

activity in prefrontal cortex, anterior cingulate, and caudate nucleus than that of other two groups.

Conclusions: Anxiety disorder commonly exists in patients with pSS. The pathology of this psychological symptom is poorly understood, which may be one of manifestation of nervous system involvement. fMRI may provide a novel insight into the pathological process accompanying subtle psychiatric disorders commonly experienced by patients with pSS.

References:

- [1] Massara A, Bonazza S, Castellino G, et al. Central nervous system involvement in Sjögren's syndrome: unusual, but not unremarkable—clinical, serological characteristics and outcomes in a large cohort of Italian patients. *Rheumatology (Oxford)*. 2010;49:1540–9.
- [2] Martinez S, Cáceres C, Mataro M, et al. Is there progressive cognitive dysfunction in Sjögren Syndrome? A preliminary study. *Acta Neurol Scand*. 2010;122:182–8.
- [3] Barbara M, Segal, Bryon A, Mueller, et al. Disruption of brain white matter microstructure in primary Sjogren's syndrome: evidence from diffusion tensor imaging. *Rheumatology* 2010;49:1530–1539.
- [4] Liu LJ, Zhang XY, He N1, et al. Genetic variation in WDR1 is associated with gout risk and gout-related metabolic indices in the Han Chinese population. *Genet Mol Res*. 2016 Apr 28;15(2). doi: 10.4238/gmr.15027381.

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AB0481 SUBCLINICAL NEUROPSYCHIATRIC DYSFUNCTIONS IN SYSTEMIC LUPUS ERYTHEMATOSUS PATIENTS -A HOSPITAL BASED STUDY

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Background: Up to 50% of systemic lupus erythematosus (SLE) patients experience neuropsychiatric manifestations (NP) during the disease course but many might have subclinical neuropsychiatric lupus (NPL), causing significant increase in morbidity and mortality. The pathogenesis of NPL is multifactorial and involves various inflammatory cytokines, autoantibodies and immune complexes¹.

Objectives: To examine for the presence of subclinical NPL, cerebral atherosclerosis and their correlation - if any to brain magnetic resonance imaging (MRI/MRA) findings and SLE disease activity.

Methods: This is a cross sectional observational study that included thirty adult female SLE patients fulfilling the updated ACR classification criteria for SLE². All were subjected to: detailed history taking, thorough clinical examination, assessment of SLE disease activity using Systemic Lupus Erythematosus Disease Activity Index (SLEDAI) score, psychometric evaluations using the Modified Mini-Mental State Examination (MMSE) to assess for cognitive dysfunction, Hamilton Depression Rating Scale (HAM-D) and Hamilton Anxiety Scale (HAM-A) to assess for the presence of depression and anxiety respectively. CBC, ESR, full blood chemistry, urine analysis, protein/creatinine ratio, brain MRI/MRA.

Results: The mean age was 31.7 years (22 to 40 years). 12 patients (40%) had positive antiphospholipid (APL) antibodies with or without clinically evident antiphospholipid syndrome (APS). 22/30 (73.33%) had different NP manifestations, 13 had depression (43.3%), 15 had anxiety (50%) and 16 had cognitive impairment (53.3%). All patients with depression and anxiety, 87.5% of patients with dementia had MRI abnormalities. All SLE patients with positive aPL were found to have MRI abnormalities, while MRI abnormalities were found in only 8 SLE patients with negative aPL (100% vs. 44.4%) ($p < 0.001$). There was a statistically significant correlation between SLE disease activity and both NP manifestations and MRI/MRA abnormalities. aPL antibodies also had a significant correlation with NP manifestations. MRI abnormalities included discrete white matter lesions (60%), cortical atrophy (25%) and gross infarctions (15%). MRA revealed atherosclerotic changes of one or more of the large intracranial vessels in (27.27%) of NPSL patients.

Conclusions: Significant number of SLE patients without overt neuropsychiatric manifestations were found to have subclinical cerebrovascular and cognitive dysfunctions, depression and anxiety by simple bedside questionnaires. SLE disease activity positively correlates with neuropsychiatric manifestations. The presence of APL antibodies is a strong risk factor for developing NPSL. Several distinct brain MRI patterns were observed in patients with active NPSL, suggestive of different pathogenetic mechanisms.

References:

- [1] Popescu A and Kao A. Neuropsychiatric systemic lupus erythematosus. *Current neuropharmacology*, 2011, 9(3):449.
- [2] Petri M, Orbai A, Alarcón G, Gordon C, Merrill J, Fortin P, Weisman M. Derivation and validation of the Systemic Lupus International Collaborating Clinics classification criteria for systemic lupus erythematosus. *Arthritis & Rheumatism*, 2012, 64(8):2677–86.

