



A Case of Bird Fancier's Lung Disease Secondary to Long-term Bird Exposure

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Abstract

Background: Bird Fancier's Lung Disease (BFLD) is a common form of hypersensitivity pneumonitis caused by repeated inhalation of avian antigens. Chronic, low-grade exposure can lead to progressive respiratory symptoms that are often nonspecific, contributing to delayed diagnosis. High-resolution computed tomography (HRCT), in combination with a detailed environmental history and pulmonary function testing, is essential for early recognition and prevention of irreversible fibrosis.

Case Presentation: We describe a 77-year-old woman with a 20-year history of exposure to a household pet parrot and long-term equine contact who presented with exertional dyspnoea and chronic cough. Physical examination was unremarkable with normal oxygen saturation. HRCT demonstrated diffuse mosaic attenuation, air trapping, and ground-glass opacities without evidence of fibrosis. Pulmonary function testing revealed normal spirometry with elevated RV/TLC and reduced diffusion capacity, consistent with gas trapping. Serologic testing showed positive budgerigar precipitins. A diagnosis of chronic hypersensitivity pneumonitis secondary to BFLD was established. The patient was treated with a tapered corticosteroid regimen and advised on antigen avoidance. She demonstrated significant symptomatic improvement and radiologic stability on serial follow-up despite continued partial exposure.

Conclusion: This case underscores the importance of considering BFLD in patients with unexplained chronic dyspnoea, particularly when exposure to birds is identified. Early diagnosis through integration of environmental history, HRCT, and physiological assessment is critical prevent progression to fibrotic disease. Corticosteroid therapy and structured surveillance can achieve clinical stability even when complete antigen avoidance is not feasible.

Keywords: Bird fancier's lung; Hypersensitivity pneumonitis; Avian antigen; High-resolution computed tomography; Air trapping

Introduction

Bird Fancier's Lung Disease (BFLD) is the most common cause of hypersensitivity pneumonitis (HP), an immune-mediated interstitial lung disease resulting from repeated inhalation of avian antigens from bird feathers, droppings, and serum. BFLD accounts for a substantial proportion of HP cases and can present in acute, subacute, or chronic forms, depending on the intensity and duration of exposure [1-3]. Risk factors include prolonged exposure to birds, indoor bird keeping, regular handling or cleaning of cages, poor ventilation, and exposure to dried droppings or feather dust. Although high-intensity exposures are classically seen in pigeon breeders and aviary workers, BFLD also occurs with chronic low-level exposure to pet birds in domestic settings. Clinical presentation is often insidious and

nonspecific, which contributes to diagnostic delay. Common symptoms include progressive exertional dyspnoea, chronic cough, fatigue, and reduced exercise tolerance, while systemic symptoms such as fever, night sweats, or weight loss are less frequent.^{3,4} Physical examination may be normal or reveal soft binasal crackles. Resting oxygen saturation is often preserved in early or non-fibrotic stages; while clubbing and cyanosis typically occur only in advanced fibrotic disease [3]. Given the nonspecific symptom profile and frequently unremarkable physical findings, early BFLD is frequently underdiagnosed. A detailed environmental exposure history, coupled with targeted imaging and pulmonary function testing, is essential for accurate diagnosis. High-resolution computed tomography (HRCT) plays a central role, as it can identify characteristic patterns such as mosaic attenuation, ground-glass opacities, centrilobular nodules,

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and areas of air trapping, which often precede development of fibrotic changes in more advanced disease [4,5]. We report a case of chronic hypersensitivity pneumonitis due to BFLD in an elderly woman with a 20-year history of exposure to an indoor pet parrot and long-term equine contact, highlighting the diagnostic challenges, importance of thorough environmental history, and favorable outcome with antigen avoidance and corticosteroid therapy.

