CHRONIC KIDNEY DISEASE AS A MANIFESTATION OF COMORBIDITY IN PATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE

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Abstract: Chronic obstructive pulmonary disease (COPD) has many systemic effects, one of which is renal dysfunction. Available studies show that patients with COPD often have risk factors for chronic kidney disease (CKD). Many risk factors for COPD are common for CKD. However, in everyday clinical practice, the incidence of renal dysfunction in patients with COPD is underestimated, while in-depth and targeted studies reveal changes in renal tissue function.
Keywords:chronic obstructive pulmonary disease, comorbidity, chronic kidney disease.

INTRODUCTION

According to foreign researchers, renal dysfunction occurs in 10.2% of patients with COPD, a significant proportion of whom are patients over 75 years of age [3]. The presence of chronic kidney disease (CKD) in patients hospitalized with an exacerbation of COPD is associated with an increased mortality rate [4]. In the studies of E.V. Bolotova, A.V. Dudnikova [1, 2] it was demonstrated that patients with COPD often have risk factors for the development of CKD. Thus, the most frequently noted were: elevated C-reactive protein values (in 100% of cases), smoking (in 92% of those examined), old age (in 78.6% of patients over 65 years of age), concomitant arterial hypertension (detected in 65.6% of patients with COPD) [1].

MATERIALS AND METHODS

Moreover, the vast majority of patients with COPD (92.6%) had a combination of three or more CKD risk factors [2]. These data are consistent with the results of studies conducted by D.A. Dolgopolova [3], who indicates that patients suffering from COPD had the following CKD risk factors: male gender (84.1%), old age (58.6%), excess body weight or obesity (49.6%), smoking (79.3%), and elevated blood pressure (59.3% of patients). D.S. Nurgazieva [4], when studying the glomerular filtration rate (GFR) in patients with COPD, notes that most patients have stage II CKD. According to E.V. Bolotova, A.V. Dudnikova [2], 37.3% of individuals with COPD have a decrease in SCF, calculated using the CKD-EPI formula taking into account serum creatinine (SCFr), within 89-60 ml/min/1.73 m2, 26.7% of patients have a decrease in SCFr to 59-45 ml/min/1.73 m2, and 3.3% of patients have SCFr within the range of 44-30 ml/min/1.73 m2. At the same time, only 4.3% of COPD patients are diagnosed with CKD at the prehospital stage [2]. D.A. Dolgopolova in her study [4] shows that among the examined individuals with COPD only every fifth had an optimal SCFr, and in 13.1% of patients the SCFr was determined to be within 59-45 ml/min/1.73 m2 (in this group of patients, those suffering from severe and extremely severe COPD predominated) [5]. About 37% of COPD patients have a persistent decrease in SCF to less than 60 ml/min/1.73 m2 [3], which exceeds the general population level, which according to F. Mallamaci is from 2 to 18% [3].

RESULTS AND DISCUSSION

A marked decrease in SCFr in patients with severe and extremely severe COPD may indicate a significant role of chronic hypoxia in the development of renal pathology [4]. There is evidence of negative correlations between the SCFr level and the thickness of the posterior wall of the left ventricle (PWL), creatinine levels and PWL. As filtration in the glomeruli of the kidneys decreases, the severity of left ventricular myocardial hypertrophy (LVH)

increases. Thus, D.A. Dolgopova found that in patients with COPD, the PWL value of >10.5 mm contributes to renal hypofiltration [5]. However, when calculating the SCF based on creatinine levels in individuals with severe and extremely severe COPD, it should be taken into account that a low body mass index in such patients is associated with a decrease in muscle mass and an increase in the severity of protein-energy malnutrition. This is important due to the presence of an inverse correlation between the creatinine value and the amount of muscle mass. Increased catabolic processes in severe and extremely severe COPD lead to a deficit of muscle tissue and, consequently, a decrease in creatinine synthesis, which demonstrates the limitations of calculating the SCF using the CKD-EPI formula using creatinine [2]. Due to the limited use of creatinine values for assessing renal function, including in patients with COPD, a search is underway for new markers that more accurately reflect the state of renal tissue. One of the markers of renal dysfunction that has been found and is currently being actively used is the cysteine proteinase inhibitor cystatin C. It is expressed by nucleated cells of the body and functions as a regulator of the activation of extra- and intracellular proteolysis. Its positive properties for studying renal function are its independence from the patient's age, muscle mass, and the absence of gender influence on its values. Cystatin C is also more accurate than creatinine because it is freely filtered in the glomeruli of the kidneys and metabolized in the renal tubules and is not secreted.

The advantages of serum cystatin C (s-cystatin C) are revealed in cases of moderate decrease in SCF (the so-called “creatinine blind area”) in the range of 90-60 ml/min/1.73 m2. In this range, there is no proportionality between creatinine and the true value of SCF, while s-cystatin C shows values closest to the real values of SCF calculated using the exogenous marker. The correlation coefficient of s-cystatin C and the true value of SCF is 0.92, while for creatinine it is 0.74 [3]. In addition, s-cystatin C is also discussed as a marker of cardiorenal syndrome (CRS) [4], which is important for assessing the state of the cardiovascular system in patients with COPD, since in the latter this lung pathology is often combined with damage to the cardiovascular system [2]. When studying the function of the cardiovascular system in individuals with CKD, T.E. Rudenko et al. [2] showed that s-cystatin C has a direct correlation with the presence of arterial hypertension and a strong correlation with the left ventricular myocardial mass index and LVH. Thus, LVH was detected in 52% of the subjects, and the frequency of its occurrence increased with the severity of renal dysfunction, assessed by the value of s-cystatin C in the blood serum.

CONCLUSION

Thus, COPD is a pathology with many systemic effects, and the comorbid background of patients suffering from this disease is extremely burdened. Currently, researchers have begun to pay great attention to the impairment of the functional state of the kidneys in patients with COPD as an important predictor of the development of cardiovascular pathology. However, to date, data fully revealing the development of renal dysfunction in individuals with COPD are insufficient, and further in-depth study of this problem is required.

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