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APPLICATION OF ULTRASOUND RENAL DOPPLER TO DETECT CHANGES IN RENAL HAEMODYNAMICS IN PATIENTS WITH COMORBIDITY OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE AND ESSENTIAL HYPERTENSION

Introduction. At the present stage of development of medicine and society, one of the most actual problems is the management of patients with comorbidities of several diseases. Comorbidity and polymorbidity are accompanied by a combination of risk factors and pathogenetic mechanisms involving other organs and systems of the body in the pathological process. This leads to accelerated disease progression, involvement of target organs and complications. The literature review and observation of cardiology and pulmonology patients have shown that essential hypertension (EH) and chronic obstructive pulmonary disease (COPD) are diseases that affect the kidneys through various pathomechanisms and lead to chronic kidney disease (CKD). Renal damage in patients with comorbidities is more advanced and clinically significant than in patients with monopathology of EH or COPD.

The aim of the study. Assessment of changes in renal blood flow at the level of the common trunk of the renal artery (CTRA), segmental branches of the renal artery (SRA) and interlobular branches of the renal artery (ILRA) in the comorbid presentation of essential hypertension (EH) stage II with chronic obstructive pulmonary disease (COPD) of stages II-III.

Materials and methods. The study included 60 patients who were divided into 3 clinical groups: group 1-15 patients with stage II essential hypertension (mean age 52.87±1.36 years; male- to-female ratio 73.33/26.67%); group 2-15 patients with stage II-III COPD (mean age 48.01±2.75 years; male-to-female ratio 86.67/13.33%); group 3 - 30 comorbid patients with COPD stage II-III and essential hypertension stage II (mean age 57.49±2.39 years, male-to-female ratio 76.67/23.33%), without signs of other clinically significant comorbidities, who did not receive systematic basic antihypertensive therapy. All groups were statistically comparable in gender proportion and demographic characteristics.

After a comprehensive clinical, laboratory and instrumental examination, it was found that the patients did not have clinically significant renal pathologies of any etiology.

A complex assessment of the patients' health status was carried out on the basis of a clinical objective examination, analysis of the results of questionnaires to determine the quality of life (SF-36), questionnaires to determine the impact of bronchial obstructive pathology on the quality of life (St George's Questionnaire, CAT), and interpretation of the results of routine laboratory and instrumental examinations. Verification of the diagnosis and determination of the degree of EH were based on 24-hour blood pressure monitoring with a CardioTens blood pressure monitor (ABPM). Verification of the diagnosis of COPD and determination of the stage of bronchial obstruction according to GOLD were performed using spirometry with a bronchomotor test on a «SPIROCOM» device. The presence of subclinical renal damage was confirmed by renal blood flow assessment using spectral Doppler US with a convex multifrequency sensor. Blood flow in the renal arteries was studied at the level of the common trunk of the renal artery (CTRA), segmental (SRA) and interlobar branches of the renal arteries (ILRA) by assessing linear velocity parameters. The following parameters were determined during spectral Doppler of the renal arteries: peak systolic flow velocity (V max.), end diastolic flow velocity (V min.), time-averaged maximum flow velocity (V avg.), resistance index (RI) and pulsatility index (PI), systolic-diastolic ratio (SDR).

Results and conclusions. The study of linear velocity indices and vascular resistance indices of renal vessels in the examined patients shows the most statistically significant reliable increase in the resistance index (RI) in the comorbid course of EH and COPD, especially at the level of segmental branches (IR in patients with comorbidity was 24.59% higher than in patients with monopathology of EH, 14.81% higher than IR in the group of patients with COPD and 36.96% higher than in the control group) and interlobular branches of the renal artery (IR elevation in patients with comorbidity of EH and COPD was 44.64% higher than in the EH group, 20.51% higher than in the COPD group, 96.77% higher than in the control group). A similar tendency to impaired renal blood flow was observed in the analysis of changes in the pulsatility index. PI values in the interlobular branches of the kidneys were statistically considerably higher in the EH and EH+COPD groups. The EH group showed an increase in PI by 46.67% compared with the COPD group, by 61.11% compared with the EH+COPD group, and by 157.14% compared with the control group. The EH+COPD group had higher values of the pulsatility index by 27.08% compared to the COPD group, by 194.29% compared to the control group. The study revealed that the minimum, average and maximum blood flow velocities progressively decreased in patients with EH, and especially in the EH+COPD group at all studied levels of the renal blood flow spectrum - interlobular branches, segmental branches and in the common trunk of the renal artery.

In summary, the analysis of parameters representing renal haemodynamics in EH and its comorbidity with COPD indicates the homogeneity and unidirectionality of haemodynamic vascular changes in cardiovascular pathology. A characteristic feature of renal blood flow changes in patients with comorbidity of EH+COPD is a marked increase in resistance and a decrease in linear blood flow parameters at the level of the microvascular vessel of the kidneys, which indicates an additional pathophysiological mechanism that is implemented in patients with bronchial obstruction - increased fibrosis of the renal parenchyma on the background of systemic inflammation and chronic hypoxia in patients with COPD. In conclusion, the combined potentiation of pathogenetic mechanisms in EH and COPD leads to the formation of rapidly progressive and clinically manifested renal dysfunction due to the simultaneous effect at the level of large and segmental branches, as well as at the level of small interlobular branches of the renal arteries.