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.