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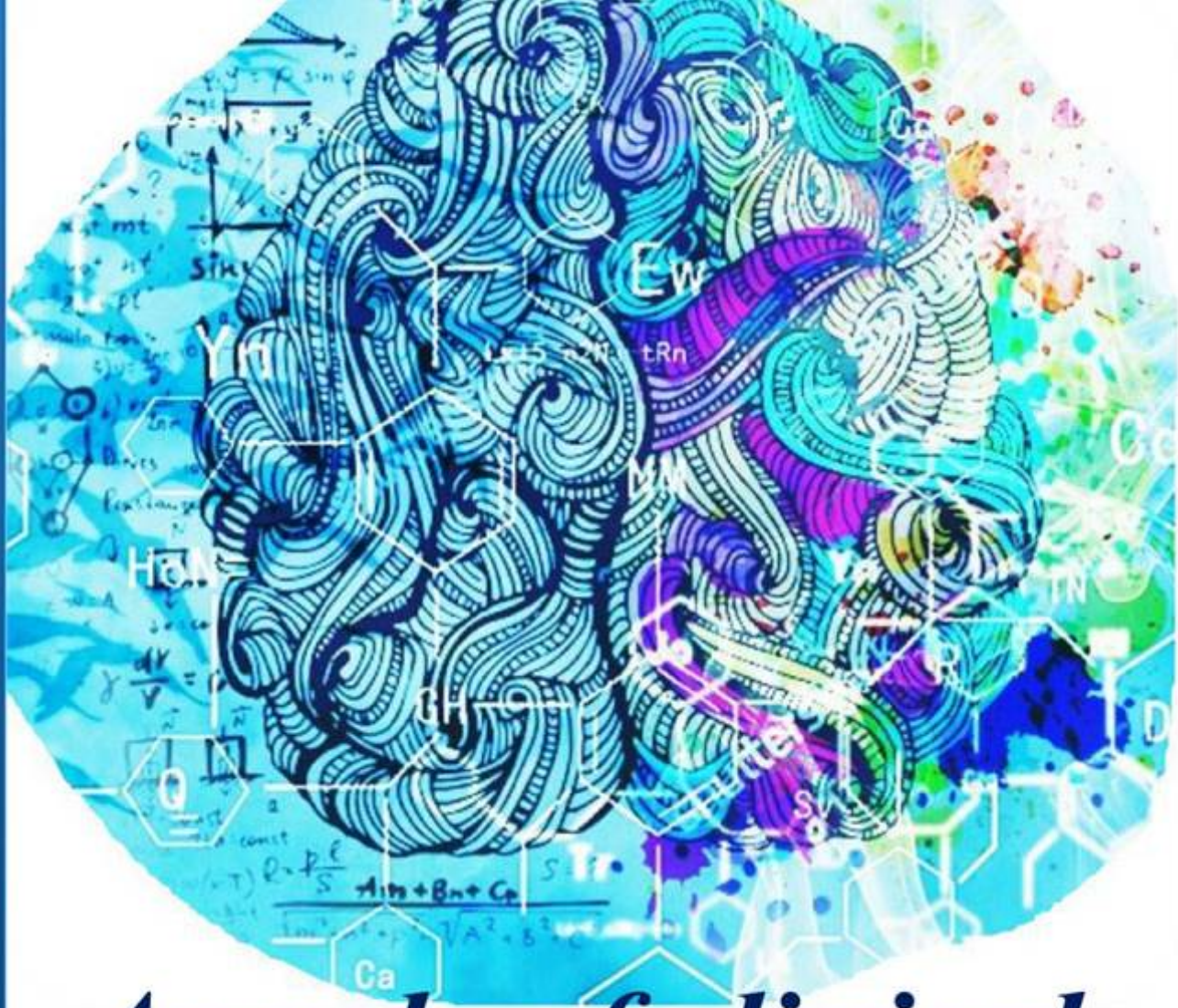

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CLINICAL AND FUNCTIONAL CHARACTERISTICS OF RENAL DYSFUNCTION IN PATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE IN COMBINATION WITH ARTERIAL HYPERTENSION

