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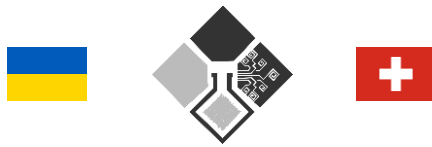
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DECLINE IN RENAL FUNCTION AND SYSTEMIC INFLAMMATION IN COPD: CLINICAL SIGNIFICANCE OF IL-6

Background

Chronic obstructive pulmonary disease (COPD) is a chronic disorder characterized by persistent and progressive bronchial obstruction and is not limited to local respiratory abnormalities, but is also accompanied by systemic effects and various comorbid conditions. In addition to local bronchopulmonary inflammation, systemic inflammation, oxidative stress, endothelial dysfunction, hypoxemia, and microcirculatory disturbances play an important role in the pathogenesis of the disease. These mechanisms may contribute to the development of various comorbid conditions in COPD, including impairment of renal functional status.

Recent evidence indicates a clinically significant relationship between COPD and chronic kidney disease. In a 2024 systematic review and meta-analysis by Liu Z. et al., patients with COPD were shown to have a higher likelihood of developing chronic kidney disease compared with controls (OR=1.54; 95% CI 1.28–1.84) [1]. These findings highlight the clinical importance of comprehensive assessment of renal function in patients with COPD.

The development of renal injury in COPD is multifactorial, with chronic hypoxemia, systemic inflammation, oxidative stress, endothelial dysfunction, and impaired renal microcirculation considered among the major pathogenetic mechanisms. These processes may lead to a reduction in glomerular filtration and the development of functional alterations in renal tissue [1,2].

Among the cytokines reflecting systemic inflammation in COPD, interleukin-6 (IL-6) is of particular importance. IL-6 is a pleiotropic cytokine involved in the initiation and maintenance of inflammatory responses, and elevated circulating levels reflect increased systemic inflammatory activity in COPD. In a meta-analysis by Wei J. et al. including 33 studies, serum IL-6 concentrations were significantly

higher in patients with stable COPD than in healthy individuals (SMD=0.65; 95% CI 0.51–0.79) [3]. The prevalence and clinical significance of renal dysfunction in COPD have also been demonstrated in several studies. In a systematic review and meta-analysis by Gaddam S. et al., chronic kidney disease was reported to occur frequently in patients with COPD [4]. In addition, Hu G. et al. showed that elevated cystatin C levels in patients hospitalized for COPD exacerbation were associated with adverse clinical outcomes [5]. These data support the importance of assessing renal function in COPD using sensitive biomarkers.

Increased systemic inflammation may adversely affect renal function through endothelial dysfunction, deterioration of renal microcirculation, and enhanced hypoxic injury to nephrons. Therefore, simultaneous assessment of IL-6 levels and renal filtration function in patients with COPD may provide a better understanding of the pathogenetic interaction between the lungs and kidneys.

Objective

To comparatively assess the relationship between serum IL-6 levels and reduced cystatin C-based estimated glomerular filtration rate (eGFR) in patients with stage II, III, and IV COPD.

Materials and Methods

The study included 214 patients with stage II–IV COPD. Patients were stratified according to disease severity and cystatin C-based estimated glomerular filtration rate. Renal functional status was assessed by measuring serum cystatin C and calculating eGFR based on cystatin C. Patients were divided into two groups: eGFR <60 mL/min/1.73 m² and eGFR ≥60 mL/min/1.73 m².

Among patients with stage II COPD, 27 had eGFR <60 mL/min/1.73 m² and 24 had eGFR ≥60 mL/min/1.73 m². In stage III COPD, the corresponding numbers were 52 and 15 patients, while in stage IV COPD they were 69 and 27 patients, respectively.

Serum IL-6 concentrations were determined by solid-phase enzyme-linked immunosorbent assay (ELISA) using the Human IL-6 ELISA Kit manufactured by Beijing Solarbio Science & Technology Co., Ltd.

The obtained data were subjected to statistical analysis. Quantitative variables were presented as mean ± standard deviation (M±SD). The statistical significance of between-group differences was assessed, with p<0.05 considered statistically significant.

Results

The results demonstrated that serum IL-6 levels were higher in COPD patients with reduced renal filtration function.

