

RELATIONSHIP BETWEEN ADIPONECTIN AND ANTHROPOMETRIC PARAMETERS, INSULIN RESISTANCE AND TRANSAMINASE LEVELS IN PATIENTS WITH NONALCOHOLIC FATTY LIVER DISEASE AND TYPE 2 DIABETES

A. Zhuravlyova

Kharkiv National Medical University, Ukraine

The basis of the pathogenesis of nonalcoholic fatty liver disease (NAFLD) is insulin resistance (IR) which appears on the background of abdominal obesity (AO) which, in turn, is a key factor in the emergence of an imbalance between adipocytokines entailing a violation of lipid and carbohydrate metabolism, which ultimately leads to damage of the liver cells, the development of inflammation, fibrosis and apoptosis.

The goal- to study features of changes in the level of adiponectin (AN), depending on the function of the liver and IR index in patients with NAFLD and diabetes mellitus type 2 and AO.

Materials and methods. 25 patients (10 men and 15 women) with NAFLD and type 2 diabetes (HbA1c <7,5%). Clinical examination included assessment of anthropometric parameters (body mass index (BMI) and waist circumference (WC)), function of the liver (ALT, AST) and index HOMA-IR.

The Results. The changes in BMI were observed in 94.5% of patients, including obesity 1st degree - in 54.6%, 2nd degree - in 31.4% and 4.6% - obesity 3rd degree. AN level was reduced compared to control ($8,7 \pm 2,4$ ng/ml vs. $15,4 \pm 2,1$ ng/ml, $p <0.05$) and correlated with the degree of obesity - $6,5 \pm 2,1$ ng/ml in patients with grade 3 obesity ($p <0.05$). There was a negative relationship between the level of AN and BMI ($r = -0,36$; $p <0.01$), WC ($r = -0,34$; $p <0.05$). The level of AN significantly decreased with increasing levels of ALT ($r = -0,44$, $p <0.001$) and AST ($r = -0,46$; $p <0.001$). An inverse relationship between the level of AN and the index HOMA-IR was determined ($r = -0,46$; $p <0.001$).

Conclusions. Hypoadiponektinemia in patients with NAFLD and type 2 diabetes is associated with AO, the deterioration of the liver function and progression of IR that contributes to the further development of metabolic abnormalities in the liver.