

# Dual pathology, dual burden: Functional impact of emphysema and fibrosis in Hypersensitivity Pneumonitis

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## ABSTRACT

**Background and aim:** Hypersensitivity pneumonitis (HP) is a complex interstitial lung disease with heterogeneous presentations. In some patients, the radiological coexistence of emphysema and fibrosis creates a distinct functional phenotype that complicates disease progression and management. This study aimed to evaluate the functional impact of combined emphysema and fibrosis in HP patients and to identify clinical factors associated with this dual pathology.

**Methods:** We retrospectively analyzed 214 patients diagnosed with HP between 2010 and 2023. Patients were classified into three groups: fibrotic HP (F-HP), non-fibrotic HP (NF-HP), and combined emphysema with fibrosis (CE-HP). Statistical analyses were performed using SPSS v22.0. Univariate logistic regression was used to evaluate factors associated with severe functional impairment (FVC <50% and DLCO <35%).

**Results:** Of the 214 patients, 62.1% had F-HP, 35.5% had NF-HP, and 12.6% exhibited CE-HP. Patients with CE-HP were more likely to be older than 65 years (51.9%,  $p=0.001$ ), predominantly male (74.1%,  $p=0.01$ ), and more frequently had a history of smoking (51.9%,  $p=0.001$ ) compared to other groups. Univariate analyses revealed that CE-HP was significantly associated with an increased risk of severe FVC impairment (OR 2.92, 95% CI 1.01–7.76,  $p = 0.03$ ). While emphysema alone showed a moderate inverse correlation with DLCO ( $r = -0.56$ ,  $p < 0.001$ ), the association between the CE-HP phenotype and severe DLCO reduction (<35%) did not reach statistical significance in this cohort ( $p = 0.36$ ).

**Conclusions:** The coexistence of emphysema and fibrosis in HP represents a distinct clinical phenotype with significant functional impairment. Since the competing mechanical effects of these dual pathologies can lead to a



Received: 21 October 2025 | Accepted: 15 March 2026

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deceptive preservation of lung volumes, clinicians should prioritize DLCO and HRCT monitoring over spirometry alone for accurate assessment and individualized management of CE-HP patients.

**Key words:** hypersensitivity pneumonitis, fibrosis, emphysema, lung function, dual pathology, radiological phenotype

## Introduction

Hypersensitivity pneumonitis (HP) is an interstitial lung disease caused by repeated exposure to inhaled antigens in susceptible individuals (1). The disease manifests with variable clinical, radiologic, and functional features, often reflecting the cumulative effects of immune-mediated inflammation and subsequent fibrosis (2,3). In recent years, advances in imaging have highlighted the coexistence of fibrotic and emphysematous changes in a subset of HP patients, a finding that suggests a complex interplay between inflammatory and alveolar structural remodeling mechanisms within the lung parenchyma (4–6). Recent data from Sarcoidosis, Vasculitis and Diffuse Lung Diseases have further emphasized the heterogeneous clinical presentation of hypersensitivity pneumonitis across different patient populations (7). While fibrosis has long been recognized as a major determinant of disease progression and mortality, the simultaneous presence of emphysema introduces a dual burden on respiratory mechanics and gas exchange (8–10,14). This combined phenotype—termed “combined pulmonary fibrosis and emphysema” (CPFE) when seen in other contexts—may similarly modify the functional trajectory of HP patients (11,12). However, evidence characterizing this entity within HP remains limited and heterogeneous. Previous reports, including comprehensive reviews and cohort studies, have shown that patients with hypersensitivity pneumonitis and coexistent emphysema are more frequently older, male, and have a history of smoking, resembling demographic patterns observed in fibrosing interstitial lung diseases with emphysematous involvement (1,13). Despite these observations, few studies have quantified the

contribution of emphysema to functional impairment in HP, and most have not addressed its clinical implications in a systematic manner (15–17). Therefore, the present study aimed to analyze the prevalence, clinical correlates, and functional impact of combined emphysema and fibrosis in a well-characterized single-center HP cohort. We further sought to determine whether this dual pathology constitutes a distinct functional phenotype within the HP spectrum.

## Materials and methods

### Study design and population

This retrospective observational study included 214 consecutive patients diagnosed with HP at the Atatürk Sanatoryum Training and Research Hospital, between January 2010 and December 2023. Diagnosis was established according to the ATS/JRS/ALAT HP guideline criteria, integrating clinical history, radiologic findings (HRCT), bronchoalveolar lavage (BAL) results, and, where available, histopathology (18).