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Abstract. The clinical and functional characteristics of renal impairment in patients with chronic obstructive pulmonary disease combined with arterial hypertension were analyzed. Among patients with chronic obstructive pulmonary disease, left atrial hypertrophy was identified in 13.9% of cases without accompanying arterial hypertension and in 9.37% of cases when arterial hypertension was present as a comorbid condition. In the general group of patients, the hypertrophic type of left ventricular diastolic dysfunction occurs in 26.7% of cases. The observed tendency toward left atrial enlargement may be related to left ventricular diastolic dysfunction. In patients with concurrent chronic obstructive pulmonary disease and arterial hypertension, structural remodeling of the right heart chambers was detected significantly more often than in those without arterial hypertension (21.3% vs. 14%). This is due to the complex picture of structural changes in the right ventricle due to hypertrophy of the interventricular septum and its displacement towards the left ventricle. In addition, the frequency of ventricular diastolic dysfunction in a combination of chronic obstructive pulmonary disease and arterial hypertension is also significantly higher than in the group of patients without arterial hypertension (30.2% versus 51.1%). With the development of chronic obstructive pulmonary disease, the level of cystatin C and microalbuminuria increases significantly. A significant association was identified between the markers of renal dysfunction and parameters of cardiac remodeling, suggesting that hemodynamic factors contribute substantially to the development of chronic kidney disease in patients with chronic obstructive pulmonary disease. Elevated cystatin C levels observed in patients with chronic obstructive pulmonary disease are consistent with findings reported in previous studies. At the same time, such deviations may be associated with the systemic effect of obstructive pulmonary disease, including chronic hypoxia, inflammatory processes, and endothelial dysfunction.

Keywords: chronic obstructive pulmonary disease, myocardial remodeling, cystatin C, microalbuminuria, arterial hypertension, indicators of renal impairment, cor pulmonale, right ventricular failure.

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КЛИНИКО-ФУНКЦИОНАЛЬНАЯ ХАРАКТЕРИСТИКА ДИСФУНКЦИИ ПОЧЕК У БОЛЬНЫХ С ХРОНИЧЕСКОЙ ОБСТРУКЦИОННОЙ БОЛЕЗНЬЮ ЛЕГКИХ В КОМБИНАЦИИ С АРТЕРИАЛЬНОЙ ГИПЕРТЕНЗИЕЙ

Аннотация. Изучены клинико-функциональные характеристики почечной дисфункции у пациентов с хроническая обструктивная болезнь лёгких и артериальная гипертензия. У больных с ХОБЛ гипертрофия левого предсердия отмечалась в 13,9% случаев при отсуствии сопуствующей артериальной гипертензии и в 9,37% случаев при её наличии. В общей выборке пациентов гипертрофический тип диастолической дисфункции левого желудщчка выявлся в 26,7% случаев. Обнаруженная склонность к дилатации левого предсердия, вероятно, связана с нарушением диастолической функции левого желудочка. Частота ремоделирования правых отделов сердца в группе больных с сопутствующей хронической обструктивной болезнью легких и артериальной гипертензией достоверно выше, чем в группе больных без артериальной гипертензии (21,3 против 14%). Это связано со сложной картиной структурных изменений правого желудочка вследствие гипертрофии межжелудочковой перегородки и ее смещения в сторону левого желудочка. Кроме того, частота диастолической дисфукции желудочков при сочетании хронической обструктивной болезни легких и артериальной гипертензии также достоверно выше, чем в группе больных без артериальной гипертензии (30,2% против 51,1%). С развитием хронической обструктивной болезни легких значительно повышается уровень цистатина С и микроальбуминурии. Установлено, что существует корреляционная связь между этими маркерами дисфункции почек и параметрами ремоделирования сердца, что свидететельствует о том, что гемодинамический фактор вносит существенный вклад в развитие хронической болезни почек у пациентов с хронической обструктивной болезнью легких. Повышенные уровни цистатина С у пациентов с хронической обструктивной болезнью легких также подтверждаются данными других исследований. Вместе с тем, такие изменения могут быть обусловлены системным влиянием заболевания, включая хроническую гипоксию, воспалительные реакции и дисфункцию эндотелия.

Ключевые слова: хроническая обструктивная болезнь легких, ремоделирование сердца, цистатин С, микроальбуминурия, артериальная гипертензия, маркеры почечной дисфункции, легочное сердце, правожелудочковая сердечная недостаточность.