Case Report

A 77-year-old woman presented with progressively worsening exercise tolerance and exertional dyspnoea when walking for approximately 100 meters or performing household chores. She also reported a chronic cough with occasional white phlegm in the mornings. She denied fevers, weight loss, night sweats, hemoptysis or recent respiratory infections. Her medical history includes ischemic heart disease requiring four coronary artery bypass grafting, paroxysmal atrial fibrillation, type 2 diabetes mellitus, hypertension, dyslipidemia, chronic kidney disease, previous thyroid cancer and hypothyroidism. She has no history of respiratory conditions. She is a non-smoker but reports regular exposure to passive cigarette smoke. She reported significant avian exposure through a pet Indian ringneck parrot at home for the past 20 years. She has also resided on a horse property with direct equine exposure for more than 20 years. She denied any previous history of contact with asbestos or dust. On examination, the patient was comfortable with no respiratory distress and oxygen saturation of 99% on room air. She had no clubbing, cyanosis or muscle wasting. Respiratory auscultation revealed clear vesicular breath sounds without crackles or wheeze. Her cardiovascular, abdominal and neurological examinations were otherwise unremarkable.

Investigations

Radiological Findings

A dedicated HRCT revealed geographic changes of mosaic attenuation throughout both lungs with areas of air trapping. Multiple patchy diffuse ground glass opacities were present without evidence of lung fibrosis or honeycombing. No mediastinal lymphadenopathy was detected.

Pulmonary Function Testing

Spirometry showed preserved volumes and FEV₁/FVC ratio. The lung volumes revealed a normal total lung capacity and elevated residual volume and RV/TLC ratio (128%), indicating moderate gas trapping. Gas transfer was mildly impaired with a DLCO of 60.9% predicted and KCO of 71% predicted.

Laboratory Tests

Laboratory investigations revealed positivity to budgerigar serum precipitins. ANA was weakly positive with a titre of 1:40 however ENA antibodies, Anti CCP and rheumatoid factor were negative. Thyroid function tests demonstrated no evidence of overt thyroid dysfunction with a TSH of 0.5 mIU/L, free T4 of 15 mIU/L and free T3 of 4.1 mIU/L. Blood counts, liver function tests, HbA1c and serum immunoglobulins were unremarkable. Renal function tests (eGFR 42 ml/min, Creatinine 108 µmol/L) and HbA1c (6%) were stable in comparison to the patient's baseline values.

Diagnostic Conclusion

The diagnosis of hypersensitivity pneumonitis secondary to Bird Fancier's Lung Disease was made on clinico-radiological grounds. HRCT demonstrated mosaic attenuation and diffuse ground-glass opacities without fibrosis (Figure 1-3), a pattern consistent with bird-related hypersensitivity pneumonitis and small airways involvement. In light of these imaging features, the exposure history was revisited and confirmed long-standing avian exposure as well as equine antigen exposure. Serological testing provided supportive evidence of sensitization, with laboratory investigations demonstrating positivity to budgerigar serum precipitins. When considered alongside physiological evidence of gas trapping and a reduced diffusion capacity, these findings supported the diagnosis and reduced the need for invasive tissue sampling. Alternative diagnoses including connective tissue disease-associated interstitial lung disease, sarcoidosis, organizing pneumonia and idiopathic pulmonary fibrosis were considered less likely in the absence of supportive clinical and radiological features.

The diagnostic impression was strengthened by the internal consistency of the radiology and immunology. The HRCT pattern - diffuse ground-glass change with mosaic attenuation and features of small airways involvement, without established fibrosis - raised strong suspicion for non-fibrotic hypersensitivity pneumonitis and prompted a targeted reappraisal of exposures. On directed questioning, the patient described long-standing and ongoing contact with birds, including indoor bird-keeping and regular handling/cleaning activities, providing a credible source of repeated avian antigen inhalation. Serum IgG precipitins were positive against avian antigens (including budgerigar antigen), supporting sensitization. While precipitin positivity alone does not confirm disease, in the setting of a compatible HRCT pattern and a clear exposure history, it provided important supportive evidence and reduced the need for invasive testing. The case was reviewed in a multidisciplinary context, and the working diagnosis of Bird Fancier's Lung Disease (bird-related hypersensitivity pneumonitis) was agreed [5-8]. Given the absence of radiological fibrosis, the preserved spirometric indices, and the lack of alternative red-flag features, bronchoalveolar

lavage or lung biopsy were not pursued. Instead, management was guided by an exposure-centred strategy and objective monitoring (symptoms, diffusion capacity and interval imaging) to confirm stability or improvement with antigen avoidance.

