Case Report

From Antigen Exposure to Right Heart Failure: Chronic Hypersensitivity Pneumonitis Due to Bird Fancier's Lung: A Case Report

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Chronic hypersensitivity pneumonitis (CHP) is an immune-mediated interstitial lung disease resulting from prolonged antigen exposure, which can lead to progressive pulmonary fibrosis, pulmonary hypertension (PH), and cor pulmonale. We present a 46-year-old female with a 30-year history of domestic poultry rearing who presented with progressive exertional dyspnea (NYHA class III), bilateral lower limb edema, and abdominal distension. Clinical examination revealed hypoxemia (SpO_2 84% on room air), elevated jugular venous pressure, bilateral basal crepitations, a parasternal heave, a loud P_2, and signs of right heart failure, including hepatomegaly and ascites. High-resolution computed tomography (HRCT) of the chest demonstrated features consistent with fibrotic interstitial lung disease, including septal thickening, ground-glass opacities, mosaic attenuation, and air trapping, alongside cardiomediastinal findings of right heart and pulmonary artery enlargement. Echocardiography confirmed severe pulmonary arterial hypertension, right atrial and ventricular dilation, paradoxical septal motion, and severe tricuspid regurgitation, establishing the diagnosis of cor pulmonale. Serological workup was negative for connective tissue disease. The final diagnosis was chronic hypersensitivity pneumonitis (bird fancier's lung) with fibrotic progression, leading to pulmonary arterial hypertension, cor pulmonale, and right-sided heart failure. Management centered on strict antigen avoidance, supplemental oxygen, diuretics, and supportive care, resulting in clinical stabilization. This case underscores the severe cardiopulmonary sequelae of advanced CHP, highlights the critical importance of early occupational and exposure history-taking, and reinforces the foundational role of antigen avoidance in management, even in established fibrotic disease, to mitigate functional decline and mortality.

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Introduction

Interstitial lung diseases (ILDs) comprise a heterogeneous group of diffuse parenchymal lung disorders characterized by varying degrees of inflammation and fibrosis of the pulmonary interstitium, alveoli, and small airways, leading to impaired gas exchange and progressive respiratory insufficiency. Among these, hypersensitivity pneumonitis (HP) also referred to as extrinsic allergic alveolitis is an immune-mediated disorder caused by repeated inhalation of a wide variety of organic and inorganic antigens in susceptible individuals. Common inciting agents include avian proteins, mold spores, agricultural dusts, and occupational exposures.

Chronic hypersensitivity pneumonitis (CHP) represents the advanced stage of the disease, resulting from prolonged or intermittent antigen exposure, often due to delayed diagnosis or ongoing environmental contact. Unlike acute and subacute forms, CHP is characterized by progressive pulmonary fibrosis, architectural distortion, traction bronchiectasis, and honeycombing, frequently mimicking idiopathic pulmonary fibrosis. The fibrotic phenotype of CHP is associated with irreversible lung damage, declining pulmonary function, and poor prognosis.

Long-standing fibrotic lung disease in CHP leads to persistent hypoxemia and pulmonary vascular remodeling, culminating in pulmonary hypertension (PH). Progressive elevation of pulmonary arterial pressures imposes a chronic pressure overload on the right ventricle, eventually resulting in cor pulmonale, defined as right ventricular dysfunction secondary to diseases affecting the structure or function of the lungs. As the disease advances, patients may develop right heart failure, manifesting clinically as peripheral edema,

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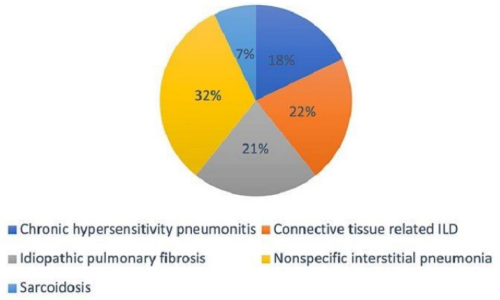

Figure 1: Type of ILD

elevated jugular venous pressure, hepatomegaly, ascites, and reduced exercise tolerance, further compounding morbidity and mortality.

The coexistence of CHP-associated ILD with cor pulmonale and right heart failure signifies an advanced stage of disease and poses significant diagnostic and therapeutic challenges. Early recognition of pulmonary hypertension and right ventricular involvement is crucial, as these complications markedly worsen functional status and survival. Despite advances in imaging, pulmonary function testing, and echocardiography, CHP remains under-recognized, particularly in regions where environmental and occupational exposures are prevalent.

This case highlights the clinical course, diagnostic challenges, and cardiopulmonary complications of chronic hypersensitivity pneumonitis progressing to fibrotic interstitial lung disease with cor pulmonale and right heart failure, underscoring the importance of early diagnosis, antigen avoidance, and timely management to prevent irreversible cardiopulmonary sequelae.