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O‘PKANING SURUNKALI OBSTRUKTIV KASALLIGI BILAN ARTERIAL GIPERTONIYA QO‘SHILGAN BEMORLARDA BUYRAK DISFUNKSIYASINING KLINIK VA FUNKSIONAL XUSUSIYATLARI

Annotatsiya. Surunkali obstruktiv o‘pka kasalligi va Arterial gipertenziya bilan og‘rigan bemorlarda buyrak disfunksiyasining klinik va funksional xususiyatlari ko‘rib chiqilgan. Surunkali

obstruktiv o‘pka kasalligi bilan og‘rigan bemorlarda yondosh arterial gipertenziyasiz chap bo‘lmacha gipertrofiyasi 13,9% ni, yondosh patologiya bilan esa 9,37% ni tashkil etishi aniqlandi. Bemorlarning umumiy guruhida chap qorincha diastolik disfunksiyasining gipertrofik turi 26,7% holatlarda uchraydi. Chap bo‘lmacha dilatatsiyasiga aniqlangan moyillik chap qorincha diastolik funksiyasining buzilishi bilan bog‘liq bo‘lishi mumkin. Surunkali obstruktiv o‘pka kasalligi va arterial gipertenziya birga kelgan bemorlar guruhida yurakning o‘ng bo‘limlari remodellanish chastotasi arterial gipertenziyasiz bemorlar guruhiga nisbatan sezilarli darajada yuqori (21,3% ga nisbatan 14%). Bu qorinchalararo to‘siq gipertrofiyasi va uning chap qorincha tomonga siljishi tufayli o‘ng qorincha strukturaviy o‘zgarishlarining murakkab manzarasi bilan bog‘liq. Bundan tashqari, surunkali obstruktiv o‘pka kasalligi va arterial gipertenziya kombinatsiyasida qorinchalar diastolik disfunksiyasi chastotasi ham arterial gipertenziyasiz bemorlar guruhiga nisbatan sezilarli darajada yuqori (51,1% ga nisbatan 30,2%). Surunkali obstruktiv o‘pka kasalligi rivojlanishi bilan sistatin C va mikroalbuminuriya darajasi sezilarli ravishda oshadi. Buyrak disfunksiyasining ushbu markerlari hamda yurak remodellanish parametrlari o‘rtasida korrelyatsion bog‘liqlik mavjudligi aniqlandi, bu esa surunkali obstruktiv o‘pka kasalligi bilan og‘rigan bemorlarda surunkali buyrak kasalligi rivojlanishida gemodinamik omil muhim hissa qo‘shishini ko‘rsatadi. Surunkali obstruktiv o‘pka kasalligida sistatin C darajasining oshishi boshqa mualliflar tadqiqotlari natijalari bilan ham mos keladi. Shu bilan birga, bunday og‘ishlar obstruktiv o‘pka kasalligining tizimli ta’siri, jumladan surunkali gipoksiya, yallig‘lanish jarayonlari va endotelial disfunksiya bilan bog‘liq bo‘lishi mumkin.

Kalit so‘zlar. o‘pkaning surunkali obstruktiv kasalligi, yurak remodellanishi, sistatin S, mikroalbuminuriya, arterial gipertenziya, buyrak disfunksiyasi markerlari, o‘pka yuragi, o‘ng qorincha yurak yetishmovchiligi.

According to the World Health Organization, 41 million people die annually from chronic non-communicable diseases, which accounts for 71% of all deaths in the world. Moreover, more than 85% of these “non-temporary” deaths occur in low and middle-income countries [34]. In the structure of mortality from chronic kidney disease (CKD), the largest share falls on cardiovascular diseases, of which 17.9 million people die annually. They are followed by respiratory diseases and diabetes mellitus (DM). Clinical-epidemiological and prognostic studies with type 2 diabetes show that for many years CKD has been in the shadow of chronic obstructive pulmonary disease (COPD) [3,4,41,59]. According to the literature, damage to the kidneys and lungs often occurs simultaneously. Thus, in 26% of patients with COPD, renal function is reduced, and in 60% of patients with CKD, obstructive sleep apnea syndrome is detected [49,60]. The development of COPD is influenced by such factors as rapid and unorganized urbanization, the globalization of an unhealthy lifestyle, and population aging.

Currently, the development of cardiac remodeling in COPD is linked to common underlying mechanisms such as pulmonary hypertension (PH) [1], increased activation of the sympathoadrenal system (SAS), the rennin-angiotensin-aldosterone system (RAAS), and systemic inflammation [9]. Earlier publications contain only limited data suggesting a high prevalence of these conditions. Likewise, previously published studies provide scarce evidence regarding the high prevalence of chronic kidney disease (CKD) in patients with COPD. The coexistence of arterial hypertension (AH) and chronic obstructive pulmonary disease (COPD) is a frequent comorbid condition, wiyh prevalence rates ranging from 6,8% to 73.3% and an average of 34.3% [3]. This combination negatively affects the clinical course, exacerbating the progression characteristic of each disease when considered separately [2]. Occurs second after kidney injury in hypertensive nephropathy.

