and median (IQR) previous maximum dose of prednisolone in patients with and without CKD ($\geq$ CKD stage 3) were 56 (46.5-66) and 41 (32-50), 191 (89%) and 615 (88%), and 40 (30-60) and 40 (30-55) mg/day, respectively. Bisphosphonate was administered in 388 (42%) patients, vitamin D supplements in 448 (49%), Ca supplements in 36 (4%), denosumab in 20 (2%) and teriparatide in 14 (2%), respectively. Of enrolled patients, any treatment for osteoporosis was not administered in 226 (25%) patients. In spite of more frequent bone damage in patients with CKD compared to those without CKD (15% vs 10%, $p=0.036$), treatment status did not differ between patients with and without CKD (bisphosphonate: 41% vs 46%, $p=0.29$; vitamin D supplements: 50% vs 44%, $p=0.14$).

Conclusion: About a quarter of patients with SLE did not take any treatment for osteoporosis. Treatment for osteoporosis might be strengthened to prevent bone damage in SLE patients with CKD.

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AB0388

IMPROVEMENT OF PATIENT-REPORTED OUTCOMES IN PATIENTS WITH PRIMARY SJÖGREN'S SYNDROME UNDERGOING CPAP-TREATMENT

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Background: Fatigue is a frequent symptom in primary Sjögren's Syndrome (pSS) determining health-related quality of life in many patients. Earlier studies could show that sleep apnea syndrome (SAS) is associated with patient-reported outcomes in pSS-patients [1].

Objectives: To investigate whether treatment of an underlying SAS improves disease activity in pSS-patients.

Methods: 14 female pSS-patients from our rheumatology outpatient clinic were enrolled and screened for SAS; continuous positive airway pressure (CPAP)-treatment was initiated in patients with a sleep apnea/hypopnea index $>5/h$ and a respiratory disturbance index $>15/h$ sleeping time (pSS treated with CPAP: pSS+; pSS untreated: pSS-). As controls, 11 SAS-patients (4 female) scheduled for initiation of CPAP-treatment were recruited from pulmonology department. In pSS, ESSDAI, ESSPRI, pain and patient global health on a visual analogue scale from 0-100mm were recorded. In both groups CRP, ESR, depression (PHQ-9), fatigue (FACIT), daytime sleepiness (EPS), and salivary gland ultrasound (SGUS) of the parotid and submandibular glands (PG; SMG) were obtained [2]. Data were collected at baseline (BL) and month 3 (M3). A p-value <0.05 was deemed significant.

Results: 6 pSS-patients were diagnosed with SAS and underwent CPAP-initiation, as well as all SAS-patients. Table 1 shows means $\pm$ SD of all assessments. EPS indicated normal sleep propensity in all groups at both time points. Fatigue was severe in all groups at BL. Numerically in SAS and pSS+, fatigue and depression trended towards improvement between BL and M3. Depression scores were moderate in all groups at BL; in SAS, PHQ-9 score showed a trend towards improvement at M3. SGUS grades 0 or 1 (i.e. normal or unspecific SGUS pattern) were found in 46.43% of PG and SMG at BL, in 40.39% at M3 in pSS, and in 100% in SAS at both time points. Grades 2 or 3 (pSS-typical US-pattern or gland destruction) were exclusively found in pSS at BL (53.57%) and M3 (59.62%). pSS-SGUS scores worsened irrespective of CPAP-treatment.

Conclusion: Even though this is only preliminary data of a small fraction of patients who completed M3 so far, there is already a clear tendency towards reduced fatigue, inflammatory markers and depression both in the CPAP-treated pSS and in the SAS-patients compared to the non-CPAP-treated pSS-patients.

References:

