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Immunological parameters in tuberculosis patient with diabetes militius and autoimmune thyroiditis with subclinical hypothyroidism

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Diabetes mellitus is a predictor of an unfavorable outcome of antituberculosis chemotherapy, and a 5-fold risk factor for a patient's death from tuberculosis. Diseases of the thyroid gland are found in patients with diabetes mellitus in 11-30%. Hypothyroidism against the background of

autoimmune thyroiditis worsens the outcomes of anti-tuberculosis chemotherapy especially in persons with concomitant diabetes mellitus due to the suppressive effect of thyroid hormone deficiency on T-cell immunity. It seems relevant to study the parameters of immunity in tuberculosis in persons suffering from diabetes mellitus with decreasing of thyroid function.

Objective of the study is to study the parameters of immunity in tuberculosis patients with diabetes mellitus and with subclinical hypothyroidism against the background of autoimmune thyroiditis.

Materials and methods. The study included 60 patients with destructive pulmonary tuberculosis (TB) with concomitant diabetes mellitus (DM), and in 30 of them the comorbid pathology was accompanied by autoimmune thyroiditis with concomitant subclinical hypothyroidism (SH) (observation group – 1 or TB/DM/SH). Comparison consisted of patients with tuberculosis and diabetes mellitus (TB/DM) without changes in thyroid homeostasis. The state of T- and B-systems of immunity, natural killers was assessed: CD3 +, T-helpers ($CD4^+$), cytotoxic T-cells (CD4 +), B-lymphocytes ($CD19^+$) and natural killer cells ($CD16^+$). The cytokine profile was determined: the levels of tumor necrosis factor-α (TNF-α), interferon-γ (INT-γ), interleukin-2 (Il-2), -6 (Il-6) and -4 (Il-4). Statistical processing of the obtained data was carried out by the method of variation statistics using a standardized package of calculations Microsoft Excel XP.

Results. The study of the immune system reveals deviations in the parameters of cellular immunity in both groups. In the observation group, there was a lower reactivity of leukocytes, a significant decrease in comparison with the control of the levels of mononuclear cells and pan-lymphocytes ($CD3^+$). A decrease in the subpopulations of T-helpers ($CD4^+$) and cells with killer-suppressive activity ($CD8^+$) indicates a deficiency of the cellular component of immunity in patients with comorbid pathology. But a more pronounced change in $CD4^+$ subpopulations was observed in patients with tuberculosis and diabetes mellitus in the presence of AIT with SH. In TB patients with DM+AIT, the level of TNF-α is half the values in patients with normal thyroid status. The level of INT-γ was 2.3 times lower in patients with AIT and SH at the background of tuberculosis and diabetes mellitus when compared with patients suffering from tuberculosis and diabetes mellitus maintaining normal thyroid state. The content of IL-2 in the systemic blood flow of TB/DM patients without thyroid pathology remains within the allowed physiological values with a decrease of 2.5 times in TB/DM patients with AIT and SH. The content of IL-6 in TB/DM patients with AIT and SH 2,5 times lower when compared with TB/DM patients without thyroid pathology – relatively and. Level of IL-4 in TB patients with AIT and SH increases compared with the control group. Higher values

of this indicator is observed in persons with AIT and SH compared with the control.

Conclusions: It has been established that in patients with diabetes mellitus, both cellular and humoral immunity is suppressed. But a significantly more significant suppression of immunity was diagnosed in patients with tuberculosis against the background of diabetes mellitus and autoimmune thyroiditis with subclinical hypothyroidism. Deficiency of thyroid hormones is the negative effect of a more significant suppression of immunity in patients with subclinical hypothyroidism.