One of the primary causes of chronic kidney disease (CKD) is diabetes mellitus [13]. Frequently, glomerular filtration rate (GFR) is estimated using creatinine as a laboratory marker of CKD, while cystatin C is less commonly employed as a biomarker of renal dysfunction (RD) and as a predictor of cardiovascular risk [4,5].

Hemodynamic factors contribute significantly to the development of chronic kidney disease (CKD) in patients with chronic obstructive pulmonary disease (COPD), remoting the onset of

cardiorenal syndrome and markedly aggravating disease progression. Consequently, investigating the role of hemodynamic factors in CKD development in COPD, as well as in the coexistence of COPD and arterial hypertension (AH), represents a highly relevant issue for prognosis assessment and the early prevention of renal dysfunction in this patient population. In chronic obstructive pulmonary disease (COPD), cardiac remodeling represents a characteristic aspect of disease progression. It is well established that in chronic pulmonary heart (CPH), structural changes occur not only in the right but also in the left heart chambers. These alterations are associated with increased stiffness of the right ventricular (RV) myocardium and the interventricular septum (IV), as well as interventricular interactions [6], in addition to toxic-allergic effects resulting from the frequent administration of various forms of beta-adrenomimetics and corticosteroids.

Purpose of the research. To identify the clinical and functional characteristics of renal dysfunction in patients with chronic obstructive pulmonary disease (COPD) and arterial hypertension (AH).

Materials and methods. A total of 134 patients with COPD stages I-IV, V, including 75 men and 59 women aged 47 to 78 years, were examined. Participants were divided into four clinical groups corresponding to disease stages and one control group (CG). The distribution was as follows: Stage I – 30 patients; Stage II – 32 patients; Stage III – 34 patients; Stage IV – 38 patients; CG – 18 individuals. Patients with diabetes mellitus, glomerulonephritis, systemic connective tissue diseases or post-infarction cardiosclerosis were excluded. All participants underwent comprehensive clinical assessment, including measurement of hemoglobin, serum creatinine (with calculation of of glomerular filtration rate, (GFR), uric acid, cholesterol, triglycerides and C-reactive preteine levels. The concentration of cystatin C in blood serum was measured using an immunoturbidimetric assay and the glomerular filtration rate (GFR) was calculated based on the Hooke formula. All patients underwent echocardiography (EchoCG) to evaluate cardiac function. Arterial hemoglobin oxygen saturation (CaO2, %) was assessed with a pulse oximeter. Assessment of external respiratory function was performed using a Spirograph device. The collected data were statistically analyzed with the Statistica 8.0 software package. Parametric variables were evaluated using Student's t-test and Pearson's chi-square test, while non-parametric data were analyzed with the Mann–Whitney U test and Spearman's rank correlation coefficient. A p-value of less than 0.05 was regarded as statistically significant.

Results and Discussion. Based on the analysis of medical history and physical examination findings, it was determined that patients had three or more risk factors for the development of CKD. Furthermore, a reduction in forced expiratory volume in one second (FEV1) was associated with the presence of microalbuminuria (MAU), elevated mean cystatin C levels and consequently, a decline in GFR calculated from cystatin C values.

The MAU levels in patients were significantly different ($p < 0.05$) compared to the control group (CG), with higher mean values observed in the control group than in patients with isolated COPD and those with COPD combined with hypertension. In the intergroup comparison, significant differences in improved GFR parameters based on creatinine and cystatin C were found only in patients with stage I and stage II COPD. ($p<0.05$).

In patients with stage III and IV with the development of bronchial obstruction, no significant difference ($p>0.05$) was found in GFR indicators for creatinine and cystatin C.

When determining GFR by creatinine at all stages of COPD, the overall frequency of cases of CKD of the first stage (C1) was 42.6%, the second stage (C2) - 56.2%, the third stage (C3) - 0.42%. When determining GFR using cystatin C, the frequency of CKD C1 was 16.7%, C2 - 42.7%, C3 - 35.4% and C4 - 1.05%.

