

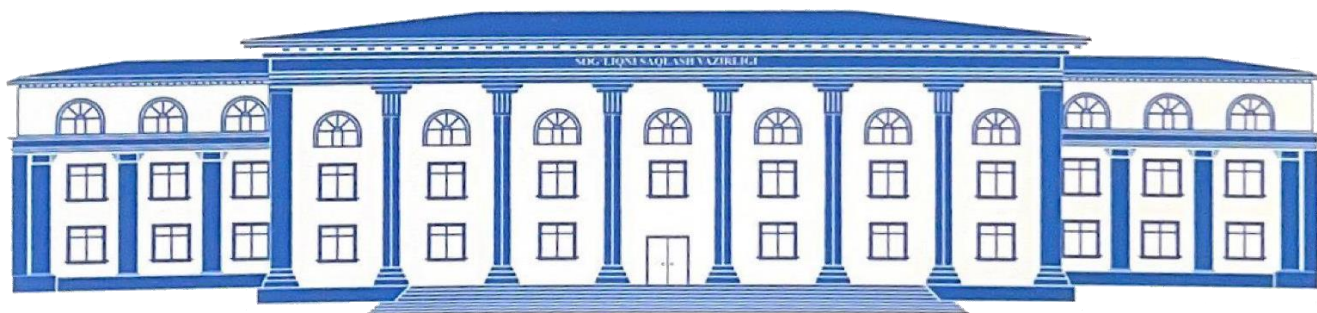
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DIAGNOSTIC SIGNIFICANCE OF GLOMERULAR FILTRATION RATE ESTIMATED USING CREATININE AND CYSTATIN C IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Салаева Муборак Саидобдуллаевна - Тошкент давлат тиббиёт университети "Клиник моделлаштириш" кафедраси доценти, т.ф.н.

e-mail: salaeva.muborak1966@gmail.com

ORCID ID: 0009-0002-4927-7054, <https://orcid.org/0009-0002-4927-7054>

Исмоилова Фазилят Рустамовна - Тошкент давлат тиббиёт университети "Клиник моделлаштириш" кафедраси катта ўқитувчиси, PhD.

e-mail: fazilat_med@mail.ru

ORCID: 0009-0004-1855-1887 <https://orcid.org/0009-0004-1855-1887>

Abstract. Objective. To assess the diagnostic value of glomerular filtration rate estimated from serum creatinine and cystatin C for detecting renal dysfunction in patients with chronic obstructive pulmonary disease (COPD).

Materials and methods. The prospective study included 214 patients with moderate to very severe COPD (grades II–IV). Serum creatinine and cystatin C levels were measured. Estimated glomerular filtration rate (eGFR) was calculated using the CKD-EPI 2021 creatinine equation and the CKD-EPI 2012 cystatin C equation. Renal filtration impairment was defined as $eGFR < 60 \text{ mL/min/1.73 m}^2$.

Results. Creatinine-based $eGFR < 60 \text{ mL/min/1.73 m}^2$ was identified in 29 patients (13.6%), whereas cystatin C-based $eGFR < 60 \text{ mL/min/1.73 m}^2$ was detected in 148 patients (69.2%). Serum cystatin C levels were significantly higher in patients with reduced eGFR across COPD grades II, III and IV ($p < 0.001$). Significant differences in serum creatinine and creatinine-based eGFR were observed predominantly in patients with grade III COPD ($p < 0.05$).

Conclusion. Creatinine-based eGFR remains an important conventional marker for assessing clinically significant impairment of renal filtration. Cystatin C-based eGFR expands the ability to detect reduced renal function in patients with COPD, including those with relatively preserved creatinine-based indices.

Keywords: chronic obstructive pulmonary disease, renal dysfunction, creatinine, cystatin C, estimated glomerular filtration rate.

