

AB0539

**DEPRESSION, SELF-ESTEEM AND QUALITY OF LIFE IN SJÖGREN'S SYNDROME**

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**Background:** Sjögren's syndrome (SS) is a connective tissue disease characterized by a triad of fatigue, pain and dryness. SS is a chronic condition with important repercussions on functioning and a source of significant suffering. Therefore, the assessment of depressive symptoms, self-esteem and quality of life (QOL) is important in the clinical and research context.

**Objectives:** We aimed to describe the clinical picture, depression, QOL and self-esteem of patients followed for SS and to study their association with disease activity.

**Methods:** This is a descriptive and analytical cross-sectional study including 42 patients with SS diagnosed based on the criteria of the American European Consensus Group of 2002 (AECG). Depression was evaluated by the Beck 13 scale, QOL by the SF-36 and self-esteem by the Rosenberg scale. Disease activity was assessed by EULAR Sjögren Syndrome Disease Activity Index (ESSDAI).

**Results:** The average age was 54.5 ± 15.67 years and the sex ratio was 0.2. Assessment of disease activity of these patients showed an average activity score of 8.23 ± 6.39 with low activity in 16 patients (38.1%), moderate activity in 18 patients (42.9%) and severe activity in 8 patients (19%). The QOL measured by SF-36 was impaired in all areas with averages ranging from 39.76 for physical limit to 66.28 for emotional limit. Depression score evaluated by the Beck scale was 8.42 ± 7.05 on average with 59.5% of the patients having depression. The self-esteem score evaluated by Rosenberg scale was 32.8 ± 5.7 on average. Self-esteem was rated as low to very low in 30.95% of patients.

Disease activity score was correlated to Beck's depression score ( $r = 0.545$ ;  $p = 0.001$ ); and Rosenberg's self-esteem score ( $r = -0.51$ ;  $p = 0.001$ ). Likewise, we found a correlation between the SF-36 score and Beck's depression score; the SF-36 score and Rosenberg's self-esteem score ( $p < 0.05$ ).

**Conclusion:** As QOL, self-esteem and depression were correlated to disease activity, taking into account these aspects in SS can improve the overall management of the disease.

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AB0540

**INTERRELATION OF HYPERURICEMIA AND BURDEN OF COMORBIDITIES IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS**

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**Background:** Comorbidities play an important role in the course and prognosis of systemic lupus erythematosus (SLE) [1]. Hyperuricemia (HP) could cause an increased burden of comorbidities in SLE patients (pts).

**Objectives:** To evaluate the interrelation between HP and comorbidities in pts with SLE.

**Methods:** We performed retrospective analysis of the data from 191 SLE pts that were collected in electronic Saint-Petersburg rheumatological register in a period from 01 Jan 2009 until 31 Dec 2020. In analysis were involved data of 85 SLE patients with hyperuricemia (serum uric acid level >360 µmol/l in 3 reports) (group 1) and of 106 SLE pts with normal uric acid level in 3 laboratory reports (≤360 µmol/l) – group 2. Pts with rheumatological diseases other than SLE, with gout, active infections, oncological diseases, with glomerular filtration rate <30 ml/min\*1.73 m<sup>2</sup> and other secondary reasons of HP were excluded. The clinical and laboratory data, presence of comorbidities, Charlson Comorbidity Index, SELENA-SLEDAI were analyzed. SPSS2020 was used to Statistics. Local ethics committee approved the study.

**Results:** SLE pts with and without HP were matched in age, sex and disease activity characteristics, Table 1 ( $p \geq 0.05$ ). Uric acids' level did not correlate with activity of SLE (SELENA-SLEDAI), Spearman'  $r = 0.06$ ,  $p \geq 0.05$ . Hypertension, obesity, hypercholesterolemia, diabetes mellitus, lower renal function were more common in SLE patients with HP than in SLE patients without HP, Table 1. Charlson comorbidity index was higher in SLE pts with HP than in SLE pts without HP ( $p < 0.05$ ).

