

THE ROLE OF OBESITY IN THE PROGRESSION OF OSTEOARTHRITIS IN YOUNG PERSONS

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It is proved that adipose tissue is an active endocrine organ. It synthesizes a large number of highly active substances that are involved in many processes in the body. Excess adipose tissue is a prognostically adverse factor for the formation of many nosologies, including osteoarthritis (OA). It is not only about increasing the production of hormone-like substances, but also the mechanical load on the joints. In recent years, it has been proven that the hormone adipose tissue apelin-13 has a negative effect on the structure of cartilage. This can be considered as one of the pathogenetic links in the formation of osteoarthritis.

Purpose: to study the effect of apelin-13 - the hormone of adipose tissue - on the course of OA in young obese patients.

Materials and methods. The study examined 96 patients with OA, in 62 cases in combination with obesity. Patients were under the age of 45 years, women predominated (69 patients). The control group included the corresponding number of people of the same gender and age.

Quantitative determination of the content of apelin-13 in the blood serum of patients was carried out by enzyme immunoassay (ELISA). Commercial test systems manufactured by Human (Germany) were used in accordance with the Labline-90 ELISA analyzer provided for the kit (Austria).

Statistical processing of the material was carried out using the STATISTICA 7 program.

Results and its discussion. According to the Kettle index, 45 people had I degree of obesity, 11 patients - II and 6 examined - III degree; 34 patients had normal BMI. The average content of apelin-13 in the serum of patients with OA was 70.23 ± 4.84 pg/ml in the control group - 56.75 ± 3.82 pg/ml. In the group of patients with OA without excess weight this indicator was equal to 62.07 ± 3.22 pg/ml. Determination of the

apelin-13 content depending on the values of the Kettle index, it was found that I degree of obesity was characterized by an increase in the rate to 73.9 ± 4.8 pg/ml, II - 62.5 ± 8.4 pg/ml and with III degree - 66.6 ± 8.7 pg/ml. Analysis of average adipokine levels in patients with OA depending on BMI did not reveal a direct correlation ($|r_{XY}| < 0,29$). The development of OA in obese individuals quite often leads to the formation of osteoporotic changes. The content of apelin-13 in patients with obesity and OA (43 patients) with osteopenic syndrome was 71.34 ± 2.04 pg/ml and with osteoporosis (14 patients) - 65.02 ± 2.64 pg/ml. Such changes in the apelin-13 index can probably be explained by the activation of the synthesis of this adipokine at the beginning of the disease formation. A decrease in the content of apelin is observed with a developed clinical picture due to depletion of its reserves. Perhaps, at more severe degree of obesity (II and III) and in the case of osteoporosis, the body makes an "attempt" to suppress the further development of dystrophic changes in the joint.

Conclusions. The presence of obesity in patients with osteoarthritis is accompanied by an increase in the synthesis of apelin-13. The content of apelin-13 becomes more expressive at its initial stage, which is probably the result of its active synthesis. The progression of obesity supports elevated apelin levels above normal, but somewhat inhibits its synthesis as a result of the depletion of its pool.