Аннотация. Мақсад. Ўпканинг сурункали обструктив касаллиги (ЎСОК) билан хасталанган беморларда креатинин ҳамда цистатин С асосида ҳисобланган коптокчалар филтрация тезлигининг буйрак дисфункциясини аниқлашдаги диагностик аҳамиятини баҳолаш.

Материал ва усуллар. Проспектив тадқиқотга ЎСОКнинг II–IV оғирлик даражаларидаги 214 нафар бемор жалб қилинди. Қон зардобиди креатинин ва цистатин С миқдори аниқланиб, коптокчалар филтрация тезлиги креатинин учун СКД-ЕПИ 2021, цистатин С учун СКД-ЕПИ 2012 формулалари асосида ҳисобланди. Буйрак филтрацион функциясининг пасайиши $хКФТ < 60 \text{ мл/дақ/1,73 м}^2$ мезони бўйича баҳоланди.

Натижалар. Креатинин асосида ҳисобланган $хКФТ < 60 \text{ мл/дақ/1,73 м}^2$ 29 нафар (13,6%) беморда, цистатин С асосида эса 148 нафар (69,2%) беморда аниқланди. Цистатин С миқдори $хКФТ < 60 \text{ мл/дақ/1,73 м}^2$ бўлган беморларда ЎСОКнинг II, III ва IV даражаларининг барчасида ишончли юқори бўлди ($p < 0,001$). Креатинин ва унинг асосида ҳисобланган $хКФТ$ бўйича ишончли фарқ асосан ЎСОКнинг III даражасида кузатилди ($p < 0,05$).

Хулоса. Креатинин асосида ҳисобланган ҳКФТ буйрак филтрацион функциясининг клиник аҳамиятли пасайишини баҳолашда муҳим анъанавий мезон ҳисобланади. Цистатин С асосида ҳисобланган ҳКФТ эса ЎСОК билан беморларда, жумладан креатинин кўрсаткичлари нисбатан сақланган ҳолатларда, буйрак филтрацион функцияси пасайишини аниқлаш имкониятини кенгайтиради.

Калит сўзлар: ўпканинг сурункали обструктив касаллиги, буйрак дисфункцияси, креатинин, цистатин С, коптокчалар филтрация тезлиги

Аннотация. Цель. Оценить диагностическое значение скорости клубочковой фильтрации, рассчитанной на основе креатинина и цистатина С, для выявления почечной дисфункции у больных хронической обструктивной болезнью лёгких (ХОБЛ).

Материал и методы. В проспективное исследование включены 214 пациентов с ХОБЛ II–IV степени тяжести. В сыворотке крови определяли уровни креатинина и цистатина С. Скорость клубочковой фильтрации рассчитывали по формуле CKD-EPI 2021 на основе креатинина и CKD-EPI 2012 на основе цистатина С. Снижение фильтрационной функции почек оценивали по уровню рСКФ < 60 мл/мин/1,73 м².

Результаты. рСКФ < 60 мл/мин/1,73 м², рассчитанная по креатинину, выявлена у 29 (13,6%) пациентов, тогда как при расчёте по цистатину С — у 148 (69,2%). Уровень цистатина С у пациентов с рСКФ < 60 мл/мин/1,73 м² был достоверно выше при II, III и IV степенях ХОБЛ ($p < 0,001$). Достоверные различия уровня креатинина и рассчитанной на его основе рСКФ преимущественно отмечались при III степени ХОБЛ ($p < 0,05$).

Заключение. рСКФ, рассчитанная на основе креатинина, остаётся важным традиционным показателем клинически значимого снижения фильтрационной функции почек. Использование цистатина С расширяет возможности выявления снижения функции почек у больных ХОБЛ, в том числе при относительно сохранённых показателях креатинина.

Ключевые слова: хроническая обструктивная болезнь лёгких, почечная дисфункция, креатинин, цистатин С, скорость клубочковой фильтрации.