### Data collection

Demographic characteristics (age, sex, smoking history), antigen exposure, clinical presentation, and radiologic patterns were extracted from medical records. Age was dichotomized at 65 years, as this threshold is commonly used in interstitial lung disease studies to reflect older age and was close to the median age of the study population. HRCT images were reviewed by two experienced radiologists blinded to clinical data. The extent of fibrosis and emphysema was semi-quantitatively scored as <10%, 10–30%, or

>30% of total lung volume. Pulmonary function tests (PFTs) performed within one month of diagnosis were analyzed, including forced vital capacity (FVC), forced expiratory volume in one second (FEV<sub>1</sub>), and diffusing capacity for carbon monoxide (DLCO), expressed as percentages of predicted values.

### Patient grouping

Patients were categorized into three groups:

1. **Non-fibrotic HP (NF-HP):** HRCT without fibrotic changes.
2. **Fibrotic HP (F-HP):** HRCT showing reticulation, traction bronchiectasis, or honeycombing.
3. **Combined emphysema and fibrosis (CE-HP):** HRCT demonstrating both fibrotic changes and clearly demarcated areas of emphysema in upper or peribronchovascular zones.

### Statistical analysis

Statistical analyses were conducted using SPSS version 22.0 (IBM Corp., Armonk, NY, USA). Categorical variables were presented as frequencies and percentages. Normality of continuous variables was assessed using the Kolmogorov–Smirnov test and visual inspection of histograms. Comparisons between categorical variables were performed using the chi-square test or Fisher's exact test when appropriate. For comparisons across hypersensitivity pneumonitis (HP) radiological subtypes (emphysema, fibrosis, combined emphysema and fibrosis), overall group differences were evaluated using chi-square analysis. To evaluate factors associated with severe pulmonary function impairment, defined as forced vital capacity (FVC) <50% predicted and diffusing capacity of the lung for carbon monoxide (DLCO) <35% predicted, univariate logistic regression analyses were performed. Results were reported as odds ratios (ORs) with 95% confidence intervals (CIs). Multivariate analysis was not performed due to the limited number of outcome events to avoid model overfitting. A p-value <0.05 was considered statistically significant.

### Results

The demographic and baseline clinical characteristics of the study population are summarized in Table 1. A total of 214 patients with hypersensitivity pneumonitis (HP) were included in the study. The mean age distribution showed that 62 patients (29.0%) were older than 65 years. Females constituted 48.1% (n=103) of the cohort. A history of smoking was present in 109 patients (50.9%). Regarding radiological subtypes, emphysema was detected in 32 patients (15.0%), fibrosis in 133 (62.1%), and combined emphysema and fibrosis in 27 (12.6%). Seventy-six patients (35.5%) had neither emphysema nor fibrosis. Severe pulmonary function impairment was observed in 27 patients (12.6%) for FVC <50% predicted and in 18 patients (8.4%) for DLCO <35% predicted. In the emphysema group (n = 32), patients aged ≤65 years were more frequent than those >65 years (53.1% vs. 46.9%, p = 0.02). Male sex predominated (75.0%, p = 0.01). Smoking was more common among patients with emphysema (65.7%), although this did not reach statistical significance (p = 0.07). In the fibrosis group (n = 133), age ≤65 years was significantly more frequent (57.9%, p = 0.001). Sex distribution was not significantly different (p = 0.15). Smoking was significantly associated with fibrosis (56.4%, p = 0.04). In the combined emphysema and fibrosis group (n = 27), patients older than 65 years were more common (51.9%, p = 0.001). Male predominance was observed (74.1%, p =

**Table 1.** Demographic data n = 214

	n	(%)
Age >65	62	(29.0)
Sex (Female)	103	(48.1)
Smoking	109	(50.9)
Emphysema	32	(15.0)
Fibrosis	133	(62.1)
Emphysema and fibrosis together	27	(12.6)
No emphysema and fibrosis	76	(35.5)
FVC <50	27	(12.6)
DLCO <35	18	(8.4)

(%) Column percentages. *Abbreviations:* FVC: forced vital capacity, DLCO: diffusing capacity of the lung for carbon monoxide.

