A Story of Hypersensitivity Pneumonitis Misdiagnosed Repeatedly as Acute Bronchitis

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***Abstract*—Hypersensitivity Pneumonitis (HP) is a rare interstitial lung disease caused by repeated exposure to environmental antigens, often misdiagnosed due to its nonspecific symptoms and radiological features. We report the case of a 19-year-old female with recurrent episodes of cough and dyspnea, repeatedly treated for acute bronchitis without improvement. High-resolution CT revealed diffuse ground-glass opacities with centrilobular nodules, and bronchoalveolar lavage showed lymphocytic alveolitis with a decreased CD4/CD8 ratio (0.38), confirming the diagnosis of HP. This case highlights the critical need for detailed exposure history, early imaging, and immunologic analysis to differentiate HP from common airway diseases. Prompt diagnosis and antigen avoidance are essential to prevent progression to chronic fibrotic forms and improve patient outcomes.**

***Index Terms*—Acute Bronchitis Mimic, Bronchoalveolar Lavage, CD4/CD8 Ratio, Environmental Antigen Exposure, Ground Glass Opacities, HRCT Thorax, Hypersensitivity Pneumonitis, Immune-Mediated Lung Disease, Interstitial Lung Disease, Misdiagnosis.**

I. INTRODUCTION

Hypersensitivity pneumonitis (HP), also known as extrinsic allergic alveolitis, is a complex immune-mediated disorder characterized by inflammation of the lung parenchyma and airways in response to repeated inhalation of environmental antigens. These antigens may be derived from occupational exposures (such as moldy hay, bird droppings, or contaminated humidifiers)[1] or from domestic sources like household molds, air conditioners, and pet birds.

The pathogenesis of HP involves a multifactorial interplay of genetic predisposition, immune dysregulation, and environmental triggers. Immunologically, HP is driven primarily by type III and IV hypersensitivity reactions, where TH1 and TH17 responses activate toll-like receptors, resulting in intense lymphocytic and neutrophilic alveolitis. Chronic exposure leads to progressive interstitial fibrosis in susceptible individuals, which significantly worsens prognosis.

Clinically, HP may manifest as an acute, subacute, or chronic condition, depending on the intensity and duration of exposure. Acute HP typically presents with sudden onset of cough, dyspnea, and flu-like symptoms within 4–8 hours of antigen exposure. Chronic HP, on the other hand, develops insidiously with persistent breathlessness, dry cough, and fatigue, often mimicking other interstitial or obstructive pulmonary diseases. Because of this overlap, it is frequently misdiagnosed as asthma, chronic bronchitis, or idiopathic pulmonary fibrosis, leading to delayed treatment and irreversible lung damage.

The diagnostic challenge of HP lies in its non-specific clinical and radiological features. High-resolution computed tomography (HRCT) of the thorax may reveal centrilobular nodules, ground-glass opacities, and interstitial thickening, while bronchoalveolar lavage (BAL) often shows a lymphocytic predominance and altered CD4/CD8 ratios. In this case, BAL analysis revealed CD4/CD8 ratio of 0.38, suggestive of HP, as opposed to sarcoidosis where the ratio is often elevated.

This case presentation highlights the clinical importance of early recognition and accurate diagnosis of HP, especially in young patients with recurrent respiratory symptoms and environmental exposures. Repeated misdiagnosis as acute bronchitis not only results in inappropriate management but also exposes the patient to unnecessary treatments and hospitalizations. Timely diagnosis with a thorough exposure history, imaging, and immunological tests can significantly alter the disease trajectory and improve outcomes.

II. CASE REPORT

A 19-year-old female with no known medical comorbidities presented to the medical outpatient department with a 10-day history of non-productive cough and progressive breathlessness for 5 days. She denied any fever, hemoptysis, weight loss, or night sweats. Notably, the patient had multiple prior hospitalizations and had been repeatedly treated for acute bronchitis at a community health center without significant improvement.