In patients with stage II moderate COPD, serum IL-6 levels were 22.6 ± 16.8 pg/mL in those with $\text{eGFR} < 60 \text{ mL/min/1.73 m}^2$ and 14.9 ± 10.7 pg/mL in those with $\text{eGFR} \geq 60 \text{ mL/min/1.73 m}^2$. Thus, IL-6 levels were higher in the group with reduced renal filtration function; however, the difference did not reach statistical significance ($p > 0.05$).

In patients with stage III severe COPD, the difference was more pronounced. IL-6 levels were 27.92 ± 19.6 pg/mL in patients with $\text{eGFR} < 60 \text{ mL/min/1.73 m}^2$ and 18.74 ± 12.41 pg/mL in those with $\text{eGFR} \geq 60 \text{ mL/min/1.73 m}^2$. The between-group difference was statistically significant ($p < 0.05$).

The highest IL-6 concentrations were observed in patients with stage IV very severe COPD. In this group, IL-6 levels were 33.87 ± 22.4 pg/mL in patients with $\text{eGFR} < 60 \text{ mL/min/1.73 m}^2$ and 23.1 ± 15.2 pg/mL in patients with preserved renal filtration function. The difference between the groups was statistically highly significant ($p < 0.01$).

Analysis of the results showed that in patients with $\text{eGFR} < 60 \text{ mL/min/1.73 m}^2$, IL-6 levels increased from 22.6 ± 16.8 pg/mL in stage II COPD to 33.87 ± 22.4 pg/mL in stage IV COPD. A similar pattern was observed among patients with $\text{eGFR} \geq 60 \text{ mL/min/1.73 m}^2$, in whom IL-6 levels increased from 14.9 ± 10.7 pg/mL to 23.1 ± 15.2 pg/mL.

These findings indicate that systemic inflammatory activity increases with greater COPD severity. At the same time, within each severity stage, IL-6 levels were higher in patients with reduced renal filtration function, suggesting a relationship between systemic inflammation and renal functional status in COPD.

Discussion

The findings indicate that increased systemic inflammation and reduced renal filtration function may occur concurrently in patients with more severe COPD. In particular, the significantly higher IL-6 levels observed in patients with $\text{eGFR} < 60 \text{ mL/min/1.73 m}^2$ at stages III and IV suggest more pronounced inflammatory activity in this subgroup.

Our findings are consistent with published evidence regarding the role of IL-6 as a marker of systemic inflammation in COPD. In the meta-analysis by Wei J. et al., data from 1,891 patients with stable COPD and 4,946 healthy controls were pooled, demonstrating higher IL-6 levels in patients with COPD [3]. At the same time, the meta-analysis did not identify a consistent relationship between IL-6 levels and spirometric severity. This suggests that IL-6 may reflect not only the degree of bronchial obstruction but also the systemic manifestations of COPD.

Renal dysfunction is considered one of the clinically relevant comorbid conditions in COPD. In the meta-analysis by Liu Z. et al., 23 studies were analyzed,

showing an increased risk of chronic kidney disease in patients with COPD and demonstrating that the presence of kidney disease is associated with poorer short- and long-term outcomes [1]. Our findings are also consistent with published data on the clinical importance of assessing renal functional status in COPD. Gaddam S. et al. reported a high prevalence of chronic kidney disease among patients with COPD [4]. Hu G. et al. found that elevated cystatin C levels in patients hospitalized for COPD exacerbation were associated with adverse in-hospital outcomes [5]. In our study, higher IL-6 levels in patients with reduced cystatin C-based eGFR, particularly in stages III–IV COPD, further support a relationship between systemic inflammation and renal dysfunction.

The relationship between IL-6 and renal functional status is also pathogenetically relevant. Chronic inflammation may aggravate endothelial dysfunction, microcirculatory disturbances, and hypoxia, thereby contributing to reduced renal perfusion. Therefore, elevated IL-6 should not be interpreted as a direct cause of renal dysfunction, but rather as a marker of systemic inflammatory activity involved in the lung–kidney interaction.

Conclusion

In patients with COPD, reduced renal filtration function may be associated with greater systemic inflammatory activity. IL-6 levels were higher across all COPD severity stages in patients with cystatin C-based eGFR <60 mL/min/1.73 m², with statistically significant differences observed in stage III ($p<0.05$) and stage IV ($p<0.01$) COPD. Combined assessment of IL-6 and cystatin C-based eGFR may provide additional value for the comprehensive evaluation of the relationship between systemic inflammation and renal functional status in COPD.

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