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Progressive dyspnea in a farmer: Chronic hypersensitivity pneumonitis due to long-term organic dust exposure

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Abstract
Background: Hypersensitivity pneumonitis (HP) is an immune-mediated interstitial lung disease resulting from repeated inhalation of environmental antigens, most commonly in occupational settings. Chronic exposure may lead to progressive fibrotic lung disease, often mimicking idiopathic pulmonary fibrosis (IPF), resulting in delayed diagnosis and suboptimal management.
Case Presentation: We report a case of a 48-year-old male farmer who presented with progressive exertional dyspnea over 18 months. He had a significant occupational exposure history of daily contact with moldy hay and grain dust for more than 25 years. Clinical examination revealed tachypnea, resting hypoxemia, and bilateral end-inspiratory crackles predominantly in the upper lung zones. There was no history of smoking, fever, weight loss, or hemoptysis. Based on the characteristic exposure history, clinical features, and examination findings, a diagnosis of chronic hypersensitivity pneumonitis was strongly suspected.
Discussion: This case highlights the insidious nature of chronic HP and the critical role of detailed occupational and environmental exposure assessment in patients presenting with unexplained progressive dyspnea. Chronic HP frequently overlaps clinically and radiologically with other fibrotic interstitial lung diseases, particularly IPF, but differs significantly in etiology, management, and prognosis. Early recognition, antigen avoidance, and appropriate therapeutic intervention are essential to prevent irreversible pulmonary fibrosis.
Conclusion: Chronic hypersensitivity pneumonitis remains an underrecognized yet preventable cause of fibrotic lung disease, particularly among agricultural workers. Heightened clinical awareness, early multidisciplinary evaluation, and incorporation of occupational health strategies are crucial to improving patient outcomes and reducing disease burden.

Keywords: Hypersensitivity pneumonitis, Farmer's Lung, occupational lung disease, interstitial lung disease, mold exposure, fibrotic hypersensitivity pneumonitis

Introduction

Hypersensitivity pneumonitis (HP), also known as extrinsic allergic alveolitis, is a complex immune-mediated interstitial lung disease (ILD) caused by repeated inhalation of a wide range of environmental antigens in susceptible individuals [1]. These inhaled antigens trigger an exaggerated immune response involving the alveoli, terminal bronchioles, and pulmonary interstitium, leading to inflammation that may progress to irreversible fibrosis if exposure persists [1, 2]. Unlike many other ILDs, HP is unique in that it is potentially preventable and partially reversible, particularly when recognized early and managed appropriately [3].

Clinically, HP represents a diagnostic challenge due to its heterogeneous presentation, variable latency between exposure and symptom onset, and significant overlap with other diffuse parenchymal lung diseases such as idiopathic pulmonary fibrosis (IPF), non-specific interstitial pneumonia (NSIP), and sarcoidosis [1, 4]. Chronic HP often presents insidiously with progressive exertional dyspnea and cough, frequently leading to delayed diagnosis and misclassification as idiopathic fibrotic lung disease [4, 5].

Agricultural exposure remains one of the most well-recognized and globally prevalent causes of HP [6]. Farmer's lung, caused by inhalation of thermophilic actinomycetes and molds present in moldy hay and grain dust, continues to be a significant occupational health problem, especially in low- and middle-income countries [6, 7]. Prolonged exposure over decades predisposes individuals to chronic HP with fibrotic remodeling of the lung parenchyma [8].

Overview and Background

Hypersensitivity pneumonitis is an immune-mediated ILD resulting from abnormal host response to inhaled antigens rather than a direct toxic effect [1, 2]. The disease primarily affects the alveoli, terminal bronchioles, and surrounding interstitium, placing it within the spectrum of small-airway–centered ILDs [9].

Pathophysiologically, HP involves a combination of type III (immune complex–mediated) and type IV (cell-mediated delayed) hypersensitivity reactions [2, 10]. Persistent antigen exposure leads to lymphocytic alveolitis, macrophage activation, poorly formed non-caseating granulomas, and progressive fibroproliferative remodeling [10, 11].

Contemporary guidelines classify HP into non-fibrotic and fibrotic forms, recognizing fibrosis as the major determinant of long-term prognosis [1, 12]. Fibrotic HP is associated with progressive lung function decline and increased mortality and may clinically resemble IPF, though with important etiological and radiological distinctions [4, 13].

Historical Aspects and Recent Incidence Trends

Hypersensitivity pneumonitis was first described over three centuries ago, with early reports emerging from agricultural communities exposed to moldy hay [6]. The term "farmer's

lung" was formally introduced in the mid-20th century following epidemiological and microbiological studies identifying thermophilic actinomycetes as causative agents [6, 14].

