**Scholars Journal of Medical Case Reports**
Abbreviated Key Title: Sch J Med Case Rep
ISSN 2347-9507 (Print) | ISSN 2347-6559 (Online)
Journal homepage: https://saspublishers.com

OPEN ACCESS

**Respiratory Diseases**

# Intra-Alveolar Hemorrhage Revealing Eosinophilic Granulomatosis with Polyangiitis (EGPA)

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**DOI:** https://doi.org/10.36347/sjmcr.2025.v13i05.006 | **Received:** 21.03.2025 | **Accepted:** 25.04.2025 | **Published:** 02.05.2025

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**Abstract** **Case Report**

Eosinophilic Granulomatosis with Polyangiitis (EGPA) is a rare systemic vasculitis that combines severe or late-onset asthma with cardiac, ear-nose-throat (ENT), neurological, or renal manifestations. Asthma, as one of the most common features of EGPA, plays a central role in the disease's progression. Intra-alveolar hemorrhage, on the other hand, is a potentially serious but rare complication. We report a case of EGPA diagnosed due to intra-alveolar hemorrhage associated with an acute asthma exacerbation and systemic manifestations in a 58-year-old patient. This case allows discussion of the diagnostic and therapeutic challenges and the prognosis of this rare and severe condition.

**Keywords:** eosinophilic granulomatosis with polyangiitis, EGPA, vasculitis, intra-alveolar hemorrhage, DAH, ANCA.



## I. INTRODUCTION

Eosinophilic Granulomatosis with Polyangiitis (EGPA), formerly known as Churg-Strauss syndrome, is a rare and serious autoimmune condition that causes inflammation in blood vessels and affects several organs, particularly the lungs, heart, and nerves. The disease is often tricky to diagnose and manage because it typically presents with both asthma and symptoms of vasculitis, which can overlap in ways that make it harder to pinpoint. While much has been studied about EGPA, some of its rarer manifestations remain underrecognized. One such example is intra-alveolar hemorrhage, which is a seldom- reported complication of the disease, despite its potential to significantly impact patient health. This case report highlights this unusual presentation, aiming to expand the understanding of EGPA and its diverse ways of affecting the body. By sharing this rare occurrence, we hope to shed light on an underexplored aspect of EGPA and encourage more attention to these less common but important complications in both diagnosis and treatment.

## II. CLINICAL CASE

A 58-year-old patient, with a childhood history of asthma initially well controlled on inhaled corticosteroids and short-acting bronchodilators, presented with worsening asthma and symptom recurrence over the past two years. Two months prior to consultation, symptoms worsened with wheezing, progressive exertional dyspnea, and bilateral hand paresthesia. One week before admission, he developed minor hemoptysis, the main reason for consultation. The symptoms occurred in a context of 7 kg weight loss, fatigue, anorexia, frequent nocturnal awakenings, and significant daily activity limitations, including cessation of work.

Clinical examination revealed a normal temperature (37.2°C), tachycardia (129 bpm), oxygen saturation at 92% on room air, and signs of respiratory distress. Urinalysis (urine test strip) was negative. Pulmonary auscultation found wheezing; skin exam showed pruritic erythematous palmoplantar lesions with a clear center (Figure 1).

