (SLE) patients^{5,6}. So far, no studies have not been reported to evaluate the relationship between anti-SS-A antibody and the risk of developing CVD in LN in a large cohort of patients with SLE in Japan.

Objectives: The aim of this study was to evaluate the association between anti-SS-A antibody and the risk of developing CVD in LN patients using a multicenter registration study [Lupus registry of nationwide institution (LUNA)] in Japan.

Methods: We identified 931 patients diagnosed with SLE in the Lupus registry of nationwide institution (LUNA), and further identified 275 LN patients with known the presence or absence of both development of CVD and presence of anti-SS-A antibody. We defined the exposure factor as anti-SS-A antibody, and the outcome as CVD. SLENA-SLEDAI score (at diagnosis), eGFR <60%, HbA1c, BMI, and steroid pulse treatment history were used as confounding factors and we analyzed using logistic regression analysis.

Results: We found 68 patients (24.7%) complicated with CVD, including pericarditis (7.3%), cerebrovascular disorder (6.2%), peripheral Arterial Disease (6.2%), Ischemic heart disease (2.9%), venous thromboembolism (2.9%), pulmonary hypertension (1.5%), valvular heart disease (1.1%), and cardiomyopathy (0.4%). In univariate analysis, there was no significant difference in the occurrence of CVD depending on the presence or absence of anti-SS-A antibody (p = 0.32), and the results of multivariate analysis showed no significant difference in anti-SS-A antibody [p = 0.23, odds: 0.41, 95% confidence interval (0.09-1.89)].

Conclusion: The association between anti-SS-A antibody and the development of CVD in LN patients in Japan has not been identified.

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AB0414

ESSPRI COMPONENTS AND SALIVARY FLOW RATE ARE RELATED TO DAILY ACTIVITY IMPAIRMENT IN PATIENTS WITH PRIMARY SJÖGREN'S SYNDROME

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