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AB0482 ELEVATION OF SERUM PROPROTEIN CONVERTASE SUBTILISIN/KEXIN TYPE 9 (PCSK9) CONCENTRATIONS AND ITS CORRELATION WITH C-REACTIVE PROTEIN, BUT NOT ATHEROGENIC LIPIDS IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS

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Background: Patients with systemic lupus erythematosus (SLE) have a tendency of accelerated atherosclerosis with controversial benefits from statin. This phenomenon can only partly be explained by traditional risk factors for cardiovascular disease. Proprotein convertase subtilisin/kexin type 9 (PCSK9) is a serine protease associated with cardiovascular risk that not only regulates cholesterol metabolism, but acts as a critical regulator of inflammatory reaction. PCSK9 inhibitors were also highly promising drugs bringing added cardiovascular benefit when administered with statin [1] [2].

Objectives: The present study firstly aimed to compare serum PCSK9 levels in SLE patients and healthy controls. The association between PCSK9 concentrations with atherogenic lipids and C-reactive protein (CRP) in SLE patients was also analyzed.

Methods: 77 individuals encompassed; 47 patients with SLE and 30 age- and sex-matched controls. Serum PCSK9, lipoproteins concentrations and CRP levels were assessed in patients and controls. Individuals with history of smoking, diabetes, infection, tumor and statin use were excluded.

Results: Serum PCSK9 levels were significantly elevated in patients with SLE, compared with healthy controls ($p=0.034$). PCSK9 positively correlated with serum levels of CRP ($r_s=0.351$, $p=0.016$); The tendency seemed more significant in female patients ($r_s=0.487$, $p=0.001$); No correlation with statistical significance between PCSK9 levels with disease activity (SLEDAI) or serum lipids parameter was found ($p > 0.05$, all) (Table 1).

Table 1. Characteristics of patients and controls and correlational analysis of PCSK9 levels and disease parameters in SLE patients. Data are shown as number or median (interquartile range), respectively

Variables	Healthy controls	SLE	r_s , p-value
Number	30	47	NA
Female/Male	24/6	42/5	NA
Age (years)	30.5 (26–39.5)	33 (28–42)	-0.014, 0.927
PCSK9 (ng/ml)	292.44 (199.87–499.93)	390.53 (305.37–525.92)	NA
SLEDAI	NA	6 (4–8)	0.092, 0.539
Cholesterol (mmol/l)	NA	4.50 (3.90–5.40)	-0.012, 0.935
LDL cholesterol (mmol/l)	NA	2.38 (1.74–3.19)	0.002, 0.989
ApoA1 (g/l)	NA	1.30 (0.97–1.59)	0.011, 0.943
ApoB (g/l)	NA	0.79 (0.63–0.94)	0.181, 0.223
Triglycerides (mmol/l)	NA	1.26 (0.79–1.64)	-0.020, 0.896
HDL cholesterol (mmol/l)	NA	1.49 (1.22–1.86)	-0.112, 0.453
CRP (mg/l)	NA	1.31 (0.50–3.60)	0.351, 0.016
CRP (mg/l) in female patients	NA	1.195 (0.500–3.568)	0.487, 0.001

Conclusions: Elevation of PCSK9 was observed in patients with SLE and correlated with CRP but not atherogenic lipids, particularly in female patients. The result was indicative of pathogenic role of PCSK9 in the low-grade inflammation which promotes the atherogenic process in SLE patients.

References:

- [1] Giugliano RP, Sabatine MS. Are PCSK9 Inhibitors the Next Breakthrough in the Cardiovascular Field?. *J Am Coll Cardiol*. 2015 Jun 23;65(24):2638–51.
- [2] Walley KR, Thain KR, Russell JA, et al. PCSK9 is a critical regulator of the innate immune response and septic shock outcome. *Sci Transl Med*. 2014 Oct 15;6(258):258ra143.

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AB0483 RESPONSIVENESS OF LUPUS IMPACT TRACKER AMONG CHINESE PATIENTS WITH LUPUS

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Background: Lupus Impact Tracker (LIT), a 10 item, patient reported outcome tool for patients with systemic lupus erythematosus (SLE) has undergone psychometric validation and responsiveness studies in the US and Europe.

Objectives: To report results on responsiveness of Lupus Impact Tracker among Chinese patients with SLE.

Methods: 430 patients with SLE meeting the ACR classification criteria were recruited in Hong Kong, China at a single center. LIT scores from two visits one year apart were analyzed for responsiveness and Minimal Clinically Important