Objectives: To verify the hypothesis about the role of SAA1 rs12218 T/C gene polymorphism in the aptitude to various clinical JIA phenotypes.

Methods: The study included 132 children, of whom 66 were diagnosed with JIA and 66 were healthy unrelated volunteers (the college students) as a control group, comparable by gender and age. The group of patients with JIA consisted of 44 girls and 22 boys, with an average age of 11.7 ± 4.2 years and an average disease duration of 4.8 ± 3.8 years. The diagnosis and classification of JIA was established according to ILAR-2004 criteria. The JIA group included 30 (45%) patients with oligoarthritis (jIA), of which 20 patients (67%) were positive for the HLA-B27 antigen (jIA-B27 +) and 10 (33%) patients with anterior uveitis (uIA); 20 (30%) patients were assigned to the group with the polyarticular variant (pJIA), while all of them were seronegative for the rheumatoid factor; 16 (24%) patients were diagnosed with JIA with a systemic onset (sJIA). The frequencies of the T/C polymorphism of the SAA1 gene were assessed using an allele-specific polymerase chain reaction in a real time mode (RT-PCR).

Results: In the group of patients diagnosed with sJIA and (JIA-B27 +), the frequency of the C allele was significantly higher compared to the control (53.3% and 57.5% versus 37.1%, p = 0.035 and 0.022, respectively). No significant differences were detected in the frequencies of the mutant C allele between sJIA and pJIA and the control group. The logistic analysis of the frequency distribution of the alleles of the SAA1 gene demonstrated an increased risk of the C allele in formation of an aptitude to the sJIA variant (OR 1.94, 95% CI 1.00-3.76, p = 0.04). In the control group, the risk of the sJIA aptitude was also increased compared to the control (OR 2.29, 95% CI 1.05-5.04, p = 0.022).

Conclusion: The data obtained confirm for the first time the involvement of the rs12218 polymorphism of the SAA1 gene in an aptitude to the oligoarthritis JIA clinical phenotype. The presented results require further replication researches using an enlarged number of patients from different population groups.

Disclosure of Interests: None declared

DOI: 10.1136/annrheumdis-2020-eular.4119

AB0020

CORRELATION BETWEEN SERUM AND SYNOVIAL CONCENTRATION OF IL-17A AND MIRNA EXPRESSION IN RHEUMATOID ARTHRITIS PATIENTS


1Medical University – Sofia, Bulgaria, Department of Internal Medicine, Clinic of Rheumatology, Sofia, Bulgaria; 2Medical University – Sofia, Department of Medical Chemistry and Biochemistry, Molecular Medicine Center, Sofia, Bulgaria; 3Clinical Immunology, University Hospital Lozenetz, Sofia, Bulgaria

Background: Interleukin 17 (IL-17) is a proinflammatory cytokine, which overproduction promotes the autocrine reaction in rheumatoid arthritis (RA). Posttranscriptional regulation of IL-17 production by specific microRNAs (miRNAs) is of great interest in the recent years. 146a was associated with IL-17 expression in IL-17 producing T-cells in the synovium when miR-155 enhanced Treg and Th17 cells differentiation and IL-17A production by directly targeting the suppressor of cytokine signaling (SOCS1) [1, 2]. It has been shown that IL-17 production in lymphocytes or its receptors [3]. To examine a possible correlation between systemic and local concentrations of IL-17A and systemic and local miR-146a, miR-155 and miR-223 expression in RA patients.

Methods: Expression levels of three miRNAs were determined in matched peripheral blood (PB) and synovial fluid (SF) samples of RA patients by relative quantification method 2-ΔΔCt. As reference control for normalization RNU6B gene was used. Concentrations of IL-17A were compared between matched serum and SF samples from 20 RA patients by Human IL-17A ELISA kit (Gene probe, Diacline, France). Healthy donors were used as controls.

Results: miR-146a, miR-155 and miR-223 showed overexpression in SF compared to HC's SF (p<0.002; p<0.001; p=0.03, respectively). The ROC curve analysis showed diagnostic accuracy for miR-146a in SF with AUC=0.769, p=0.006, AUC for SF miR-155 was 0.858, p=0.001 and AUC for SF miR-223 was 0.841, p=0.002. SF levels of miR-146a and miR-155 were overexpressed in 52.17% and in 76.09% of the RA patients compared to its systemic levels. The opposite changes of IL-17A and miR-223 systemic and local levels confirms the data about the possible role of miR-223 in regulating IL-17 production. The opposite changes of IL-17A and miR-223 systemic and local levels confirms the data about the possible role of miR-223 in regulating IL-17 function. Further analysis with larger sets is needed to confirm these results.

References: