therapy group were found to be significantly lower (-0.41±0.29 mg/dL, P<0.001). However, the serum uric acid levels in the estrogen monotherapy and tibolone groups did not differ significantly from the control group level.

Conclusions: Serum uric acid levels decreased in response to estrogen-progestogen combination therapy in postmenopausal women. We attribute our findings to the effects of progesterone, rather than estrogen.

References:

[1] Roddy E, Doherty M. Epidemiology of gout. Arthritis Res Ther. 2010;12:223.
[2] Hak AE, Curhan GC, Grodstein F, Choi HK. Menopause, postmenopausal hormone use and risk of incident gout. Ann Rheum Dis. 2010;69:1305–9.
[3] Hak AE, Choi HK. Menopause, postmenopausal hormone use and serum uric acid levels in US women–the Third National Health and Nutrition Examination Survey. Arthritis Res Ther. 2008;10:R116.
[4] Bruderer SG, Bodmer M, Jick SS, Meier CR. Association of hormone therapy and incident gout: population-based case-control study. Menopause. 2015;22:1335–42.

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SAT0694 SUBGROUPING OF EARLY RA-PATIENTS IDENTIFIED CLUSTER OF PATIENTS WITH HIGH LEVELS OF PAIN, FATIGUE AND PSYCHOSOCIAL DISTRESS 3 YEARS AFTER DIAGNOSIS

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Background: The wide range of effective treatment alternatives for rheumatoid arthritis (RA) makes treating the disease to inflammatory remission a feasible goal for a majority of patients. However, earlier studies have reported that symptoms other than inflammatory disease activity causes a substantial burden of illness for RA-patients. These unmet needs include persistent pain, fatigue, impaired physical function and mental health status (1).

Objectives: To identify clusters of early RA-patients based on pain, fatigue, sleep, physical function, mental health status and perceptions of quality of life, 3 years after diagnosis. Withal investigate associations between clusters and clinical parameters at the time of diagnosis.

Methods: Data was compiled from the Swedish case-control cohort Epidemiological Investigation of Rheumatoid Arthritis (EIRA) and linked to the Swedish Rheumatology Quality Register (SRQ). All patients were diagnosed with RA according to the 1987 ACR criteria. Early RA-patients with clinical data from diagnosis and 3 year follow-up questionnaire data were included (N=618; 74% women, median age at diagnosis 58 years). Measurements of pain, fatigue, sleep problems, physical and mental functioning and quality of life was entered into a hierarchical agglomerative clustering procedure using Ward's method of squared Euclidian distances. Number of clusters was determined by largest changes in distances at which clusters were formed. Associations between clusters and clinical variables at diagnosis were calculated using contingency analysis. All statistical analysis was performed using jmp statistical software (SAS, US). p<0.05 was considered significant.

Results: The cluster analysis identified three distinct clusters. Cluster 1 consisted of 178 patients (29%) doing significantly worse for all included variables. Cluster 3 consisted of 209 patients (34%) doing very well and cluster 2, consisting of 231 patients (37%), constituted an intermediate group doing fairly well. Cluster 1 was associated with female sex (p=0.0007) and lower education level (p=0.0003) compared to cluster 3. Cluster 1 was also associated to higher HAQ (p<0,0001), higher patient global assessment of health (p<0.0001), higher pain ratings (p<0.0001) and lower swollen/tender joint count ratio (STR) (p=0.0065) at the time of diagnosis compared to cluster 3.

Conclusions: Through cluster analysis, we could identify a subgroup of almost a third of the RA-patients with high levels of pain, fatigue, sleep problems and poor physical and mental health related quality of life 3 years after RA-diagnosis. These symptoms are indicative of a central sensitization syndrome and these findings indicate that other factors than inflammatory disease activity causes a significant burden of illness also at an early stage of RA and that there is a need of additional intervention strategies for these patients.

References:

[1] Taylor PC, Moore A, Vasilescu R, Alvir J, Tarallo M. A structured literature review of the burden of illness and unmet needs in patients with rheumatoid arthritis: a current perspective. Rheumatol Int. 2016;36(5):685–95.

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SAT0695 NO ASSOCIATION BETWEEN VITAMIN D LEVELS AND CARDIOVASCULAR DISEASES IN INFLAMMATORY JOINT DISEASES AND SYSTEMIC AUTOIMMUNE DISEASES – A SYSTEMATIC REVIEW

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Background: In recent years, vitamin D deficiency has been linked to disease activity and pathogenesis of systemic autoimmune diseases (SAD) like systemic lupus erythematosus (SLE) and inflammatory joint diseases (IJD) such as rheumatoid arthritis (RA). In the general population, the association between vitamin D with risk for cardiovascular diseases (CVD) is still debatable. While people with IJD and SAD tend to be vitamin D deficient and suffer from an elevated CVD burden, the effect of vitamin D on their cardiometabolic risk factors is of much interest.

