

Disclosure of Interest: None declared
DOI: 10.1136/annrheumdis-2017-eular.3006

AB0508 EFFECT OF ALCOHOL CONSUMPTION AND SMOKING ON DISEASE DAMAGE IN SYSTEMIC LUPUS ERYTHEMATOSUS: DATA FROM KOREAN LUPUS NETWORK (KORNET) REGISTRY

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Objectives: We assessed correlations of smoking habits and alcohol consumption with disease activity or damage in patients with systemic lupus erythematosus (SLE).

Methods: A total of 505 patients with SLE were enrolled in the KORnet lupus Network (KORNET) SLE registry from January 2014 to January 2016. Disease activity and organ damage were measured by the Systemic Lupus Erythematosus Disease Activity Index 2000 (SLEDAI-2K) and the Systemic Lupus International Collaborating Clinics/American College of Rheumatology (SLICC/ACR) damage index, respectively. Multivariate logistic regression analysis was used to analyze associations with cutaneous lesions.

Results: There are no differences in SLEDAI-2K and SLICC/ACR damage indexes according to either smoking status or alcohol consumption. More frequent cutaneous damage was observed in current alcohol drinkers compared to non-current alcohol drinkers ($p=0.020$). Cutaneous damage was significantly associated with alcohol consumption [Odds ratio (OR) 4.048, 95% confidence interval (CI) 1.251 – 12.102, $p=0.020$]. Both low (1–5 glasses/week) and high (≥ 6 glasses/week) amounts of alcohol consumption had a significant impact on cutaneous damage compared to the absence of current alcohol consumption ($p=0.033$ and $p=0.027$, respectively). Pairwise comparison of alcohol consumption and smoking status with cutaneous damage showed that only alcohol consumption was significantly associated with the presence of cutaneous damage, compared to non-current alcohol consumption and non-current smoking (OR 3.513, 95% CI 1.130 – 10.920, $p=0.030$).

Conclusions: Current alcohol consumption, but not smoking, might influence the development of cutaneous damage in patients with SLE.

Disclosure of Interest: None declared
DOI: 10.1136/annrheumdis-2017-eular.3070

AB0509 IDENTIFICATION OF NOVEL BIOMARKERS ASSOCIATED WITH DISEASE ACTIVITY OF PRIMARY SJÖGREN'S SYNDROME AND CLINICAL RESPONSE TO VAY736

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Background: Overexpression of B cell activating factor (BAFF) contributes to the pathogenesis of primary Sjögren's syndrome (pSS) [1]. Treatment of pSS patients with VAY736, an anti-human BAFF receptor mAb, appears promising and was associated with a positive therapeutic effect [2]. Given the complexity and heterogeneity of pSS, there is a need to further identify molecular mechanisms involved in pSS and in response to new therapeutics.

Objectives: To address this question, we assessed a panel of biomarkers in 27 patients from a clinical trial and tested their associations with pSS activity and clinical response to VAY736.

Methods: This study comprised 27 pSS patients treated with a single intravenous dose of VAY736 at 10 mg/kg ($n=12$), 3 mg/kg ($n=6$), or placebo ($n=9$). The disease activity scores included EULAR Sjögren's Syndrome Disease Activity Index (ESSDAI) and Patient Reported Index (ESSPRI), patient's and physician's reported visual analog scales (VAS), Short-Form 36 and Multidimensional Fatigue Inventory (MFI). BAFF and a panel of chemokines in serum and saliva were assessed using immunoassays. Circulating B cells and B cell subsets were measured by flow cytometry. High frequency ultrasound (US) of the parotid and sub-mandibular glands measured gland thickness and quality using a 4 point scoring (de Vita) [3]. Shear wave elastography of the parotid glands was also measured. All biomarkers were measured at baseline (BL) and post-treatment w6, w12, w24. The effect of VAY736 on biomarker levels was assessed by descriptive statistics. Correlations between biomarkers and disease activity scores were calculated at BL and w6, w12, and w24 using levels and relative changes from BL.

Results: In addition to B cell depletion, serum BAFF increase, and improvements in US and elastography measures [2], a subset of serum chemokine tended to be reduced nine weeks after VAY736 treatment. Pooling all 27 patients, salivary BAFF levels correlated with parotid De Vita scores at BL (left: $r=0.75$, right: $r=0.72$, $p<10^{-4}$ for both) and w6 (left: $r=0.72$; right: $r=0.78$, $p<10^{-4}$ for both) but not at later time points. Pooling the 18 VAY736 treated patients, increase in salivary BAFF correlated with decrease in MFI at w6 ($r=-0.83$, $p=3\times 10^{-4}$) and high levels of one of the serum chemokines at BL correlated with decrease in ESSPRI at w24 ($r=-0.76$, $p=3\times 10^{-4}$). In the same patients, the B cell count at BL

correlated with changes in several clinical outcomes at w12: ESSPRI ($r=-0.65$, $p=0.01$), Physician's VAS ($r=-0.6$, $p=0.01$), shear wave ($r=-0.63$, $p=0.02$), and parotid thickness ($r=-0.6$, $p=0.03$).

