Postinfectious Pulmonary Complications: What Pneumonia Leaves

Moon Seong Baek

Organization Department of Internal Medicine, Chung-Ang University Hospital, Chung-Ang University
Current Position Associate Professor

Educational background

2019-2023	Ph.D., University of Ulsan
2014-2016	M.A., Internal Medicine, Kangwon National University Graduate School
2000-2006	M.D., Wonkwang University College of Medicine

Professional experience

2020-Present	Associate Professor, Chung-Ang University College of Medicine
2018-2020	Assistant Professor, Hallym University Dongtan Sacred Heart Hospital
2015-2018	Clinical Fellow, Asan Medical Center
2011-2015	Residency in Internal Medicine, Wonkwang University Sanbon Hospital
2006-2007	Medical Internship, Ilsan Paik Hospital

Pulmonary sequelae that develop in survivors of lower respiratory tract infections are referred to as postinfectious pulmonary complications (PIPCs). While the long-term decline in lung function and increased morbidity among survivors of pulmonary tuberculosis are well recognized, the COVID-19 pandemic has heightened awareness of the broader spectrum of long-term pulmonary complications following pneumonia. Infection-related lung damage results from both pathogen-induced tissue destruction and the ensuing host immune response. Although the host immune response plays a critical role in pathogen clearance, an excessive or dysregulated response may paradoxically lead to local tissue injury, chronic inflammation, and aberrant remodeling and fibrosis. PIPCs can affect nearly all compartments of the lung, including the parenchyma, both large and small airways, as well as the pulmonary vasculature and endothelium. As a result, they may present with diverse manifestations such as pulmonary fibrosis, cavitary lesions, nodules, bronchiectasis, thrombosis, pleural thickening, and scarring.

Pulmonary injury mechanisms may differ depending on the pathogen. Respiratory viruses can directly damage lung tissue, disrupt the alveolar–capillary barrier, and trigger inflammatory responses that exacerbate tissue injury. There is a paucity of research on pathogen-specific factors underlying PIPCs following bacterial pneumonia. Neutrophils clear pathogens via phagocytosis, formation of neutrophil extracellular traps (NETs), degranulation, and production of reactive oxygen species; however, excessive activation can cause significant tissue injury. In addition, although it remains unclear whether Mycobacterium tuberculosis (Mtb) directly causes chronic lung damage, its virulence factors likely contribute indirectly by modulating host responses. Although the underlying mechanisms of PIPCs remain poorly understood, further research is needed to address these gaps and improve long-term outcomes.