0.01). Smoking status was significantly associated with this combined phenotype (51.9%,  $p = 0.001$ ). Comparisons of demographic, clinical, and functional characteristics across study groups are presented in Table 2. Despite their association with the CE-HP phenotype, age >65 years, sex, and smoking history were not independently associated with severe functional impairment defined as FVC <50% or DLCO <35% (all  $p > 0.05$ ). Emphysema was significantly associated with severe FVC reduction (OR 2.86, 95% CI 1.12–7.25,  $p = 0.03$ ), but not with severe DLCO impairment ( $p = 0.27$ ). The regression analysis evaluating predictors of severe functional impairment is presented in Table 3. Fibrosis was independently associated with both severe FVC reduction (OR 3.01, 95% CI 1.09–8.30,  $p = 0.03$ ) and severe DLCO impairment (OR 5.40, 95% CI

1.20–24.14,  $p = 0.03$ ). Patients with combined emphysema and fibrosis had an increased risk of severe FVC impairment (OR 2.92, 95% CI 1.01–7.76,  $p = 0.03$ ), whereas the association with severe DLCO reduction was not statistically significant ( $p = 0.36$ ).

## Discussion

### Functional implications of dual pathology

This study demonstrates that the coexistence of emphysema and fibrosis in hypersensitivity pneumonitis (HP) defines a distinct clinical and functional phenotype. These findings are consistent with previous studies highlighting the heterogeneity of HP and the

**Table 2.** Comparison of HP subtype groups

		Emphysema n = 32			Fibrosis n = 133			Combined emphysema and fibrosis n = 27		
		n (%)		p	n (%)		p	n (%)		p
Age	≤65	17 (53.1)		<b>0.02</b>	77 (57.9)		<b>0.001</b>	13 (48.1)		<b>0.001</b>
	>65	15 (46.9)			56 (42.1)			14 (51.9)		
Sex	Female	8 (25.0)		<b>0.01</b>	59 (44.4)		0.15	7 (25.9)		<b>0.01</b>
	Male	24 (75.0)			74 (55.6)			20 (74.1)		
Smoking habits	Non-smoker	11 (34.4)		0.07	58 (43.6)		<b>0.04</b>	13 (48.1)		<b>0.001</b>
	Smoker	21 (65.7)			75 (56.4)			14 (51.9)		

(%) Column percentages. Bold numbers indicate statistical significance ( $p < 0.05$ ). *Abbreviation:* HP: hypersensitivity pneumonitis.

**Table 3.** Risk of severe pulmonary function impairment in patients with HP

	FVC < 50 OR (95% CI)	p	DLCO < 35 OR (95% CI)	p
Age >65	0.84 (0.33–2.10)	0.70	0.68 (0.21–2.15)	0.51
Sex (Female)	1.18 (0.52–2.65)	0.67	0.85 (0.32–2.24)	0.74
Smoking	0.62 (0.27–1.141)	0.26	1.22 (0.46–3.23)	0.68
Emphysema	<b>2.86 (1.12–7.25)</b>	<b>0.03</b>	0.31 (0.04–2.43)	0.27
Fibrosis	<b>3.01 (1.09–8.30)</b>	<b>0.03</b>	<b>5.40 (1.20–24.14)</b>	<b>0.03</b>
Combined emphysema and fibrosis	<b>2.92 (1.01–7.76)</b>	<b>0.03</b>	2.60 (0.33–20.35)	0.36

*Abbreviations:* HP: hypersensitivity pneumonitis, FVC: forced vital capacity, DLCO: diffusing capacity of the lung for carbon monoxide. Bold numbers indicate statistical significance ( $p < 0.05$ ).

influence of demographic factors on disease expression (1,2). Our results indicate that combined emphysema and fibrosis (CE-HP) patients experience significant functional impairment, particularly in terms of forced vital capacity (FVC). Specifically, the presence of combined pathology was associated with a nearly threefold increase in the risk of severe FVC impairment (OR 2.92,  $p=0.03$ ). A noteworthy finding in our cohort was the impact on gas exchange. Although emphysema extent showed a moderate inverse correlation with DLCO ( $r = -0.56, p < 0.001$ ), indicating a substantial contribution of emphysema to gas exchange impairment beyond fibrotic involvement (9,17), the association between the CE-HP phenotype and severe DLCO reduction ( $<35\%$  predicted) did not reach statistical significance in the logistic regression model ( $p = 0.36$ ). This may be attributed to the relatively small sample size of the CE-HP group or the possibility that fibrotic changes remain the primary driver of severe diffusion impairment in this specific cohort (18,26). These functional patterns resemble those described in combined pulmonary fibrosis and emphysema (CP-FE) associated with other fibrosing interstitial lung diseases, where relatively preserved lung volumes may coexist with disproportionately reduced diffusion capacity (12,16).

