CHANGES IN SOME OF THE IMMUNE CELL POPULATIONS IN PATIENTS WITH ANKYLOSING SPONDYLITIS ON SECUKINUMAB THERAPY - PRELIMINARY DATA

Ekaterina Ivanova-Todorova[1] Mariana Goycheva[2], Rumen Stoilov[2] , Dobroslav Kyurkchiev[1]

[1]*Laboratory of Clinical immunology, University hospital "St. Ivan Rilski";*
Department of Clinical immunology, MF, Medical University of Sofia, Bulgaria
[2]*Clinic of Rheumatology, University hospital "St. Ivan Rilski";*
Department of Internal medicine, MF, Medical University of Sofia, Bulgaria

Aim: Ankylosing spondylitis (AS) is the most common autoimmune disease in the group of spondyloarthropathies. In 94% of the patients, HLA-B27 is strongly associated with the disease and shows an unusual tendency to form so-called homodimers from its heavy chain - ("B272"). These homodimers bind to a specific KIR3DL2 receptor expressed by Th17 and mucosa-associated invariant (MAIT) Tc17 lymphocytes. IL-17 is known to play a crucial role in AS, and therefore, anti-IL-17A therapy is an effective treatment for AS. The aim of the presented study was to monitor Th17, MAIT as well as the expression of KIR3DL2 during secukinumab therapy.

Materials and Methods: The study included 27 patients who were HLA-B27 +. Changes in Th17, MAIT, and KIR3DL2 expression in peripheral venous blood prior to therapy at six months and one year were determined by flow cytometry.

Results: The mean percentage of Th17 (12.2%) lymphocytes as well as CD8 +, CD161 ++ MAIT (8.23%) in peripheral blood before therapy were in the reference range similar to that in healthy controls. During the course of tretment, these cell populations did not show any significant changes. Increased expression of KIR3DL2 on CD3 +, CD4 +, CD161 +, CD169 + Th17 was confirmed in patients with AS, with a tendency of the expression to decrease during therapy. An increased percentage of KIR3DL2 expressing MAIT cells was found, which decreased significantly in patients responding to the therapy ($p < 0.05$). A significant decrease in CD161 + CD8 + lymphocytes was also noted.

Conclusion: Our preliminary data showed that during anti-IL-17A therapy, Th17 and MAIT-expressing KIR3DL2 subpopulations showed significant changes that could elucidate some of the immunopathogenic mechanisms associated with AS and the disease therapy.