ROLE OF IL-17A IN THE PATHOGENESIS OF REACTIVE ARTHRITIS

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Reactive arthritis usually develops two to four weeks after infection and typically follows a limited course, with most people recovering from its symptoms in three to 12 months.A tendency does exist for more severe and long-term disease in patients who test positive for HLA-B27, as well as in those who have a family history of spondyloarthritis.In about 15 to 20 percent of people with ReA, the condition recurs, sometimes brought on by reinfection. There is also a possibility of developing a chronic form of arthritis. Though the chronic arthritis brought on by ReA is usually mild, a minority of people develop a more severe form of arthritis, or spondyloarthritis.

The objective was to study the effectiveness of treatment aimed at slowing down the damage to the structural progression of the musculoskeletal system and bone remodeling in the joints of the spine in patients with ReA.

Materials and methods. In the period from 2021-2023, 30 patients diagnosed with ReA with spondylitis were examined in the 3rd city clinical hospital in Tashkent, the average duration of the disease was 5.8±2.4 years. The control group consisted of 15 healthy volunteers of the corresponding average age. Average age was 36.76±2.37 years old.

Results. During the year of treatment, the peripheral manifestations of spondylitis in patients with ReA decreased. The initial concentrations of IL-17A in the blood serum of patients with ReA exceeded the concentrations of patients in the control group. The average concentration of IL-17A in the blood serum of patients with ReA was initially higher than in patients of the control group (28.4±14.4 and 2.4±2.1 pg/ml, respectively; $p<0.0001$). It should be noted that the final mean concentration of IL-17A did not differ in patients who achieved an ASAS20 response (n=24) and those who did not (n=6): 27.5±11.6 and 29.1±14.5 pg/ ml, respectively ($p=0.77$). At the same time, in patients with ReA who achieved (n=12) and did not achieve partial remission of ASAS (n=18), both initial and final concentrations of IL-17A differed. The initial and final concentrations of IL-17A in patients who subsequently achieved remission were statistically significantly lower than in patients who did not achieve partial remission of ASAS.

Conclusion. Thus, treatment of patients with ReA with this method indicates the important role of IL-17A in the pathogenesis of ReA and makes it possible to use the inhibition of this cytokine as a therapeutic method. Secukinumab has low immunogenicity, the presence of antibodies to it is not associated with a decrease in the effectiveness of treatment.