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Research Article

Clinical Interplay Between Chronic Obstructive Pulmonary Disease and Chronic Kidney Disease: A Cross-Sectional Analysis of 79 Patients

Petar Avramovski[1]*, Igor Nedelkovski[2], Liljana Todorovska[3], Maja Avramovska[4], Zorica Nikleski[5], Stefan Talev[6], Svetlana Stefanovska[7] and Biljana Taleva[8]

[1]Department of Internal Medicine, Clinical Hospital - Bitola, St. Clement of Ohrid University, Bitola, North Macedonia

[2]Department of Anesthesia and Intensive Care, Clinical Hospital - Bitola, North Macedonia

[3]Department of Nuclear Medicine, Clinical Hospital - Bitola, North Macedonia

[4]Department of Gynecology and Obstetrics, Clinical Hospital - Bitola, North Macedonia

[5]Medico - Legal Department, MedAssess, Sydney, New South Wales, Australia

[6]Department of Chirurgie, Clinical Hospital - Bitola, North Macedonia

[7]Department of Internal Medicine and Department of Cardiology, Clinical Hospital - Bitola, Bitola, North Macedonia

[8]Department of Vascular Surgery, PHI University Clinic for Surgical Diseases "St. Naum Ohridski - Skopje", Skopje, North Macedonia

***Corresponding Author:** Petar Avramovski, Department of Internal Medicine, Clinical Hospital - Bitola, St. Clement of Ohrid University, Bitola, North Macedonia.

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Abstract

Background: Chronic Obstructive Pulmonary Disease (COPD) and Chronic Kidney Disease (CKD) often coexist, sharing risk factors and pathophysiological mechanisms such as systemic inflammation and oxidative stress. Their overlap may worsen disease progression and complicate management.

Objective: To evaluate the clinical, laboratory, and functional characteristics of patients with coexisting COPD and mild CKD, and to assess correlations between renal and pulmonary function.

Methods: A cross-sectional study included 79 patients with mildly impaired renal function (eGFR 78.7 ± 10.3 mL/min/1.73m^2) and respiratory symptoms. Demographics, laboratory data (urea, creatinine, hemoglobin, erythrocytes), and spirometric parameters-FVC, FEV1, FEV1/FVC, PEF, ELA, FEV25-75, FET, FIVC, FEV1/VC-were analyzed. Pearson correlation evaluated associations between renal and pulmonary function.

Results: Patients had a mean age of 43.2 ± 15.6 year. Laboratory results confirmed mild renal impairment (urea 6.74 ± 1.93 mmol/L, creatinine 95.2 ± 13.8 µmol/L, hemoglobin 98.1 ± 13.2 g/L, erythrocytes 4.51 ± 0.46 ×10^{12}/L). Pulmonary function was reduced (FVC 1.49 ± 0.13 L, FEV1 1.21 ± 0.09 L, PEF 2.42 ± 0.31 L/s, FEV25-75 1.16 ± 0.19 L/s). Significant positive correlations were observed between eGFR and FVC ($r = 0.271$, $p = 0.015$), FEV1 ($r = 0.299$, $p = 0.007$), PEF ($r = 0.227$, $p = 0.044$), FIVC ($r = 0.241$, $p = 0.032$), FEV25-75 ($r = 0.332$, $p = 0.003$), and ELA ($r = 0.286$, $p = 0.011$).

Conclusion: Mild CKD in patients with COPD is associated with measurable declines in pulmonary function. These findings highlight the clinical interplay between kidney and lung health, supporting early recognition and integrated multidisciplinary management to optimize outcomes.

Keywords: *Chronic Kidney Disease; Chronic Obstructive Pulmonary Disease; Spirometry; Estimated Glomerular Filtration Rate, Pulmonary Age*

Introduction

Chronic obstructive pulmonary disease (COPD) and chronic kidney disease (CKD) are prevalent chronic conditions, particularly among older adults with multiple comorbidities. These two diseases frequently coexist, and growing evidence suggests a complex bidirectional interaction between pulmonary and renal function. Patients with COPD often exhibit systemic inflammation, oxidative stress, and cardiovascular comorbidities, all of which can contribute to the development or progression of renal dysfunction. Conversely, CKD can exacerbate respiratory impairment through fluid overload, metabolic derangements, anemia, and uremic myopathy, ultimately reducing respiratory reserve and exercise tolerance [112].

