181. HLA POLYMORPHISMS IN TAKAYASUS'S ARTERITIS PATIENTS IN SERBIA

Maja Stojanovic[1,2], Zorana Andric[3], Dusan Popadic[4], Marija Stankovic[5], Aleksandra Peric-Popadic[1,2], Rada Miskovic[1,2], Jasna Bolpacic[1,2], Dragana Jovanovic[1], Vesna Tomic-Spiric[1,2] and Sanvila Raskovic[1,2]
[1]*Clinic of Allergy and Immunology, Clinical Center of Serbia, Belgrade, Serbia,* [2]*Faculty of Medicine, University of Belgrade, Belgrade, Serbia,* [3]*Tissue Typing Department, Blood Transfusion Institute of Serbia, Belgrade, Serbia,* [4]*Institute of Microbiology and Immunology, Faculty of Medicine, University of Belgrade, Belgrade, Serbia,* [5]*Department of Pathophysiology, Faculty of Medicine, University of Belgrade, Belgrade, Serbia*

Background: Takayasu's arteritis (TA) is a rare systemic vasculitis that affects aorta, its major branches, and occasionally pulmonary arteries. Genetic factors, such as certain human leukocyte antigen (HLA) seem to play an important role in the development of TA. The aim of our study was to examine HLA polymorphisms and its correlation with clinical characteristics of TA patients in Serbia.
Methods: DNA was extracted from blood samples of 25 patients with confirmed diagnosis of TA by a fully automated system with Maxwell 16 Purification Kit. The allelic groups of HLA-A*, -B*, -C*, -DRB1* and -DQB1* loci were typed by polymerase chain reaction sequence-specific oligonucleotide probe using a LuminexTM platform. The allele frequencies were compared with a control group consisted of 1992 unrelated healthy potential bone marrow donors. We used a 2X2 contingency table and Fisher exact test to test the differences of allele and haplotype frequencies in the control and patient groups. The association of HLA-B*52 allele with clinical covariates was evaluated with ordinal logistic regression, X square and Fisher's exact test. $p < 0.05$ was considered to be statistically significant.
Results: A significant association of TA with HLA-B*52 was found [20% of patients (5/25) with 10% HLA-B*52 alleles frequency (5/50) vs 1.2% (46/3884) in healthy controls]; $p = 0.0003962$, p adj =0,011. The presence of HLA-B*52 was associated with an earlier disease onset, poorer clinical outcomes and respond to treatment. A higher frequency, but without statistical significance after p-value correction, for HLA-A*32 ($p = 0.012$, p adj 0.2), HLA-B*15 ($p = 0.012$, p adj 0.326), HLA-B*57 ($p = 0.018$, p adj 0.483), HLA-C*03 ($p = 0.009$, p adj 0.121) allelic group and DRB1*15-DQB1*05 haplotype ($p = 0.039$, p adj 0.583) was found. In contrast to susceptibility alleles, HLA-C*03 allelic group, found in 32% (8/25) of TA patients, was present in patients with a milder clinical form of the disease.
Conclusion: Our study has shown the strong association between HLA-B*52 and TA. The HLA-A*32, -B*15 and -B*57 allelic groups and DRB1*15:02-DQB1*05 haplotype, as the susceptibility factors, and HLA-C*03 as a putative protective allelic group in TA patients need to be confirmed in a larger study population.
Disclosures: None