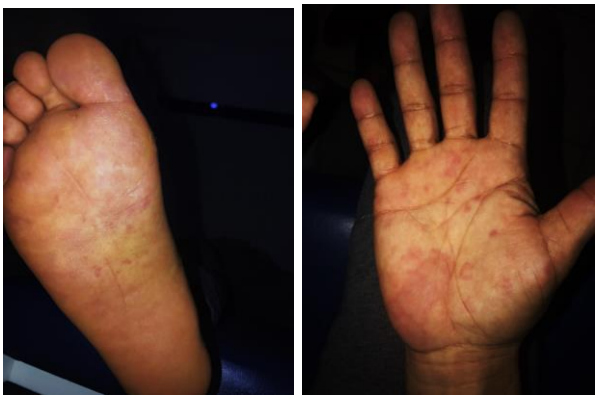


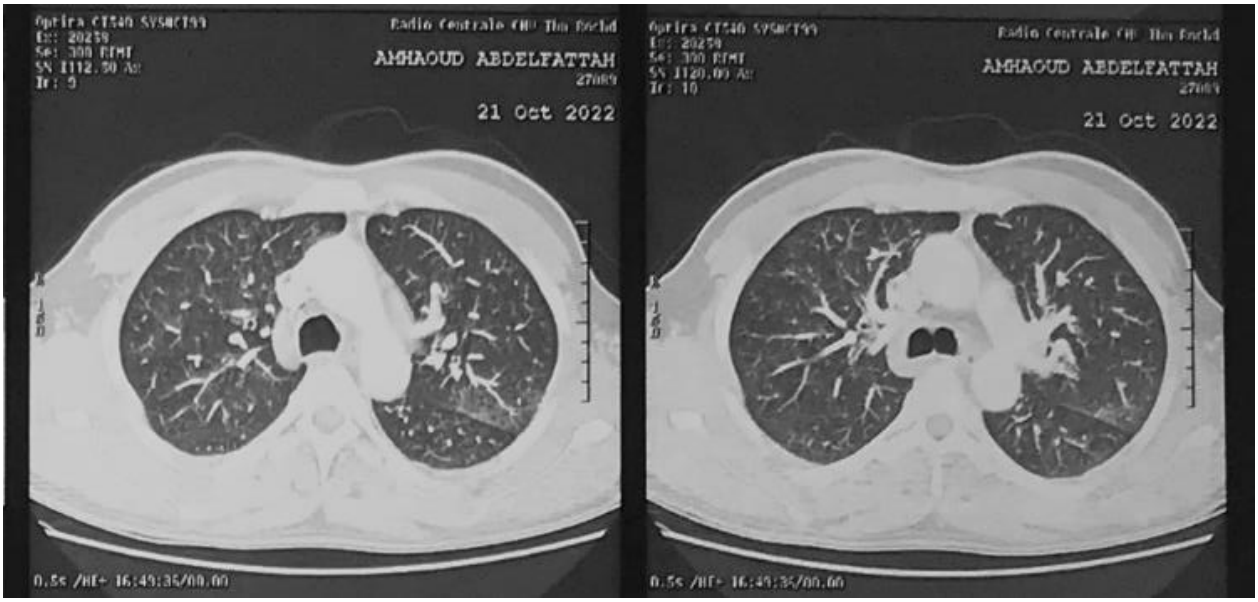
**Figure 1: Erythematous, clear-centered, pruritic palmoplantar skin lesions**

Lab tests showed marked eosinophilia (PNE 14 500/mm³), with previous records confirming persistent eosinophilia for over a month (12 750 - 15 752/mm³) (Figure 2). There was normocytic normochromic anemia and elevated CRP (120 mg/L). Bone marrow exam showed eosinophilia without dysplasia. Renal function and 24h proteinuria were normal. Chest CT showed bilateral ground-glass opacities (Figure 3). Bronchoalveolar lavage (BAL) fluid was hemorrhagic, confirming alveolar hemorrhage, with >38% eosinophils (Figure 4). Bronchial biopsies showed eosinophilic inflammatory infiltrates suggestive of bronchial eosinophilia.

Jour de l'admission

| | 04/10 | 13/10 | 25/10 | 10/11 | 12/11 |
|---|---|---|---|---|---|
| GB (/mm³) | 29 170 | 25 580 | 28 540 | 29 350 | 22 440 |
| PNE (/mm³) | 15 752 | 13455 | 12 750 | 14 500 | 6870 |
| Hb (g/dl) | 13,5 | 13,7 | 13,9 | 10,2 | 7,9 |