Despite the clinical significance of this lung-kidney interplay, data on the correlations between renal function and COPD severity remain limited. Understanding these associations is essential for comprehensive risk stratification and individualized patient management. Estimated glomerular filtration rate (eGFR), a widely used marker of renal function, may provide valuable prognostic information in patients with COPD, while standard spirometric indices such as FEV_1, FVC, and GOLD stage reflect pulmonary impairment.

In this context, the present study aimed to evaluate the relationship between renal function and pulmonary parameters in a cohort of 79 patients with COPD, providing insights into the clinical and pathophysiological link between CKD and COPD. By identifying key laboratory and functional correlations, this study seeks to inform clinical practice and highlight the need for integrated assessment of kidney function in patients with chronic respiratory disease.

Aim of the Study

The aim of this study was to assess the correlation between renal function, measured by eGFR, and pulmonary function in COPD patients. Spirometric parameters analyzed included Forced Vital Capacity (FVC), Forced Expiratory Volume in 1 second (FEV1), FEV1/Forced Vital Capacity ratio (FEV1/FVC), Peak Expiratory Flow (PEF), Effective Lung Age (ELA), Forced Expiratory Flow at 25-75% of pulmonary volume (FEV2575), Forced Expiratory Time (FET), Forced Inspiratory Vital Capacity (FIVC), and FEV1/Vital Capacity ratio (FEV1/VC), aiming to evaluate how renal impairment may affect lung function.

Methods

We examined 79 patients aged 39 to 60 years (median age 44) with mild renal impairment, as measured by estimated glomerular filtration rate (eGFR), who presented with respiratory symptoms. Laboratory analyses included Urea (mmol/L), Creatinine (μmol/L), Hemoglobin (g/L), and Erythrocytes. All patients underwent spirometry, and the results were compared with their creatinine values to evaluate the relationship between renal function and pulmonary performance. All laboratory analyses were performed following standard laboratory procedures. Pulmonary function tests were conducted using a Spirobank II new spirometer (S/N Y14726) according to the manufacturer's instructions.

Results

The demographic and laboratory analyses for the 79 patients with mildly impaired renal function are summarized in table 1. The cohort had a mean age of XX ± XX years, with a balanced distribution of males and females. Laboratory results demonstrated a mean urea of 6.74 ± 1.93 mmol/L and creatinine of 95.2 ± 13.8 μmol/L, consistent with mild renal impairment. Hemoglobin levels averaged 98.1 ± 13.2 g/L, and erythrocyte counts were 4.51 ± 0.46 $\times 10^{12}$/L, indicating no severe anemia in the cohort. The mean estimated glomerular filtration rate (eGFR) was 78.7 ± 10.3 mL/min/1.73m^2, confirming mild CKD according to standard guidelines. Overall, these results reflect a patient population with relatively preserved renal function but measurable laboratory deviations indicative of early renal dysfunction.

Parameter (N = 79)	Mean ± SD	Reference range
Age (years)	43.2 ± 15.6	/
Urea (mmol/L)	6.74 ± 1.93	2.5 - 7.5
Creatinine (μmol/L)	95.2 ± 13.8	60 - 110
Hemoglobin (g/L)	98.1 ± 13.2	120 - 155
Erythrocytes ($\times 10^{12}$/L)	4.51 ± 0.46	4.0 - 5.2
eGFR (ml/min/1.73m^2)	78.7 ± 10.3	> 90 (60 - 80 mild)
eGFR: Estimated Glomerular Filtration Rate		
The results are presented as mean ± SD (standard deviation)		

Table 1: *Demographic and laboratory characteristics of 79 patients with mildly impaired renal function.*

A significant relationship was observed between renal function and several spirometric parameters in the COPD cohort. Higher eGFR values correlated positively with FVC (r = 0.271; p = 0.015), FEV1 (r = 0.299; p = 0.007), PEF (r = 0.227; p = 0.044), ELA (r = 0.286; p = 0.011), FEF25-75 (r = 0.332; p = 0.003), and FIVC (r = 0.241; p = 0.032). These findings suggest that preserved renal function is associated with improved lung volumes, better expiratory airflow, and younger estimated lung age. No significant associations were found for FEV1/FVC (r = 0.116; p = 0.308) or FEV1/VC (r = 0.135; p = 0.235). The FET showed a negative but non-significant correlation (r = -0.271; p = 0.063). The overall pattern indicates that even early reductions in renal filtration capacity may contribute to spirometric decline, particularly affecting small-airway flow and expiratory performance. Table 2 summarizes the results of 79 COPD patients with impaired renal function based on eGFR, and the Pearson correlations between COPD parameters and eGFR are presented.