[1] Usmani ZM Sleep Medicine 2012; 2. Jousse-Joulin S Ann Rheum Dis. 2019

		Baseline (mean $\pm$ SD)	Month 3 (mean $\pm$ SD)	p-Values
CRP	pSS+	4.58 $\pm$ 2.98	2.79 $\pm$ 0.56*	*p=0.014
	pSS-	6.53 $\pm$ 5.07	4.08 $\pm$ 3.69	
	SAS	4.27 $\pm$ 1.80	4.76 $\pm$ 3.50*	
ESR	pSS+	30.50 $\pm$ 21.92	24.75 $\pm$ 10.69*	*p=0.031 ^p=0.013
	pSS-	34.75 $\pm$ 37.92	47.29 $\pm$ 37.82^	
	SAS	20.67 $\pm$ 17.56	11.67 $\pm$ 11.38 [^]	
ESSDAI	pSS+	10.67 $\pm$ 4.97	11.00 $\pm$ 6.08	*p=0.037
	pSS-	11.63 $\pm$ 3.25	9.43 $\pm$ 4.72	
	SAS	6.75 $\pm$ 1.44*	6.47 $\pm$ 1.75	
ESSPRI	pSS+	5.18 $\pm$ 1.32*	5.46 $\pm$ 1.77	*p=0.037
	pSS-	5.58 $\pm$ 2.61°	54.17 $\pm$ 24.60	
	SAS	27.50 $\pm$ 26.33°	34.25 $\pm$ 26.87	
VAS pain	pSS+	58.50 $\pm$ 11.67	56.00 $\pm$ 19.54	^p=0.059
	pSS-	52.88 $\pm$ 30.86	53.00 $\pm$ 23.05	
	SAS	24.00 $\pm$ 6.03	29.00 $\pm$ 10.56 [^]	
FACIT	pSS+	27.88 $\pm$ 14.75	30.50 $\pm$ 12.54	^p=0.054
	pSS-	29.36 $\pm$ 10.46 [^]	37.82 $\pm$ 10.73 [^]	
	SAS	11.50 $\pm$ 3.02	9.33 $\pm$ 3.88 [^]	
PHQ-9	pSS+	10.86 $\pm$ 5.05	9.00 $\pm$ 2.51@	*p=0.059; @p=0.068; °p=0.087
	pSS-	10.11 $\pm$ 5.33x	6.55 $\pm$ 7.5x [^]	
	SAS			

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AB0389

GLUCOCORTICOID WITHDRAWAL IN SYSTEMIC LUPUS ERYTHEMATOSUS: ANALYSIS OF 750 SLE PATIENTS FROM THE RUSSIAN AND KYRGYZ COHORTS.

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Background:

Objectives: Glucocorticoids (GCs) have been the mainstream of systemic lupus erythematosus (SLE) treatment for the last 70 years. GCs allow to achieve effective control over SLE activity quite rapidly – both in mild and severe disease. The majority of SLE patients have received GC therapy; in some cohorts up to 80% - 100% of patients continue on low maintenance GCs doses < 7.5 mg/day for many years, perhaps some of them are treated indefinitely. It is clear that cumulative GCs dose is responsible for adverse effects. But it remains still unclear whether GCs should be continued indefinitely and, if not, when and how this treatment should be discontinued. On the other hand, treat-to-target SLE recommendations suggest GC withdrawal where possible as an important target of the treatment plan.

Methods: Patients who attempted GCs withdrawal were included in the Eurasian SLE RENAISSANCE cohort. A retrospective analysis of 350 patients from Russia and 400 patients from Kyrgyzstan was conducted. The following information was assessed during withdrawal attempts: SLE duration, disease activity at the onset and initiation of GCs dose reduction, therapy at SLE onset, the duration of the last flare, activity and therapy at the end of FUP, and duration of remission after GCs withdrawal. Definitions of remission were applied to GCs withdrawal in line with European consensus criteria.

Results: Out of 750 patients with a follow-up of about 6 years (IQR 1-23), GCs withdrawal due to persistent remission was documented in 15 patients (2.0%). In 14 out of these 15, SLE onset was associated with high disease activity based on SLEDAI 2K > 8 . High level of anti-DNA and a decrease in C3 \ C4 complement were present in 12, 4 patients had nephritis with preserved renal function, 4 patients manifested signs of CNS damage (convulsions, headaches, sleep disturbances, memory issues, neuropathy, hallucinations), and another 5 had vasculitis. 10 patients were administered pulse therapy with 3g methylprednisolone due to high disease activity. Initiation of GCs dose reduction with intent to discontinue in 7 patients was substantiated by prolonged clinical remission, meanwhile SLE duration in this group varied from 2 to 20 years, and duration of the last flare - from 6 to 165 months. Acute onset with high disease activity reaching 12-23 scores by SLEDAI 2 K was documented in 8 cases of early SLE with disease duration varying from 1.5 to 6 months. These patients were prescribed the most aggressive induction therapy, including cascade plasma filtration in combination with pulse therapy, cyclophosphamide and Rituximab at 1g dose. Remission (SLEDAI2K 0-2 scores) was achieved 4-6-8 months later after termination of aggressive induction therapy. The duration of remission after GCs withdrawal in all 15 patients ranged from 3.5 to 240 months. In 8 patients with aggressive induction therapy, remission lasted from 18 to 240 months. In 2