A detailed environmental history was elicited. The patient resides in a semi-urban area and reported frequent exposure to poultry birds at home, as well as indoor mold growth, raising the suspicion of a domestic antigen-induced hypersensitivity[2] pneumonitis.

Clinical Examination

General Condition: Conscious, alert, oriented; vitals stable

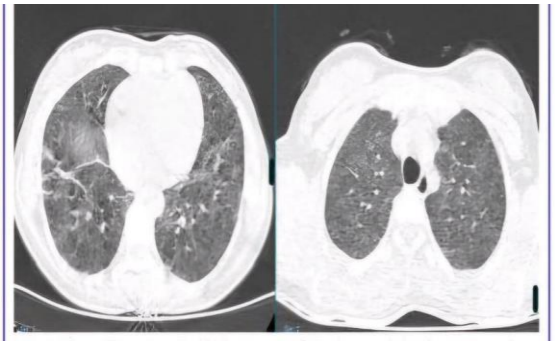
- Respiratory System: Normal vesicular breath sounds with bilateral infrascapular crepitations
- Cardiovascular System: S1, S2 audible; no murmurs
- Abdomen: Soft, non-tender, no organomegaly
- CNS: No focal neurological deficits, higher mental functions intact

III. INVESTIGATIONS

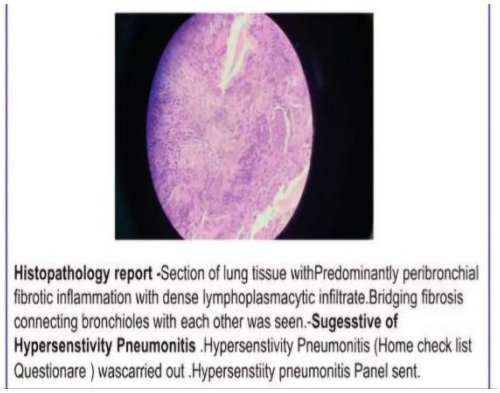
- Complete Blood Picture (CBP):
  - Total Count (TC): 19,800 cells/mm³
  - Neutrophils: 89%
  - Lymphocytes: 6%
  - Monocytes: 3%
  - Eosinophils: 1.8%
  - Basophils: 0.2%
- Hemoglobin: 11.0 g/dL
- Peripheral Smear: Normocytic normochromic blood picture with neutrophilic leukocytosis

Radiological Findings

- High-Resolution Computed Tomography (HRCT) Thorax revealed:
  - Diffuse ground-glass opacities
  - Mild inter- and intralobular septal thickening
  - Centrilobular nodules in bilateral lung fields, typical of subacute/chronic HP[3]



HRCT Chest showing :Indistinct centrilobular nodules in all over lung fields



Histopathology report -Section of lung tissue withPredominantly peribronchial fibrotic inflammation with dense lymphoplasmacytic infiltrate.Bridging fibrosis connecting bronchioles with each other was seen.-**Sugesstive of Hypersenstivity Pneumonitis** .Hypersenstivity Pneumonitis (Home check list Questionare) wascarried out .Hypersenstiity pneumonitis Panel sent.

Bronchoalveolar Lavage (BAL) Analysis

- Cell Differentiation:
  - Lymphocytes: 50%
  - Neutrophils: 40%
  - Macrophages: 10%
- Microscopy: Inflammatory pathology evident
- Flow Cytometry – Lymphocyte Subtyping:
  - CD3+ T cells: 91.58% (↑; normal 60.5–76.9%)
  - CD4+ T cells: 22.96% (↓; normal 29.3–44.9%)
  - CD8+ T cells: 60.94% (↑; normal 25.2–42.8%)
  - CD4/CD8 ratio: 0.38 (↓; normal 0.82–3.21)

A low CD4/CD8 ratio in BAL fluid is suggestive of HP[4] (unlike sarcoidosis, which usually has a high ratio).