Pulmonary Function Test	Parameters (best)	eGFR [r]	P
FVC (L)	1.49 ± 0.13	0.271	0.015
FEV1 (L)	1.21 ± 0.09	0.299	0.007
FEV1/FVC (%)	81.2 ± 7.64	0.116	0.308
PEF (L/s)	2.42 ± 0.31	0.227	0.044
ELA (years)	91 ± 1.32	0.286	0.011
FEV25-75 (L/s)	1.16 ± 0.19	0.332	0.003
FET (s)	2.31 ± 0.29	-0.271	0.063
FIVC (L)	1.61 ± 1.93	0.241	0.032
FEV1/VC (%)	61.4 ± 0.76	0.135	0.235
FVC: Forced Vital Capacity; FEV1: Forced Expiratory Volume in one second; PEF: Peak Expiratory Flow; ELA: Estimated Lung Age; FEF25-75: Forced Expiratory Flow between 25% and 75% of FVC; FET: Forced Expiratory Time; FIVC: Forced Expiratory Vital Capacity; VC: Vital Capacity; eGFR: Estimated Glomerular Filtration rate			
The results are presented as mean ± SD (standard deviation), P-value (two-tailed probability)			

Table 2: *Correlation between spirometric parameters and estimated glomerular filtration rate (eGFR) in patients with chronic obstructive pulmonary disease.*

Discussion

Chronic obstructive pulmonary disease (COPD) and chronic kidney disease (CKD) frequently coexist, particularly in older adults with multiple comorbidities. In the present study, 79 COPD patients demonstrated varying degrees of impaired renal function as measured by eGFR. The observed relationships between COPD parameters and eGFR highlight an important interplay between respiratory and renal physiology [1,2].

Several mechanisms may explain why reduced kidney function negatively impacts pulmonary performance. First, CKD is associated with systemic inflammation, oxidative stress, and endothelial dysfunction-all of which contribute to accelerated decline in lung function. Studies have shown that elevated inflammatory markers, including CRP and IL-6, correlate with worse FEV1 and more severe COPD exacerbations [36]. Persistent inflammation in CKD can aggravate airway remodeling and impair gas exchange [7,8].

Second, impaired renal function promotes fluid retention, metabolic acidosis, and anemia. Fluid overload increases pulmonary vascular pressures and may worsen dyspnea, particularly in hyperinflated COPD lungs. Metabolic acidosis stimulates compensatory hyperventilation, increasing respiratory muscle workload and fatigue [911]. Additionally, anemia reduces oxygen-carrying capacity and contributes to reduced exercise tolerance; its prevalence increases significantly when eGFR falls below 60 mL/min/1.73m^2 [9,10]. Together, these factors decrease overall respiratory reserve. Figure 1 illustrates the pathophysiological links between renal dysfunction and COPD, highlighting how impairment in kidney function may influence respiratory decline and vice versa.

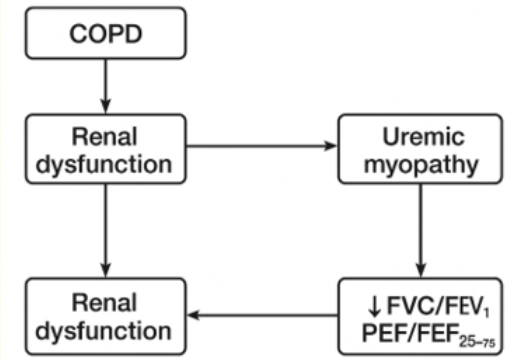

Figure 1: *Pathophysiological interplay between COPD, renal disfunction and spirometric decline.*

COPD: Chronic Obstructive Pulmonary Disease; FVC: Forced Vital Capacity; FEV1: Forced Expiratory Volume in One Second; PEF: Peak Expiratory Flow.

Bronchial and respiratory muscle weakness may also partly explain the observed correlation. CKD-related uremic myopathy affects both peripheral and respiratory muscles, reducing maximal inspiratory and expiratory pressures [12]. When superimposed onto COPD, this dual impairment can worsen airflow limitation and hyperinflation. Furthermore, shared risk factors-age, smoking, cardiovascular disease, hypertension, and diabetes-contribute to both COPD progression and renal decline. Several cohort studies have reported that lower eGFR is independently associated with decreased FEV1 and increased respiratory symptoms, even after adjusting for confounders. This suggests that kidney dysfunction has a direct pathophysiological influence on pulmonary health rather than merely reflecting comorbidity burden [1,2].

