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

M. Seng¹, B. C. Frye², L. Vollmer², F. Kollert³, R. Voll⁴, S. Finzel⁵. ¹Medical Centre - University of Freiburg, Faculty of Medicine, Department of Rheumatology and Clinical Immunology, Medical Centre, 79106, Germany; ²Medical Centre - University of Freiburg, Faculty of Medicine, Department of Pneumology, Freiburg, Germany; ³University Hospital Bern, Department of Rheumatology, Immunology, and Allergology, Bern, Switzerland; ⁴Medical Centre - University of Freiburg, Faculty of Medicine, Department of Rheumatology and Clinical Immunology, Medical Centre, Freiburg im Breisgau, Germany; ⁵Medical Centre - University of Freiburg, Faculty of Medicine, Department of Rheumatology and Clinical Immunology, Medical Centre, Freiburg, Germany

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.

S. Solovyev¹, E. Aseeva¹, G. Koilubaeva², E. Nasonov¹, A. Lila¹, N. Nikishina¹. ¹V.A. Nasonova Research Institute of Rheumatology, Moscow, Russia, Intensive Care Department, Moscow, Russian Federation; ²National Center for Cardiology and Internal Medicine named after Academician M. Mirrakhimov, Rheumatology Department, Bishkek, Kyrgyzstan

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

remaining patients, remission lasted for 9 and 16 months after GCs withdrawal. Each flare required intake of low prednisolone doses for 3-4 weeks.

Conclusion: GCs withdrawal is an achievable goal in SLE and may be attempted after a long-term remission, and possibly after aggressive intensive care in the early stages of SLE.

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AB0390

ASSOCIATION BETWEEN INITIAL SERUM "TUMOR NECROSIS FACTOR-LIKE WEAK INDUCER OF APOPTOSIS" (TWEAK) LEVEL AND TREATMENT RESPONSE IN SYSTEMIC LUPUS ERYTHEMATOSUS (SLE) NEPHROPATHY

Z. Wailinn¹, C. Soe², H. M. Win³, ¹Yangon Specialist Hospital (500 Bedded), Rheumatology Department, Yangon, Myanmar, Burma; ²Yangon Specialist Hospital (500 Bedded), Rheumatology Department, Yangon, Myanmar, Burma; ³Yangon General Hospital, Medical Department, Yangon, Myanmar, Burma

Background: SLE is a chronic inflammatory immunologic abnormalities disease which produce a number of antinuclear antibodies. The SLE renal involvement is clinically apparent in approximately 50% patients (Norby et al., 2017). It is very important to introduce the prompt treatment to prevent the permanent end stage renal disease.

Objectives: This study aimed to identify the serum biomarkers that correlate with pretreatment disease activity in patients with SLE nephropathy and predict the treatment outcome so that we may identify the unresponsive cases and switch to the other biologic agents like anti-TWEAK monoclonal antibody in the future.

Methods: This was a hospital-based prospective analytical study conducted from January 2018 to November 2019 in Rheumatology Department, Yangon Specialty Hospital. 88 SLE nephropathy patients with 24-hour urinary protein above 0.5g/day planned to have 6 months course of IV cyclophosphamide were enrolled. The paired serum sample of each patient was analyzed by ELISA twice to get the mean serum TWEAK value. Pretreatment SLE disease activity was assessed by the SLEDAI 2k. After the completion of 6 months of aggressive treatment, the treatment response was assessed by measuring the 24 hour urinary protein.

Results: Among the 88 patients, 63 patients (71.6%) had completed total 6-months course and 25 patients (28.4%) had not completed: 11 patients (12.5%) expired and 2 patients (2.27%) had been changed to other DMARD and 12 patients (13.63%) did not attend the follow up clinic. The mean serum TWEAK level was 856 ± 77 pg/ml in 88 patients. According to the range of serum TWEAK level, most of the patients had serum TWEAK level of 601-900 pg/ml (53.4% of the study population). There was positive correlation between pre-treatment SLEDAI 2k score and pretreatment serum TWEAK level ($r=0.464$ and $P<0.001$). When the SLEDAI 2k score was grouped into mild, moderate, high and very high disease activity, the serum TWEAK level also had positive association with the different levels of disease activity ($p<0.001$). Among 63 treatment completed patients, 55 patients (87.3%) were the treatment responders but 8 patients (12.7%) were treatment non-responders. There was significant difference in the pretreatment SLEDAI 2k in terms of disease activity between treatment responder and treatment non-responder ($p<0.001$). There was significant difference in the pretreatment SLEDAI 2k in terms of reduction in 24-hours urinary protein between treatment responder and treatment non-responder ($p<0.001$). There was no significant difference in the level of pretreatment serum TWEAK level between treatment responders and treatment non responders ($p=1.000$). There was also no significant difference in the pretreatment serum TWEAK level between treatment responders and treatment non-responders in terms of reduction in 24 hours urinary protein ($p=0.804$).