Case Report

A 46-year-old female presented with progressive breathlessness on exertion (NYHA class III), bilateral lower limb swelling, and abdominal distension for two months.

Occupational and Exposure History

On detailed history, the patient reported rearing chickens at home for over 30 years, with daily exposure to bird droppings, feathers, and poultry shed dust in poorly ventilated conditions. She recalled intermittent episodes of dry cough and breathlessness over the preceding several years, which were initially mild and ignored.

Clinical Progression

Initial phase

Recurrent cough and exertional dyspnea suggestive of hypersensitivity pneumonitis

Intermediate phase

Persistent symptoms with reduced exercise tolerance, consistent with progression to fibrotic interstitial lung disease

Late phase

Development of pedal edema, ascites, and signs of right-sided heart failure due to pulmonary arterial hypertension and cor pulmonale

Past history

- Hypothyroidism on irregular treatment
- No history of pulmonary tuberculosis, connective tissue disease, smoking, or biomass fuel exposure

General Examination

- Conscious and oriented
- Blood pressure: 110/70 mmHg
- Pulse rate: 110/min
- Oxygen saturation: 84% on room air
- Raised jugular venous pressure
- Bilateral pitting pedal edema

Systemic Examination

- Respiratory: Bilateral basal end-inspiratory crepitations
- Cardiovascular: Normal precordium, epigastric pulsations present, parasternal heave present; loud P2, S1S2 present, pansystolic murmur at tricuspid area was audible.
- Abdomen: Soft, not distended, RHC tender on palpation, Liver palpable just below the RCM- Grade I hepatomegaly present. Ascites present
- CNS: Normal

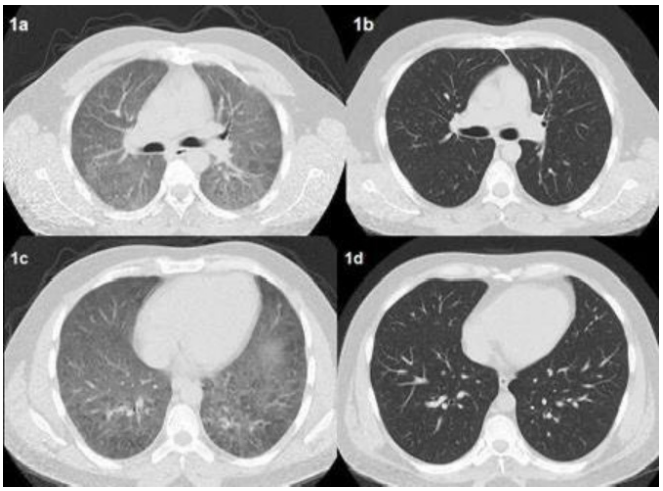

Figure 2: HRCT Chest

Figure 3: HRCT Chest

Investigations

Laboratory Findings

- Hemoglobin: 10 g/dL
- ESR: Mildly elevated
- Liver function tests: Hypoalbuminemia
- Thyroid profile: Elevated TSH
- ANA: Negative
- Rheumatoid factor: Negative
- Ascitic fluid: Lymphocyte predominant, no malignant cells
- Viral markers: Non-reactive

Radiological Findings

HRCT chest (Figures 2 and 3)

- HRCT chest revealed:
- Inter- and intralobular septal thickening
- Patchy ground-glass opacities
- Mosaic attenuation pattern
- Air trapping on expiratory images
- Features consistent with chronic hypersensitivity pneumonitis with fibrotic ILD
- Cardiac and vascular findings included:
- Dilated right atrium and right ventricle
- Enlarged pulmonary artery
- Mild bilateral pleural effusion
- Ascites and subcutaneous edema

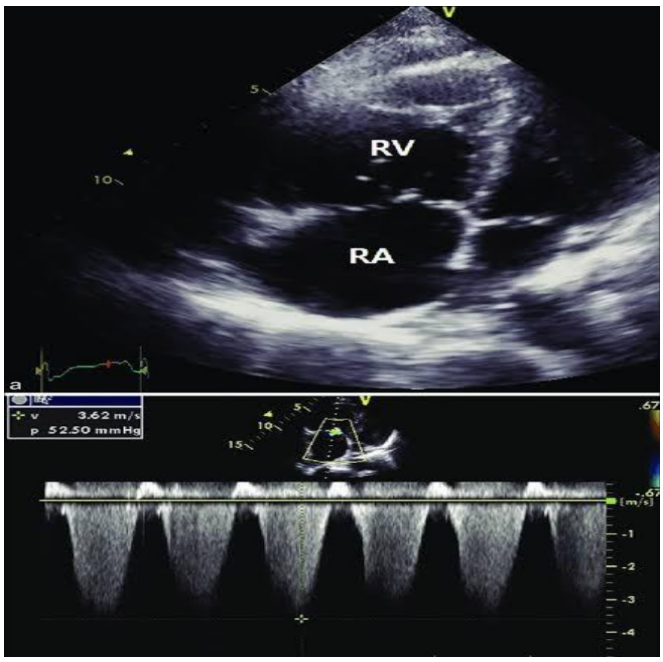

Figure 4: Echocardiography

Echocardiography (Figure 4)

