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(CASE REPORT)

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Hypersensitivity pneumonitis revealed by severe asthma: A report of 2 cases

O. Abouobayd *, M.A. Eddahioui, C. Rachid, O. Fikri, M. Ijim and L. Amro

Department of Pulmonology, Arrazi Hospital, Mohammed VI University Hospital, LRMS Laboratory, FMPM, UCA, Marrakesh, Morocco.

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Abstract

Hypersensitivity pneumonitis (HP) is a complex interstitial lung disease characterized by a heterogeneous clinical presentation that frequently leads to diagnostic delays. While chronic fibrotic forms are well described, clinical presentations mimicking asthma remain a formidable diagnostic pitfall. We report two cases of patients initially followed for refractory severe asthma, in whom thoracic CT imaging and an environmental survey ultimately corrected the diagnosis to HP (specifically "Farmer's Lung" and "Domestic Lung"). These cases illustrate the necessity of investigating for bronchiole-alveolar involvement in any difficult-to-treat asthma: cross-sectional imaging and bronchoalveolar lavage constitute the cornerstones of this re-evaluation.

Keywords: Hypersensitivity pneumonitis; Severe asthma; Computed tomography; Bronchoalveolar lavage; Antigen avoidance

1. Introduction

Hypersensitivity pneumonitis (HP) constitutes a heterogeneous group of immune-mediated lung diseases caused by repeated inhalation of organic or chemical antigens in sensitized individuals [1, 2]. Long classified in a binary manner (acute/chronic), recent international guidelines from the ATS/JRS/ALAT now favor a distinction based on the presence or absence of fibrosis, a major prognostic factor [3].

However, the true clinical challenge often lies in the initial diagnosis. The presentation of HP can be particularly deceptive: cough, dyspnea, and wheezing are frequent, closely mimicking a classic obstructive pathology [4]. Retrospective studies highlight that nearly 20% of patients with HP received an initial erroneous diagnosis of asthma or COPD [5]. This symptom overlap often leads to inappropriate prescription of inhaled corticosteroids or biologics for "severe asthma," delaying essential antigen avoidance [6].

Through two clinical observations from the Department of Pulmonology at the Mohammed VI University Hospital in Marrakesh, we illustrate this "pseudo-asthmatic" form of HP and discuss the key elements allowing for diagnostic correction.

* Corresponding author: O. Abouobayd

2. Clinical Cases

2.1. Case N°1

The first case involves a 49-year-old female patient with no known history of atopy, followed for two years for asthma labeled as severe. Despite therapeutic escalation and supposedly optimal adherence, disease control remained precarious. The clinical history progressively evolved with exertional dyspnea and repeated exacerbations, contrasting with an initially unremarkable pleuropulmonary examination.

Given this clinical-functional discrepancy, a chest radiograph was performed, revealing a diffuse interstitial infiltrate. The thoracic CT scan confirmed parenchymal involvement with a mosaic attenuation pattern, combining expiratory air trapping and diffuse micronodules. While Pulmonary Function Tests (PFTs) showed a decrease in resistance after bronchodilation (mimicking asthma), bronchoscopy corrected the diagnosis by revealing diffuse grade II inflammation.

Bronchoalveolar Lavage (BAL) demonstrated lymphocytic alveolitis with a CD4/CD8 ratio < 2.5. The environmental survey finally uncovered the etiology: chronic exposure to livestock and poultry. Positive precipitins for "Farmer's Lung" confirmed the diagnosis. Antigen avoidance and corticosteroid therapy led to clinical and functional remission.

2.2. Case N°2

The second patient, aged 47, had been treated for severe asthma for five years. Recent worsening of her dyspnea and the onset of a debilitating dry cough prompted a re-evaluation. Unlike the first case, clinical examination revealed crackles associated with wheezing in both lung fields.

High-resolution CT imaging highlighted diffuse ground-glass opacities associated with centrilobular and subpleural micronodules, a pattern suggestive of micronodular HP. Although PFTs confirmed a reversible obstructive ventilatory defect, BAL analysis revealed a lympho-histiocytic pattern with a CD4/CD8 ratio of 2.08.

The investigation identified domestic exposure to molds in a damp environment. The diagnosis of "Domestic Lung" (non-fibrotic HP) was retained. The outcome was favorable following antigen avoidance and anti-inflammatory treatment.