According to echocardiography data, the left ventricular mass index was significantly higher in patients with renal dysfunction (108.4±12.6 g/m2 versus 132.7 ± 15.2; $p< 0.001$). The diameter of the left atrium was also greater (3.8±0.4 sm versus 4.4± 0.5; $p=0.002$). The E/A ratio for diastolic function indicators decreased (0.92±0.18 versus 0.71± 0.15; $p<0.01$) and the E/e index increased (9.8± 1.6 versus 14.2±2.4; $p<0.001$). Systolic pressure in the pulmonary artery was higher in the group with renal dysfunction (32.5±6.4 mm Hg versus 41.8± 8.1; $p<0.001$). The TAPSE indicator

was lower (19.4±2.1mm versus 16.8±2.5; p=0.01). In the correlation analysis, GFR showed a negative correlation with: left ventricular mass index (r=-0.58; p<0.001), E/e'(r=-0.62; p<0.001), pulmonary artery pressure (r=-0.49; p=0.003). A positive correlation was found with TAPSE (r=0.44; p=0.01). In the multivariate regression model, E/e' (β =-0.41; p=0.002) and pulmonary artery systolic pressure (β=-0.36; p=0.01) were identified as independent predictors of GFR (R2=0.52). A moderate positive correlation was found in patients with COPD without concomitant hypertension.

On one hand, a correlation was identified between MAU and cystatin C. On the other hand, a moderate negative association was observed between BPFVC, LVEF, SLAD, and DLA. Additionally, a moderate inverse relationship was found between the RV E/A and LV E/A ratios and LVEDD.

In patients with COPD combined with AH, cystatin C levels showed a positive correlation with VCVD and a negative correlation with left ventricular dimensions and AR E/A (Table 1). Furthermore, MAU demonstrated a direct association with OBQAT, CHQODQ, QATQ, SLAD and DLA, while inverse correlations were observed between MAU and OBQAT. These heterogeneous findings may reflect the specific features of cardiac remodeling occurring in the presence of AH.

Laboratory and anamnestic indicators of patients with COPD

Indicator		Stages of COPD				CG
		I	II	III	IV	
Age, years		51,38±8,96	54±12,4	62±11,57	63,26±9,7	40,94±11,8
M/W		17\10	17\12	22\14	18\11	10/7
BMI, kg/m2		25,06±3,67	27,9±10,6	27,57±4,02	24,67±6,04	22,9±4,27
Smoking, %		27,5	43,1	36	64	0
Hyperlipidemia, %		34	41,1	54,8	31	0
Hyperuricemia,%		32	58,9	48	54	0
Proteinuria, %		0	0	7	9	0
AH, %		13,3	25,3	57,8	24	0
OFV1, % (Me 25; 75)		77,25 (65,3; 81.4)	61,8 (49; 75)	42,25 (24; 82)	27,14 (17; 32)	91 (86; 100,5)
SatO2,% (Me 25; 75.)		97 (96; 98)	96 (95;97)	93 (92;96)	88 (86;91)	97 (96;99)
MAU, mg/l	AG siz	27,5±10,4	24±8,07	42,57±22,3	69±31,61	8,92±4,15
	AG	30,42±11,4	25,6±2,78	45,31±13,2	52,75±17,46	
Creatinin, mkmol/l	AG siz	62,44±7,37	72,6±16,2	81,4±15,6	82,94±14,69	64±12,4
	AG	64,74±7,38	76±4,35	82,7±13,8	89,8±8,8	

Sistatin S, mg/l	AG siz	0,83±0,05	1,08±0,15	1,12±0,3	1,32±0,474	0,86±0,1
	AG	1,11±0,07	1,22±0,14	1,33±0,24	1,186±0,155	
KFT Creatinin ml/min	AG siz	104,7±8,31	88,4±21,5	82,15±17,1	82,4±18,8	111,5±14,8
	AG	94,5±15,84	78±10,5	79,7+11,1	75,62±13,6	
KFT sistatin S, ml/min	AG siz	85,58±11,4	71,6±19	68,24±22,43	56,6±18,53	103,7±14,2
	AG	68,4±6,8	58,3±7,73	53,7±13,2	61,17±14,22	

Note: M - men; J - women; BMI - body mass index.

The obtained results showed that renal dysfunction in patients with COPD and arterial hypertension is closely related to structural and functional changes in the heart. In patients with impaired renal function, markedly more pronounced left ventricular hypertrophy and diastolic dysfunction were identified. Elevated pulmonary artery pressure along with reduced TAPSE suggests that right ventricular dysfunction associated with COPD also exerts a detrimental effect on renal function.

Conclusion. Therefore, in the coexistence of COPD and arterial hypertension, a pathogenetic interaction develops within the cardiopulmonary-renal system. Cardiac remodeling and diastolic dysfunction serve as significant prognostic indicators of declining renal function. From a practical point of view, early echocardiographic examination in this category of patients, the detection of diastolic dysfunction and the assessment of pulmonary hypertension are important for the prevention of kidney damage and the optimization of a comprehensive treatment stragy.

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