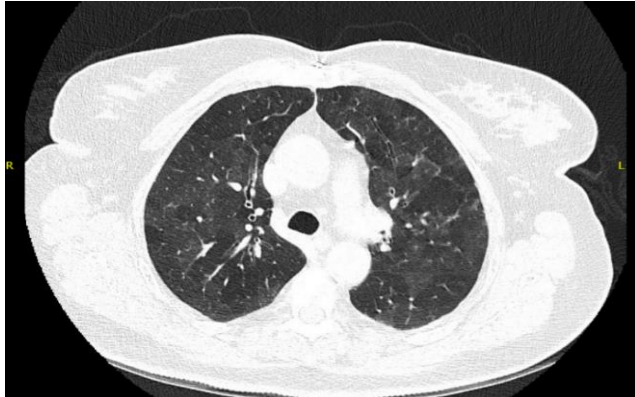


Figure 1: HRCT showing mosaic attenuation and diffuse ground-glass opacities without fibrosis in the upper lung zones.

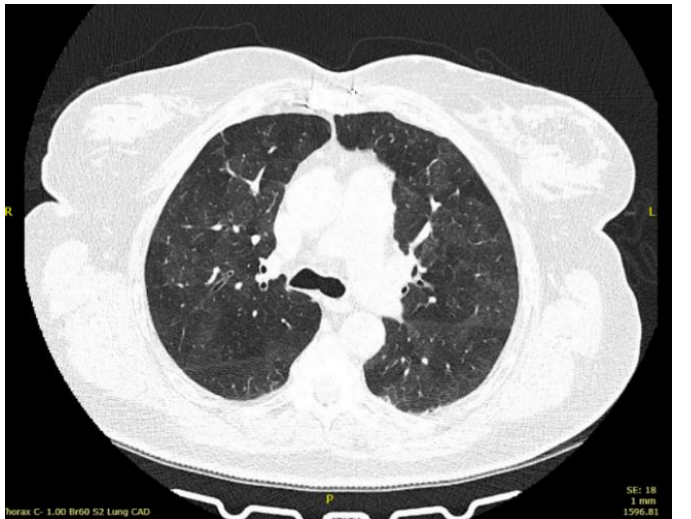
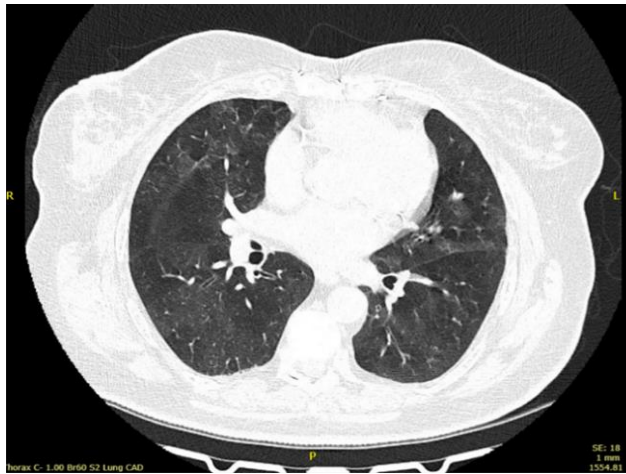


Figure 2: HRCT showing mosaic attenuation and diffuse ground-glass opacities without fibrosis in the mid lung zones.

