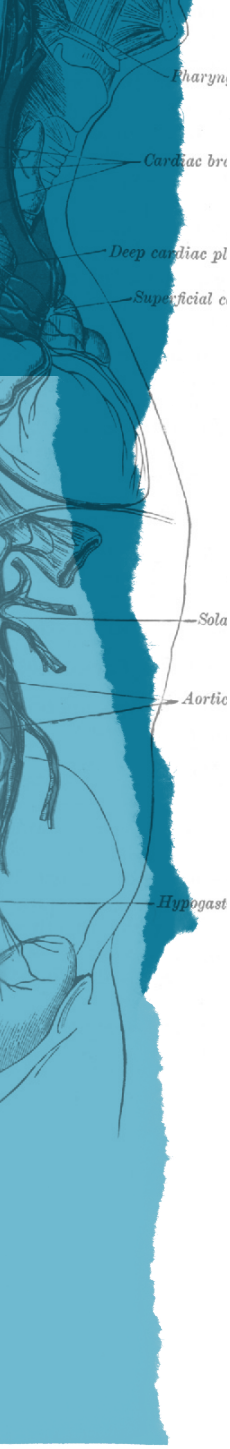

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Markers of Inflammation, Tissue Damage, and Fibrosis in Individuals Diagnosed with Human Immunodeficiency Virus and Pneumonia: A Cohort Study

Katherine Peña-Valencia[1,2,3], Will Riaño[2,4], Mariana Herrera-Diaz[5], Lucelly López[2,6], Diana Marín[2,6], Sandra Gonzalez[5,7], Olga Agudelo-García[3], Iván Arturo Rodríguez-Sabogal[4,8], Lázaro Vélez[8], Zulma Vanessa Rueda[2,5], Yoav Keynan[5,9,10,11]

[1] Escuela de Microbiología, Universidad de Antioquia, Medellín (Colombia).
[2] Grupo de Investigación en Salud Pública, Facultad de Medicina, Universidad Pontificia Bolivariana, Medellín (Colombia).
[3] Grupo Bacterias & Cáncer, School of Medicine, Universidad de Antioquia, Medellín (Colombia).
[4] School of Medicine, Universidad de Antioquia, Medellín (Colombia).
[5] Department of Medical Microbiology and Infectious Disease, University of Manitoba, Winnipeg (Canada).
[6] School of Medicine, Universidad Pontificia Bolivariana, Medellín (Colombia).
[7] JC Wilt Infectious Diseases Research Center, Winnipeg (Canada).
[8] Infectious Diseases Section, Hospital Universitario San Vicente Fundación, Medellín (Colombia).
[9] Grupo Investigador de Problemas en Enfermedades Infecciosas (GRIPE), Facultad de Medicina, Universidad de Antioquia, Medellín (Colombia).
[10] Department of Internal Medicine, University of Manitoba, Winnipeg (Canada).
[11] Department of Community Health Sciences, University of Manitoba, Winnipeg (Canada).

Correspondence: Katherine Peña-Valencia. katherine.penav@udea.edu.co

Katherine Peña-Valencia, Will Riaño, Mariana Herrera-Diaz, Lucelly López, Diana Marín, Sandra Gonzalez, Olga Agudelo-García, Iván Arturo Rodríguez-Sabogal, Lázaro Vélez, Zulma Vanessa Rueda, Yoav Keynan

ABSTRACT

Introduction: Previous studies have noted that persons living with human immunodeficiency virus (HIV) experience persistent lung dysfunction after an episode of community-acquired pneumonia (CAP), although the underlying mechanisms remain unclear. We hypothesized that inflammation during pneumonia triggers increased tissue damage and accelerated pulmonary fibrosis, resulting in a gradual loss of lung function.

Methods: We carried out a prospective cohort study of people diagnosed with CAP and/or HIV between 2016 and 2018 in three clinical institutions in Medellín, Colombia. Clinical data, blood samples, and pulmonary function tests (PFTs) were collected at baseline.

Results: Forty-one patients were included, divided into two groups: HIV and CAP (n = 17) and HIV alone (n = 24). We compared the concentrations of 17 molecules and PFT values between the groups. Notably, patients with HIV and pneumonia presented elevated levels of cytokines and chemokines (IL-6, IL-8, IL-18, IL-1RA, IL-10, IP-10, MCP-1, and MIP-1β) compared to those with only HIV. A marked pulmonary dysfunction was evidenced by significant reductions in FEF25, FEF25-75, and FEV1. Moreover, the correlation between these immune mediators and lung function parameters supports the connection between pneumonia associated inflammation and end-organ lung dysfunction. A low CD4 cell count (< 200 cells/μL) predicted inflammation and lung dysfunction.

Conclusions: These results underscore the need for targeted clinical approaches to mitigate the adverse impacts of CAP on lung function in this population.

Keywords: inflammation, cytokines, pulmonary dysfunction, HIV, pneumonia.