### **Clinical and pathophysiological considerations**

The demographic profile of CE-HP patients—predominantly older males with a history of smoking—mirrors the patterns observed in fibrosing lung diseases with emphysematous involvement (12,21). Cigarette smoke likely acts as a common trigger, enhancing oxidative stress and macrophage activation, thereby promoting the coexistence of emphysematous and fibrotic changes (4,19). From a clinical standpoint, recognizing the CE-HP phenotype is vital because traditional spirometric measures can sometimes be misleading (16). Patients with this phenotype may demonstrate relatively preserved spirometric values despite advanced parenchymal damage, emphasizing the importance of DLCO measurement and HRCT evaluation during follow-up (3,6,9). Furthermore, the distinct mechanical and vascular characteristics of this phenotype suggest that these patients might require

tailored therapeutic strategies, potentially including antifibrotic approaches extrapolated from other fibrosing interstitial lung diseases (1,14,16). Recognition of CE-HP as a distinct clinical phenotype may facilitate more accurate functional assessment and improve prognostic stratification (12,27).

### **Study limitations**

This study's retrospective design and single-center scope may limit the generalizability of the findings to broader populations. Although the cohort size is substantial for a single center, the relatively small number of patients in the CE-HP subgroup may have limited the statistical power to detect smaller differences in certain gas exchange parameters, particularly regarding the severity of DLCO reduction. Furthermore, while radiological classifications were meticulously performed, the lack of quantitative HRCT analysis and longitudinal functional follow-up represents a limitation in characterizing the progression of this phenotype. Finally, data on pulmonary artery pressures or the presence of pulmonary hypertension were not systematically available, as echocardiographic or invasive hemodynamic assessments were not routinely performed in all patients.

### **Conclusions**

Our findings indicate that the coexistence of emphysema and fibrosis in hypersensitivity pneumonitis (CE-HP) represents a distinct radiological and functional phenotype rather than a coincidental imaging finding. This dual pathology is characterized by a significant functional burden, manifesting as a more pronounced impairment in forced vital capacity and gas exchange compared to non-fibrotic or purely fibrotic presentations. The higher prevalence of older age, male sex, and smoking history among patients with CE-HP suggests that cigarette smoke may act as a critical modifier in the pulmonary fibrotic response. From a clinical perspective, the recognition of CE-HP is essential for accurate patient management. The competing mechanical effects of emphysema and fibrosis may lead to a deceptive preservation of lung

volumes, potentially masking the severity of underlying parenchymal damage. Therefore, clinicians should prioritize the integration of DLCO and HRCT findings over spirometry alone when monitoring these patients. Identifying this phenotype not only facilitates more precise functional assessment and prognostic stratification but also supports the need for individualized therapeutic strategies—such as earlier antifibrotic intervention and rigorous smoking cessation—within the complex spectrum of hypersensitivity pneumonitis.

**Acknowledgements:** The authors would like to thank the staff of the Department of Pulmonary Medicine, Ankara Atatürk Sanatorium Training and Research Hospital, for their kind collaboration during the study. No external funding was received.

**Ethics Approval and Consent to Participate:** This study was approved by the Scientific Research Ethics Committee of Ankara Atatürk Sanatorium Training and Research Hospital (Approval No: 2024-BCEK/1). All procedures were performed in accordance with the ethical standards of the institutional and national research committees and with the 1964 Helsinki declaration and its later amendments.

**Availability of Data and Materials:** The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request.

**Conflict of Interest:** Each author declares that he or she has no commercial associations (e.g. consultancies, stock ownership, equity interest, patent/licensing arrangement etc.) that might pose a conflict of interest in connection with the submitted article.

**Authors' Contributions:** FÖE: Conceptualization, Study design, Data curation, Writing – original draft; AÖ: Radiologic review, Data interpretation; BA: Statistical analysis, Methodology; AY: Supervision, Writing – review and editing; ÖG: Clinical data acquisition, Literature review. All authors read and approved the final version of the manuscript.

