МІНІСТЕРСТВО ОХОРОНИ ЗДОРОВ'Я УКРАЇНИ
ВИЩИЙ ДЕРЖАВНИЙ НАВЧАЛЬНИЙ ЗАКЛАД УКРАЇНИ
«БУКОВИНСЬКИЙ ДЕРЖАВНИЙ МЕДИЧНИЙ УНІВЕРСИТЕТ»

М А Т Е Р І А Л И

101 – ї
підсумкової наукової конференції
професорсько-викладацького персоналу
Вищого державного навчального закладу України
«БУКОВИНСЬКИЙ ДЕРЖАВНИЙ МЕДИЧНИЙ УНІВЕРСИТЕТ»
10, 12, 17 лютого 2020 року

Чернівці – 2020

УДК 001:378.12(477.85)
ББК 72:74.58
М 34

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.

ББК 72:74.58

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.

ISBN 978-966-697-843-4

Khukhlina O.S.

THE CONTENTS OF CYTOKERATIN 18, ADIPONECTIN AND LEPTIN IN PATIENTS WITH COMORBID COURSE OF NONALCOHOLIC STEATOHEPATITIS AND CORONARY HEART DESEASE

Department of Internal Medicine, Clinical Pharmacology and Occupational Diseases
Higher state educational establishment of Ukraine
"Bukovinian State Medical University"

One of the most informative markers of the inflammation and liver fibrosis is the presence of the cytokeratin 18 (CK 18). CK 18 is a fragment of intermediate phylums of cells cytoskeleton, which is cut by the effector caspase 3 because of the process of apoptosis of hepatocytes. This peptide is detected in the blood even before the morphological signs of apoptosis occur. It allows to use CK-18 as a non-invasive biomarker of NASH.

The objectives of the study was to determine the level of cytokeratin 18, adiponectin and leptin in patients' plasma of with NASH and comorbid coronary heart disease (CHD) and their association with the degree of cytolysis, disorders of carbohydrate and lipid metabolism.

We examined 60 patients with NASH, which were divided into 2 groups: group 1 - 30 patients with NASH on the background of obesity of I-II degree, group 2 - 30 patients with NASH and comorbid CHD (the I and II functional classes of the stable angina pectoris) and obesity of I-II degrees. The average age of patients was 55.13 ± 4.34 years. The control group consisted of 20 practically healthy persons (PHP). Serum CK18, adiponectin and leptin levels were determined by the immunoassay analysis.

Our research showed that NASH is closely associated with disorders of adipocytokine homeostasis. In patients with NASH and comorbid CHD and obesity of I-II degree there is an increase level of CK 18, proinflammatory adipokine leptin and a decrease in the level of adiponectin.

Thus, the significant increase of liver aminotransferase activity and the level of CK 18 fragment in patients with NASH, their close positive correlation relationship suggest that the leading pathophysiological mechanism of progression of NAFLD is the necrosis and apoptosis of hepatocytes. The determination of serum fragments of CK 18 can be used as a non-invasive test for diagnostic of NASH and liver steatosis.

Kolodnitska T.L.

CURRENT VIEWS ON PATHOGENESIS OF THE ADVERSE PARTICULAR INFLUENCE OF ULTRADISPERSE PARTICLES ON THE CARDIOVASCULAR SYSTEM

Department of Internal Medicine, Physical Rehabilitation and Sports Medicine
Higher State Educational Establishment of Ukraine
"Bukovinian State Medical University"

Evidence on the health effects of ultrafine particles (UFP) is still limited as they are usually not monitored routinely. Organs that might be affected by fine and ultrafine particles are not only the lungs but also the cardiovascular system and other organs such as the brain. Although epidemiological studies by nature can never prove hypothesized pathways, the large number of studies forms a sound basis for evidence and the main pathways described are now widely accepted.

Aim: To analyze the current views on pathogenesis of the harmful effects of UFP on the cardiovascular system. Research methods: informational-analytical, content-analysis.

The analysis of the results showed that despite the fact that the precise mechanisms by which UFP affect the cardiovascular system are under study, several probable pathways have already been described. Thus, three generalized intermediate mechanisms through which UFP can affect the cardiovascular system have been proposed, but none of them can work separately, and the complex combination and interaction of the mechanisms is not yet fully understood.