Introduction. Chronic obstructive pulmonary disease (COPD) is associated not only with persistent airflow limitation but also with systemic pathological processes and a wide range of comorbid conditions. Renal dysfunction in patients with COPD is multifactorial in origin. Chronic hypoxemia, systemic inflammation, endothelial dysfunction, oxidative stress, activation of the renin–angiotensin–aldosterone system, impaired microcirculation, and cardiovascular comorbidities may adversely affect the functional state of the nephrons [1, 4]. The development of renal dysfunction in patients with COPD may aggravate the clinical course of the disease and increase the risk of complications and adverse clinical outcomes [2, 5].

Glomerular filtration rate (GFR) is one of the principal functional indices used to assess renal function. In routine clinical practice, serum creatinine is the most widely used endogenous marker for estimating GFR [6, 7]. Its broad availability, relatively low cost, and the existence of standardized equations have established creatinine-based estimated GFR (eGFR) as one of the main laboratory tools for the initial assessment of renal filtration function [6].

However, serum creatinine concentration is influenced not only by glomerular filtration but also by age, sex, muscle mass, nutritional status, and other non-renal factors [7–9]. This limitation is particularly relevant in patients with COPD, since progression of the disease may be accompanied by loss of muscle mass, nutritional impairment, and reduced physical activity. Consequently, serum creatinine may remain within a relatively normal range despite a reduction in renal filtration function, and creatinine-based eGFR may overestimate actual kidney function in some patients [3, 8].

Cystatin C has gained particular importance as an alternative endogenous filtration marker for the assessment of renal function. Cystatin C is a low-molecular-weight protein produced at a relatively constant rate by nucleated cells and freely filtered through the glomeruli. Compared with creatinine, its serum concentration is less dependent on muscle mass and nutritional status [8–10].

Current clinical recommendations support the initial assessment of GFR using serum creatinine; however, the use of cystatin C may provide important additional diagnostic information in clinical situations in which the accuracy of creatinine-based assessment is potentially limited [6]. Cystatin C-based eGFR may be particularly useful in elderly patients, individuals with reduced muscle mass, and patients with multiple comorbidities, thereby improving the detection of impaired renal function [8–10].

Previous studies in patients with COPD have demonstrated discrepancies between GFR estimates based on creatinine and those based on cystatin C. In several studies, the use of cystatin C identified a greater proportion of patients with reduced renal filtration function than creatinine-based assessment [3, 11]. These findings highlight the need to evaluate the diagnostic capabilities of each filtration marker separately when identifying latent renal dysfunction in patients with COPD.

Therefore, separate assessment of the diagnostic value of creatinine-based and cystatin C-based eGFR in patients with COPD is relevant for the timely detection of renal dysfunction and for improving strategies of dynamic clinical monitoring.

Objective. To separately evaluate the diagnostic significance of glomerular filtration rate estimated from serum creatinine and from cystatin C in patients with chronic obstructive pulmonary disease.

Materials and methods. The study was conducted at the Pulmonology Department of the Multidisciplinary Clinic of Tashkent State Medical University in 2023–2024. A total of 214 patients hospitalized with COPD of grades II, III, and IV severity were included in the prospective analysis.

According to COPD severity, 51 patients ($23.8 \pm 2.9\%$) had grade II disease, 67 patients ($31.3 \pm 3.2\%$) had grade III disease, and 96 patients ($44.9 \pm 3.4\%$) had grade IV disease. According to the clinical phenotype of COPD, the bronchitic phenotype was observed in 86 patients ($40.2 \pm 3.4\%$), the emphysematous phenotype in 73 patients ($34.1 \pm 3.2\%$), and the mixed phenotype in 55 patients ($25.7 \pm 3.0\%$). The mean age of the patients was 64.7 ± 0.8 years. The study population included 137 men (64.0%) and 77 women (36.0%). The mean age of men was 65.8 ± 1.1 years, whereas the mean age of women was 62.8 ± 1.4 years.

All patients underwent standard clinical examination, and COPD severity was assessed using clinical and spirometric parameters. To evaluate renal functional status, serum creatinine and cystatin C concentrations were measured in venous blood samples using laboratory methods.