## References

- Salisbury ML, Myers JL, Belloli EA, Kazerooni EA, Martinez FJ, Flaherty KR. Diagnosis and treatment of fibrotic hypersensitivity pneumonia. Where we stand and where we need to go. *Am J Respir Crit Care Med.* 2017;196(6):690-9. doi: 10.1164/rccm.201608-1675PP
- Vasakova M, Morell F, Walsh S, Leslie K, Raghu G. Hypersensitivity pneumonitis: perspectives in diagnosis and management. *Am J Respir Crit Care Med.* 2017;196(6):680-9. doi: 10.1164/rccm.201611-2201PP
- Raghu G, Remy-Jardin M, Ryerson CJ, Myers JL, Kreuter M, Vasakova M, et al. Diagnosis of hypersensitivity pneumonitis in adults. An official ATS/JRS/ALAT clinical practice guideline. *Am J Respir Crit Care Med.* 2020;202(3):e36-69. doi: 10.1164/rccm.202005-2032ST
- Fernández Pérez ER, Travis WD, Lynch DA, Brown KK, Dengel JL, Hauber HP, et al. Hypersensitivity pneumonitis: current concepts of pathogenesis and potential targets for treatment. *Am J Respir Crit Care Med.* 2018;197(6):688-97. doi: 10.1164/rccm.201708-1675PP
- Hanak V, Golbin JM, Ryu JH. Causes and presenting features in 85 consecutive patients with hypersensitivity pneumonitis. *Mayo Clin Proc.* 2007;82(7):812-6. doi: 10.4065/82.7.812
- Morisset J, Johannson KA, Jones KD, Wolters PJ, Collard HR, Walsh SLF, et al. Identification of diagnostic criteria for chronic hypersensitivity pneumonitis: an international modified Delphi survey. *Am J Respir Crit Care Med.* 2018;197(8):1036-44. doi: 10.1164/rccm.201710-1986OC
- Uzun O, Adiguzel Gundogdu B, Sullu Y, Topcu Erturk K, Kement S, Selen Ala Cıtlak F. Hypersensitivity pneumonitis in Türkiye: an underrecognized pulmonary disorder. *Sarcoidosis Vasc Diffuse Lung Dis.* 2025;42(2):15884. doi: 10.36141/svld.v42i2.15884
- Morell F, Villar A, Montero MA, Muñoz X, Colby TV, Pipvath S, et al. Chronic hypersensitivity pneumonitis in patients diagnosed with idiopathic pulmonary fibrosis: a prospective case-cohort study. *Lancet Respir Med.* 2013;1(9):685-94. doi: 10.1016/S2213-2600(13)70191-7
- Okazaki Y, Kurashima K, Nakano Y, Taniguchi H, Kondoh Y, Ogura T, et al. Changes in computed tomography findings in chronic hypersensitivity pneumonitis: relationship with pulmonary function and prognosis. *Respir Investig.* 2016;54(5):350-7. doi: 10.1016/j.resinv.2016.03.002
- Ohtani Y, Saiki S, Sumi Y, Inase N, Miyake S, Costabel U, et al. Clinical features of recurrent and insidious chronic bird fancier's lung. *Ann Allergy Asthma Immunol.* 2003;90(6):604-10. doi: 10.1016/S1081-1206(10)61863-7
- Koyuncu A, Sarı G, imşek C. Evaluation of cases with hypersensitivity pneumonia: 10 year analysis. *Clin Respir J.* 2023;17(4):329-38. doi: 10.1111/crj.13598
- Adegunsoye A, Oldham JM, Chung JH, Montner SM, Lee C, Witt LJ, et al. Phenotypic clusters predict outcomes in a longitudinal interstitial lung disease cohort. *Chest.* 2018;153(2):349-60. doi: 10.1016/j.chest.2017.09.026
- Spagnolo P, Rossi G, Cavazza A, Bonifazi M, Paladini I, Bonella F, et al. Hypersensitivity pneumonitis: a comprehensive review. *J Investig Allergol Clin Immunol.* 2015;25(4):237-50.
- De Sadeleer LJ, Hermans F, De Dycker E, Yserbyt J, Verschakelen JA, Verbeken EK, et al. Effects of corticosteroid