**Table 1. Clinical characteristics and occurrence of comorbidities in SLE pts.**

Patients and Disease' characteristics	Group 1 (n=85)	Group 2 (n=106)	P-value
Age, years, mean ± SD	47.05±13.51	44.84±12.11	≥0.05
Female, n (%)	69 (81.18)	95 (89.62)	≥0.05
SELENA-SLEDAI, mean ± SD	7.33±5.03	6.15±3.92	≥0.05
SLE duration, years, mean ± SD	4.73±5.01	0.97±2.15	<0.05
Serum uric acid, µmol/l, mean ± SD	434.6±63.16	238.38±59.36	<0.05
Hypertension, n (%)	52 (61.18)	30 (28.3)	<0.05
Obesity, n (%)	15 (17.65)	5 (4.72)	<0.05
Hypercholesterolemia, n (%)	54 (63.53)	34 (32.08)	<0.05
Diabetes mellitus, n (%)	14 (16.47)	3 (2.83)	<0.05
Glomerular filtration rate, ml/min*1.73 m <sup>2</sup> , mean ± SD	80.29±28.35	95.81±23.38	<0.05

SD - standard deviation

**Conclusion:** Elevated serum uric acid levels in SLE patients are associated with a higher incidence of hypertension, hypercholesterolemia, obesity, diabetes mellitus, decreased glomerular filtration rate, with a higher Charlson comorbidity index, but not with disease activity.

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AB0541

**NEUROPSYCHIATRIC MANIFESTATIONS IN SYSTEMIC LUPUS ERYTHEMATOSUS: PREVALENCE, MANAGEMENT AND ASSOCIATED FACTORS**

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**Background:** Neuropsychiatric involvement is one of the major causes of morbidity and mortality in systemic lupus erythematosus (SLE). Its prevalence varies in the literature because of its clinical polymorphism.

**Objectives:** The aim of our study is to evaluate the clinical, biological, immunological and therapeutic characteristics of neuropsychiatric complications and to analyze the associated factors.

**Methods:** This is a retrospective descriptive study including patients followed for SLE (ACR 1997/ SLICC 2012) during the period between 2010 and 2021 at the Rheumatology and Internal Medicine departments in Mahdia, Tunisia.

**Results:** Our study included 82 SLE patients among whom 29 had neuropsychiatric manifestations (NPM) with a frequency of 35.4%. They were 28 women and 1 man. Their mean age was 52.41 ± 18.11 years [21 - 85]. The mean disease duration was 2.4 ± 3 years [15 days-15 years]. The mean

SLEDAI was  $9.3 \pm 7.85$  [0-32]. NPM were inaugural of SLE in 14 cases (17%). Central neurological manifestations consisted of cerebral vasculitis ( $n=11$ ), epileptic seizures ( $n=1$ ), ischemic stroke ( $n=1$ ), aseptic meningitis ( $n=1$ ) and transverse myelitis ( $n=1$ ). Peripheral neurological manifestations of mono/polyneuropathy were found in 6 cases. Cranial nerves involvement was described in 2 cases (Optic neuropathy and vestibular nerve damage). Psychiatric manifestations were present in 7 cases (25%): Depression in 4 cases and psychosis in 3 cases. The most frequent associated clinical manifestations were: musculoskeletal (100%), dermatological (89.7%), hematological (72.5%) and cardiovascular (24.1%). Biological findings included: leucopenia (31%), lymphopenia (44.8%), anemia (44.8%) and biological inflammatory syndrome (41.4%). The mean sedimentation rate (ESR) was  $53.75 \pm 34$  mm [2-130]. The mean C-reactive Protein was  $13.6 \pm 27.8$  mg/dL [0-130]. Anti-nuclear antibodies and anti-DNA were positive in 100% and 41.4% of cases, respectively. Anti-Sm antibodies were positive in 13.5%, anti-SSA in 24.1% and anti-SSB in 13.8% of cases. Antiphospholipid was positive in 8 patients. C3 and C4 consumption was found in 20.7% and 27.6% of cases, respectively. Brain magnetic resonance imaging showed T2 white matter hyperintensities, particularly in the periventricular area, in 8 cases and ischemic brain lesions in 2 cases. The treatment of NPM required: Glucocorticoids (75.9%), Azathioprine (AZT) (24.1%), Cyclophosphamide (13.7%), Mycophenolate Mofetil (3.4%), anticoagulants and antiplatelet agents (40%). The evolution was favorable for our patients except for one patient who developed AZT-induced hepatotoxicity. Statistical analysis showed that NPM were significantly correlated with older age ( $p=0.003$ ), hematological involvement ( $p=0.02$ ), lymphopenia ( $p=0.004$ ), ESR ( $p=0.01$ ), anti-Sm antibodies ( $p=0.03$ ) and SLEDAI score ( $p=0.008$ ).

