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Recurrent Eosinophilia in Severe Allergic Asthma Managed with Biological Therapy

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Introduction

Severe allergic asthma is a chronic respiratory condition characterized by heightened airway inflammation, bronchial hyperresponsiveness, and frequent exacerbations. Eosinophilia, defined as an elevated number of eosinophils in the blood or tissues, plays a critical role in the pathophysiology of allergic asthma. Biological therapies targeting eosinophilic inflammation have transformed the management of severe asthma, providing significant improvements in symptom control and reducing the frequency of exacerbations. However, some patients experience relapsing eosinophilia despite ongoing biological therapy, raising questions about the underlying mechanisms and optimal management strategies. This report describes the clinical course of a patient with severe allergic asthma who experienced recurrent eosinophilia while receiving biological therapy. The patient was a 45-year-old female with a long-standing history of allergic asthma diagnosed in her late teens. Her asthma was classified as severe due to persistent symptoms, frequent exacerbations requiring oral corticosteroids, and a history of hospitalizations for acute respiratory distress. Initial evaluation revealed elevated serum IgE levels, peripheral eosinophilia, and sensitivity to multiple environmental allergens, including dust mites and pollen. Pulmonary function tests showed a reduced forced expiratory volume in one second (FEV1) with significant reversibility after bronchodilator administration, consistent with asthma. Despite optimized treatment with high-dose inhaled corticosteroids, long-acting β-agonists, and leukotriene receptor antagonists, her asthma remained poorly controlled.

Description

Given the refractory nature of her disease, the patient was started on omalizumab, an anti-IgE monoclonal antibody. Initial response to therapy was favorable, with a reduction in exacerbations and improved symptom control. However, over time, she began experiencing intermittent relapses of eosinophilia accompanied by worsening asthma symptoms. Peripheral blood eosinophil counts repeatedly rose above 500 cells/μL during these episodes, prompting concerns about treatment efficacy. A detailed review of her medical history, medication adherence, and potential environmental triggers failed to identify a clear explanation for these relapses. The patient was subsequently switched to mepolizumab, an anti-IL-5 monoclonal antibody specifically targeting eosinophilic inflammation. Following initiation of mepolizumab, her eosinophil counts normalized, and asthma control improved significantly. However, after 12 months of treatment, she experienced a relapse characterized by peripheral eosinophilia exceeding 1000 cells/μL, increased wheezing, nocturnal symptoms, and reduced exercise tolerance. High-resolution computed tomography of the chest was performed to exclude alternative diagnoses such as allergic bronchopulmonary aspergillosis (ABPA) or eosinophilic granulomatosis with polyangiitis (EGPA), both of which were ruled out based on imaging findings and serological markers [1].

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The recurrence of eosinophilia raised questions about potential mechanisms of resistance or escape from anti-IL-5 therapy. Further laboratory investigations revealed elevated levels of thymic stromal lymphopoietin (TSLP) and IL-33, cytokines involved in the upstream activation of eosinophils and other inflammatory cells. These findings suggested that her disease was not solely driven by IL-5-dependent pathways and highlighted the complex interplay of multiple inflammatory mediators in severe asthma. The patient's treatment was escalated to include dupilumab, an anti-IL-4 receptor α monoclonal antibody that inhibits IL-4 and IL-13 signaling. This approach aimed to target broader type 2 inflammatory pathways contributing to her eosinophilic asthma. After starting dupilumab, the patient experienced a marked reduction in eosinophil counts and significant clinical improvement. Her asthma symptoms stabilized, with fewer nocturnal awakenings, improved FEV1, and no further exacerbations requiring systemic corticosteroids. Serial measurements of inflammatory biomarkers, including fractional exhaled nitric oxide (FeNO) and serum periostin, showed a consistent decline, reflecting reduced type 2 inflammation. The patient's quality of life improved substantially, and she was able to resume normal daily activities without limitations [2,3].

The case highlights several important considerations in the management of severe allergic asthma with recurrent eosinophilia. First, it underscores the heterogeneity of asthma and the need for personalized treatment approaches. Although biological therapies targeting specific inflammatory pathways have revolutionized asthma care, some patients may exhibit incomplete or transient responses due to the involvement of alternative pathways or compensatory mechanisms. Understanding the molecular drivers of inflammation in individual patients is critical for selecting the most appropriate therapy. Second, the case illustrates the potential for disease progression or changes in inflammatory phenotypes over time. Longitudinal monitoring of biomarkers and clinical parameters is essential to detect such changes and guide treatment adjustments. In this patient, the transition from an IL-5-dominant phenotype to a broader type 2 inflammatory profile necessitated a shift in therapeutic strategy from anti-IL-5 to anti-IL-4/IL-13 therapy [4,5].

Conclusion

Third, the report emphasizes the importance of ruling out other causes of eosinophilia in patients with severe asthma. Conditions such as ABPA, EGPA, parasitic infections, and drug reactions can mimic or exacerbate asthma-related eosinophilic inflammation. Comprehensive diagnostic evaluations, including imaging, serology, and microbiological testing, are crucial for excluding these conditions and ensuring accurate diagnosis and treatment Finally, this case highlights the role of emerging biomarkers in understanding and managing severe asthma. Elevated TSLP and IL-33 levels in this patient provided valuable insights into the underlying inflammatory pathways driving her disease and informed the decision to initiate dupilumab. As the field of asthma research continues to evolve, the development of novel biomarkers and targeted therapies holds promise for improving outcomes in patients with refractory disease. In conclusion, recurrent eosinophilia in patients with severe allergic asthma receiving biological therapy presents a challenging clinical scenario. This case demonstrates the need for a comprehensive and individualized approach to diagnosis and treatment, incorporating advanced diagnostics, longitudinal monitoring, and flexible therapeutic strategies. By addressing the multifaceted nature of eosinophilic inflammation, clinicians can optimize care for patients with severe asthma and improve their quality of life.

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Conflict of Interest

None.

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