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Disease Overshadowed by Asthma and Thromboembolism: Hypereosinophilic Syndrome

Astım ve Pulmoner Embolinin Gölgesinde Kalan Bir Hastalık: Hipereozinofilik Sendrom

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Abstract: Hypereosinophilic syndrome is a rare condition characterized by persistent eosinophilia and multisystem involvement. Diagnosis is established after excluding secondary causes, and it can easily be overlooked when clinical features overlap with other diseases. Consequently, the diagnostic process becomes more challenging in patients with coexisting chronic respiratory disorders and a history of thromboembolic events. In our case, we present the diagnostic course of a patient with a 10-year history of asthma and two episodes of presumed idiopathic pulmonary thromboembolism within the past two years, who developed new bilateral pulmonary consolidations and a morphea-like skin lesion. Cases encountered in clinical practice do not always exhibit the classic presentations described in the literature; therefore, assessing each patient within their unique context through a holistic approach is essential for achieving accurate diagnosis and appropriate management.
Keywords: Hypereosinophilia, Asthma, Pulmonary Thromboembolism

Özet: Hipereozinofilik sendrom, kalıcı eozinofili ve çoklu organ tutulumu ile seyreden, nadir görülen bir hastalıktır. Tanı, sekonder nedenlerin dışlanması ile konur ve klinik belirtiler başka hastalıklarla örtüştüğünde kolayca gözden kaçabilir. Bu nedenle, özellikle eşlik eden kronik solunum hastalıkları ve tromboembolik öyküsü olan olgularda tanı güçleşir. Olgumuzda, 10 yıldır astım tanısı ile izlenen ve son iki yılda iki kez idiopatik olduğu düşünülen pulmoner tromboemboli geçiren bir hastada, yeni gelişen bilateral akciğer infiltratları ve ciltte morfea benzeri lezyon ile ortaya çıkan tanı süreci aktarılmıştır. Klinik pratiğe yansıyan vakalar, her zaman literatürde tarif edilen klasik görünümleri sergilemeyebilir; bu nedenle her hastayı kendi bağlamında bütüncül bir yaklaşımla değerlendirmek, doğru tanı ve tedaviye ulaşmada kritik öneme sahiptir.
Anahtar Kelimeler: Hipereozinofili, Astım, Pulmoner Tromboemboli

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1. Introduction

Eosinophils are polymorphonuclear leukocytes that contribute to host defense against helminthic parasites and play a central role in allergic inflammation. Upon activation, they release cytotoxic mediators capable of causing tissue injury, particularly in the lungs (1). Eosinophilia is defined as an absolute eosinophil count exceeding >500/μL, while counts ≥1500/μL are classified as hypereosinophilia (2, 3). Hypereosinophilic syndrome (HES) is defined by persistent eosinophilia ≥1500/μL for more than six months, absence of identifiable secondary causes (e.g., parasitic infection, allergic disease, drug reaction, autoimmune disorder, malignancy), and organ damage attributable to eosinophilia (4). The condition is rare, with a male predominance (9:1) and peak incidence between 20 and 50 years of age (5). Asthma–HES coexistence is uncommon. In patients with asthma and organ involvement, eosinophilic granulomatosis with polyangiitis (EGPA) is often considered first, as both can present with eosinophilia and multisystem disease (4). Distinguishing between them requires careful clinical, laboratory, and radiological assessment.

2. Case Report

A 45-year-old woman presented to the pulmonology outpatient clinic with a one-week history of worsening dyspnea, productive cough, and fever. Posteroanterior chest radiography demonstrated bilateral infiltrates, predominantly in the left lower lung zone. She was admitted to the Pulmonology Department with the preliminary diagnosis of pneumonia.

Her medical history included asthma (10 years), two episodes of presumed idiopathic pulmonary thromboembolism (PTE) within the past two years, and hypothyroidism. Asthma was managed with regular inhaled corticosteroid/long-acting beta-agonist (ICS/LABA) therapy and a leukotriene receptor antagonist (LTRA). Both PTE episodes, confirmed by ventilation–perfusion scintigraphy at different anatomical sites, prompted extensive evaluation genetic testing, collagen vascular markers, and rheumatologic screening all of which were unremarkable. She was diagnosed with idiopathic recurrent PTE. Three months after the first PTE episode, and prior to the second, she was hospitalized for pneumonia. Family history revealed that her father had developed pleural effusion with 60% eosinophils in pleural fluid. Although video-assisted thoracoscopic surgery was recommended, he declined and died five years later.

On examination, vital signs were stable. Auscultation revealed bilateral diffuse rhonchi and coarse crackles in the lower lung zones. Dermatological inspection showed a firm, hypopigmented, atrophic macular lesion (7 cm) on the lower right back, reported to have appeared three months earlier.

