Table 1. Comparison of comorbidities between Antiphospholipid syndrome (APS) vs. matched Rheumatoid Arthritis (RA) patients and between primary APS (PAPS) or Systemic Lupus Erythematosus-APS (SLE-APS) vs matched RA patients

	APS	RA	OR*	PAPS	RA	OR	SLE-APS	RA	OR
n (%)	326	652		161	322		165	330	
Hypertension	97 (29.8)	136 (21)	1.61 (1.19-2.18)	40 (25)	75 (23.3)	1.09 (0.70-1.69)	57 (34.6)	61 (18.5)	2.33 (1.52-3.56)
Smoking	175 (53.7)	264 (40.5)	1.70 (1.30-2.22)	87 (54)	142 (44)	1.49 (1.02-2.18)	88 (53.3)	122 (37)	1.95 (1.33-2.85)
Hyperlipidemia	79 (24.2)	135 (20.7)	1.23 (0.89-1.68)	40 (24.8)	62 (19.3)	1.39 (0.88-2.18)	39 (23.6)	73 (22)	1.09 (0.70-1.70)
Obesity	48 (20.5)	105 (19.5)	1.06 (0.73-1.56)	20 (17)	51 (19)	0.86 (0.49-1.52)	28 (24)	54 (19.7)	1.28 (0.76-2.15)
Stroke [‡]	66 (20.3)	9 (1.4)	13.8 (6.5-29.1)	36 (22.4)	4 (1.2)	19.9 (6.6-59.9)	30 (18.2)	5 (1.5)	7.8 (2.7-22.6)
Coronary disease [‡]	16 (4.9)	13 (2)	3.14 (1.17-8.45)	2 (1.2)	7 (2.2)	0.46 (0.04-4.77)	14 (8.5)	6 (1.8)	10.9 (2.7-44.3)
Osteoporosis [‡]	66 (20.3)	92 (14)	1.45 (1.01-2.06)	19 (11.8)	42 (13)	0.96 (0.54-1.73)	47 (28.5)	50 (15)	1.91 (1.20-3.05)
Diabetes [‡]	18 (5.5)	58 (9)	0.58 (0.33-1.01)	5 (3)	29 (9)	0.34 (0.13-0.89)	13 (8)	29 (9)	0.88 (0.44-1.79)
COPD [‡]	11 (3.4)	14 (2.2)	1.26 (0.56-2.84)	3 (1.9)	6 (2)	0.96 (0.23-4.0)	8 (5)	8 (2.4)	1.28 (0.44-3.72)
Depression [#]	53 (16.3)	66 (10)	1.70 (1.15-2.53)	23 (14)	30 (9.3)	1.69 (0.93-3.05)	30 (18.2)	36 (10.9)	1.65 (0.96-2.84)
Neoplasms [‡]	14 (4.3)	27 (4.1)	1.05 (0.54-2.06)	5 (3)	12 (3.7)	0.84 (0.28-2.52)	9 (5.5)	15 (4.6)	1.31 (0.55-3.1)

* OR: Odds ratio, crude or adjusted for:

± age, sex, smoking, hypertension, hyperlipidemia, BMI, corticosteroid (Cs) duration

× Cs duration

± smoking, Cs duration

± sex, disease duration, Cs duration

± age, disease duration

Conclusion: Comorbidity burden in APS (PAPS and SLE-APS) is comparable or even higher to that in RA, entailing a high level of diligence for CV risk prevention, awareness for depression and corticosteroid exposure minimization.

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FRI0148 SEROLOGICAL PROFILES IN PRIMARY SJÖGREN SYNDROME, SJOGRENSER PROJECT

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Objectives: Primary Sjögren's Syndrome (pSS) is characterized by hyperactivity of the B cell, with the consequent production of autoantibodies. The presence of anti-Ro and anti-La has been associated with extraglandular manifestations and lymphoma development. The aim of this study is to evaluate the frequency of different serological profiles in patients of SJOGRENSER registry and to assess whether the combination of antibodies and serological markers are associated with the development of systemic disease and the systemic illness and/or the development of systemic disease and complications.

