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Sepsis associations with HLA-DR and -DQ genes

Linda Bridina and **Angelika Krumina**
Riga Stradins University, Latvia

Aim: Despite the extended laboratory and clinical study of sepsis, its diagnosis remains a clinical challenge. The initiation of sepsis activates many different biochemical and immunological pathways, which is expressed with alterations of many molecules on human tissues. The aim of this research was to investigate the genetically determined predisposition to developed sepsis by analysis of distribution of human leukocyte antigen HLA class II genes. We summarize the evidence for a genetic susceptibility to develop the sepsis and unfavorable outcome of sepsis. We consider the candidate genes are likely to be involved in the pathogenesis of sepsis and based on genetic variability.

Method: This was a single-center study at Pauls Stradins Clinical University Hospital in Latvia. The study group consisted of patients (n=62) who had sepsis who were enrolled during an 8-month period. The immunogenetic part of the study was done and 62 sepsis patient and control group, samples of 100 healthy individuals who were genotyped for HLA-DRB1; DQB1 and DQA1 using RT-PCR with sequence-specific primers.

Results: Summarized results shows that the alleles: DRB1*04:01 (OR=5.54; 95%CI=1.88-16.29; p=0.001); DRB1*07:01 (OR=19.03; 95%CI=2.37-152.82; p=0.001); DQA1*05:01 (OR=14.17; 95%CI=5.67-35.4; p<0.001); DQB1*02:01 (OR=50.00; 95%CI=2.90-861.81; p<0.001) were significantly increased in patients with sepsis compared to the control group patients. Comparing these alleles who were significantly increased in patients with sepsis compared to the control group patients with the most common final clinical diagnosis was pneumonia 66% (n=41).

Conclusion: Undoubtedly, our preliminary data shows that development of sepsis can have association with opedelenny alleles of genes HLA class II. These results have to be confirmed prospectively in a large population.

linda.bridina@gmail.com