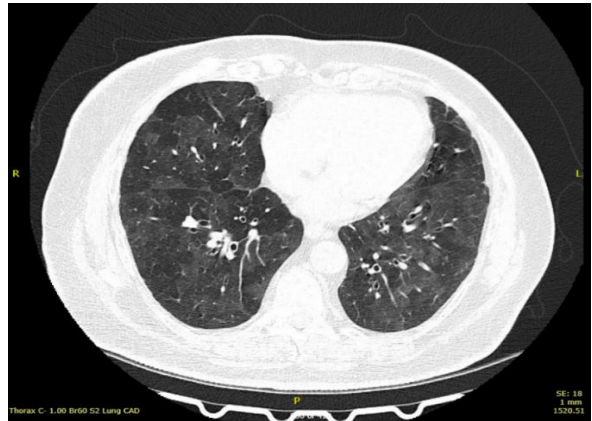
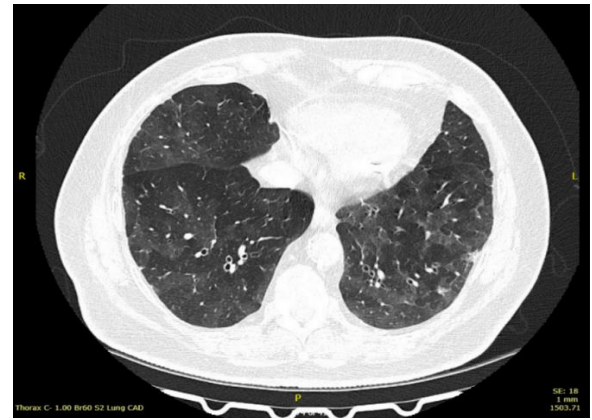


Figure 3: HRCT showing mosaic attenuation and diffuse ground-glass opacities without fibrose.

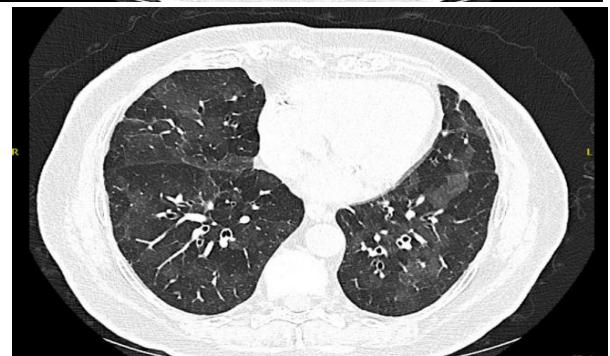


Figure 4: Follow-up HRCT demonstrating stable disease with interval decrease in mosaic perfusion and air trapping; scattered ground-glass change persists without fibrosis, predominantly in the lower lobes.

Management

The patient received counselling regarding strict avoidance of offending antigens, including her pet bird and horse, however she was reluctant to limit exposure to her pet bird. In accordance with current guidelines, she was commenced on a tapered course of corticosteroids 3 to 6 months to reduce inflammation and prevent disease progression. She was educated on the risks of steroid use, particularly the importance of blood sugar level and blood pressure monitoring in the setting of her comorbidities. Vitamin D and calcium supplementation were initiated to support bone health. At the 6-month review, her symptoms had improved significantly, and corticosteroids were ceased. She required an additional short course of corticosteroids at the 12-month review for worsening of symptoms, however repeat HRCT and lung function testing showed no evidence of progression warranting immunosuppressive therapy. She was commenced on a structured surveillance plan with 6-12 monthly lung function testing and HRCT, with further counselling on limiting antigen exposure. Subsequent assessment revealed stable disease on imaging with mild obstructive lung disease and gas trapping on spirometry. She remained on serial surveillance with HRCT and lung function testing thereafter.

Discussion

BFLD represents a chronic form of HP caused by repeated inhalation of avian antigens, with clinical manifestations that vary according to antigen intensity and duration [1-3]. Acute HP typically presents with abrupt flu-like symptoms several hours after exposure, whereas chronic forms progress gradually over months to years and may remain unnoticed until HRCT or pulmonary function abnormalities emerge [3,5]. This wide clinical spectrum contributes to delayed diagnosis, particularly in non-occupational, low-level household exposure settings [1,3,9-25]. Radiologically, chronic bird-related HP demonstrates characteristic HRCT findings of mosaic attenuation, ground-glass opacities, centrilobular nodules, and air trapping, which reflect small-airway inflammation, bronchiolitis, and ventilation-perfusion mismatch [4-6]. These features were evident in our patient, whose imaging showed diffuse mosaic attenuation and gas trapping without fibrosis, consistent with a non-fibrotic type. The absence of fibrotic features is associated with a more favorable prognosis and potential reversibility following antigen avoidance or corticosteroid therapy [4,7]. Conversely, reticulation, traction bronchiectasis, or honeycombing signal progression to chronic fibrotic HP, which carries poorer outcomes [4,5,10,15,16,19]. Physiologically, BFLD often presents with impaired diffusion capacity and gas trapping, even when spirometry remains normal, a pattern reported in multiple chronic HP studies [4,8]. This underscores the importance of full lung