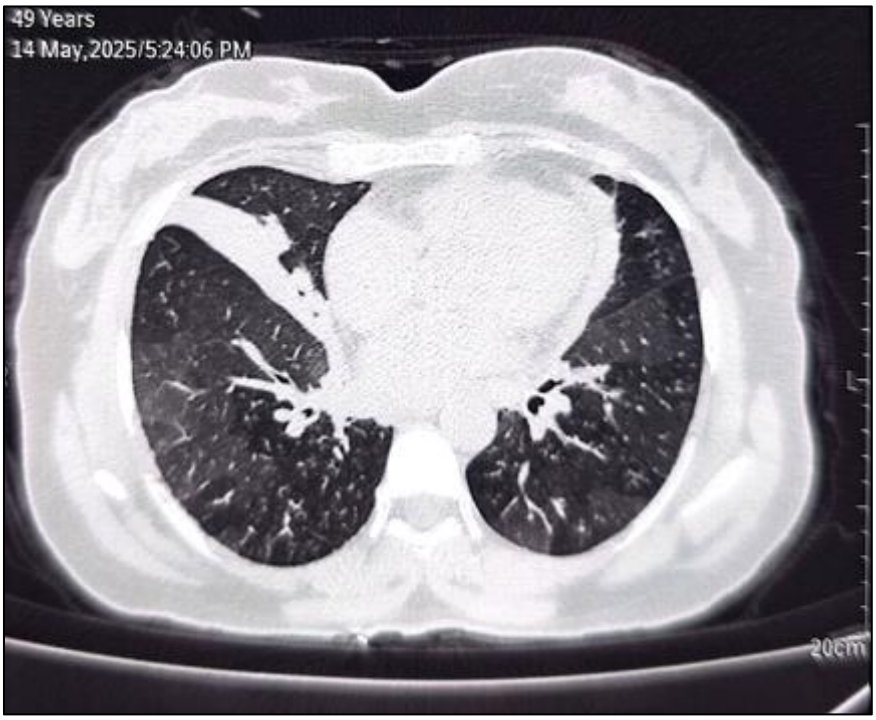

Figure 1 CT aspect of diffuse mosaic attenuation with expiratory trapping

Figure 2 Frontal chest X-ray showing diffuse bilateral interstitial syndrome

Figure 3 CT aspect of micronodular HP

3. Discussion

Distinguishing between severe asthma and hypersensitivity pneumonitis (HP) represents a major diagnostic challenge due to often similar clinical and functional presentations. Patients with HP frequently present with exertional dyspnea, cough, and wheezing, mimicking the clinical picture of asthma [6]. Functionally, the small airway involvement

(bronchiolitis) inherent to HP can lead to an obstructive ventilatory defect with a positive response to bronchodilators, thus fulfilling the reversibility criteria usually specific to asthma [4, 7]. In our observations, this similarity led to an initial classification of "uncontrolled severe asthma," motivating ineffective therapeutic escalation.

However, thoracic imaging serves as a guide for diagnosis: while an asthmatic's CT is often normal or shows simple air trapping, HP typically associates areas of air trapping (hypodense) with ground-glass opacities (hyperdense), creating a "mosaic" appearance [8]. The concomitant presence of centrilobular or ill-defined micronodules, observed in our two patients, constitutes a discriminating radiological sign favouring active interstitial disease [3, 9].

Diagnostic confirmation relies on cytological analysis of bronchoalveolar lavage (BAL). While severe asthma is predominantly associated with eosinophilic inflammation (T2-high profile), HP is characterized by marked lymphocytic alveolitis, often exceeding 20-30% [10]. Analysis of lymphocyte subpopulations provides an additional argument: a predominance of CD8+ T lymphocytes (inversion of the CD4/CD8 ratio), although inconstant, is evocative, as noted in our cases.

Differentiating between these two entities is decisive for the therapeutic strategy and prognosis [11]. Asthma treatment relies on controlling bronchial inflammation and remodelling, whereas HP management imperatively requires the identification and avoidance of the causal antigen (livestock, Molds, etc.). Failure to avoid the antigen exposes the patient to the risk of progression towards irreversible pulmonary fibrosis, the morbidity of which exceeds that of asthma [1, 5].

4. Conclusion

These two observations underscore the importance of questioning a diagnosis of severe asthma when the therapeutic response is atypical. HP must be systematically considered in the presence of bronchial obstruction associated with interstitial CT anomalies (ground-glass, mosaic). High-resolution thoracic CT and a rigorous environmental survey remain the cornerstones for avoiding this diagnostic pitfall and preventing progression to fibrosis.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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