Laboratory findings: C-reactive protein: 131 mg/L; hemoglobin: 12.1 g/dL; neutrophils: 8.94 × 10^3/μL; white blood cells: 13.45 × 10^3/μL; eosinophils: 1.58 × 10^3/μL; ESR: 54 mm/h. Liver enzymes, electrolytes, renal function, thyroid function, anemia panel, and tumor markers were normal. Stool examination for parasites was negative; sputum and blood cultures were sterile.

Thoracic CT revealed bilateral consolidations right upper and left lower lobes surrounded by scattered micronodular opacities. No filling defect suggestive of pulmonary embolism was present (Figure 1A–B).

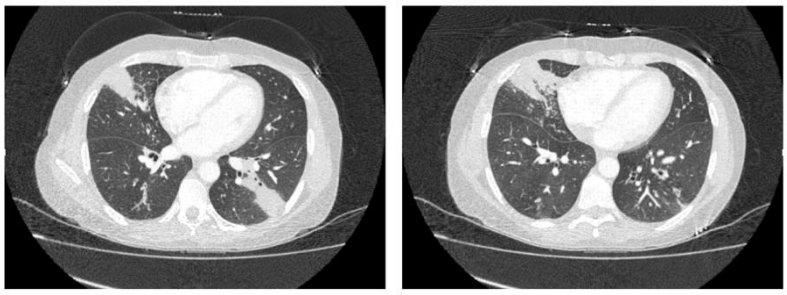

Figure 1A Figure 1B

Figure 1A–B. Axial thoracic CT images at different levels demonstrate bilateral consolidations in the right upper and left lower lobes, surrounded by scattered micronodular opacities.

Ampicillin–sulbactam and a macrolide were continued. By day 3, infection markers improved, but eosinophils rose. Peripheral smear confirmed hypereosinophilia. Hematology recommended bone marrow biopsy, transthoracic biopsy of the lung lesion, endoscopy, colonoscopy, and echocardiography. Dermatology biopsy of the skin lesion confirmed morphea.

Follow-up CT before lung biopsy showed lesion regression, so biopsy was canceled. Endoscopy revealed only hyperemic gastropathy; colonoscopy was normal. Echocardiography showed LVEF 65% with normal chamber sizes. Bone marrow biopsy demonstrated marked eosinophilia, confirming HES. Methylprednisolone 1 mg/kg/day was initiated, and genetic testing ordered.

3. Discussion and Conclusion

HES is a rare leukoproliferative disorder characterized by persistent eosinophilia in the peripheral blood and tissues, leading to eosinophil-mediated tissue damage and potential multi-organ dysfunction (1). The diagnosis requires exclusion of secondary causes of eosinophilia, such as parasitic infections, allergic diseases, drug reactions, autoimmune disorders, and malignancies. In the present case, the patient's history of asthma, recurrent PTE, and previous episodes of pneumonia initially suggested acute pneumonia, asthma exacerbation, or recurrent PTE as the most likely causes of her current presentation. However, the coexistence of bilateral pulmonary consolidations, elevated eosinophil counts, and a markedly increased ESR (Erythrocyte Sedimentation Rate) raised suspicion for an underlying systemic disorder beyond these initial differential diagnoses.

In patients with longstanding eosinophilia and uncontrolled asthma, the presence of bilateral pulmonary infiltrates often raises suspicion for eosinophilic granulomatosis with polyangiitis (EGPA). However, in the present case, neither the clinical findings nor the radiological assessments revealed any evidence of vasculitis.

Reviewing the patient's eosinophil curve over a decade revealed levels that consistently remained below 1000/μL, likely suppressed by chronic use of corticosteroids, leukotriene receptor antagonists, and antihistamines (7). This pharmacologically masked elevation contributed to delayed diagnosis, illustrating how standard thresholds can be misleading in treated asthmatic patients.

Importantly, although hypereosinophilia is not commonly considered a risk factor for venous thromboembolism (VTE), recent studies suggest this may be an underappreciated mechanism. Hypereosinophilia can damage the endothelium and promote a hypercoagulable state through eosinophil-derived toxic proteins and prothrombotic phospholipid surfaces(8). In fact, retrospective data demonstrate that 25% of patients with idiopathic hypereosinophilia or HES develop VTE, including pulmonary embolism (PE), especially when eosinophil counts and disease duration are elevated(6). These findings reinforce the importance of considering HES in patients with recurrent, idiopathic PTE.

This case underscores the value of recognizing multisystem involvement. The combination of pulmonary findings and a morphea-like skin lesion signaled widespread eosinophilic activity. Even though gastrointestinal, cardiac, and neurologic systems were unremarkable, a multidisciplinary evaluation remains essential in HES to detect hidden organ involvemen (9).

Overall, this case powerfully illustrates how HES can be masked by comorbid conditions—such as asthma and recurrent PTE—and how systemic organ involvement may only become apparent after comprehensive evaluation. It highlights the imperative of a systematic, holistic approach to diagnosis and the value of early immunosuppressive therapy (e.g., corticosteroids), which can mitigate tissue damage and thrombotic risk.

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