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(CASE REPORT)

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# Fatal progression of fibrotic hypersensitivity pneumonitis driven by persistent antigen exposure and socioeconomic barriers: A case report

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## Abstract

Hypersensitivity pneumonitis (HP) is an immunological interstitial lung disease. The fibrotic form is associated with a severe prognosis, particularly in the absence of antigen avoidance.

We report the case of a 58-year-old female patient, a former wool weaver, exposed to domestic mold for 15 years. She presented with progressive exertional dyspnea evolving toward Sadoul stage IV, associated with digital clubbing and 'velcro' crackles. Computed tomography (CT) showed signs of advanced fibrosis (honeycombing, traction bronchiectasis). Bronchoalveolar lavage (BAL) revealed a macrophagic predominance (90%) and precipitins to domestic mold were positive. Despite treatment with corticosteroids, the impossibility of environmental avoidance led to the patient's death.

This case highlights the need for early recognition of domestic exposure sources to prevent irreversible progression to fibrosis.

**Keywords:** Hypersensitivity pneumonitis; Domestic mold; Pulmonary fibrosis; Antigen avoidance; Interstitial lung disease

## 1. Introduction

Hypersensitivity pneumonitis (HP) results from an immune reaction to the chronic inhalation of environmental antigens. The transition to a chronic fibrotic form represents a major prognostic turning point, making the disease clinically and radiologically similar to other idiopathic pulmonary fibroses. This case is particularly unique as it illustrates the tragic intersection of clinical severity and socioeconomic barriers; it highlights how the inability to achieve definitive antigen avoidance—due to financial constraints—acts as the primary driver of disease non-control, leading to frequent exacerbations and a fatal outcome despite specialized medical interventions. [1, 4, 6]

## 2. Case Report

The patient was a 58-year-old female, a former wool weaver and current merchant, presenting with progressive exertional dyspnea that had evolved over seven months to Sadoul stage IV, associated with a dry cough and deterioration of general condition (anorexia, asthenia, weight loss) in a context of chronic exposure to domestic mold for 15 years.

Physical examination revealed an oxygen saturation of 93% on room air, digital clubbing, and diffuse bilateral 'velcro-type' crackles. An initial AP chest X-ray showed diffuse bilateral reticulomicronodular opacities (Figure 1). high-

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resolution chest computed tomography (HRCT) showed signs of advanced fibrosis, including traction bronchiectasis, regular thickening of septal lines, and a characteristic honeycombing appearance (Figure 2). Biological workup confirmed the presence of positive precipitins to domestic mold, while the immunological profile (ANA, anti-dsDNA, SSA, SSB) and angiotensin-converting enzyme levels were negative. Bronchoscopy revealed diffuse grade 2 inflammation with bronchoalveolar lavage (BAL) fluid showing a macrophagic predominance (90%). Functionally, the 6-minute walk test documented severe exertional desaturation, dropping from 75% to 67% within the first minute. These elements led to the diagnosis of chronic fibrotic domestic hypersensitivity pneumonitis (HP). The case was submitted to a Multidisciplinary Discussion, where the progressive fibrosing profile of the hypersensitivity pneumonitis (HP) was definitively established. Following the identification of progressive phenotype, the initiation of anti-fibrotic therapy was formally recommended. However, while waiting to establish the formal indication and initiate anti-fibrotic treatment, the patient's lack of financial means made domestic antigen avoidance—the most critical therapeutic step—impossible (Figure 3). This persistent exposure led to non-control of the disease and frequent respiratory exacerbations. Although corticosteroid therapy at 40 mg/day was initiated, the inability to modify her environment resulted in an irreversible decline, and the outcome was ultimately fatal.