These three main mechanisms are: Systemic inflammation: Numerous experimental and epidemiological studies have shown that inhalation of UFP causes increased accumulation and

activation of inflammatory cells (eg, neutrophils, T-lymphocytes, macrophages, mast cells) and the generation of large numbers of prooxide and / or proinflammatory mediators. Such mediators are cytokines (e.g, interleukin-6), acute-phase proteins (e.g, C-reactive protein and fibrinogen), vasoactive hormones (e.g, endothelin), and activated leukocytes, which can lead to endothelial dysfunction and pro-coagulation state with formation thrombus and progression of atherosclerotic lesions. Changing the balance of the autonomic nervous system: inhaled particles deposited in the pulmonary tree can directly stimulate the pulmonary reflexes through irritation of the receptors. This, in turn, may alter the balance of the autonomic nervous system (inhibition of the parasympathetic nervous system and / or activation of the sympathetic nervous system). These changes can also be indirectly caused by oxidative stress and inflammation in the lungs, or a combination of both. Changes in autonomic tone can contribute to instability of vascular plaques or initiate cardiac arrhythmias. Direct effect of UFP and / or soluble particle components in the bloodstream: after inhalation, these particles can quickly penetrate the bloodstream and directly affect the cardiovascular system. UFP or soluble compounds can provoke local inflammation and oxidative stress, as well as affect vascular endothelium and atherosclerotic plaques.

In recent years, a number of specific biological mechanisms have been proposed that can directly explain the triggering of cardiovascular events. These include vascular dysfunction or vasoconstriction, increased thrombosis or coagulation potential, increased blood pressure, progression of atherosclerosis, or platelet vulnerability and arrhythmia. For example, a major cause of coronary syndrome and cardiovascular death is damage to the atherosclerotic plaque and the formation of a blood clot. Therefore, air pollution and acute cardiovascular events may be related to changes in blood clot formation or vessel wall behavior.

Thus, at the present stage, there are three main mechanisms of UFP influence on the cardiovascular system: systemic inflammation, change in the balance of the autonomic nervous system, the direct effect of UFP on the vessel wall.

Kotsubiychuck Z.Ya.

METHOD OF BLOOD PRESSURE CORRECTION IN PATIENTS WITH NON-ALCOHOLIC STEATOHEPATITIS AND DIABETIC NEPHROPATHY AGAINST OBESITY

Department of Internal Medicine, Clinical Pharmacology and Occupational Diseases
Higher state educational establishment of Ukraine
"Bukovinian State Medical University"

Nonalcoholic steatohepatitis (NASH) is one of the major problems of internal medicine, has a general medical and social importance. Diabetic kidney disease - kidney damage in patients with type 2 diabetes, the evolution of which is characterized by the stability of functional and morphological changes in the kidneys, reduced GFR. Currently, the increase in the incidence of non-alcoholic fatty liver disease (NAFLD) and type 2 diabetes is that they are an essential component of metabolic syndrome and a risk factor for cardiovascular and dysmetabolic complications.

We aimed to investigate the effect of ramipril on blood pressure in patients with NASH, diabetic nephropathy and obesity in their comorbidities, as well as the efficacy of energy in different combinations of treatment in patients with comorbid course of these diseases.

40 patients with isolated NASH and with a comorbid course of diabetic nephropathy against obesity were examined. There were 2 groups of patients, each randomized by age, sex, degree of obesity, and cytolytic syndrome activity. The mean age of the patients was 45.1 ± 5.2 years. There were 28 men and 12 women. The main group (20 people) received a hypocaloric diet, metmorphine 500 mg twice daily, energies as a hepatoprotector 1 capsule 3 times a day, rosuvastatin 10 mg once a day as a lipid-lowering agent and ramipril 10 mg daily for 20 days. The control group (20 people) received a hypocaloric diet, metmorphine 500 mg twice daily, energies 1 capsule 3 times daily, rosuvastatin 10 mg daily and fosinopril 10 mg daily for 20 days.