

AN IMBALANCE OF THE HORMONES OF FATTY TISSUE IN PATIENTS WITH TYPE 2 DIABETES MELLITUS DEPENDING ON THE BODY MASS INDEX

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Purpose. The study was designed to assess the level of resistin and tumor necrosis factor-alpha (TNF- α) and its relationship with body mass index (BMI) in patients with type 2 diabetes mellitus (DM-2) with normal body weight and obesity.

Materials and methods. The study was performed on 70 patients with DM-2 with normal weight and obesity. All patients were divided into 2 groups: group 1 (n=20) patients with DM-2 and normal body weight and group 2 (n=50) patients with DM-2 and obesity. The controls (n=20) were apparently healthy individuals. The BMI, levels of resistin and TNF- α were determined.

Results. The mean level of resistin was significantly ($p < 0.001$) increased in all groups in comparison with the controls (4.87 ± 0.11 ng/ml), the level in group 2 (10.0 ± 0.11 ng/ml) was significantly different from that in group 1 (8.06 ± 0.23 ng/ml). The mean level of TNF- α also was significantly ($p < 0.001$) increased in all groups of patients (group 1 - 86.4 ± 1.21 pg / ml, group - 2 96.65 ± 0.72 pg / ml) in comparison with the controls (24.19 ± 1.06 pg / ml), the level in group 2 was significantly ($p < 0.001$) different from that in group 1 group. In both groups of patients group the direct correlation was established between resistin and BMI ($r = 0.36$, $p < 0.05$ – group 1, $r = 0.84$, $p < 0.05$ – group 2) and between TNF- α and BMI ($r = 0.39$, $p < 0.05$ – group 1, $r = 0.69$, $p < 0.05$ – group 2).

Conclusions. There is an imbalance in the production of fatty tissue hormones in patients with DM-2, especially with concomitant obesity. This hormonal imbalance is manifested by an increase of the level of resistin and TNF- α . It is important to note that the correlation between this adipokines and BMI was more pronounced in patients of group 2. It confirms the fact that obesity is the leading etiological factor in the pathogenesis of molecular-cellular mechanisms of interaction of immune and metabolic processes in patients with DM-2.