volumes and diffusion measurements, as spirometry alone may underestimate disease severity. Serologic testing for avian precipitins offers supportive evidence of sensitisation but is not diagnostic in isolation [1,3,9]. Positive precipitins confirm antigen exposure but cannot distinguish between sensitised individuals with or without clinical disease, and false negatives occur due to poor assay sensitivity. In this case, budgerigar precipitins supported the diagnosis when interpreted alongside HRCT findings and a significant exposure history [11,14]. The patient's normal physical examination of clear auscultation, preserved oxygen saturation, and absent clubbing is common in many BFLD cases, where clinical findings remain subtle until fibrosis or advanced airway obstruction occurs [1,3,5]. This highlights the essential role of a thorough environmental history in patients with chronic cough or exertional dyspnoea. Antigen avoidance remains the cornerstone of management and the strongest predictor of long-term outcome. However, complete avoidance may be challenging when the antigen source is a household pet bird. Corticosteroids reduce inflammation and improve symptoms, particularly in cases of ongoing partial exposure [4-6]. Long-term monitoring with serial HRCT and pulmonary function tests is recommended to detect early progression toward fibrosis [4-7]. Studies indicate that structural deterioration may occur despite symptomatic improvement if antigen exposure persists [5]. In this case, surveillance imaging demonstrated stable disease without fibrotic evolution, suggesting effective inflammatory control, though continued exposure poses a future risk of exacerbation [17,18].

Pathobiology and antigen sources

Bird-related hypersensitivity pneumonitis is an immune-mediated interstitial lung disease triggered by repeated inhalation of avian proteins, most commonly from feather bloom, droppings and dried secretions that become aerosolised during close contact, cage cleaning and disturbance of bedding. The inflammatory response typically involves the terminal bronchioles and adjacent interstitium, explaining why small airways disease (air-trapping and mosaic attenuation) often sits alongside ground-glass change on HRCT. Clinically meaningful exposure can occur even when bird contact is perceived as 'low level' or occurs intermittently, and antigen can persist in the domestic environment, so symptom-exposure relationships may be subtle.

Clinical phenotypes and what to look for Bird fancier's lung may present as acute, subacute or chronic disease, although overlap is common. Acute episodes may occur hours after exposure, whereas chronic disease often evolves insidiously with exertional dyspnoea, cough, fatigue and weight loss. Physical examination may be normal early, and spirometry can remain preserved; a disproportionate reduction in diffusion capacity, exertional desaturation, or persistent symptoms despite asthma/COPD

therapies should prompt evaluation for HP. Features that increase concern for fibrotic HP include crackles, progressive decline in

FVC/DLCO, and HRCT evidence of traction bronchiectasis, architectural distortion or honeycombing.

Table 1: Summary of features supporting the diagnosis of Bird Fancier’s Lung Disease in this case.

Domain	Findings in this case (supporting bird-related hypersensitivity pneumonitis)
Exposure	Long-standing close contact with birds, including indoor exposure; ongoing handling/cleaning activities. Equine exposure was also present but avian exposure was the dominant, biologically plausible trigger.
Symptoms	Chronic cough and exertional dyspnoea with a nonspecific clinical presentation.
Physiology	Preserved spirometry with a disproportionate reduction in diffusion capacity (DLCO 60.9% predicted; KCO 71% predicted), consistent with gas transfer impairment.
HRCT pattern	Diffuse ground-glass change with mosaic attenuation and small airways involvement, without established fibrosis, traction bronchiectasis or honeycombing (non-fibrotic phenotype).
Immunology	Positive serum IgG precipitins against avian antigens (including budgerigar antigen), interpreted as supportive when integrated with imaging and exposure history.