Methods: SJOGRENSER is a transversal multicenter study with pSS patients (patients who meet 2002 pSS classification criteria); it is collected demographic, clinical, serological data and the activity index: ESSDAI and ESSPRI. It was established three serological profiles: 1. ANA/Ro+ with La/RF-; 2. ANA/Ro/La+ with RF-; 3. ANA/Ro/La/RF+. A descriptive analysis was done, with means and standard deviations, frequencies and proportions. Student t for quantitative variables and Chi2 for qualitative ones were performed to evaluate differences between groups, it was considered a p < 0.05 as significant. The association was analyzed by multinomial analysis.

Results: Four hundred and thirty-seven patients were included, the most frequent serological patterns observed were: ANA/Ro/La/RF+ 221 patients (50.5%), ANA/Ro/La+ 61 patients (13.9%) and ANA/Ro+ 60 patients (13.7%). There were only two patients with ANA/La+. The ESSDAI score was significantly associated with the profile ANA/Ro/La/RF+ (6 vs 4, p=0.007) and the group with the ANA/Ro/La+ profile reached an ESSDAI < 5 (79%, p=0.043) (Table 1).

Table 1. Serological markers associated with the development of systemic disease

Involvement	Profile 1 ANA + Anti-Ro + Anti-La - RF - n=60 Nº (%)	Profile 2 ANA + Anti-Ro + Anti-La + RF - n= 61 Nº (%)	Profile3 ANA + Anti-Ro + Anti-La + RF + n= 221 Nº (%)	p
ESSDAI index	4	4	6	0.007
ESSDAI < 5	44 (73.3)	48 (78.7)	139 (63.2)	0.043
ESSPRI index	6.1	5.3	5.0	0.007
Constitutional syndrome	5 (8.3)	9 (14.8)	51 (23.1)	0.023
Lymphadenopathy	8 (13.3)	9 (14.8)	37 (16.7)	0.790
Glandular	14 (23.3)	16 (26.2)	87 (39.4)	0.024
Articular	49 (81.7)	40 (65.6)	187 (84.6)	0.004
Lung	3 (5.0)	4 (6.6)	29 (13.2)	0.100
Kidney	0 (0)	5 (8.2)	31 (14.1)	0.006
Peripheral nervous system	5 (8.3)	6 (9.8)	18 (8.2)	0.918
Central nervous system	4 (6.7)	7 (11.5)	13 (5.9)	0.320
Hematological	24 (40.0)	26 (42.6)	147 (66.5)	<0.001
Biologic	5 (8.3)	4 (6.6)	26 (11.8)	0.428
Lymphoma	1 (1.7)	2 (3.3)	3 (1.4)	0.598

In contrast to the ESSPRI index was lower significantly associated with the profile ANA/Ro/La/RF+ and higher in the ANA/Ro+ (5 vs 6.1, p=0.007). The presence of antiLa and/or RF to the profile ANA/Ro+ rises the percentage of patients with constitutional syndrome, glandular, kidney and hematological affectation with statistically significant differences. The percentage of patients with articular involvement it was similar in the profiles ANA/Ro+ and ANA/Ro/La/RF+.

The multinomial analysis showed that the profile ANA/Ro/La/RF+ was associated with higher ESSDAI values (OR=1.09 (1.01-1.17)). The hematological involvement was associated with the profiles ANA/Ro/La/RF+ (OR= 2.76 (1.46-5.22)). Higher scoring of ESSPRI was associated with the profile ANA/Ro+ (OR=0.79 (0.69-0.91)).

Conclusion: The most frequent serological profile was ANA/Ro/La/RF+ and it was associated with higher ESSDAI score. The presence of antiLa and/or RF with an ANA/Ro+ increased the presence of systemic involvement. The ANA/Ro+ profile was associated with a higher level of ESSPRI.

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FRI0149 THE CLINICAL FEATURES OF SJÖGREN'S SYNDROME PATIENTS WITH EARLY AND LATE DISEASE ONSET

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Background: Sjögren's syndrome (SS) affects mainly individuals of the 4th or 5th decade of life, although patients with early (≤ 35 years old) or late (≥ 65 years old) disease onset have been described in the literature. The clinical spectrum of the disease extends from mild dryness to severe systemic vasculitis and lymphoproliferative disorders. The phenotypic diversity of SS is defined by many factors, including age, since many parameters related to age may affect the clinical expression of the disease. Few studies have been conducted to study the effect of age on the clinical phenotype of SS, though with limited number of patients. Large and well-defined groups of SS are required to address such questions.