During the latter half of the 20th century, recognition of additional occupational and environmental exposures expanded the disease spectrum to include bird fancier's lung, mushroom worker's lung, humidifier lung, and air-conditioner–related HP [2, 15]. The introduction of high-resolution computed tomography (HRCT) and bronchoalveolar lavage (BAL) analysis in the late 20th century significantly improved diagnostic accuracy [9, 16].

Recent ILD registry data suggest that fibrotic HP accounts for 10–20% of fibrotic ILDs previously labeled as idiopathic, reflecting improved recognition and reclassification [4, 5, 13]. The incidence of HP is now increasing not only in rural settings but also in urban environments due to exposure to water-damaged buildings, metalworking fluids, and bioaerosols [3, 17].

Epidemiology

HP accounts for approximately 5–15% of all interstitial lung diseases globally, with significant geographic variation depending on occupational and environmental exposure patterns [1, 3, 6]. Agricultural workers remain the most affected group worldwide, particularly in regions with traditional farming practices and limited occupational health measures [6, 7].

The disease predominantly affects individuals in the fourth to sixth decades of life, with a male predominance reflecting occupational exposure rather than biological susceptibility [2, 6].

Smoking appears to reduce the incidence of acute HP but may worsen outcomes in chronic fibrotic disease [18].

Underdiagnosis is particularly pronounced in developing countries due to lack of awareness, limited access to HRCT, and absence of multidisciplinary ILD clinics [7].

Clinical Manifestations

Clinical presentation depends on antigen load, exposure duration, and host immune response [1, 2].

Symptoms

Patients with chronic or fibrotic HP commonly present with:

- Progressive exertional dyspnea
- Persistent dry cough
- Fatigue and reduced exercise tolerance
- Absence of fever or weight loss, unlike acute HP [1, 6]

Physical Examination

Common findings include:

- Tachypnea
- Resting or exertional hypoxemia
- Fine end-inspiratory crackles, often upper or mid-zone predominant
- Digital clubbing is uncommon early but may appear in advanced fibrotic disease [9, 13]

Differential Diagnosis

The differential diagnosis of chronic HP includes several fibrotic ILDs and occupational lung diseases [1, 4]:

- **Idiopathic Pulmonary Fibrosis (IPF):** Basal-predominant, subpleural fibrosis with honeycombing and absence of exposure history [4, 19]
- **Non-Specific Interstitial Pneumonia (NSIP):** Uniform fibrosis often associated with connective tissue disease [20]
- **Sarcoidosis:** Lymphadenopathy, extrapulmonary involvement, and well-formed granulomas [21]
- Pneumoconiosis and organic dust lung disease [22]

Distinguishing HP from IPF is critical due to major differences in treatment and prognosis [4, 19].

Treatment Strategy

Antigen Avoidance

Antigen avoidance is the most important disease-modifying intervention and should be implemented immediately upon suspicion of HP [1, 6, 12].

Pharmacological Therapy

- Corticosteroids may accelerate symptomatic improvement in inflammatory disease but do not prevent fibrosis if exposure persists [6, 23].
- Immunosuppressive agents such as azathioprine or mycophenolate mofetil may be used in selected cases [20, 23].
- Antifibrotic therapy has demonstrated benefit in progressive fibrotic HP, reflecting evolving management paradigms [24, 25].

Supportive Care

Pulmonary rehabilitation, oxygen therapy, vaccination, and management of comorbidities are essential components of care [12].

Discussion

This case illustrates a classical presentation of chronic farmer's lung disease resulting from prolonged exposure to moldy hay and grain dust. The insidious symptom onset and absence of systemic features contributed to delayed diagnosis, a well-documented challenge in HP [2, 4, 6].

Failure to recognize HP early allows progression to fibrotic disease, emphasizing the importance of detailed occupational history and early referral to ILD specialists [1, 12].

Summary

Hypersensitivity pneumonitis is a preventable, exposure-driven ILD with significant potential for progression to fibrosis when diagnosis is delayed [1, 6]. Agricultural workers represent a high-risk population requiring targeted surveillance.

Conclusion

Chronic hypersensitivity pneumonitis is an underrecognized cause of fibrotic lung disease. Early diagnosis, prompt antigen avoidance, and appropriate therapy can significantly alter disease trajectory and improve outcomes [1, 12, 24].

Way Forward and Implications

Clinical

- Mandatory exposure history in all ILD patients
- Early HRCT and multidisciplinary evaluation [1, 12]

Academic

- Enhanced training on occupational lung diseases

- Research into biomarkers and exposure quantification [5, 16]

Policy

- Occupational health surveillance for agricultural workers
- Integration of ILD screening into rural health programs [7, 22]

Take-Home Messages

- Always enquire about occupational exposure in ILD patients
- Upper-lobe crackles with mosaic attenuation suggest HP
- Chronic HP can mimic IPF but has different management
- Early diagnosis can prevent irreversible lung damage

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