Conclusion: Although the pretreatment serum TWEAK level had a positive correlation with pretreatment disease activity of SLEDAI 2k, it did not reflect the outcome of the responsiveness to the intensive therapy.

References:

[1] Norby, et al (2017) Outcome in biopsy-proven lupus nephritis: evaluation of biopsies from the Norwegian kidney biopsy registry. *Lupus*; 26:88

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AB0391

EFFECT OF HCQ ON LLDAS ACHIEVEMENT IN SLE PATIENTS

R. Wakiya¹, K. Ueda¹, H. Shimada¹, S. Nakashima¹, M. Mahmoud Fahmy Mansour¹, M. Kato¹, T. Miyagi¹, Y. Ushio¹, K. Sugihara¹, T. Kameda¹, H. Dobashi¹, ¹Kagawa University, Division of Hematology, Rheumatology and Respiratory Medicine, Department of Internal Medicine, Kagawa, Japan

Background: HCQ for SLE in Japan has been administered in many cases after approval. Therefore, the effect of additional administration of HCQ on low disease activity of SLE was considered to be clearer.

Objectives: To clarify the effect of HCQ treatment on the control of disease activity in SLE patients.

Methods: All SLE patients with low disease activity (LDA) enrolled in this study started additional HCQ treatment from January 2016. All patients with LDA enrolled in this study started HCQ treatment and had been receiving oral HCQ continuously for at least 3 months without using other immunosuppressive treatments or glucocorticoids. Disease activity was evaluated by SLEDAI, CLASI, and LLDAS, and serum complement values, anti-DNA antibodies, and pro-inflammatory cytokines were analyzed as immunological biomarkers before and after HCQ treatment.

Results: 52 of 100 patients were enrolled in this study (M:F; 4:48, average age; 40.6 ± 13.4). 24 lupus nephritis patients were in sustained remission. 29 patients (56%) achieved LLDAS and 3 patients (6%) achieved clinical remission (CR) before HCQ administration.

Of the 20 patients (38%) who did not achieve LLDAS before HCQ administration, the LLDAS achievement rates at 3, 6, and 12 months after additional HCQ were 47%, 59%, and 81% (including 12.5% of CR achievement rates), respectively. Serum levels of MRP8, MRP14, TNF- α , IL-6, VEGF-A, IL-1 α , MIP-1 α and IL-2 decreased significantly 3 months after additional HCQ treatment. In addition, serum levels of MRP8, MRP14, TNF- α , IL-6 and IL-2 also decreased significantly 3 months after additional HCQ treatment despite achieving LLDAS or CR. The expressions of IFN- α didn't decrease significantly in 9 cases that could be detected. The magnitude of the changes in serum MRP8, MRP14, IL-8 and IL-1 α levels in patients with a history of LN was significantly higher than in those without a history of LN. The magnitude of the reduction in serum MCP-1 levels in patients not achieving LLDAS with a history of LN was significantly higher than in those without a history of LN ($p=0.046$).

The change of CLASI activity score was correlated with the change in serum levels of MRP14 and MCP-1 with univariate analysis (MRP14: $r=-0.41$, $p=0.017$, MCP-1: $r=-0.58$, $p=0.0006$). The change of serum C3 levels had a negative correlation with MCP-1 ($r=-0.33$, $p=0.022$).

The magnitude of the change in serum levels of MRP14, TNF- α , IL-8, MCP-1, MIP-1 α and IL-1 α in patients achieving LLDAS were correlated with the change of CLASI activity score with univariate analysis (MRP14: $r=-0.49$, $p=0.041$, TNF- α : $r=0.74$, $p=0.0038$, IL-1 α : $r=0.66$, $p=0.038$, MIP-1 α : $r=0.63$, $p=0.037$, Figure 1). Moreover, the change of serum C3 and C4 levels in them had a negative correlation with the change of serum MCP-1 levels (Figure 2).

Figure 1. Correlation between change of CLASI activity scores and serum MCP-1 levels in SLE patients with LLDAS (IL-8: $r=0.77$, $p=0.0007$, MCP-1: $r=0.80$, $p=0.0001$).

Figure 2. Correlation between change of serum C3 and C4 levels and serum MCP-1 levels in SLE patients with LLDAS (C3: $r=-0.40$, $p=0.028$, C4: $r=-0.37$, $p=0.047$).

Conclusion: Additional administration of HCQ is useful for cytokine control even in LLDAS-achieved cases, and particularly contributes to the improvement of skin lesion.

In addition, regulation of IL-8 and MCP-1 is important for control of renal lesions of SLE, and more control of the activity of SLE

The effect of HCQ on IL-8 and MCP-1 is related to the control of renal lesions in SLE, so that disease activity of more SLE patients might be more controlled disease activity.