Renal filtration function was assessed by calculating GFR separately on the basis of serum creatinine and cystatin C. Creatinine-based eGFR was calculated using the CKD-EPI 2021 creatinine equation, taking into account serum creatinine, age, and sex. Cystatin C-based eGFR was calculated using the CKD-EPI 2012 cystatin C equation, taking into account serum cystatin C, age, and sex.

GFR was expressed as mL/min/1.73 m². An eGFR of <60 mL/min/1.73 m² was considered indicative of clinically significant impairment of renal filtration function. Quantitative variables were expressed using measures of central tendency and dispersion. Depending on the distribution of the data, appropriate parametric and non-parametric statistical tests were used to assess between-group differences. A value of $p < 0.05$ was considered statistically significant.

Results. The biochemical parameters of patients with COPD included in the prospective study were comparatively analyzed according to disease severity. To assess renal function, patients were divided into two groups according to cystatin C-based eGFR: Group I included patients with $\text{eGFR} < 60 \text{ mL/min/1.73 m}^2$, whereas Group II included patients with $\text{eGFR} \geq 60 \text{ mL/min/1.73 m}^2$. Serum creatinine and cystatin C levels, as well as creatinine-based and cystatin C-based eGFR values, were compared between these groups.

Separate assessment of renal filtration function using creatinine- and cystatin C-based equations demonstrated a marked difference in the detection of reduced eGFR within the same cohort of patients (Figure 1).

According to creatinine-based eGFR, 29 patients (13.6%) had an $\text{eGFR} < 60 \text{ mL/min/1.73 m}^2$, whereas 185 patients (86.4%) had an $\text{eGFR} \geq 60 \text{ mL/min/1.73 m}^2$. These findings indicate that creatinine-based eGFR is clinically useful primarily for identifying patients with more evident impairment of renal filtration function.

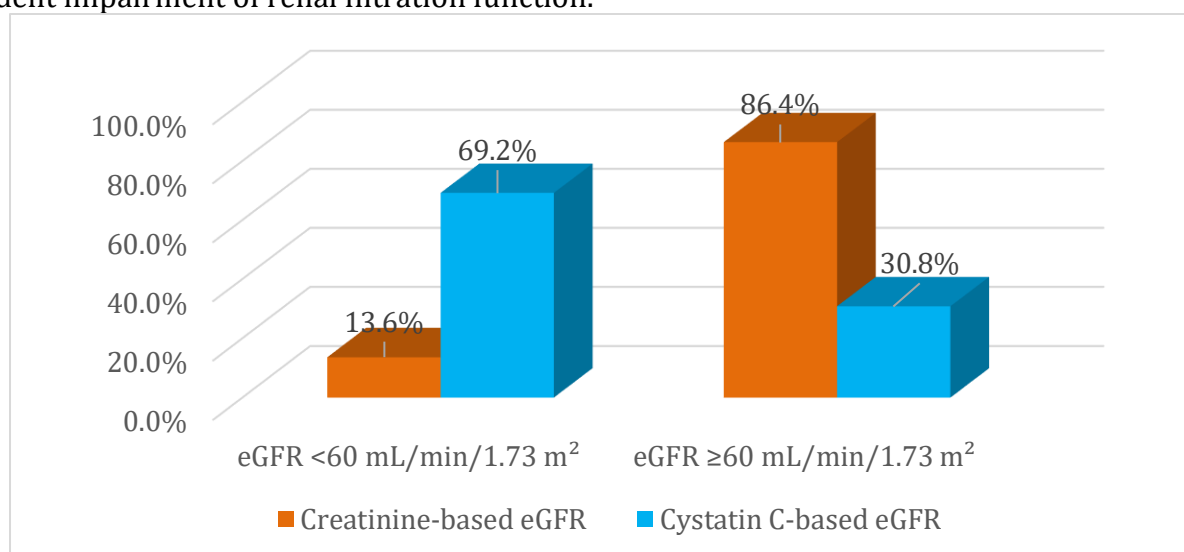


Figure 1. Distribution of estimated glomerular filtration rate (eGFR) calculated on the basis of creatinine and cystatin C in patients with chronic obstructive pulmonary disease

However, in patients with COPD, serum creatinine levels may be influenced by factors such as reduced muscle mass and impaired nutritional status. Therefore, preserved creatinine-based eGFR does not completely exclude the presence of early changes in renal function.

