ГАСТРОЕНТЕРОЛОГІЯ
GASTROENTEROLOGY

УДК 616.33+613.06.055.1 DOI: https://doi.org/10.22141/2308-2097.54.4.2020.216717

Тези науково-практичної конференції з міжнародною участю «VIII наукова сесія Інституту гастроентерології НАМН України. Новітні технології в теоретичній та клінічній гастроентерології» (26–27 листопада 2020 року)

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The intensity of lipid destress syndrome in patients with non-alcoholic fatty liver disease on the background of obesity and chronic kidney disease

The purpose of the study was to find out the likely interaction of the blood lipid profile on the clinical course of non-alcoholic fatty liver disease (NAFLD) on the background of obesity, depending on its form and the presence of comorbid chronic kidney disease (CKD).

Material and methods. 384 patients with NAFLD were examined: 84 of them were NAFLD with obesity 1st degree (1 group), which contained 2 subgroups: 32 patients with non-alcoholic steatosis (NAS) and 52 patients with non-alcoholic steatohepatitis (NASH); 270 patients with NAFLD with comorbid obesity of the I degree and CKD I–III stage (group 2), including 110 patients with NAS and 160 patients with NASH. The control group consisted of 90 patients with of the I–III stage with normal body weight (group 3). To determine the dependence of the NAFLD course on the form and stage of CKD, the group of patients was randomized according to age, sex, degree of obesity, and activity of NASH. The average age of patients was (45.80 ± 3.81) years.

Results. Significant metabolic prerequisites for the development of NASH against the background of obesity and CKD are likely postprandial hyperglycemia, hyperinsulinemia, increase in the degree of glycosylation of hemoglobin, the primary tissue insulin resistance. The reason for the progression of the metabolic syndrome on the background of NASH and CKD is lipid distress syndrome with an increase in blood total cholesterol, proatherogenic LDL, HDL antiatherogenic deficiency. The leading role in the development and progression of steatohepatitis is the disorders of the hepatic circulation that results in an TG increase in blood.

Conclusions. Thus, the development of NASH in patients with CKD and obesity is accompanied by a significant disorder of hyperlipidemia with the highest among the groups compared with the increase in the content of cholesterol and low density proatherogenic lipoproteins, the probable decrease in anti-atherogenic high-density lipoproteins and the increase in the atherogenicity index.

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The fibrinolytic activity of blood in pathogenesis of non-alcoholic fatty liver disease with obesity and chronic kidney disease

The purpose of the study was to establish the features of changes fibrinolytic activity of blood in patients with non-alcoholic fatty liver disease (NAFLD) with comorbid obesity and chronic kidney disease (CKD).

Material and methods. 444 patients were examined: 84 of them were with NAFLD and obesity I degree (group 1), which contained 2 subgroups: 32 patients with non-alcoholic steatosis (NAS) and 52 patients with non-alcoholic steatohepatitis (NASH); 270 patients with NAFLD with comorbid obesity of the I degree and CKD I–III stage (group 2), including 110 patients with NAS and 160 patients with NASH. The control group consisted of 90 patients with CKD of I–III stage with normal body weight (group 3).

Results. The study of fibrinolytic activity of blood showed that total fibrinolytic activity (TFA) of blood plasma in patients of all groups was significantly lower than the control indexes: in patients with NAS — by 7.1 %, patients with NAS with CKD — by 14.9 %, patients with NASH — by 17.2 %, patients with NASH with CKD — by 18.9 %, patients with CKD — by 10.6 % ($p < 0.05$) with the presence of a probable intergroup difference between groups with comorbidity and

isolated course of CKD ($p < 0.05$). The suppression of TFA occurred at the expense of the decrease of EF: in patients with NAS the index is significantly lower than the control in 1.2 times, in patients with NAS with CKD — in 1.4 times, in patients with NASH — in 1.7 times, in the group of patients with NASH and CKD — by 1.9 times, while in the group of patients with CKD, the suppression of EF was registered — 1.3 times ($p < 0.05$). At the same time, the NEF in patients of all groups increased in comparison with the PHP group: in patients with NAS, in 1.2 times, in patients with NAS with CKD — in 1.3 times, in patients with NASH — in 1.4 times, in the group of patients with NASH with CKD — 1.5 times, while in the group of patients with CKD the activation of NEF was registered 1.2 times ($p < 0.05$), with the presence of a probable difference between the groups with comorbidity and isolated course of CKD ($p < 0.05$).

