cholesterol. The received data claims that the level of hepatic sortilin affects on low-density lipoprotein cholesterol that are associated with their suppression and degradation.

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CHANGES OF CALPROTECTINE, PARAMETERS OF IMMUNE INFLAMMATION WITH PARAMETERS OF LIPID AND CARBON EXCHANGE IN PATIENTS WITH ACUTE MYOCARDIAL INFACTION

Aim is to assess the state of immune inflammation based on the study of serum calprotectin level, as well as to analyze the presence and character of links with carbohydrate metabolism parameters based on the study of blood glucose, insulin and insulin resistance in patients with acute MI and DM type 2.

Materials and methods. The study involved examination of 110 patients (mean age 65.25 ± 0.09 years) who underwent treatment at myocardial infarction department of Kharkiv City Clinical Hospital No.27 and Kharkiv Railway Clinical Hospital No.1. The main group included 64 patients (average age 65. 31 ± 1.62 years) with acute MI and concomitant DM type 2. Blood glucose concentration was determined by glucose oxidase method. Insulin level was determined by immunoassay using test system EIA-2935, Insulin ELISA. Serum calprotectin level was established by immunoassay using the MRP8 / 14 ELISA KIT test system.

The level of carbohydrate metabolism disruption was assessed by calculating homeostasis model assessment (HOMA), QUICKI, Caro indices of insulin resistance by the following mathematical formulas.

Results.The direct correlation between the content of calprotectin and the index of insulin resistance of HOMA ($R = 0.52$; $p < 0.05$), insulinemia ($R = 0.57$; $p < 0.05$), fasting glycemia ($R = 0, 59$; $p < 0.05$), as well as inverse correlation relationships with the Caro index ($R = 0.68$; $p < 0.05$) and the QUICKI index ($R = 0.59$; $p < 0.05$). In addition, a direct correlation was found between calprotectin and triglyceride levels (R

= 0.31; p <0.05), and negative correlation with high density lipoprotein (R = 0.35; p <0.05). The level of total cholesterol, low density lipoprotein showed no significant association with the proinflammatory factor (R = 0.12; p> 0.05 and R = 0.18; p> 0.05, respectively).

Conclusions: Such connections indicate an increase in the activity of the immunoinflammatory parameter of calprotectin in proportion to weight gain, which may be an additional factor confirming the involvement of adipocytic tissue with chronic inflammation.

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ABDOMINAL OBESITY AS A PREDICTOR OF GERD DEVELOPMENT

Introduction. Obesity is one of the most complicated medical and social problems of our time. Its high prevalence, deterioration of quality of life and extremely high mortality from its effects require the combined efforts of physicians of different specialties. Gastroesophageal reflux disease (GERD) is an urgent problem in modern gastroenterology, due to its high frequency of occurrence, wide range of complaints expressed by the patients, development of severe complications and need for long-term therapy. There are a lot of risk factors for development of GERD and an important role is taken by abdominal obesity (AO) which, in addition to mechanical increase in intra-abdominal pressure, is accompanied by systemic chronic inflammation due to ability of adipocytes and adipose macrophages to produce a number of proinflammatory cytokines. According to the literature, C-reactive protein (CRP) can serve as a marker of the inflammatory process in obesity.

Aim. The aim of the study was to determine the relationship between the presence of excess body weight and AO, and the incidence of GERD.

Materials and methods. A retrospective analysis of 52 case histories of patients in the gastroenterology department of Kharkiv Regional Clinical Hospital was conducted.