Objectives: To provide an overall conclusion on whether vitamin D deficiency contributes to an increased cardiovascular morbidity in these patients, a systematic literature review was done.

Methods: A systematic literature search was done in PubMed/MEDLINE and EMBASE to identify all articles that assessed the association of vitamin D with cardiovascular disease and its risk factors in patients with IJD (rheumatoid arthritis (RA), psoriatic arthritis (PsA), ankylosing spondylitis) and SAD (systemic lupus erythematosus (SLE), Behcet's disease, vasculitis, Sjogren syndromes, systemic sclerosis). Eligible studies were assessed for quality and risk of bias according to the Cochrane Handbook Chapter 13.5.2.1. (Higgins JPT. GS. Cochrane Handbook for Systematic Review of Interventions The Cochrane Collaboration 2011.)

Results: In total 3273 abstracts were identified. After screening, selection and quality assessment, 16 studies were included (6 case-control and 10 cohort studies), which described only RA and SLE except for one study which focused on PsA. Therefore, this study focused on RA and SLE because they are the most frequent IJD and with highest CVD risk respectively. In RA patients (n=812) vitamin D deficiency was associated with presence of (components)of metabolic syndrome (OR =1.8 (95% CI: 1.3; 2.5), P=0.001) in RA, especially dyslipidemia (OR 1.7; 95% CI:1.1–2.5; P=0.013) and obesity. No studies with prospective design in RA have assessed CVD risk in relation to vitamin D. In SLE patients (n=1850) the only prospective study observed no association between vitamin D deficiency and CVD, although weak associations with dyslipidemia and obesity were observed in some studies.

Conclusions: No clear association between vitamin D deficiency and CVD was found in patients with RA and SLE, probably due to large heterogeneity in terms of sample sizes, designs, analyses and outcome measures. As conclusions were mainly drawn on cross-sectional data, there is an urgent need for adequate prospective studies to assess if vitamin D levels are associated with cardiovascular outcomes.

References:

[1] Higgins JPT, Green S (editors). Cochrane Handbook for Systematic Reviews of Interventions Version 5.1.0 [updated March 2011]. The Cochrane Collaboration, 2011. Available from www.handbook.cochrane.org.

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SAT0696 ENDOTHELIAL NITRIC OXIDE SYNTHASE T-786C GENE PROMOTER POLYMORPHISM IS A POTENTIAL PREDICTOR OF LOW RESPONSE TO THE THERAPY IN PATIENTS WITH RHEUMATOID ARTHRITIS, UKRAINE POPULATION

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Background: Recently endothelial nitric oxide synthase (eNOS) T-786C gene promoter polymorphism was considered to be a factor of a high severity of RA [1,2]. It is possible that the eNOS T-786C gene promoter polymorphism can modify the efficacy and safety of treatment in patients with RA, but there is no information on the subject.

Objectives: To evaluate efficacy of treatment of patients with RA in accordance to the eNOS T-786C gene promoter polymorphism.

Methods: Patients who were enrolled in the study satisfied follows criteria: MTX 10–15 mg/week during the past 12 weeks, stable oral NSAIDs and corticosteroids (CCS), ≤10 mg/day prednisone or equivalent during the past 2 weeks, DAS28 ≥3.2, >6 tender and swollen joints. We excluded patients with previous biologic agent treatment history. All patients received treatment that included MTX, folic acid, CCS, NSAIDs. We evaluated RA activity (DAS28), number of swollen and tender joints, ESR, CRP, and HAQ before enrollment in the study and at the 12 week. Efficacy of the treatment was assessed by the ACR 20; 50; 70 at the end of the study.

The 12-week study completed 148 patients with RA, 100% female, aged 45.7±8.54 years (mean ± SD), with moderate (DAS28 3.2–5.1; 34.5%) and high disease activity (DAS28 >5.1; 65.5%). Among the patients with RA, seropositive were 81.8% and 84.5% by the RF and the anti-CCP, respectively. Polymorphism of NOS3 T786C gene (rs2070744) was performed by Real-Time PCR. All studied polymorphism satisfied Hardy-Weinberg equilibrium. The study was conducted in accordance with the Declaration of Helsinki of the World Medical Association