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Case Report

Bird Fancier's Lung in an 8-Year-Old Girl: A Pediatric Diagnostic Challenge

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KEYWORDS

Hypersensitivity
pneumonitis; Bird
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exposure; Child;
Interstitial lung
disease; Asthma
misdiagnosis

Abstract

Background:

Hypersensitivity pneumonitis (HP) is an uncommon immune-mediated interstitial lung disease in children, frequently misdiagnosed as asthma. Avian antigens are the predominant cause.

Case presentation:

An 8-year-old girl presented with an 8-month history of dry cough, exertional dyspnea, and weight loss, unresponsive to inhaled therapy. HRCT revealed diffuse ground-glass opacities. Bronchoalveolar lavage showed lymphocytic alveolitis, and serology confirmed elevated pigeon-specific IgG. Chronic exposure to pigeons on an adjacent balcony established the diagnosis of bird fancier's lung. Strict antigen avoidance and systemic corticosteroids (intravenous pulses followed by oral prednisone) resulted in rapid clinical improvement and near-complete radiologic resolution at 12 months.

Conclusion:

Early recognition through targeted environmental inquiry, HRCT, and immunologic testing is critical in children with refractory respiratory symptoms. Antigen avoidance and timely corticosteroid therapy ensure an excellent prognosis

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Introduction

Hypersensitivity pneumonitis (HP), also known as extrinsic allergic alveolitis, is a diffuse interstitial lung disease of immunologic origin triggered by recurrent inhalation of organic or chemical antigens [1]. The condition results from a complex interplay of type III (immune complex-mediated) and type IV (T-cell-mediated) hypersensitivity reactions, leading to alveolitis, granulomatous inflammation, and—when exposure persists—progressive fibrosis [2].

HP is uncommon in children, with an estimated prevalence of four cases per million and an annual incidence of two cases [3]. However, the number of pediatric cases reported worldwide has been steadily increasing, reflecting heightened clinical awareness and improved access to high-resolution imaging [4]. Avian antigens remain the leading cause, with bird fancier's lung accounting for approximately two-thirds of cases [5,6].

We report a pediatric case of HP secondary to chronic pigeon exposure. This case highlights the diagnostic complexity of HP in children and integrates recent advances in diagnostic and therapeutic approaches from international guidelines.

Case Presentation

An 8-year-old girl, living in an urban environment and born to non-consanguineous parents, was referred for progressive exertional dyspnea and weight loss. Her symptoms had begun eight months earlier with a persistent dry cough, initially managed as asthma with inhaled corticosteroids and bronchodilators, without clinical improvement. Over time, asthenia, anorexia, and exercise intolerance progressively worsened. There was no history of tuberculosis exposure, atopy, or recurrent respiratory infections.

On examination, the child was underweight (−3 SD), tachypneic (35 breaths/min) with mild suprasternal retractions, and had an oxygen saturation of 90% at rest and 85% on exertion. Digital clubbing and fine bilateral crackles were present, while cardiovascular examination was unremarkable.

Chest radiography revealed bilateral reticulonodular opacities (Fig. 1). High-resolution computed tomography (HRCT) demonstrated diffuse ground-glass opacities consistent with active alveolitis (Fig. 2). A diagnosis of diffuse interstitial lung disease (ILD) was considered, prompting further evaluation.

Pulmonary function testing showed a mixed restrictive-obstructive pattern (FVC 70% predicted, FEV₁ 65% predicted, FEV₁/FVC 80%, TLC 68% predicted). Bronchoalveolar lavage (BAL) revealed lymphocytic alveolitis (36% lymphocytes). Tests for *Mycobacterium*

tuberculosis, primary immunodeficiency, and sarcoidosis were negative.

A detailed environmental history disclosed chronic exposure to pigeons kept on the balcony adjacent to the child's bedroom, with daily contact over several years. Serologic testing demonstrated markedly elevated pigeon-specific IgG (141 mgA/L; hemagglutination titer 1:6400) and three precipitin arcs to pigeon dust antigens, confirming sensitization.