A markedly different pattern was observed when eGFR was calculated on the basis of cystatin C: 148 patients (69.2%) had an $\text{eGFR} < 60 \text{ mL/min/1.73 m}^2$, whereas only 66 patients (30.8%) had an $\text{eGFR} \geq 60 \text{ mL/min/1.73 m}^2$.

Thus, assessment based on cystatin C identified reduced renal filtration function in a considerably larger proportion of patients than creatinine-based assessment. These findings indicate that cystatin C has greater diagnostic potential for detecting renal dysfunction in patients with COPD when impairment is not clearly reflected by creatinine-based indices.

When the diagnostic significance of cystatin C was analyzed according to COPD severity, cystatin C-based $\text{eGFR} < 60 \text{ mL/min/1.73 m}^2$ was detected in 52.9% of patients with grade II COPD, 77.6% of those with grade III COPD, and 71.9% of those with grade IV COPD. These findings demonstrate that impaired renal filtration function is highly prevalent in patients with more severe stages of COPD.

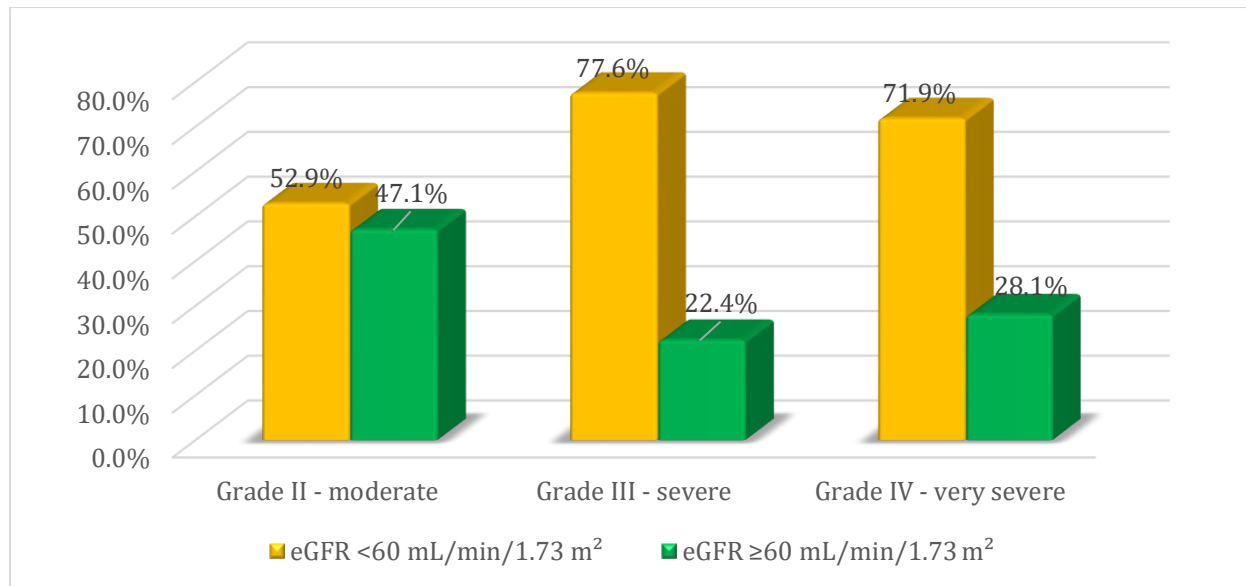


Figure 2. Distribution of patients according to cystatin C-based estimated glomerular filtration rate across different COPD severity grades