Objectives: To study the clinical phenotype of SS patients with early and late disease onset and to explore the association of age with lymphoma development in a unified multicenter cohort.

Methods: From a total cohort of 1997 consecutive SS patients who fulfill the 2016 EULAR/ACR criteria and are followed up in 5 clinical centers ([Universities of Udine, Pisa and Athens, Harokopio and Ioannina, (UPAH)], those with either early (≤ 35 years) or late (≥ 65 years) disease onset were identified and matched according to gender and disease duration with middle aged controls whose disease onset was at the 4th or 5th decade of life. Glandular manifestations, extra-glandular manifestations, serologic characteristics and histologic features were compared between the 2 age groups and the middle-aged control groups. Statistical analysis for categorical variables was performed by Fisher exact or chi-square tests and for continuous variables with t test or Mann-Whitney accordingly.

Results: Three hundred seventy-nine (19%) SS patients with early and 293 (15%) with late disease onset were identified and compared with 353 and 285 middle aged SS controls respectively. The median disease duration of patients with early onset was 12 years (range: 0-68) and for those with late disease onset was 5 years (range: 0-27). SS patients with early disease onset had statistically significant higher frequency of Raynaud's phenomenon, lymphadenopathy, hypergammaglobulinemia, anti-Ro/SSA, anti-La/SSB, rheumatoid factor, salivary gland enlargement, low C4 complement levels, leukopenia and lymphoma (10.3% vs 5.7%, $p = 0.03$, OR = 1.91, 95% CI: 1.11-3.27) while SS patients with late disease onset had more frequently dry mouth, interstitial lung disease and lymphoma (6.8% vs 2.1%, $p = 0.01$, OR = 3.4, 95% CI: 1.35-1.81).

Conclusion: In a multicenter cohort of 1997 consecutive SS patients, those with early and late disease onset comprise more than one third of the total SS population. Patients with early disease onset, exhibit robust B cell responses with traditional risk factors for lymphoma as opposed to patients with late disease onset. Both age groups have increased lymphoma prevalence but presumably for different reasons, since late onset patients lack classical predictors of lymphoma. Therefore, these predictors deserve further study in different disease subsets.

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FRI0150

USEFULNESS OF SALIVARY GLAND ULTRASONOGRAPHY IN THE ASSESSMENT OF SALIVARY GLAND ACTIVITY AND DAMAGE IN PRIMARY SJÖGREN'S SYNDROME: A SINGLE CENTER EXPERIENCE

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Background: Salivary gland ultrasonography (SGUS) has an emerging role in the diagnosis of primary Sjögren's syndrome (pSS); however, it is still an open issue whether distinct sonographic abnormalities may indicate reversible glandular activity or irreversible disease-induced damage.

Objectives: To assess the association between SGUS abnormalities, salivary gland disease activity and loss of function in pSS patients over a long-term follow-up.

Methods: Patients with pSS fulfilling the AECG 2002 criteria were included in this observational study. Both parotid and submandibular glands were examined at the time of the study inclusion and during the follow-up. SGUS findings (i.e gland size, echogenicity, homogeneity, hyperechoic bands, number and location of the hypoechoic/anechoic areas, number of lymph nodes, calcification, posterior border visibility) were defined according to previous studies and monitored over the time. Patients demographics, clinical, histological and laboratory data were routinely collected. ESSDAI and ESSPRI were used to assess disease activity and PROs.