Imaging hallmarks and interpretation of follow-up HRCT

HRCT is central to recognizing HP and differentiating non-fibrotic from fibrotic disease. Typical/compatible patterns include centrilobular ground-glass nodules, patchy ground-glass opacities, mosaic attenuation and expiratory air-trapping; the combination of areas of increased attenuation (ground-glass), normal lung and low attenuation (air-trapping) may produce the ‘three-density’ pattern. In this case, the baseline HRCT demonstrated diffuse ground-glass change with mosaic attenuation without established fibrosis, supporting a non-fibrotic phenotype. On interval imaging, the decrease in ground-glass opacities was consistent with improvement in active inflammation following treatment and exposure reduction, while persistent mosaic attenuation likely reflected ongoing small airways involvement. Serial HRCT is useful to confirm resolution of inflammatory change and to detect any evolution towards fibrosis in patients with continued or recurrent exposure.

Where avian precipitins fit (and their limitations)

Serum IgG precipitins against avian antigens support exposure and sensitization and can be helpful when exposure history is not initially volunteered or when multiple potential antigens exist. However, precipitin testing is neither perfectly sensitive nor

specific: false negatives occur (e.g., early disease, assay variability), and false positives occur in exposed but unaffected individuals. Guideline-based approaches emphasize integrating exposure assessment, HRCT pattern and (when needed) bronchoalveolar lavage lymphocytosis and histopathology, rather than relying on serology in isolation. In this patient, the positive avian precipitins were therefore interpreted as supportive evidence that cohered with the HRCT pattern and exposure history, allowing a confident diagnosis without invasive sampling.

How this case adds value

This case reinforces three practical lessons: (i) chronic bird-related HP can present with nonspecific symptoms and minimal examination findings; (ii) diffusion impairment may be the dominant physiological abnormality even when spirometry is preserved; and (iii) a compatible HRCT pattern should trigger a focused exposure history and targeted serology, which together may establish a confident diagnosis without invasive sampling.

Conclusion

This case highlights the diagnostic challenges posed by chronic Bird Fancier’s Lung Disease, particularly in individuals with prolonged low-grade domestic exposure where symptoms are subtle and unremarkable physical examination findings. Accurate

diagnosis relies on a high level of suspicion, detailed environmental history, and integration of HRCT imaging, pulmonary function testing, and serologic assessment. The characteristic radiologic patterns of mosaic attenuation, air trapping, and patchy ground-glass opacities were instrumental in establishing the diagnosis in this patient without the need for invasive biopsy. Early identification and reduction of antigen exposure remain central to preventing long-term complications, including progression to fibrotic hypersensitivity pneumonitis. Although complete antigen avoidance was challenging in this case due to ongoing contact with a pet bird, the patient demonstrated clinical improvement and radiologic stability following corticosteroid therapy and structured surveillance. This underscores that, even in the context of incomplete avoidance, timely initiation of anti-inflammatory treatment and close monitoring can help mitigate disease progression. Clinicians should maintain vigilance for BFLD in patients with chronic cough and exertional dyspnoea, particularly non-smokers or those with persistent respiratory symptoms despite normal examination findings. Prompt recognition and comprehensive evaluation are vital to optimize outcomes and prevent irreversible lung injury in this underdiagnosed condition.

Declarations

Patient consent: Written informed consent for publication of this case report and accompanying images was obtained from the patient.

Ethics approval: Not applicable. This case report was prepared in accordance with local institutional requirements for publication of de-identified clinical information.

Conflict of interest: The authors declare no competing interests.

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Author contributions: All authors contributed to clinical care and/or data collection, drafting and critical revision of the manuscript, and approved the final version.

Data availability: All relevant data are included within the article.

Abbreviations: BFLD, Bird Fancier's Lung Disease; HP, hypersensitivity pneumonitis; HRCT, high-resolution computed tomography; FEV₁, forced expiratory volume in 1 second; FVC, forced vital capacity; RV, residual volume; TLC, total lung capacity; DLCO, diffusing capacity of the lung for carbon monoxide; KCO, transfer coefficient; ANA, antinuclear antibody; ENA, extractable nuclear antigen; anti-CCP, anti-cyclic citrullinated peptide; eGFR, estimated glomerular filtration rate; TSH, thyroid-stimulating hormone.

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SUNTEXT REVIEWS

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