Pulmonary Vascular Phenotype in COPD: Definition and Clinical Implications

Jieun Kang

Organization Seoul National University, Seoul National University Bundang Hospital, Department of Internal Medicine
Current Position Clinical Professor

Educational background

2018-2020	Ph.D., University of Ulsan College of Medicine
2016-2018	M.A., University of Ulsan College of Medicine
2006-2013	B.A., Ajou University School of Medicine

Professional experience

2025-Present	Clinical Professor, Seoul National University Bundang Hospital, Department of Internal Medicine
2020-2024	Assistant Professor, Inje University College of Medicine, Ilsan Paik Hospital, Department of Internal Medicine
2018-2020	Fellow, Asan Medical Center, Department of Pulmonary and Critical Care Medicine
2014-2018	Resident, Asan Medical Center, Department of Internal Medicine

Chronic obstructive pulmonary disease (COPD) is classically defined by persistent airflow limitation due to chronic inflammation of the airways and lung parenchyma. Increasing evidence indicates that pulmonary vascular alterations constitute a significant, though frequently underrecognized, component of the disease. The vascular phenotype of COPD represents a distinct clinical and pathobiological entity, characterized by disproportionate pulmonary vascular remodeling and severe precapillary pulmonary hypertension (PH) that cannot be fully explained by the degree of airflow obstruction.

Patients with this phenotype typically present with severe PH, profound hypoxemia, and markedly reduced diffusing capacity for carbon monoxide, despite only mild-to-moderate airflow limitation. Histopathological examination reveals pronounced remodeling of the small pulmonary arteries, including intimal hyperplasia and luminal narrowing. Clinically, this phenotype is associated with adverse outcomes, including increased mortality, higher incidence of right heart failure, reduced exercise capacity, and more frequent hospitalizations compared with COPD patients without severe PH. The presence of severe PH is also associated with an elevated burden of cardiovascular comorbidities and serves as a predictor of poor survival.

Therapeutic options remain limited. Conventional pulmonary vasodilators used for pulmonary arterial hypertension are generally not recommended in COPD due to their limited efficacy and potential for worsening gas exchange. Notably, inhaled treprostinil, which demonstrated benefit in interstitial lung disease-associated PH, failed to improve exercise capacity in PH-COPD and was associated with increased serious adverse events, resulting in premature termination of clinical trials. Oxygen therapy remains the cornerstone for patients with chronic hypoxemia, while pulmonary rehabilitation may contribute to improved exercise tolerance. Optimal management requires a multidisciplinary approach, focusing on comorbidity control and individualized therapy. Continued research is needed to refine the phenotypic definition, identify reliable biomarkers, and establish evidence-based therapeutic strategies aimed at improving outcomes in this high-risk population.