Results: We included 419 (402 F:17M) pSS patients: 206/419 at the diagnosis and 213/419 with a median disease duration of 7 (IQR 4-11) years. SGUS

examination was repeated in 81/206 and in 108/213 patients, after a median follow-up of 30 (IQR 12-42) months, respectively. Noteworthy, 18/419 pSS patients were treated with rituximab (RTX) during the study period. The overall SGUS score correlated directly with the minor salivary focus score ($r = 0.366$, $p = 0.000$) and with the ESSDAI ($r = 0.482$, $p = 0.000$); the parotid inhomogeneity score correlated directly with the glandular domain of the ESSDAI ($r = 0.530$, $p = 0.000$). The unstimulated salivary flow rate (USFR) correlated inversely with the overall SGUS score ($r = -0.257$, $p = 0.000$) and with the presence of hyperechoic bands in both the parotid ($r = -0.210$, $p = 0.03$) and the submandibular glands ($r = -0.316$, $p = 0.000$). When compared to patients with an established pSS, newly diagnosed patients presented less frequently a gross inhomogeneity in their parotid glands (30/206, 14.6% vs 53/213, 24.9%, $p = 0.01$) and less hyperechoic bands in both their parotid (33/206, 16% vs 61/213, 29%, $p = 0.001$) and submandibular glands (53/206, 26% vs 110/213, 52%, $p = 0.001$). However, over a median 30 month-follow-up we did not observe any significant change neither in the number of hypo-anechoic areas nor in the inhomogeneity score in both newly diagnosed patients and in those with an established disease. Out of the 18 pSS patients treated with RTX, 14 (78%) presented at the baseline a moderate to gross inhomogeneity in their glands: no changes in the number of hypo-anechoic areas were observed also in these patients with the exception of a variation in the number of intra-parotid lymph nodes.

Conclusion: SGUS abnormalities appeared to be associated to both salivary gland disease activity and damage. Namely, the presence of hyperechoic bands significantly correlated with salivary loss function. Diffuse-scattered hypoechoic areas did not change over a median 30-month follow-up indicating that additional studies are required to better elucidate the correlation between SGUS abnormalities and the corresponding histopathologic lesions.

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FRI0151

COMORBIDITY IN INCIDENT SYSTEMIC LUPUS ERYTHEMATOSUS: ANALYSIS OF A CLAIMS DATA COHORT

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Background: Systemic lupus erythematosus (SLE) targets many tissues including heart, kidney, bone and brain, causing a variety of comorbidities. Epidemiological studies assessing comorbidity in SLE facilitate earlier detection to limit its progression.

Objectives: To analyze comorbidities in individuals with an incident diagnosis of systemic lupus erythematosus (SLE) in claims data in comparison to matched controls.

Methods: Individuals with ≥ 2 outpatient or 1 inpatient SLE diagnosis (M32.1, M32.8, M32.9, ICD10) in 2016-2018 and no diagnosis in 2015 or 2014 were identified in a German statutory health insurance fund covering 7.2 million people > 18 years old. Comorbidities defined by the Elixhauser index, medical prescriptions, hospitalization and sick leave were analyzed in the year prior to diagnosis and during a two year follow-up in comparison to age and sex-matched controls without related autoimmune diseases (1:10). To account for detection bias, a second age and sex-matched control group with incident diabetes mellitus (E10-14) was drawn (1:5). To consider diagnostic uncertainty, two sensitivity analyses were performed, namely (i) excluding cases with ICD diagnosis of Sjögren's syndrome, mixed connective tissue disease, systemic sclerosis and tubulointerstitial nephritis and (ii) excluding cases without any prescription of antimalarials.

Results: A total of 559 persons were included (87% female, mean age 55 years). The mean Elixhauser score increased from 1.1 in 2015 to 4.2 in the index year and to 4.5 in the 2nd year after diagnosis. Hypertension (47%), depression (31%), hyperlipidemia (25%), osteoarthritis (25%) and osteoporosis (21%) were among the most frequent comorbidities in the index year. Drug prescriptions (mean 9.7, $\Delta +6.3$), among these glucocorticoids (56%), HCQ (50%) and azathioprine (13%), and hospitalization (40%, $\Delta +26\%$) increased in the 1st year after the SLE diagnosis. The increase in comorbidity diagnoses was low in controls without related autoimmune disease, while controls with incident diabetes showed a more pronounced increase in cardiovascular risk factors and a lesser increase in osteoporosis and cerebrovascular disease (figure 1). Comparable results were found in the sensitivity analyses, where patients with HCQ were younger and had less general comorbidity.

Figure 1. Comorbidity claims in persons with incident systemic lupus erythematosus in 2016 (N=559, M32.1,8,9) and age and sex-matched controls (N=5,590 without related autoimmune disease, N=2,795 with incident diabetes, E10-14)