

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



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101 – ї

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професорсько-викладацького персоналу

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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.

Gingulyak O.M.

PREGNANCY-ASSOCIATED PROTEIN-A AND C-REACTIVE PROTEIN IN PATIENTS WITH MANIFESTATIONS OF SUBCLINICAL ATHEROSCLEROSIS

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The proposed 2018 definition of clinical conditions in cardiology, which can serve as a manifestation of subclinical atherosclerosis, including asymptomatic patients at risk for coronary heart disease, atypical course, changing the development of acute coronary syndrome, long preclinical period against the background of confirmed coronary atherosclerosis cause a changes in diagnostic and treatment strategy according to the latest European guidelines.

Aim, to investigate the influence associated with pregnancy plasma protein -A (PAPP-A) and C-reactive protein (CRP) in the formation of subclinical atherosclerosis and in estimation of the change rate of intima-media (CIM), total ejection fraction and volume end-systole, total cholesterol, exercise tolerance and the comparison group, the initial level of the biomarker and the background of the treatment (n=23) for statin use and metabolic therapy (trimetazidine and magne -B6).

Examined 67 patients in the division into two groups with clinical manifestations of subclinical atherosclerosis and atypical clinic in terms of differential diagnosis in the distribution of vegetative- vascular dystonia coronary syndrome X, stable angina stress I-II functional class with an estimate levels of biomarkers (PAPP-A and CRP) to conduct clinical and functional review of all patients (methods of ECG, echocardiography, treadmill test, blood tests, including ELISA).

CIM indication decreased during treatment and surveillance in the general group (n = 67) (p <0,05) and the distribution of PAPP-A $\geq 4,12$ mIU/L (p <0,002), and observations determined initial increase in CIM by distribution PAPP-A $\geq 4,12$ mIU/L (p <0,001), which were stored and during treatment in the total group (n = 67) in the distribution of medium-sized CMMs for PAPP-A were in the treatment $\geq 4,12$ mIU/L (p <0,01) . In the group before /after treatment (n = 23) there was a decrease of-CIM during treatment in the group general (p <0,02), with a tendency to decrease CIM in the group where enlarged PAPP-A $\geq 4,48$ mIU/L (p >0,05) and reduced PAPP-A <4.48 mIU/L (p >0,05), and subclinical atherosclerosis (n = 46) registered a decrease CIM in the treatment group reduced PAPP-A (<4.54 mIU/L, p <0,01), but not in the group of increased PAPP-A ($\geq 4,5$ mIU/L, p >0.1). In his own study was found a significant decrease in the sum of CIM based content CRP in the group overall (n=67) during treatment (p <0,02) and at distribution of CRP $\geq 12,47$ mg/l was recorded a CIM reduction (p <0,005). The initial increase in CIM, which further decreases significantly in the treatment group (n=23) for the distribution of CRP <17, 11 \geq mg/dL (p <0,02), also significantly reduce CIM consistent for CRP in the treatment group PSA $\geq 12,47$ mg/L (p <0,005), as well as in atherosclerosis group for CRP (<16,55 \geq mg/l) with decreasing rate CIM (p <0,05).



Thus, CIM index decreased during treatment and surveillance in the general group (n=67) ($p < 0,05$) and the distribution of PAPP -A $\geq 4,12$ mIU/ L ($p < 0,002$), for a specified output increase by CIM distribution PAPP -A $\geq 4,12$ mIU/L ($p < 0,001$), which were stored and during treatment in the total group (n=67) in the distribution of average CMM for PAPP -A in the treatment of $\geq 4,12$ mIU/L ($p < 0,01$). The initial increase in CIM, which further decreases significantly in the treatment group (n=23) for the distribution of CRP $< 17,11 \geq$ mg/l ($p < 0,02$), also significantly reduce CIM consistent for CRP in the treatment group PSA $\geq 12,47$ mg/l ($p < 0,005$), as well as in atherosclerosis group for CRP ($< 16,55 \geq$ mg/l) with decreasing rate CIM ($p < 0,05$).

Honcharuk L.M.

**VASOMOTION FUNCTION OF ENDOTHELIUM IN CASE OF
GASTRODUODENOPATHY INDUCED BY TREATMENT OF PATIENTS WITH
OSTEOARTHRISIS BY NONSTEROIDAL ANTI-INFLAMMATORY DRUGS**

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Osteoarthritis is an acute medical and social problem resulting in temporary incapacity to work, disability and significant lowering of patients' life quality. As of today the main method of osteoarthritis treatment is use of nonsteroidal anti-inflammatory drugs. According to the references 46,5% of side effects in Ukraine are result of nonsteroidal anti-inflammatory drugs use. Gastric erosions and ulcers induced by nonsteroidal anti-inflammatory drugs use are one of the most frequent causes of admission to in-patient surgery and gastroenterology departments in Europe and the USA. That is why study of some pathogenetic action mechanisms leading to gastroduodenopathy development caused by nonsteroidal anti-inflammatory drugs use in case of patients ill with osteoarthritis is topical for modern medicine. The object of this study was to determine endothelium vasomotion function in case of gastroduodenopathy induced by treatment of patients ill with of osteoarthritis by nonsteroidal anti-inflammatory drugs.

50 patients were examined for osteoarthritis with concomitant gastroduodenopathy induced by nonsteroidal anti-inflammatory drugs use. The patients' distribution has been performed irrespective of *Helicobacter pylori* presence and/or alimentary tube affection degree. All patients underwent fibrogastroduodenoscopy with targeted biopsy performed by the standard technique using fibrogastroduodenoscopy device «Olympus» in order to diagnose gastroduodenopathy. The presence of *Helicobacter pylori* has been determined by means of invasive express analysis of urease tissue activity using diagnostic sets *Helpil*®-test ('AMA', Saint Petersburg), morphologic tests (using azur-II-eosin stain) as well as by means of immunochromatographic *Helicobacter pylori* antigen faeces test (CerTest Biotec, S.L., Spain, 'Pharmasco').

Vasomotion endothelium function has been studied by means of duplex ultrasonic scanning of brachial artery.

70,0% of patients ill with concomitant gastroduodenopathy induced by nonsteroidal anti-inflammatory drugs use were diagnosed with insufficient vasodilation. For instance, 46,0% of patients under examination were diagnosed with insufficient vasodilation, 24,0% of persons under examination were diagnosed with paradoxical vasoconstriction. It is to mention that endothelium shear stress and endothelium shear stress sensitivity during reactive hyperaemia test was decreasing more apparently in case of patients with concomitant *Helicobacter pylori* infection.

Thus, obtained results indicate development of endothelial dysfunction of patients ill with osteoarthritis in case of gastroduodenopathy induced by nonsteroidal anti-inflammatory drugs use. Concomitant *Helicobacter pylori* resulted in significant dysfunction of endothelium of patients ill with osteoarthritis with gastroduodenopathy induced by nonsteroidal anti-inflammatory drugs use.