In this context, the significant Pearson correlations found between COPD measures [e.g. FEV1, FVC, SpO_2, Global Initiative for Chronic Obstructive Lung Disease (GOLD) stage] and eGFR in our cohort further support the concept of a bidirectional lung-kidney axis. Declining

kidney function may amplify COPD severity through metabolic, inflammatory, and cardiovascular pathways, while chronic hypoxia and pulmonary hypertension in advanced COPD may impair renal perfusion and promote renal decline. These findings reinforce the need for early identification of renal impairment in COPD patients. Incorporating routine assessments of kidney function into COPD management may improve risk stratification, guide individualized therapy, and potentially reduce morbidity. Future studies with larger multicenter cohorts are warranted to further clarify causal mechanisms and evaluate whether interventions targeting CKD can improve respiratory outcomes.

Arterial stiffness is known to progress more rapidly in patients with chronic kidney disease due to vascular calcification, endothelial dysfunction, and chronic inflammation. Studies also show that individuals with COPD exhibit significantly increased aortic stiffness, likely driven by systemic inflammation and oxidative stress. These overlapping mechanisms suggest that COPD may further amplify arterial stiffening in patients with impaired renal function, contributing to a higher cardiovascular burden [12,13].

Limitation of the Study

This study has several limitations. The sample size was relatively small, and other parameters of chronic kidney disease or patient comorbidities were not included in the analysis. Data on hypertension, diabetes, and other relevant conditions were unavailable, which may affect the interpretation of the observed associations.

Conclusion

Our findings demonstrate a significant interplay between COPD and CKD. Even mild renal impairment is associated with reduced pulmonary function, likely mediated by inflammation, fluid retention, anemia, and muscle weakness. The correlations between spirometric parameters and eGFR support a bidirectional lung-kidney axis. Early monitoring of kidney function in COPD patients may help guide management and reduce morbidity.

Conflict of Interest

The authors declare that there are no conflicts of interest related to this manuscript.

Bibliography

1. Liu Z., *et al*. "Association between COPD and CKD: a systematic review and meta analysis". *Frontiers in Public Health* 12 (2024): 1494291.

2. Corrado Pelaia., *et al*. "Predictors of renal function worsening in patients with chronic obstructive pulmonary disease (COPD). Multicenter study, 707 patients". *Nutrients* 13.8 (2021): 2811.

3. Jia Wei., *et al*. "Association between serum interleukin 6 concentrations and chronic obstructive pulmonary disease: a meta-analysis". *PeerJ* 3 (2015): e1199.

4. Francisco Garcia-Rio., *et al*. "Systemic inflammation in chronic obstructive pulmonary disease: a population based study". *Respiratory Research* 11.1 (2010): 63.

5. Renata Ferrari., *et al*. "Three-year follow-up of interleukin 6 and C-reactive protein in COPD". *Respiratory Research* 14 (2013): 24.

6. Hui Huang., *et al*. "Interleukin 6 is a strong predictor of the frequency of COPD exacerbation within 1 year". *International Journal of Chronic Obstructive Pulmonary Disease* 16 (2021): 2945-2951.

7. Francesca Tinti., *et al*. "Chronic kidney disease as a systemic inflammatory syndrome: update on mechanisms involved and potential treatment". *Life (Basel)* 11 (2021): 419.

8. Victor M Baião., *et al*. "Effects of exercise on inflammatory markers in individuals with chronic kidney disease: a systematic review and meta analysis". *Metabolites* 13.7 (2023): 795.

9. Michal L Melamed and Kalani L Raphael. "Metabolic acidosis in CKD: A review of recent findings". *Kidney Medicine* 3.2 (2021): 267-277.

10. Donald E Wesson., *et al*. "Mechanisms of metabolic acidosis-induced kidney injury in chronic kidney disease". *Journal of the American Society of Nephrology* 31.3 (2020): 469-482.

11. Hyo Jin Kim. "Metabolic acidosis in chronic kidney disease: pathogenesis, clinical consequences, and treatment". *Electrolytes and Blood Pressure* 19.2 (2021): 29-37.

12. Chatrenet A., *et al*. "Analysis of the rate of force development reveals high neuromuscular fatigability in elderly patients with chronic kidney disease". ar Xiv. (2023): 2307.10691.

13. Avramovski P., *et al*. "The effect of chronic pulmonary disease and aortic stiffness". *EC Pulmonology and Respiratory Medicine* 4.2 (2017): 59-66.

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