- 2D-echocardiography showed:
- Dilated right atrium and right ventricle
- Severe tricuspid regurgitation
- Pulmonary arterial hypertension
- Paradoxical interventricular septal motion
- Left ventricular ejection fraction: 48–50%
- Findings were consistent with cor pulmonale secondary to chronic lung disease.

Final diagnosis

- Chronic hypersensitivity pneumonitis (bird fancier's lung)
- Fibrotic interstitial lung disease
- Pulmonary arterial hypertension
- Cor pulmonale
- Right-sided heart failure
- Hypothyroidism

Management and outcome

The patient was advised of strict antigen avoidance, including cessation of poultry rearing. She was managed with oxygen therapy, diuretics, thyroid hormone replacement, bronchodilators, and supportive care. Clinical improvement was noted, and she was discharged in stable condition with advice for long-term follow-up.

Discussion

Hypersensitivity Pneumonitis (HP)

A progressive fibrotic ILD hypersensitivity pneumonitis is an immune-mediated interstitial lung disease caused by repeated inhalation of specific antigens, leading to diffuse alveolar and interstitial inflammation. A common form is bird fancier's lung, resulting from sensitization to avian proteins.

Pathophysiology

Sensitization to fibrosis Inhalation triggers a combined type III (immune complex) and type IV (cell-mediated)

hypersensitivity response. The acute phase features lymphocytic alveolitis and non-necrotizing granulomas. Sustained exposure causes recurrent epithelial injury, dysregulated repair, and persistent inflammation, activating profibrotic pathways like TGF-β. This culminates in pathologic extracellular matrix deposition, resulting in:

- Interstitial expansion and fibrosis
- Alveolar architectural destruction
- Traction bronchiectasis and honeycombing
- Reduced lung compliance and impaired gas diffusion

Progression to Cor Pulmonale

In advanced chronic HP, chronic hypoxia and capillary loss from fibrosis induce pulmonary vascular remodeling, increased vascular resistance, and pulmonary hypertension (PH). This imposes chronic pressure overload on the right ventricle, leading to compensatory hypertrophy, dilation, and failure (cor pulmonale), indicating a poor prognosis.

Diagnostic Framework

Diagnosis requires multidisciplinary integration.

Clinical history

Identification of prolonged, repetitive antigen exposure.

Pulmonary function tests

Restrictive defect with disproportionately reduced DLCO; exertional hypoxemia.

HRCT

Fibrotic pattern with mid-to-upper zone reticulation, mosaic attenuation, traction bronchiectasis, and honeycombing.

Serology

Serum-specific IgG antibodies indicate sensitization but lack diagnostic specificity.

BAL

Lymphocytic alveolitis (>20-30% lymphocytes) supports diagnosis.

Cardiac evaluation

Echocardiography for PH screening; ECG for right heart strain; NT-proBNP as a biomarker.

Therapeutic strategies

Management targets immunopathology, fibrosis, and cardiopulmonary complications.

Antigen avoidance complete, permanent cessation is fundamental at all stages.

Immunomodulation

Systemic corticosteroids for inflammatory components; steroid-sparing agents (e.g., azathioprine) considered.

Antifibrotic therapy

Fibrotic HP is classified as progressive pulmonary fibrosis (PPF). Nintedanib reduces FVC decline; pirfenidone may be considered.

PH & RV failure management

Supportive with supplemental oxygen and diuretics; role of pulmonary vasodilators is uncertain.

Advanced care

Pulmonary rehabilitation; lung transplantation for eligible patients.

Prognostic Role of Antigen Avoidance

Avoidance cannot reverse established fibrosis but is critical for long-term outcomes. Continued exposure accelerates functional decline and increases mortality. In fibrotic chronic HP, rigorous cessation can promote stabilization, reduce acute exacerbations, and improve symptom control, especially with antifibrotic therapy. Complete antigen removal remains imperative regardless of disease stage.

Conclusion

Chronic bird-related hypersensitivity pneumonitis represents a preventable cause of progressive fibrotic ILD. Early recognition and antigen avoidance are critical to altering disease trajectory. Once fibrosis and cor pulmonale develop, management focuses on disease stabilization, symptom control, and slowing progression using antifibrotic agents and supportive cardiopulmonary care.

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