IV. DIAGNOSIS

Based on clinical features, HRCT findings, BAL lymphocytosis with a reduced CD4/CD8 ratio, and a strong exposure history, a diagnosis of Hypersensitivity Pneumonitis (subacute to chronic form) was made.

V. MANAGEMENT AND OUTCOME

- Environmental Intervention: Advised strict antigen avoidance, including removal from exposure to birds and mold remediation.
- Pharmacologic Therapy: Initiated oral corticosteroids (Prednisolone) in a tapering dose regimen.
- Follow-Up: Within 2 weeks, significant symptomatic improvement in breathlessness and cough was reported. A repeat chest examination showed resolution of basal crepitations.

VI. CONCLUSION

This case underscores the clinical complexity and diagnostic challenges associated with Hypersensitivity Pneumonitis (HP), particularly in young patients with nonspecific respiratory symptoms and no significant comorbidities. The patient's repeated misdiagnosis as acute bronchitis highlights a common clinical pitfall where environmental and occupational exposure histories are overlooked. Timely diagnosis[1,3], guided by high-resolution imaging and bronchoalveolar lavage analysis—including a low CD4/CD8 ratio—was critical in distinguishing HP from other respiratory conditions. Early initiation of antigen avoidance and corticosteroid therapy led to marked clinical improvement. This case reinforces the importance of maintaining a high index of suspicion for HP in patients with recurrent respiratory symptoms, especially when standard treatment[1,3]s fail. Accurate diagnosis and early intervention are essential to prevent progression to chronic fibrotic lung disease and to improve long-term outcomes.

APPENDIX

Appendix A: Laboratory Investigations

Test	Result	Reference Range	Interpretation
Total Leukocyte Count	19,800 /mm³	4,000–11,000 /mm³	Elevated – Neutrophilic leukocytosis
Hemoglobin	11.0 g/dL	12–16 g/dL	Mild anemia
Differential Count	N: 89%, L: 6%, M: 3%, E: 1.8%, B: 0.2%	N: 50–70%, L: 20–40%	Neutrophil predominance
Peripheral Smear	Normocytic, normochromic	—	Consistent with mild anemia

Appendix B: Radiological Findings (HRCT Thorax)

- Diffuse ground-glass opacities
- Centrilobular nodules in bilateral lung fields
- Mild inter- and intralobular septal thickening

Interpretation: Classic imaging pattern suggestive of subacute/chronic Hypersensitivity Pneumonitis.

Appendix C: Bronchoalveolar Lavage (BAL) Analysis

Parameter	Result	Normal Range	Interpretation
Lymphocytes	50%	<15%	Elevated – consistent with HP
Neutrophils	40%	<3%	Markedly elevated – mixed inflammation
Macrophages	10%	>80%	Decreased – suggests active alveolitis
CD3+ T Lymphocytes	91.58%	60.5–76.9%	Elevated
CD4+ T Lymphocytes	22.96%	29.3–44.9%	Decreased
CD8+ T Lymphocytes	60.94%	25.2–42.8%	Elevated
CD4/CD8 Ratio	0.38	0.82–3.21	Decreased – highly suggestive of HP

Appendix D: Key Clinical Timeline

Timeline	Clinical Events
Month 1–2	Repeated episodes of cough and breathlessness; treated as acute bronchitis
Month 3	Worsening symptoms; hospital visit with crepitations on auscultation
Month 3	HRCT and BAL performed; features suggestive of HP
Post-diagnosis	Environmental modification and corticosteroid therapy initiated
Follow-up (2 weeks)	Symptomatic improvement and resolution of auscultatory findings

Appendix E: Diagnostic Criteria Met for HP (Based on CHEST 2021 Guidelines)

- Exposure to known inciting antigen: Yes (birds, mold)

- Compatible radiologic findings on HRCT: Yes
- BAL lymphocytosis: Yes (>30%)
- Decreased CD4/CD8 ratio: Yes (<1.0)
- Clinical improvement after antigen avoidance: Yes

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