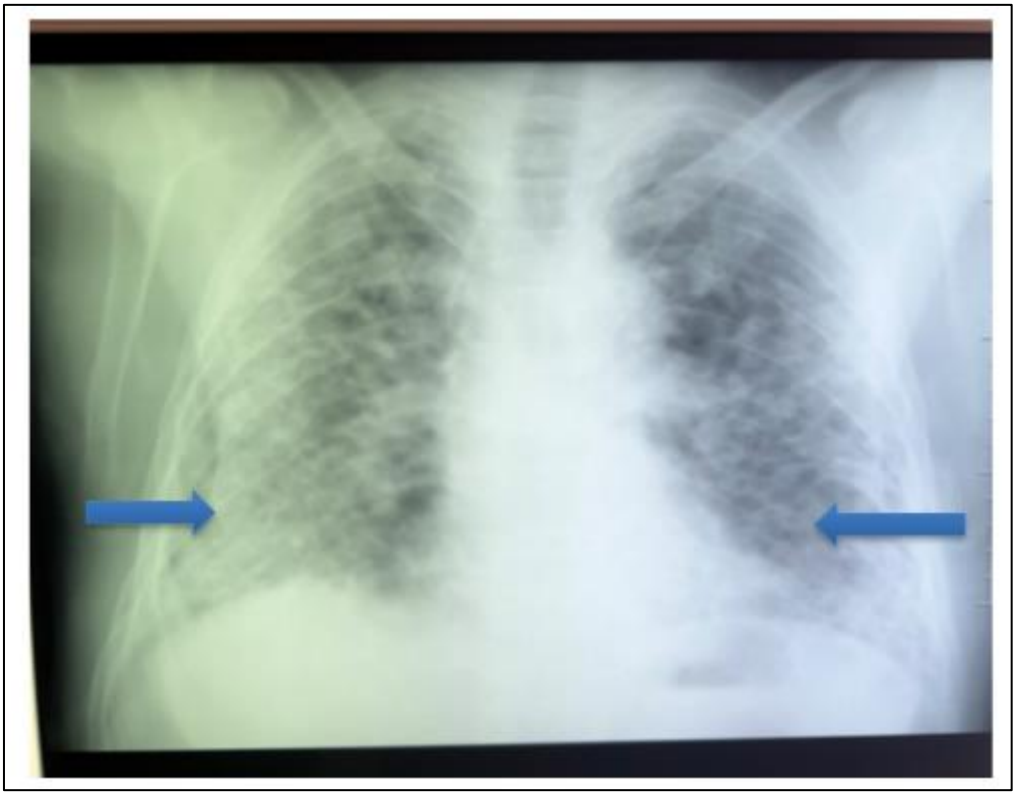

**Figure 1** AP Chest X-ray showing diffuse bilateral reticulomicronodular opacities

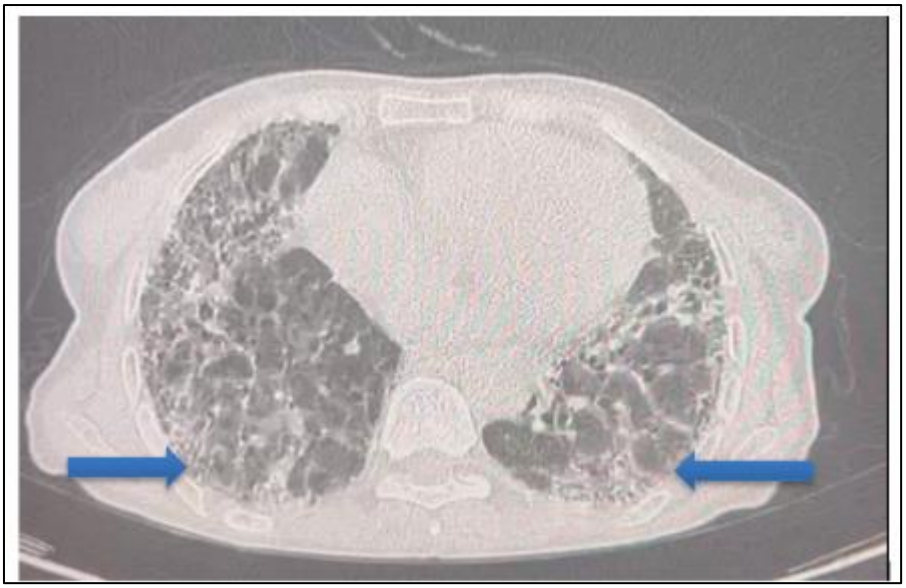

**Figure 2** Axial HRCT scan showing advanced fibrotic changes with honeycombing and traction bronchiectasis.

**Figure 3** Significant domestic mold infestation and severe wall dampness identified as the primary antigenic source

## 3. Discussion

Fibrotic hypersensitivity pneumonitis (HP) is a complex immunological lung disease whose diagnosis and management remain a major clinical challenge. According to the international guidelines of the ATS/JRS/ALAT (2020) [1], diagnosis relies on a multidisciplinary approach combining the identification of exposure, radiologic phenotype, and bronchoalveolar lavage (BAL) cellularity. In this case, the presence of positive serum precipitins to domestic mold correlated with a 15-year environmental exposure confirms the etiology. The patient's CT phenotype, marked by honeycombing and traction bronchiectasis, is a strong predictor of mortality, with a median survival often less than five years, approaching that observed in idiopathic pulmonary fibrosis (IPF) [1, 4].

A crucial diagnostic aspect in this observation is the BAL cellularity. While non-fibrotic HP is classically characterized by marked alveolar lymphocytosis (> 30%), the fibrotic form may present less specific cellularity. In our patient, the 90% macrophagic predominance perfectly illustrates what Vasakova et al. (2017) [2] describe as an immunological transition where lymphocytic inflammation gives way to an irreversible fibroblastic remodeling process. The absence of lymphocytosis should not rule out the diagnosis of HP at an advanced stage. The undisputed therapeutic mainstay of

HP is early and definitive antigen avoidance [6]. In this case, the patient's inability to achieve domestic avoidance sustained the immunopathological process. Although corticosteroid therapy at 40 mg/day was initiated, it often proves insufficient at the fibrotic stage. In this regard, the INBUILD study (2019) [7] opened new perspectives by showing that anti-fibrotic agents, such as nintedanib, can slow the decline in forced vital capacity in progressive fibrosing interstitial lung diseases, including HP. However, the fatal outcome in our patient emphasizes that these therapies, while useful, cannot substitute for antigen avoidance.

## 4. Conclusion

In conclusion, this case illustrates the clinical severity of domestic hypersensitivity pneumonitis (HP) in its chronic fibrotic form. The observation highlights that prolonged exposure to domestic mold, confirmed by positive serum precipitins, can lead to irreversible structural remodeling such as honeycombing seen on imaging. The fatal evolution of this 58-year-old patient, despite corticosteroid therapy at 40 mg/day, demonstrates that antigen avoidance is the indispensable pillar of treatment. Once the stage of advanced fibrosis is reached and in the absence of environmental modification, the prognosis remains grim. This case report recalls the necessity of early diagnosis based on a rigorous environmental investigation and highlights the importance of public health interventions to improve housing hygiene to prevent these terminal respiratory pathologies.

## Compliance with ethical standards

### *Disclosure of conflict of interest*

The author declares no conflict of interest

### *Statement of informed consent*

Informed consent was obtained from all individual participants included in the study.

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