Conclusions. Analysis of hemostasis and fibrinolysis indices in examined patients with NASH, depending on the stage of CKD showed that with the growth of the CKD stage, the activity of the cohort increases, with the exception of the fibrinogen content (most likely due to coagulopathy consumption), the activity of the anticoagulants decreases, the total and enzymatic activity of fibrinolysis is reduced, and non-enzymatic compensator increases. Thus, metabolic intoxication, oxidative stress, which accompany the flow of NAFLD with obesity and CKD, promote the activation of the kallikrein-kinin system, the formation of plasma and thrombin, with subsequent disturbance of equilibrium between them, the development of stasis, slag phenomenon, the formation of platelet and erythrocyte aggregates in blood circulation system. The consequence of significant activation of hemocoagulation against the suppression of total fibrinolytic activity (TFA) is the local clotting of blood in the arteries.

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The features of biochemical markers of liver fibrosis with non-alcoholic steatohepatitis in patients with I–II degree obesity and chronic kidney disease I–III stage

The purpose of the research — to find out the features of biochemical markers of liver fibrosis with non-alcoholic steatohepatitis in patients with I–II degree obesity and chronic kidney disease I–III stage, to establish the effectiveness of Heparhizine influence on the state of carbohydrate-protein components of the connective tissue of the extracellular matrix of the liver and kidneys.

Material and methods. 98 patients with non-alcoholic steatohepatitis on the background of I–II degree obesity were examined: 52 patients with non-alcoholic steatohepatitis (1st group) (without accompanying chronic kidney disease), 46 patients with non-alcoholic steatohepatitis with a comorbid chronic kidney disease I–III stage (2nd group). The control group consisted of 20 practically healthy persons (PHPs) with the corresponding age and sex. Biopsy of the liver was performed on 32 patients with non-alcoholic steatohepatitis with the accompanying of chronic kidney disease I–III stage, 28 patients with non-alcoholic steatohepatitis without chronic kidney disease. Patients on both groups of non-alcoholic steatohepatitis received Heparhizine treatment (glycyrrhizin 40 mg, glycine 400 mg, L-cysteine hydrochloride 20 mg) (Valartin Pharma) by intravenous administration of 20 ml of the drug for 10 days followed by enteral administration of 2 tablets of Heparhizine (1 tablet: glycyrrhizin 25 mg, glycine — 25 mg, methionine — 25 mg) 3 times a day for 80 days. Patients with non-alcoholic steatohepatitis with a comorbid flow of non-alcoholic steatohepatitis, obesity and chronic kidney disease of the I–III stage, except heparisin, they received baseline therapy of chronic kidney disease I–III stage: chronic pyelonephritis (course of antibacterial drugs, uroseptics, cainfron). The examinations were carried out prior to treatment and on the 90th day of treatment.

Results. The study showed that in the case of non-alcoholic steatohepatitis that develops on the background of obesity and chronic kidney disease on the I–III stage, the presence of fibrotic changes in the liver tissue was established, which according to the biochemical index of fibrosis, exceeds those in patients with non-alcoholic steatohepatitis without comorbidity with kidney pathology. in patients with non-alcoholic steatohepatitis, which was accompanied by obesity, a significant increase in the synthesis of collagen and glycosaminoglycans which was accompanied with an ineffective resorption of newly formed collagen due to inhibition of the collagenolytic activity of blood plasma, due to significant activation of proteinase inhibitors (α2-MG) was observed with a significant imbalance in the system of connective tissue metabolism.

Conclusions. Under the conditions of the comorbidity of non-alcoholic steatohepatitis with chronic kidney disease I–III stage, collagen synthesis and resorption are activated, but the anabolism processes predominate, in spite of the compensatory activation of collagenolysis, a substantial hyperproduction of actinic-phase proteins, fibronectin, glycosaminoglycans, fibroblast growth factor and lead to progressive fibrosis of the liver and disturbance of its functions.

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The pathogenetic features of nonalcoholic steatohepatitis course with obesity and chronic kidney disease

The purpose of the study was to determine the pathogenetic role of the bacterial endotoxin content in the blood on the hepatocytes damage markers, the degree of steatosis and liver fibrosis in patients with NASH with obesity, depending on the form and stage of CKD and their progression.

Materials and methods. To realize this goal 170 patients with NASH aged 40–55 years were examined. All patients were distributed as follows. Group 1 consisted of 70 patients with NASH with concomitant obesity 1st degree.