The convergence of clinical, radiologic, immunologic, and environmental findings established the diagnosis of hypersensitivity pneumonitis secondary to avian exposure (bird fancier's lung).

Immediate and strict antigen avoidance was implemented, including removal of the birds and thorough environmental cleaning. Treatment consisted of intravenous methylprednisolone pulses followed by oral prednisone (0.8 mg/kg/day) tapered over six months. Clinical improvement was rapid: within two months, oxygen saturation and exercise tolerance normalized, weight gain resumed, and dyspnea resolved. At 12 months, follow-up HRCT showed near-complete resolution with only minimal residual changes.

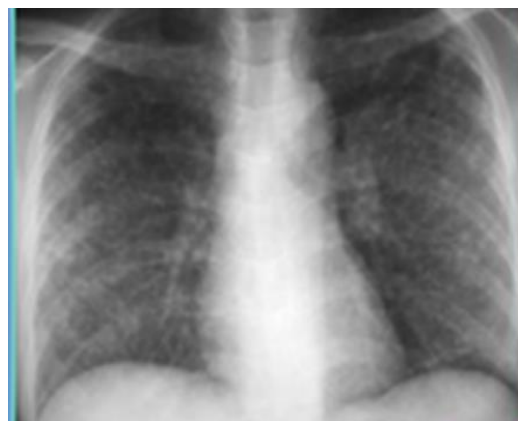


Figure 1. Chest radiograph showing bilateral reticulonodular opacities.



Figure 2. High-resolution computed tomography (HRCT) demonstrating diffuse ground-glass opacities.

Discussion

Pediatric HP is rare but likely underdiagnosed. The median age at onset ranges from 8 to 10 years, with no clear sex predominance [4,5]. The main antigenic sources include avian proteins, thermophilic actinomycetes, and molds such as *Aspergillus* species [4–8].

The clinical presentation is heterogeneous and depends on the intensity and duration of antigen exposure [7, 8]. The traditional classification into acute, subacute, and chronic forms has been replaced by the clinico–radiologic–pathologic classification proposed by Vasakova et al. [1], which distinguishes two main entities: (a) an acute/inflammatory form (<6 months, potentially reversible), characterized by dyspnea, dry cough, and weight loss; and (b) a chronic/fibrotic form, which is irreversible and carries a poorer prognosis. In children, the non-fibrotic inflammatory phenotype predominates, and the prognosis is excellent when diagnosis and antigen avoidance occur early [4–6].

Host susceptibility factors—including HLA-DRB1 polymorphisms, MUC5B mutations, and telomeric abnormalities—as well as environmental cofactors such as viral infections and air pollution, may modulate fibrotic progression risk [7,8].

According to current international recommendations [4–8], diagnosis relies on a combination of: (1) a compatible clinical presentation; (2) documented antigen exposure; (3) suggestive HRCT features; (4) BAL lymphocytosis; and (5) supportive histopathology when necessary. HRCT remains the imaging modality of choice.

In pediatric cases, subacute HP typically shows ground-glass opacities, centrilobular nodules, or mosaic attenuation with air trapping [1,4–8]. Fibrotic forms are characterized by reticulation, traction bronchiectasis, and honeycombing, often with upper-lobe predominance [1,4–8].

The presence of avian-specific IgG antibodies confirms exposure but is not diagnostic in isolation, as elevated titers may persist after antigen avoidance [4–8].

A BAL lymphocytosis >30% provides strong diagnostic support [6,8]. HP frequently mimics other respiratory disorders such as asthma, atypical pneumonia, sarcoidosis, cystic fibrosis, or connective tissue–associated interstitial pneumonias [4–6]. In our case, the initial misdiagnosis of refractory asthma delayed recognition for eight months—a common pitfall in pediatric practice [4–6].

Antigen avoidance remains the cornerstone of management [4–8]. Beyond this, corticosteroids (prednisone 0.5–1 mg/kg/day for 4–8 weeks) hasten recovery [4,5]. Intravenous corticosteroid pulses should be considered in cases of persistent symptoms or severe impairment on pulmonary function tests and HRCT [4–6].