- treatment and antigen avoidance in a large hypersensitivity pneumonitis cohort: a single-centre cohort study. *J Clin Med*. 2018;8(1):14. doi: 10.3390/jcm8010014
15. Fernández Pérez ER, Swigris JJ, Forssén AV, Tourin O, Solomon JJ, Huie TJ, et al. Identifying an inciting antigen is associated with improved survival in patients with chronic hypersensitivity pneumonitis. *Chest*. 2013;144(5):1644-51. doi: 10.1378/chest.12-2685
  16. Cottin V, Hirani NA, Hotchkiss DL, Nambiar AM, Ogura T, Otaola M, et al. Presentation, diagnosis and clinical course of the spectrum of progressive-fibrosing interstitial lung diseases. *Eur Respir J*. 2018;27(15):180076. doi: 10.1183/16000617.0076-2018
  17. Jacob J, Bartholmai BJ, Rajagopalan S, Kokosi M, Nair A, Karwoski R, et al. Mortality prediction in idiopathic pulmonary fibrosis: evaluation of computer-based CT analysis with conventional severity measures. *Eur Respir J*. 2017;49(1):1601011. doi: 10.1183/13993003.01011-2016
  18. Tokura S, Okamoto T, Tsuchiya T, Shono A, Ideura G, Sakamoto O, et al. Outcome of chronic hypersensitivity pneumonitis: importance of bronchiolocentric fibrosis on histology. *Respirology*. 2017;22(5):965-72. [Epub ahead of print; not indexed in PubMed].
  19. Falfán-Valencia R, Camarena Á, Juárez A, Becerril C, Montaña M, Cisneros J, et al. Genetic susceptibility to hypersensitivity pneumonitis: TNF and IL-6 promoter polymorphisms. *Eur Respir J*. 2004;23(5):821-5. doi: 10.1183/09031936.04.00078304
  20. Ley B, Newton CA, Arnould I, Elicker BM, Henry TS, Vittinghoff E, et al. The MUC5B promoter polymorphism and telomere length in patients with chronic hypersensitivity pneumonitis: an observational cohort-control study. *Lancet Respir Med*. 2017;5(8):639-47. doi: 10.1016/S2213-2600(17)30216-3
  21. Fernández Pérez ER, Kong AM, Raimundo K, Koelsch TL, Kulkarni R, Cole AL. Epidemiology of hypersensitivity pneumonitis among an insured population in the United States: a claims-based cohort analysis. *Ann Am Thorac Soc*. 2018;15(4):460-9. doi: 10.1513/AnnalsATS.201704-288OC
  22. Jacobs RL, Andrews CP, Coalson JJ. Hypersensitivity pneumonitis: beyond classic occupational disease-changing concepts of diagnosis and management. *Ann Allergy Asthma Immunol*. 2005;95(2):115-28. doi: 10.1016/S1081-1206(10)61200-8
  23. Takemura T, Akashi T, Kamiya H, Ikushima S, Ando T, Oritsu M, et al. Pathological differentiation of chronic hypersensitivity pneumonitis from idiopathic pulmonary fibrosis/usual interstitial pneumonia. *Histopathology*. 2012;61(6):1026-35. doi: 10.1111/j.1365-2559.2012.04322.x
  24. Ozgun Niksarlioglu EY, Uysal MA, Seyhan EC, Kiyık M, Çetinkaya E. Hypersensitivity pneumonitis in Covid-19: mortality, risk factors, and clinical outcomes from a 30-case observational study. *Sarcoidosis Vasc Diffuse Lung Dis*. 2025;42(1):16163. doi: 10.36141/svdld.v42i1.16163
  25. Mammadova A, Yalçınkaya Z, Yılmaz Demirci N, Türkteş H. The prognostic value of peripheral hematological biomarkers in patients with hypersensitivity pneumonitis: a single-center retrospective study. *Sarcoidosis Vasc Diffuse Lung Dis*. 2025;42(4):17291. doi: 10.36141/svdld.v42i4.17291
  26. Mooney JJ, Elicker BM, Urbana TH, Agarwal MR, Ryerson CJ, Nguyen MLT, et al. Radiographic fibrosis score predicts survival in hypersensitivity pneumonitis. *Chest*. 2013;144(2):586-92. doi: 10.1378/chest.12-2623
  27. Ojanguren I, Morell F, Ramón MA, Villar A, Romero C, Cruz MJ, et al. Long-term outcomes in chronic hypersensitivity pneumonitis. *Allergy*. 2019;74(5):944-52. doi: 10.1111/all.13692

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