According to the data presented in Table 1, a statistically significant difference in serum creatinine levels was observed in patients with grade III COPD. Serum creatinine was 87.8 ± 6.96 $\mu\text{mol/L}$ in patients with $\text{eGFR} < 60 \text{ mL/min/1.73 m}^2$ and 68.8 ± 4.32 $\mu\text{mol/L}$ in those with $\text{eGFR} \geq 60 \text{ mL/min/1.73 m}^2$ ($p=0.024$; $p<0.05$). At the other COPD severity grades, between-group differences in creatinine levels did not reach statistical significance. In grade II COPD, serum creatinine values were nearly comparable between the groups ($p>0.05$), whereas in grade IV COPD creatinine levels were higher in patients with $\text{eGFR} < 60 \text{ mL/min/1.73 m}^2$, although the difference was not statistically significant ($p=0.212$). These findings indicate that creatinine remains an important conventional marker of clinically significant impairment of renal filtration function; however, its diagnostic sensitivity for detecting early and latent renal dysfunction in COPD may be limited.

In contrast, cystatin C showed a clear association with reduced eGFR across COPD grades II–IV and demonstrated greater potential for identifying relatively early impairment of renal filtration function.

Table 1. Comparative analysis of biochemical parameters in patients with cystatin C-based $\text{eGFR} < 60$ and $\geq 60 \text{ mL/min/1.73 m}^2$ across different COPD severity grades

Parameters	Grade II - moderate		Grade III - severe		Grade IV - very severe	
	eGFR <60 n=27	eGFR ≥60 n=24	eGFR <60 n=52	eGFR ≥60 n=15	eGFR <60 n=69	eGFR ≥60 n=27
Creatinine, $\mu\text{mol/L}$	71.1 ± 4.47	72.6 ± 2.54	87.8 ± 6.96	68.8 ± 4.32	88.0 ± 4.23	80.4 ± 4.34
p	>0.05		<0.05		>0.05	
Cystatin C, mg/L	1.71 ± 0.12	0.82 ± 0.06	1.54 ± 0.04	0.92 ± 0.05	1.64 ± 0.04	0.74 ± 0.06
p	<0.001		<0.001		<0.001	
Creatinine-based eGFR, mL/min/1.73 m ²	92.0 ± 3.29	97.4 ± 2.88	81.9 ± 3.48	94.6 ± 4.24	79.5 ± 2.76	85.8 ± 3.24
p	>0.05		<0.05		>0.05	
Cystatin C-based eGFR, mL/min/1.73 m ²	41.1 ± 2.40	107.7 ± 12.29	43.7 ± 1.24	84.7 ± 6.21	40.8 ± 1.14	106.9 ± 7.86
p	<0.001		<0.001		<0.001	

Across all analyzed COPD severity grades, patients with $\text{eGFR} < 60 \text{ mL/min/1.73 m}^2$ had significantly higher serum cystatin C levels than those with ($p < 0.001$), indicating a close association between increased cystatin C levels and impaired glomerular filtration.

In patients with grade II COPD, serum cystatin C levels were 1.71 ± 0.12 versus $0.82 \pm 0.06 \text{ mg/L}$; in grade III COPD, 1.54 ± 0.04 versus $0.92 \pm 0.05 \text{ mg/L}$; and in grade IV COPD, 1.64 ± 0.04 versus $0.74 \pm 0.06 \text{ mg/L}$, with highly significant between-group differences in all cases ($p < 0.001$). Thus, across all analyzed COPD severity grades, increased cystatin C levels were clearly associated with impaired renal filtration function.