Immunosuppressive agents such as azathioprine or mycophenolate mofetil are reserved for relapsing or steroid-dependent disease [4–6]. In adults, antifibrotic agents such as nintedanib have demonstrated benefit in progressive fibrosing HP [7,8], though pediatric data remain limited. Supportive measures include vaccination, pulmonary rehabilitation, and nutritional follow-up [4–6].

The prognosis of non-fibrotic pediatric HP is generally excellent, with complete clinical and radiologic recovery in most cases [4–6].

Diagnostic delay and ongoing antigen exposure are the principal risk factors for fibrotic progression and chronic respiratory failure [4–6]. The favorable outcome in our patient after antigen avoidance and corticosteroid therapy confirms the reversibility of early inflammatory forms.

Our case aligns with recent pediatric reports. Maggiolino et al. (2024) described a 5-year-old girl exposed to doves who achieved complete recovery after corticosteroid therapy and antigen avoidance [9]. Zahraldin et al. (2023) reported an adolescent with severe HP secondary to parrot and pigeon exposure, requiring high-flow oxygen and intravenous methylprednisolone pulses, with multiple avian precipitins identified [10]. These cases, together with ours, emphasize the critical importance of a systematic and repeated environmental assessment in children with chronic respiratory symptoms unresponsive to standard asthma treatment. Early recognition and prompt intervention ensure full reversibility of the disease.

Conclusion

Although uncommon, HP should be considered in any child presenting with chronic cough or exertional dyspnea unresponsive to standard asthma therapy.

Systematic environmental assessment, together with high-resolution computed tomography, bronchoalveolar lavage, and serologic testing, is essential for accurate diagnosis.

Our case of Bird Fancier's Lung highlights the diagnostic pitfalls and underscores the critical importance of thorough environmental evaluation. When recognized early and managed with strict antigen avoidance and timely systemic corticosteroid therapy, pediatric HP carries an excellent prognosis.

CONFLICT OF INTEREST STATEMENT

No conflicts of interest to be disclosed.

References

1. Vasakova M, Morell F, Walsh S, Leslie K, et al. Hypersensitivity Pneumonitis: Perspectives in Diagnosis and Management. *Am J Respir Crit Care Med*. 2017;196 : 680–9.
2. Selman M, Buendía-Roldan I. Immunopathology, diagnosis, and management of hypersensitivity pneumonitis. *Semin Respir Crit Care Med*. 2012;33:543–54.
3. Buchvald F, Petersen B, Damgaard K, Deterding R, et al. Frequency, treatment, and functional outcome in children with hypersensitivity pneumonitis. *Pediatr Pulmonol*. 2011;46:1098–107.
4. Mastroiilli C, Pecoraro L, Arasi S, Barni S, et al. Pediatric hypersensitivity pneumonitis: literature update and proposal of a diagnostic algorithm. *Ital J Pediatr*. 2022;48:51.
5. Soumagne T, Dalphin M, Dalphin J, et al. Hypersensitivity pneumonitis in children. *Rev Mal Respir*. 2019;36:495–507.
6. Wawszczak M, et al. Hypersensitivity pneumonitis in children. *Ann Agric Environ Med*. 2021;28:214–9.
7. Fernández Pérez ER, Travis WD, Lynch DA, MB, Evans R B, et al. Diagnosis and Evaluation of Hypersensitivity Pneumonitis. *CHEST Guideline*. *Chest*. 2021;160:e97–156.
8. Koschel D, Behr J, Berger M, Bonella F, Hamer O, et al. Diagnosis and Treatment of Hypersensitivity Pneumonitis: S2k Guideline of the German Respiratory Society and the German Society for Allergology and Clinical Immunology. *Respiration*. 2025;104:485–528.
9. Maggiolino Q, et al. Hypersensitivity Pneumonitis in a 5-Year-Old Girl due to Turtledove: A Case Report. *Arch Bronconeumol*. 2024;60:656–7.
10. Zahraldin K, et al. Bird Fancier's Lung in an Adolescent Male: A Case Report. *Am J Respir Crit Care Med*. 2023;207: A3643. Titre du site web. Disponible en ligne : URL (consulté le jour mois année).