**Conclusion:** NPM during SLE are a serious complication of the disease. Our study shows the frequency and variety of neuropsychiatric presentations during SLE. They must be systematically sought, especially if the disease is active, with older patients and anti-Sm antibodies positivity.

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AB0542

#### ANTIPHOSPHOLIPID ANTIBODY CARRIERS WITH THROMBOCYTOPENIA COULD BE AN INDEPENDENT PHENOTYPE OF PRIMARY ANTIPHOSPHOLIPID SYNDROME

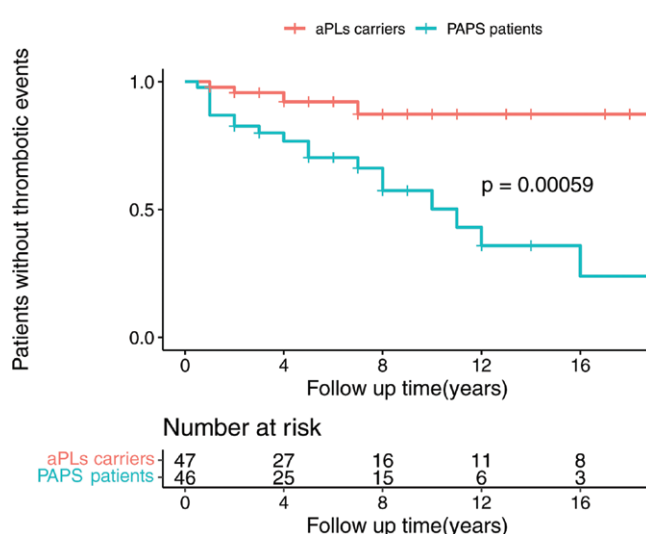
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**Background:** Among patients with immune thrombocytopenic (ITP), 10-20% of them were found with positive antiphospholipid antibodies (aPLs) but without typical clinical manifestations of antiphospholipid syndrome (APS), especially thrombotic events<sup>1</sup>.

**Objectives:** To compare the clinical characteristics and prognosis between aPLs carriers and patients with APS.

**Methods:** This is a single center prospective cohort study consecutively enrolling thrombocytopenic patients with continuous positive aPLs. Patients developing thrombotic events are classified as the APS group. The exclusion criteria are other underlying connective tissue diseases such as lupus and other causes that might manifest as thrombocytopenia, such as virus infection, hypersplenism, etc.