Cystatin C-based eGFR also differed significantly between the study groups. In grade II COPD, eGFR values were 41.1 ± 2.40 and $107.7 \pm 12.29 \text{ mL/min/1.73 m}^2$, respectively; in grade III COPD, 43.7 ± 1.24 and $84.7 \pm 6.21 \text{ mL/min/1.73 m}^2$; and in grade IV COPD, 40.8 ± 1.14 and $106.9 \pm 7.86 \text{ mL/min/1.73 m}^2$. Highly significant between-group differences were observed at all COPD severity grades ($p < 0.001$).

Creatinine-based eGFR in patients with grade II COPD was $92.0 \pm 3.29 \text{ mL/min/1.73 m}^2$ in Group I and $97.4 \pm 2.88 \text{ mL/min/1.73 m}^2$ in Group II, with no statistically significant difference ($p > 0.05$). In grade III COPD, the corresponding values were 81.9 ± 3.48 and $94.6 \pm 4.24 \text{ mL/min/1.73 m}^2$, and the difference was statistically significant ($p < 0.05$). In grade IV COPD, creatinine-based eGFR values were 79.5 ± 2.76 and $85.8 \pm 3.24 \text{ mL/min/1.73 m}^2$, respectively, with no statistically significant difference ($p > 0.05$).

Discussion. The study findings demonstrated that the diagnostic capabilities of creatinine-based and cystatin C-based estimated glomerular filtration rate (eGFR) are not equivalent when assessing renal filtration function in patients with COPD. Creatinine is a widely used and readily available marker in routine clinical practice and remains important for identifying clinically significant impairment of renal function. However, because serum creatinine concentration is influenced by age, sex, muscle mass, and nutritional status, it may not always adequately reflect early stages of renal dysfunction in patients with COPD [6–9].

In the present study, statistically significant differences in serum creatinine and creatinine-based eGFR were not observed across all COPD severity grades, with statistical significance being detected mainly in patients with grade III COPD ($p < 0.05$). This may be explained by the reduction in muscle mass and metabolic alterations associated with severe COPD, which can influence serum creatinine levels [3, 8].

In contrast, cystatin C demonstrated consistent and statistically significant differences across grades II, III, and IV COPD ($p < 0.001$). Cystatin C-based eGFR also clearly distinguished patients with impaired renal filtration function at all analyzed disease severity grades. These findings may be explained by the lower dependence of cystatin C on muscle mass compared with creatinine and by its greater sensitivity to early changes in renal function [7–10].

The present findings are consistent with the results reported by Gjerde B. et al. and Yoshizawa T. et al., who showed that some cases of renal dysfunction in patients with COPD may remain undetected when assessment is based solely on creatinine, whereas the use of cystatin C may improve the detection of impaired renal function [2, 11].

At the same time, cystatin C should not be regarded as an absolutely specific marker, since its concentration may be affected by systemic inflammation, glucocorticoid therapy, thyroid dysfunction, and other non-renal factors [9, 10]. Therefore, cystatin C results should be interpreted in conjunction with the patient's clinical condition and other laboratory findings.

Thus, creatinine-based eGFR has practical value for the initial and routine assessment of renal function in patients with COPD, whereas cystatin C-based eGFR may facilitate earlier detection of latent renal dysfunction when creatinine-based indices remain relatively preserved. Therefore, in patients with COPD—particularly those with grades II–IV disease, advanced age, hypoxemia, reduced muscle mass, or other comorbid conditions—it may be appropriate not to

rely exclusively on creatinine-based assessment and, when indicated, to additionally determine cystatin C-based eGFR. This approach may improve early detection of renal dysfunction, identification of high-risk patients, and optimization of follow-up strategies.

Conclusion. In patients with COPD, serum creatinine and creatinine-based eGFR are clinically relevant for assessing impaired renal filtration function; however, statistically significant differences were observed mainly in grade III COPD ($p < 0.05$), while no significant differences were found in grades II and IV.

Cystatin C and cystatin C-based eGFR reliably distinguished patients with reduced renal filtration function across grades II–IV COPD ($p < 0.001$), indicating the high diagnostic value of cystatin C for detecting latent and early renal dysfunction in COPD.

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