Lupus arthritis was more common in SLE СТ-genotype carriers than in other SLE patients (χ²=5.9, p=0.015; p=0.027). We revealed significant increase of CT genotype (RUNX1 rs9979383) in healthy donors vs SLE patients (χ²=4.4; p=0.042; OR=0.53 (Cl95% 0.29-0.98); LR+ =0.69 (Cl95% 0.45-0.99); LR- =1.3 (Cl95% 1.01-1.56)). Lupus arthritis was more common in ST genotype carriers than in other SLE patients (χ²=4.66 p=0.031; p=0.058).

Significant differences in IL6 rs1800795, IL6 rs2282114 and IL6 rs4584618 genotypes distribution between studied groups were not found (χ²=0.427, p=0.559 and p=0.407 correspondingly) but GG-genotype (IL6 rs1800795) carriage in SLE patients was associated with increased APS frequency (χ²=4.45, p=0.035; OR=0.19 (Cl95% 0.04-0.9); LR+ =0.28 (Cl95% 0.07-0.93); LR- =1.41 (Cl95% 1.03-1.64).

Conclusion: Our data suggest the susceptibility to SLE in TT genotype of STAT4 rs7574865 polymorphism, protective role of CT genotype of RUNX1 rs9979383 for SLE and association between GG-genotype of IL6 rs1800795 and APS in SLE patients in Belarusian population. Lupus arthritis was associated with TT genotype of STAT4 rs7574865 and CT genotype of RUNX1 rs9979383.

REFERENCES:

Disclosure of Interests: None declared
DOI: 10.1136/annrheumdis-2021-eular.2023

AB0010
ASSOCIATION OF SAA1 GENE POLYMORPHISM -13T/C (RS12218) WITH ANKYLOSING SPONDYLITIS IN RUSSIAN POPULATION

I. Guseva1, K. Sakharova2, M. Krylov1, E. Samarkina1, S. Erdes2,1

1. VA. Nasonova Research Institute of Rheumatology, Laboratory of Immunology and Molecular Biology of Rheumatic Diseases, Moscow, Russian Federation; 2. VA. Nasonova Research Institute of Rheumatology, Laboratory of Spondyloarthritis, Moscow, Russian Federation

Background: Ankylosing spondylitis is a chronic systemic inflammatory disease. Inflammation and high levels of serum amyloid A (SAA) protein are predisposing factors for secondary AA amyloidosis. The role of SAA1 gene polymorphisms in AS is not well understood.

Objectives: To investigate the association of SAA1 gene polymorphism -13T/C (rs12218) with ankylosing spondylitis and to evaluate the influence of this polymorphism on SAA protein concentration.

Methods: 123 AS patients (72 males, 51 females; age - M (SD) 37.51 (12.77) years; disease duration - 14.28 (11.22) years; BASDAI - 5.59 (1.13); B27-positive - 111 (90.2%) pts) and 95 gender, age matched healthy individuals (control group) were included in this study. SAA1 gene polymorphism -13T/C was genotyped using allele-specific RT-PCR assay. SAA protein concentration was measured using nephelometry in AS patients.

Results: The distribution of genotypes TT, TC and CC differed statistically between AS and control groups (24.4%, 56.1%, 19.5% and 41.1%, 42.4%, 14.7% respectively, χ²=6.9, p=0.03). The presence of the C allele was associated with the development of AS (OR=1.55 [CI 1.04-2.33], p=0.03). The SAA1 -13T/C polymorphism tended to be associated with SAA protein value in AS patients: TT+TC genotypes -13.8 mg/l [4.2; 91.0], CC genotype -7.8 mg/l [1.6; 29.6], p=0.07 ESR, CRP and BASDAI values did not correlate with SAA1 -13T/C polymorphism (p=0.6, p=0.4, p=0.4 respectively).

Conclusion: The results of our study demonstrated for the first time that SAA1 gene polymorphism -13T/C (rs12218) is associated with susceptibility to AS. It is also shown that this polymorphism can affect the SAA protein level. Our findings need to be verified in AS patients with high levels of SAA protein in various ethnic and population groups.

Disclosure of Interests: None declared
DOI: 10.1136/annrheumdis-2021-eular.2029