**Results:** This cohort included 47 thrombocytopenic patients with continuous positive aPLs and 55 with diagnosed primary APS. The proportion of thrombotic high risk demographic characteristics including smoking, hypertension, and higher level of homocysteine are higher in the APS group ( $p = 0.03$ ,  $0.04$ ,  $0.03$ , respectively). The prevalence of nephropathy was significantly higher in patients with diagnosed APS [0 vs 7 (12.7%),  $p = 0.01$ ]. Laboratory results and antibody profiles are presented in Table 1. The platelet count of aPLs carriers at admission was lower than APS patients [ $26 \times 10^9/L$  ( $9 \times 10^9/L$ ,  $46 \times 10^9/L$ ) vs  $64 \times 10^9/L$  ( $24 \times 10^9/L$ ,  $89 \times 10^9/L$ ),  $p = 0.0002$ ]. The proportion of positive anti- $\beta_2$ -glycoprotein I, anticardiolipin and lupus anticoagulant separately was similar, but triple aPLs positivity is more common in primary APS patients with thrombocytopenia [24 (51.1%) vs 40 (72.7%),  $p = 0.04$ ]. There is no significant differences over the complement levels between the two groups [ $p = 0.2$  for low complement 3 (C3),  $p = 0.8$  for low C4]. Regarding the treatment response, the complete response (CR) rate is similar between aPLs carriers and primary APS patients with thrombocytopenia ( $p = 0.2$ ). Nonetheless, the proportion of response, no response and relapse differed significantly between the two groups [13 (27.7%) vs 4 (7.3%),  $p < 0.0001$ ; 5 (10.6%) vs 8 (14.5%),  $p < 0.0001$ ; 5 (10.6%) vs 8 (14.5%),  $p < 0.0001$ , respectively]. In Kaplan-Meier analysis (Figure 1), primary APS patients had significantly more thrombotic events than aPLs carriers ( $p = 0.0006$ ). aPLs carriers with thrombocytopenia share similar clinical manifestations with primary APS patients but hardly develop thrombotic events.



**Figure 1.** Kaplan-Meier analysis of thrombotic events. aPLs: antiphospholipid antibodies; PAPS: primary antiphospholipid syndrome.

**Table 1.** Baseline characteristics of laboratory results and antibody profiles.

Characteristics	aPLs carriers (n=47)	Primary APS with thrombocytopenia (n=55)	p-value
Laboratory results, median (Q1, Q3)			
Platelet count ( $\times 10^9/L$ )	26 (9, 46)	64 (24, 89)	0.0002*
hsCRP (mg/L)	1.39 (0.475, 2.845)	1.08 (0.41, 2.565)	0.8
ESR (mm/h)	5.5 (2.75, 10.75)	7.5 (5, 14.25)	0.06
LDH (U/L)	221 (176, 252)	228 (184.8, 282)	0.3
Low C3, n (%)	3 (6.4)	9 (16.4)	0.2
Low C4, n (%)	5 (10.6)	8 (14.5)	0.8
Antibody profiles, n (%)			
Positive aCL	45 (95.7)	33 (69.1)	0.3
Positive Anti- $\beta_2$ GP I	38 (80.9)	48 (87.3)	0.5
Positive LA	35 (74.5)	50 (90.9)	0.05
Triple positivity	24 (51.1)	40 (72.7)	0.04*
Positive Coombs	6 (12.8)	10 (18.2)	0.4

\* $p < 0.05$ , statistically significant. aPLs: antiphospholipid antibodies; APS: antiphospholipid syndrome; Q1/3: quantile 1/3; hsCRP: hypersensitive C-reactive protein; ESR: erythrocyte sedimentation rate; LDH: lactate dehydrogenase; C3/4: complement 3/4; aCL: anticardiolipin; GP: glycoprotein; LA: lupus anticoagulant.

**Conclusion:** In the absence of other high risk factor for thrombosis, Thrombocytopenia could be an independent and long-lasting clinical phenotype for aPLs carriers.

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AB0543

#### HIGHER LEFT VENTRICULAR MASS INDEX IN PATIENTS WITH LUPUS NEPHRITIS

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**Background:** Systemic lupus erythematosus (SLE) patients have a worse cardiovascular prognosis than the general population. It is estimated that approximately 40% of SLE patients develop lupus nephritis (LN) throughout the evolution of the disease (1). Patients with LN had 8 times more risk of myocardial infarction and 4 times more risk of cardiovascular mortality than SLE patients without LN (2).

**Objectives:** To compare the echocardiographic parameters between SLE patients with and without LN.

**Methods:** This was a cross-sectional study nested of a SLE cohort. We recruited patients with SLE diagnosis, according to the 2019 EULAR/ACR