

autoimmune thyroiditis - 27%, rheumatoid arthritis - 12%, autoimmune polyglandular syndrome - 7%, Sjogren's syndrome – 7,5%, systemic lupus erythematosus – 6,5%, diabetes mellitus type 1 - 2.5%, rheumatic polymyalgia - 2%, Crohn's disease - 1.5%, ulcerative colitis - 1%, vitiligo - 1%. Two clinical cases were selected among KhRCH patients with diagnoses: 1 - AIH and PBH, 2 - AIH and vitiligo. As a result, we revealed the presence of significant differences in the combined course of AP. Such differences are due to the fact that in case 1, mutations of the HLA-DR4 gene are usually detected, whereas in case 2, the HLA-DR3 gene mutation plays a major role. However, one should take into account the mutations in the CTLA4 gene, which are responsible for many AD, as well as epigenomic factors.

Discussion and Conclusions: The difference in the prevalence of combined autoimmune pathology is most likely due to mutations of different HLA genes. However, we suggest an existence of single mechanism for AIH and AD combinations.

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THE ESTIMATION OF RELATION BETWEEN SORTILIN AND LIPID METABOLISM INDICATORS IN PATIENTS WITH CORONARY HEART DISEASE AND DIABETES MELLITUS TYPE 2

Introduction. The problem of lipid metabolism destruction is becoming the subject of scientific research more often. Particular attention is paid to the problem of disease comorbidity, as well as the search for new early markers of asymptomatic diagnosis of lipid metabolism disorders. It is well-known that the lipid metabolism disorders play an important role in the development of complications and affect the prognosis of life in patients with coronary heart disease (CHD) and diabetes mellitus (DM) type 2. Sortilin is a new marker for the diagnosis of lipid metabolism disorders, which has received special attention lately.

Aim. To evaluate the association between sortilin and lipid metabolism in patients with coronary heart disease and diabetes mellitus type 2.

Materials and methods. A comprehensive examination of 105 patients with CHD and DM type 2 who were treated in the cardiology Department of Kharkiv city clinical hospital № 27 was provided. The patients were divided into two groups. The main group included 75 patients with CHD and DM type 2, the comparison group consisted of 30 patients with CHD without diabetes. The level of sortilin was determined by enzyme-linked immunosorbent assay using a commercial test system manufactured by Human SORT 1 ELISA Kit (USA). The study of the lipid profile parameters included the determination of total cholesterol (CHC), triglycerides (TG), high density lipoprotein cholesterol (HDL cholesterol) by enzymatic method according to standard biochemical methods. The low-density lipoprotein cholesterol content of LDL cholesterol was calculated by the formula of W. T. Friedewald: $LDL\ cholesterol = CHC - (HDL\ cholesterol + TG / 2.22)$, where $TG / 2.22$ is the low-density lipoprotein (LDL) cholesterol content. The results obtained are given as mean \pm standard deviation from mean ($M \pm \sigma$). Statistical data processing was performed using Statistica package version 6.0. The differences between the groups in the distribution close to normal were estimated using the Pearson test. The differences at $p < 0.05$ were considered statistically significant. Correlation analysis was performed to establish the nature of the relationship between the level of sortilin and lipid metabolism in patients with CHD and DM type 2.

Results. The study revealed direct correlation between the level of sortilin and such indicators of the lipid profile as LDL cholesterol ($r = 0.65$; $p < 0.05$), TG ($r = 0.46$; $p < 0.05$), CHD ($r = 0.22$; $p < 0.05$) with significant inverse with HDL cholesterol ($r = -0.42$; $p < 0.05$).

Conclusions. The study of the nature of the relations between the level of sortilin and lipid metabolism suggests that as the level of sortilin increases, there is a corresponding increase in very low-density of lipoprotein cholesterol, total cholesterol, triglycerides, which is typical for type 2 diabetes. Thus, there is a dependence of the sortilinemia level increasing with disorders of lipid metabolism. The overexpression of sortilin in cells increases the binding of low-density lipoprotein cholesterol surface and the absorption of sortilin, which is localized in the plasma membrane, and deficiency, which leads to impaired cell surface interaction between sortilin and low-protein lipoprotein

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 INFARCTION

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