

**МІНІСТЕРСТВО ОХОРОНИ ЗДОРОВ'Я УКРАЇНИ
БУКОВИНСЬКИЙ ДЕРЖАВНИЙ МЕДИЧНИЙ УНІВЕРСИТЕТ»**



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STATE OF THE CYTOKINE LINK ACCORDING TO IL-18 AND IL-10 IN PATIENTS WITH OSTEOARTHRITIS IN COMBINATION WITH CHRONIC PANCREATITIS

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Introduction. Osteoarthritis is one of most world wide known damage of joints. About 60% of all arthritis is known as osteoarthritis. Approximately 20% of all gastrointestinal pathology is chronic pancreatitis. This makes this comorbidity very actual.

The aim of the study. To evaluate the state of the cytokine link, based on IL-18 and IL-10 indicators, in patients with chronic pancreatitis in combination with osteoarthritis, since pro-inflammatory and anti-inflammatory cytokines should balance the immune homeostasis.

Material and methods. 52 patients, aged from 37 to 65 years, were examined, including 38 women and 14 men. An average duration of chronic pancreatitis was 14.9 years and osteoarthritis was 8.1 years. The group of practically healthy individuals consisted of 10 people. The study of interleukin 10 and 18 was carried out by the Platinum ELISA solid-phase immunoenzyme method using the appropriate kits.

Results. Patients with chronic pancreatitis were dominated by exacerbation of osteoarthritis. At the same time, the clinical manifestation was characterized by pain during active and passive movements, in the morning or after significant overloading, limitation of the amplitude of movements in the joints, their deformation due to proliferative changes, which was indicative of osteoarthritis advance. Analysis of the results of IL-18 indicators showed a tendency to increase in 18 patients, the indicators were significantly increased in 34 patients (compared to the group of practically healthy people ($p < 0.001$)). They were correlated with indicators of malondialdehyde and CRP ($r = 0.52$; $p < 0.05$). Indicators of IL-10 (which is an antioxidant cytokine) in 18 patients did not exceed those in practically healthy ones, which can be considered as reflection and compensation of immune response processes. In the second group, they significantly increased in comparison with the group of practically healthy individuals ($p < 0.001$). Increase in IL-18 and IL-10 indicators can simultaneously indicate the activation of the cytokine defense system. Such a reaction can be considered as aimed at compensation in the new conditions of the course of chronic systemic inflammation.

Conclusions. The immune system in patients with chronic pancreatitis and osteoarthritis is aimed at compensation, balance between the processes of activation of the cytokine chain and the processes of protection (according to IL-18 and IL-10 indicators), ensuring the permanence of low-grade chronic inflammation.

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COVID-19 AND CHRONIC KIDNEY DISEASE

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Introduction. The recent outbreak of COVID-19 is spreading rapidly on a global scale. To date, there are no effective treatments for COVID-19, raising concerns about the impact of risk factors, such as clinical course, pathophysiological parameters, and the presence of comorbidities, on disease severity and treatment outcome in patients with COVID-19.

The aim of the study was to conduct a meta-analysis to examine the relationship between existing chronic kidney disease (CKD) and disease severity in patients with COVID-19.

Material and methods. Analysis of the published clinical data available.

Results. We identified several key clinical characteristics associated with increased disease severity and mortality among patients with COVID-19. In particular, pre-existing chronic conditions such as hypertension, cardiovascular disease, chronic kidney disease and diabetes are strongly associated with an increased risk of severe COVID-19. Chronic kidney disease and acute kidney injury are strongly correlated with increased disease severity in patients with COVID-19.

A recent clinical study of 59 patients with COVID-19 found that 32 of 51 patients (63%) had proteinuria, an indicator of impaired kidney function. Regarding other renal parameters, the authors also found that 19% and 27% of patients with COVID-19 had elevated levels of plasma creatinine and urea nitrogen, and CT scans showed that 100% of the 27 patients with COVID-19 examined had impaired kidney function. Another study by Zhejiang University School of Medicine involving 52 patients with COVID-19 (20 survivors and 32 non-survivors) found that 15 patients (29%) presented with acute renal failure. In general, almost 9.4% of critically ill patients were hospitalized with SARS-CoV-2 (55 out of 585 patients) had CKD. Taken together, these data indicate that renal function should be carefully monitored during treatment of patients with COVID-19, especially in patients with pre-existing CKD and/or abnormal levels of serum creatinine, blood urea nitrogen, and proteinuria. In addition, early continuous renal replacement therapy is indicated in the treatment of patients with severe symptoms such as hyperkalemia, acidosis, electrolyte imbalance, and acid-base imbalance in patients with COVID-19.

Conclusions. Summarizing the results of our analysis, it should be noted that patients with chronic renal failure have an increased risk of developing a severe form of COVID-19.

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ENDOTHELIAL DYSFUNCTION AS A FACTOR IN THE PROGRESSION OF CHRONIC CHOLECYSTITIS IN PATIENTS WITH CORONARY HEART DISEASE

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Introduction. Chronic inflammatory diseases are associated with accelerated development of atherosclerosis and increased risk of cardiovascular diseases. As the pathogenesis of atherosclerosis is increasingly recognized as an inflammatory process, the similarity between atherosclerosis and systemic inflammatory diseases such as inflammatory bowel disease, including chronic cholecystitis, has become a topic of current interest to many researchers. Endothelial dysfunction is a key step in the initiation and acceleration of atherosclerosis and may serve as a marker for future risk of cardiovascular events. In patients with chronic inflammatory diseases, endothelial dysfunction is often detected in the early stages of the disease. Thus, the mechanisms linking systemic inflammatory diseases and atherosclerosis can be best understood based on the study of numerous markers of endothelial dysfunction that directly or indirectly activate endothelial cells, leading to impaired vascular relaxation, increased leukocyte adhesion, increased endothelial permeability and generation of a pro-thrombotic state.

The aim of the study. The study aimed to determine the degree of development and the role of endothelial dysfunction in the development and progression of chronic cholecystitis (CC) in patients with coronary heart disease (CHD) and obesity.

Material and methods. 136 patients were examined: Group 1 (n=28) - CC; Group 2 (n=30) - CC against the backdrop of CHD; Group 3 (n=30) - CC against the backdrop of CHD and 1-2 grade obesity; Group 4 (n=30) - CC, cholesterosis of the gallbladder (CG), IHD, obesity 1-2 grade; Group 5 (n=18) - CC and CG. The functional state of the endothelium was studied by blood levels of stable metabolites of nitrogen monoxide (NO), the activity of endothelial (eNOS) and inducible (iNOS) NO-synthase and endothelin-1 (ET-1) by ELISA.

Results. Results of the study showed that 97,8% of examined patients with CC found a significant increase in the content of stable NO metabolites in the blood ($p<0,05$). Patients of the 3rd group experienced substantial growth content of NO in the blood (2,4-fold) compared to the 1st group (1,9-fold) and 2nd group (1,6-fold) ($p<0,05$). It was established that the intensity of stress increased as a result of the addition of coronary heart disease and obesity with CC and cholesterosis (an increase of 2,8 times to 2,1 times, $p<0,05$). In the 4th group: the content of NO in blood exceeded compared to the 1st group by 17,7% ($p<0,05$). The 4th group revealed the most pronounced indicators: overproduction of iNOS (5.2-fold increase) and eNOS deficit (down by 53,0%) ($p<0,05$).