**Figure 2: Major blood hypereosinophilia persisting for more than one month**



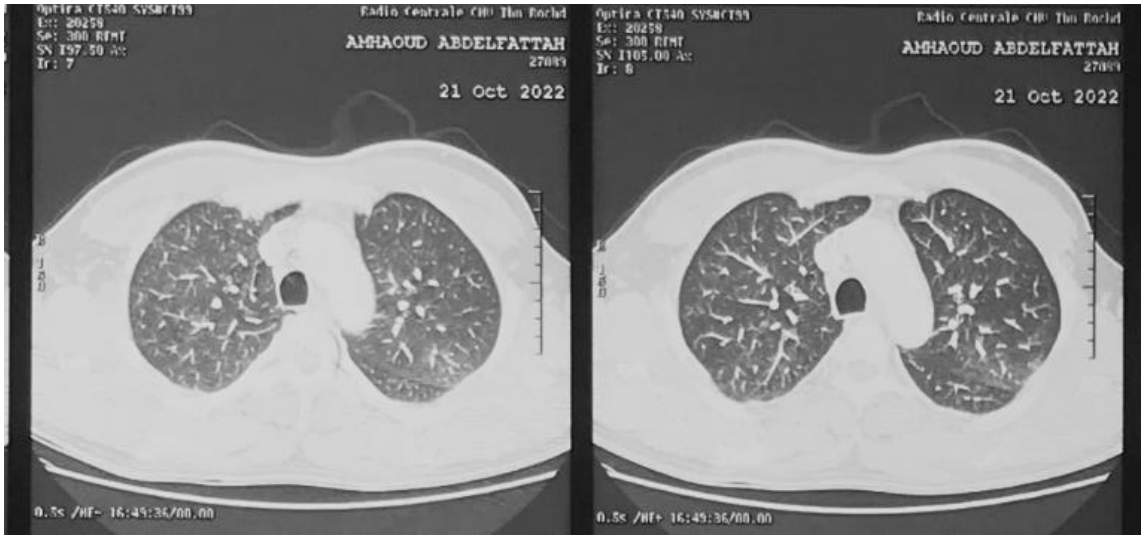
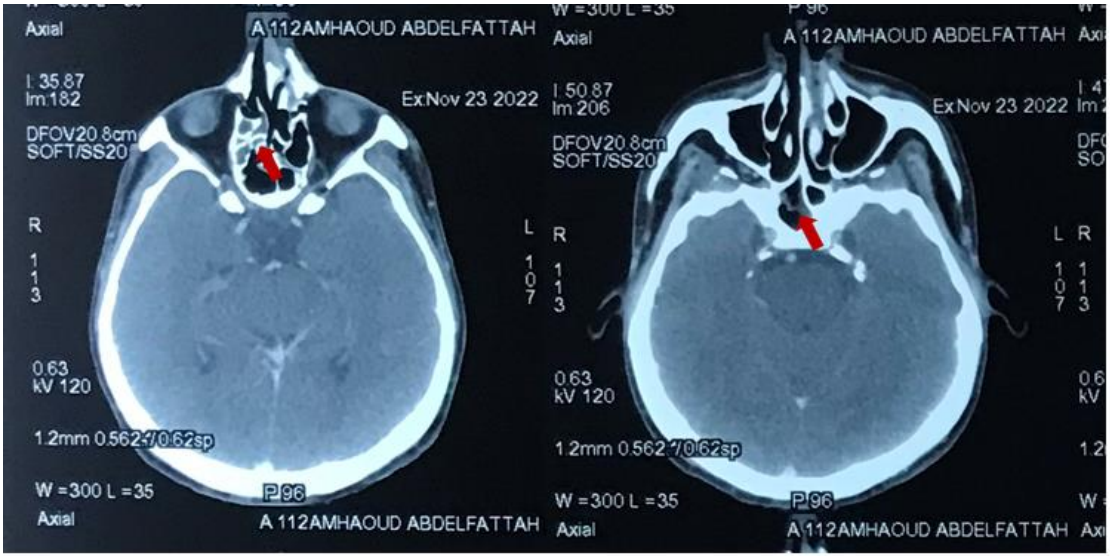



**Figure 3: Chest CT showing bilateral ground glass image**

**Figure 4: Bronchoalveolar lavage fluid with a frankly hemorrhagic appearance**

Immunology showed positive pANCA (anti-myeloperoxidase), negative cANCA and IgE elevated at 1417.04 IU/mL. Nasal endoscopy showed inflamed pituitary mucosa with purulent discharge; sinus CT showed chronic ethmoidal, sphenoidal, and right maxillary sinusitis (Figure 5). Neurological and cutaneous signs did not warrant further testing.



