distributed as follows: 1 group - to 3 subgroups: CKD of the I stage - 51 patients, CKD of the II stage - 53 patients, CKD of the III stage - 41 patients. The 2nd group was divided into 3 subgroups: CKD of the I stage - 32 patients, CKD of the II stage - 35 patients, CKD of the III stage - 28 patients. The control group consisted of 30 practically healthy individuals (PHPs).

The analysis of kidneys functional state indicators showed that the creatinine content in blood in patients of group 1 exceeded the data in the PHPs in 1,5 times ($p < 0,05$), in 2 groups - in 1,3 times ($p < 0,05$). Accordingly, in patients with CKD of the III stage group 1 the creatinine exceeded the data in PHPs by 2.3 times ($p < 0.05$), in group 2 - by 1.9 times ($p < 0.05$). Thus, comorbidity with NASH significantly affects the kidneys functional state indicators, in particular, their nitrogen-excretory function. Thus, the content of blood urea in patients with CKD I stage exceeded the indicators in PHPs, respectively, in 1 and 2 groups - in 2,4 and 2,2 times ($p < 0,05$). In patients with CKD II stage in group 1 the urea content exceeded the index in PHPs by 2.5 times compared with 2.4 times in group 2 ($p < 0.05$). As a result of the established changes, a significant decrease in GFR (Glomerular filtration rate) was obtained for creatinine clearance using the Cockroft-Gaulta formula. Thus, the indicator of creatinine clearance by the Cockroft-Gaulta formula in patients with CKD I stage was lower than that in PHPs only in group 1 patients (11.8%) ($p < 0.05$); in patients of group 2, changes were unlikely and no significant difference was found between the groups ($p > 0.05$). In patients with CKD II stage in group 1, the creatinine clearance score was lower than the PHPs index by 39.2% versus a decrease of 25.5% in group 2 ($p < 0.05$) with a confirmation of statistically significant difference between the groups ($p < 0.05$). At the same time, patients with CKH III stage, the rate of creatinine clearance in patients in group 1 was lower than the normative at 55.9% ($p < 0.05$), in group 2 - by 44.1% ($p < 0.05$), with the presence of a probable difference between patients with a combined course NASH and CKD in comparison with patients with CKD without comorbid diseases ($p < 0,05$).

Non-alcoholic steatohepatitis significantly aggravates the course of chronic kidney disease of I-III stages with a possible decrease in nitrogen excretory function, glomerular filtration rate, hypopaluminemia than in the isolated course of chronic kidney disease.

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CONDITION OF HEMOSTASIS SYSTEM IN PATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE AND CHRONIC PANCREATITIS

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We observe the comorbidity during COPD and chronic pancreatitis (CP) quite often, which is due to the presence of a number of pathogenetic mechanisms of mutual encumbrance. It can be assumed that the comorbidity course of COPD and CP can enhance the clinical symptoms of both diseases and lead to frequent relapses of the pathological process.

Objectives – to establish the features of some indicators of hemocoagulation hemostasis in patients with COPD and concomitant CP. 60 patients were examined, including 15 patients with COPD (GOLD 2, B) with an isolated course (group 1), 15 patients with COPD (GOLD 2, B) with accompanying CP in the acute phase (group 2), and 15 patients with CP with the isolated course (group 3). The mean age of the patients was 46.2±4.3 years. The control group consisted of 15 practically healthy individuals (PHI) of the appropriate age and gender.

Analysis of results of studying the 2nd phase of coagulation hemostasis showed that prothrombin time (PTT) was significantly reduced in all observation groups. The maximum similar decline in the indices was observed in patients of group 2 – by 39.5% compared to the index in the PHP ($p < 0.05$) in the absence of intergroup differences; in patients of group 1 PTT decreased by 19.5% compared with those in PHI; and in patients of group 3 there was a decrease of PTT by 30.9% ($p < 0.05$). Studying the 3rd phase of coagulation hemostasis considering the content of fibrinogen in the blood suggests that in patients of all observation groups this figure was significantly reduced: in patients of the 1st group – by 11.0%, group 3 – by 17.5%, group 2 – by 26.6% and it was significantly different when compared in the intergroup aspect ($p<0.05$). While

analyzing the blood anticoagulant potential we found a reduction in thrombin time in all groups of patients with the highest percentage of decline in the patients of group 2 – by 37.6% ($p < 0.05$) compared with group of PHP, but in the patients of group 1 thrombin time decreased reliably too by 21.8%, in group 3 by28.2% with the reliable difference between groups 1, 2 and group 3.

The intensity of plasma proteolysis processes in inflammatory conditions tends to increase in a variety of internal pathology and is controlled by a number of tissue and plasma proteinase inhibitors (α2-macroglobulin, α1-proteinase inhibitor, Antithrombin III, etc.). An imbalance of these systems can lead to a predominance of protein catabolism processes that perform structural (components of cell membranes, coagulation hemostasis) and transport functions that is also a powerful disturbing factor. Analysis of the study results shows that all patients with COPD had an increase in the intensity of lysis of low-molecular proteins ($p<0.05$), while in patients of group 1, ILAA exceeded the indicator in the PHI 1.3 times, and in groups 2 and 3 – 1.6 times. Maximum activation rates of systemic proteolysis were recorded in COPD patients with concomitant CP, and minimal – in patients with isolated COPD. That is, the raising of plasma proteolytic activity due to an inflammatory process in the pancreas is a predictor of COPD progression for this comorbidity.

Finding of a more pronounced decrease fibrinogen in blood of the patients suffering from COPD with concomitant CP is indicative of consumption coagulopathy, that is, the use of fibrinogen in the process of intravascular coagulation with simultaneous depletion of the circulating pool of the I factor. Defined suppression of the activity of the anti-coagulation system factors and enzymatic, Hageman-factor-dependent fibrinolysis indicates the formation of hypercoagulation syndrome in chronic obstructive pulmonary disease patients with accompanying chronic pancreatitis.

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THE ROLE OF BONE DISORDERS IN CHRONIC KIDNEY DISEASE AND SYSTEMIC CONNECTIVE TISSUE DISEASES PROGRESSION, EVALUATION AND THERAPEUTIC APPROACHES

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Chronic kidney disease (CKD) is defined as a structural or functional kidney abnormality lasting for 3 or more months. The global prevalence of CKD is estimated to be more than 10%, and CKD has emerged as a public health problem. Adverse outcomes of CKD such as kidney failure, cardiovascular disease, and premature death can be prevented or delayed when treatment is initiated in the early stages of disease. As the earlier stages are often asymptomatic, CKD is usually detected during laboratory evaluation of comorbid conditions.

Chronic kidney disease (CKD) and systemic connective tissue diseases (CTD) are systemic disorders that leads to vascular calcification and accelerated progression. Uric acid has been shown to associate with vascular calcification and with carotid intima-media thickness (CIMT) and to suppress the 1 α-hydroxylase enzyme leading to lower 1,25-dihydroxyvitamin D (1,25(OH)2D) and higher intact parathyroid hormone (iPTH) levels.

These data suggest that factors other than uric acid may play a more important role in the regulation of CKD- CTD including vascular calcification and vitamin D metabolism in patients with CKD.

Thus, the authors present and discuss available data regarding potential role of hyperuricaemia, hyperphosphatemia in CKD-CTD incidence and progression. Possible therapeutic approaches are also being discussed.