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Матеріали 101 – ї підсумкової наукової конференції професорсько-викладацького персоналу вищого державного навчального закладу України «Буковинський державний медичний університет» (м. Чернівці, 10, 12, 17 лютого 2020 р.) – Чернівці: Медуніверситет, 2020. – 488 с. іл.

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У збірнику представлені матеріали 101 – ї підсумкової наукової конференції професорсько-викладацького персоналу вищого державного навчального закладу України «Буковинський державний медичний університет» (м.Чернівці, 10, 12, 17 лютого 2020 р.) із стилістикою та орфографією у авторській редакції. Публікації присвячені актуальним проблемам фундаментальної, теоретичної та клінічної медицини.

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terminal stage. A large number of researches have shown that circulatory dysfunction plays a key role in the pathophysiology of hepatorenal syndrome. In recent years it has become increasingly apparent that cirrhosis is a condition with a pronounced systemic inflammatory response, which increases with progression of the disease and strongly influences the patient's prognosis. Some clinical studies have evaluated inflammation in cirrhosis by measuring leukocyte counts or C-reactive protein levels; however, these methods are not accurate enough to evaluate the inflammation. In addition, studies on experimental animals have also demonstrated the presence of systemic inflammatory response syndrome, which is more noticeable in animals with ascites in comparison with others. Therefore, it has been hypothesized that cirrhosis is a disease that is accompanied by significant and progressive inflammation, which plays an important role in the development of complications. However, for the present, there is insufficient evidence regarding the presence, extent, and significance of inflammation in patients with hepatorenal syndrome, but such information may be relevant not only for pathogenesis but also for identifying potential therapy targets to prevent disease progression.

The objective of this research was to assess the level of markers of the systemic inflammatory response syndrome in patients with hepatorenal syndrome against a background of alcoholic liver cirrhosis, depending on the type of hepatorenal syndrome and presence or absence of infectious complications.

To achieve this goal, 165 patients with decompensated alcoholic liver cirrhosis were examined and divided into three groups according to the renal function: group 1 - alcoholic liver cirrhosis without hepatorenal syndrome (n=44), group 2 - alcoholic liver cirrhosis with hepatorenal syndrome type 2 (n=63), group 3 - alcoholic liver cirrhosis with hepatorenal syndrome type 1 (n=58).

Systemic inflammatory response syndrome prevalence, leukocyte counts, and C-reactive protein levels were higher in patients with hepatorenal syndrome type 1 compared to the other two groups. In the hepatorenal syndrome type 1 group, the cytokine profile had significantly higher levels of MCP-1 in urine and serum IL-6, TNF-α, VCAM-1, IL-8, and lower levels of MIP1-α and fractalkine. Of the 5 cytokines that were significantly elevated in patients with hepatorenal syndrome type 1, only plasma IL-6 was significantly higher in patients with hepatorenal syndrome type 1 associated with infections compared with patients without infections - 59,26±13,41 vs 23,15±11,34 pg/ml, respectively ($p<0,05$), which testifies to the hypothesis of hepatorenal syndrome as the cause of the development of systemic inflammatory response syndrome in this case.

It has been found that in patients with alcoholic liver cirrhosis complicated with hepatorenal syndrome, the levels of systemic inflammatory markers and cytokines are higher than in patients without such complications, that is evidence of a significant role of the inflammatory response in its pathogenesis. Increased levels of MIP1-α, MCP-1, IL-8 plasma, and uMCP-1 (pg/ml) urine can be used as a differential sign of hepatorenal syndrome type 1, being important in the choice of therapeutic tactics.

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BLOOD PRESSURE CHANGES AS A RESULT OF TAKING NONSTEROIDAL ANTI-INFLAMMATORY DRUGS AMONG THE PATIENTS WHO SUFFER FROM OSTEOARTHRITIS WITH CONCOMITANT HYPERTENSION

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An integral part in the treatment of patients with osteoarthritis (OA) is taking nonsteroidal anti-inflammatory drugs (NSAIDs). In many cases, NSAIDs as symptomatic drugs do not affect the fundamental pathogenic mechanisms underlying these processes. Due to the anti-inflammatory and pain relieving effects, they can be taken for a long time, but even short-term intake of NSAIDs at low doses can lead to the development of side effects, which on the whole are detected in about

35% of cases. There is a high risk of side effects among the patients with concomitant arterial hypertension (AH).

Objective: to study the changes in blood pressure (BP) among OA patients with hypertension within 24 hours during their treatment with NSAIDs.

Bibliographic, descriptive, medical statistical and sociological methods of research were used in this work.

According to 24-hour blood pressure monitoring, it can be assumed that associationof osteoarthritis with AH worsens the course of hypertension. Activation of the inflammatory process in the joints facilitates blood pressureincrease. Patients who have had the most pronounced inflammatory joint symptoms observed higher levels of average SBP and DBP. The degree of the surveyed patients' night-time reduction of blood pressure significantly differed. It was statistically lower among patients with osteoarthritis, than among patients who had no pronounced articular manifestations. This was accompanied by increased headache, cardialgia, discomfort with changes of weather and tendency to dizziness.

The results of the conducted medical researches indicate that the treatment of OA in many cases leads to the progression of hypertension. Thus, most nonselective NSAIDs are capable of causing an increase in blood pressure due to disorders in synthesis of a natural vasodilator - prostacyclin – in the daytime. On the contrary, the selective representatives of the rofecoxib group, nabumetone did not cause significant changes in blood pressure in the daytime, but caused a significant increase at night, which led to the leveling of physiological daily variation. There is also an increased risk of developing myocardial infarction among patients taking selective inhibitors of cyclooxygenase-2 (celecoxib) and some non-selective NSAIDs (ibuprofen, diclofenac).

The influence of NSAIDs on the antihypertensive effect among patients with arterial hypertension associated with osteoarthrosis of the knee joints is an urgent problem of the present and needs further research.

Sydorchuk L.P.

ALDOSTERONE SYNTHASE CYP11B2 (-344C/T) GENE POLYMORPHISM AS A POSSIBLE MARKER OF KIDNEY FAILURE DEVELOPMENT IN HYPERTENSIVE PATIENTS

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Nowadays the numerous genes encode the renin-angiotensin-aldosterone system (RAAS) activity and have pleiotropic effects on cardiovascular and metabolic disorders. Cytochrome 11B2 aldosterone synthase gene (CYP11B2) codes the activity of aldosterone synthase in the suprarenal adrenal cortex influencing the Aldosterone synthesis level and RAAS activity as well.

The aim of the study was to analyze the association of aldosterone synthase gene polymorphism (CYP11B2, -344C/T) with renal function in patients with primary arterial hypertension (PAH).

The study involved 100 patients suffering from PAH with a target-organ damaging, a moderate, high or very high cardiovascular risk. Among them there were 79.0% (79) women and 21.0% (21) men, whose average age was 59.87±8.02 yo. Case-control study included besides 48 practically healthy persons of relevant age ($p>0.05$). All enrolled / screened patients signed the Informed Consent to participate in the research. Kidney function was studied by the glomerular filtration rate (GFR) after Creatinine or Cystatin-C serum levels depending on gender. Chronic Kidney Disease (CKD) was determined by GFR decrease <60 ml/min/1,73m^2 with clinical course of renal functions impairment for ≥3 months. Aldosterone level was determined by Immuno-enzyme method ELISA. Gene polymorphism CYP11B2 (-344C/T) was evaluated by polymerase chain reaction.

The Creatinine and Cystatin-C serum levels as well as Aldosterone concentration were significantly higher in hypertensive patients with TT-genotype of CYP11B2 gene than in the