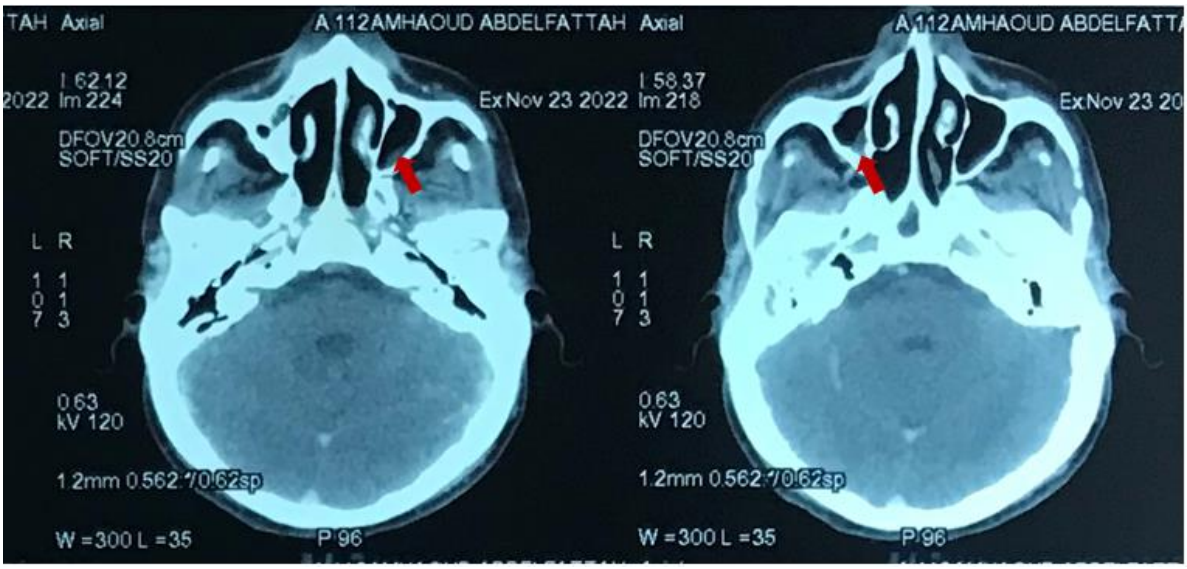



**Figure 5: CT scan of the sinuses showing chronic anterior ethmoidal and sphenoidal rhinosinusitis and right maxillary sinuses**

The ACR/EULAR 2022 score was 10, confirming EGPA. Despite pANCA positivity (suggesting less aggressive disease) and a Five Factor Score (FFS) of 0, prognosis was guarded due to intra-alveolar hemorrhage, a poor prognostic indicator. ECG and echocardiography were normal. After ruling out infection, high-dose corticosteroids (initially IV, then oral) were started along with cyclophosphamide pulses, leading to clinical, biological, and radiological improvement. Close monitoring was implemented.

## III. DISCUSSION

EGPA is a rare systemic vasculitis, often associated with eosinophilia and pulmonary/systemic involvement. This case highlights the importance of considering EGPA in patients with intra-alveolar hemorrhage, particularly in the presence of severe, poorly controlled asthma and systemic symptoms.

EGPA pathophysiology involves genetic (HLA system) and environmental factors (infections, exposure to grains or pigeons). Drugs like leukotriene antagonists and macrolides may also trigger it.

Asthma in EGPA is often longstanding but worsens with additional respiratory/systemic signs, as seen in this case. Severe allergic rhinitis and reduced daily activity are typical signs. Reassessing asthma diagnosis is crucial when EGPA is suspected.

Intra-alveolar hemorrhage, a rare but serious EGPA complication, occurs in 10–20% of cases. Eosinophils, through pro-inflammatory cytokines and enzymes, promote inflammation, granuloma formation, and in severe cases, vascular damage leading to hemorrhage. Prompt evaluation and immunosuppressive therapy are essential.

Blood eosinophilia (>1,500/mm³) is key to EGPA diagnosis, especially when associated with respiratory and systemic symptoms. Though not pathognomonic, it strongly suggests EGPA.

pANCA (anti-MPO) antibodies, present in 60–80% of EGPA cases, are associated with a better prognosis. Testing for ANCA is useful in patients with moderate to high EGPA suspicion. Complementary exams like biopsies and BAL confirm pulmonary eosinophilia. Sinus CT often reveals chronic sinusitis, a common finding in EGPA.

Treatment is based on immunosuppressants to control inflammation and eosinophilia. High-dose systemic corticosteroids are the first-line treatment. Immunosuppressants like azathioprine or methotrexate are used for maintenance and relapse prevention.

Bronchodilators and inhaled corticosteroids are important for asthma symptom control. A combined approach—treating both EGPA and asthma—is essential.

EGPA prognosis depends on age, disease severity, and treatment response. The Five Factor Score (2011) evaluates long-term risk, considering cardiac, renal, and neurological involvement. This patient's FFS of 0 suggests low risk for major cardiac events, but the intra-alveolar hemorrhage implies a more reserved long-term outlook.

Long-term follow-up is crucial to manage EGPA-related complications and prevent recurrence. Irreversible pulmonary damage and quality-of-life deterioration highlight the need for early, aggressive treatment.

## IV. CONCLUSION

This case illustrates the diagnostic challenge of EGPA, a rare but potentially life-threatening condition,

especially when presenting with severe pulmonary symptoms such as intra-alveolar hemorrhage. Early diagnosis and prompt immunosuppressive treatment are key to preventing respiratory complications and improving long-term outcomes.

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