Conclusions: We identified a set of markers correlated with clinical outcomes in pSS after treatment with VAY736, which have the potential to provide additional insight in pSS and treatment-modifying effects. Further large-scale studies are necessary to confirm the value of these markers.

References:

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Disclosure of Interest: J. Doucet Employee of: Novartis AG, R. Kazma Employee of: Novartis AG, M. Cabanski Employee of: Novartis AG, E. Kamphausen Employee of: Novartis AG, P. Maguire Employee of: Novartis AG, A. Avrameas Employee of: Novartis AG, M.-A. Valentin: None declared, Y. Li Employee of: Novartis AG, A. Auger-Sarrazin Employee of: Novartis AG, S. Kaiser Employee of: Novartis AG, P. Follet Employee of: Novartis AG, S. Oliver Employee of: Novartis AG, A. Vitaliti Employee of: Novartis AG

DOI: 10.1136/annrheumdis-2017-eular.4736

AB0510 NEUROPSYCHIATRIC MANIFESTATIONS AND DISEASE ACTIVITY IN POLISH COHORT OF SYSTEMIC LUPUS ERYTHEMATOSUS PATIENTS

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Background: Neuropsychiatric systemic lupus erythematosus (NPSLE) is defined as wide range of neurological and psychiatric symptoms due to inflammation and ischemic processes. It is difficult to recognize primary NPSLE because of multiple central and peripheral symptoms.

Objectives: The aim of the study was to identify and classified the group of NPSLE patients with evaluation of disease activity.

Methods: We observed clinical neuropsychiatric (NP) manifestations in the cohort of 128 Polish patients with SLE. All patients with suspicion of NP symptoms had neuropsychological and imaging examinations. Symptoms of NPSLE were observed in 38 (30%) patients (34 female and 4 male) with average age 38 ± 6 years (range 18–61 yrs), average disease duration 6.6 ± 5.6 years (range 1.0 - 18.0 yrs). Patients were treated with oral and pulse glucocorticoids (GC) and 89% of them standard immunosuppressive drugs (CYC, MMF, AZA, MTX, CsA). As a background therapy 82% of these patients were on chloroquine or hydroxychloroquine (CQ/HCQ). All patients were assessed according to Systemic Lupus Erythematosus Disease Activity Index by SLEDAI (version 2000), Physical Global Assessment (PGA) and damage index (SDI).

Results: Central and peripheral NPSLE symptoms were recognized and categorized (Tab 1). All NPSLE patients had symptoms from central nervous system, but only 16% ($n=6$) of them had peripheral lupus manifestations. Mean SLEDAI score at NP event was very high 29 ± 9.6 , but mean SLEDAI score without NP symptoms was 15 ± 8.3 and was connected with musculoskeletal, mucocutaneous, renal and hematological domains respectively $n=29$, 76%; $n=23$, 60%; $n=11$, 29%; $n=8$, 21%. Low disease activity was estimated at 3% of patients examined. Most of lupus patients ($n=37$, 97%) had moderate or high disease activity regardless of NP symptoms. In our study group lupus patients during NPSLE symptoms were immunologically active with increased anti-dsDNA antibodies ($n=30$, 78%) and/or lower complements C3 and/or C4 levels ($n=21$, 55%).

Conclusions: In Polish lupus cohort we observed more frequently lupus-related primary neuropsychiatric symptoms from central nervous system, especially cognitive dysfunctions, mood disorders, cerebrovascular events. Clinical activity of NPSLE patients was rather high and definitely most of patients were immunologically active despite aggressive immunosuppressive treatment and with standard background therapy.

Disclosure of Interest: None declared
DOI: 10.1136/annrheumdis-2017-eular.4265

AB0511 VITAMIN D: POTENTIAL ROLE IN ANTIPHOSPHOLIPID SYNDROME

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Background: Vitamin D, due to its immunoregulatory properties, has been implicated in the pathogenesis of autoimmune diseases, such as antiphospholipid syndrome (APS).

Objectives: A) To determine vitamin D levels in patients with primary APS and to compare them with patients with positive antiphospholipid antibodies (aPL), not meeting clinical criteria for APS, and with healthy controls. B) To analyze the association of the vitamin D levels with both the clinical manifestations and the immunological profile of patients with primary APS.

Methods: We conducted a retrospective study including patients attended at the rheumatology clinic from a tertiary facility in Northern Spain. We included 74