CASE REPORT

A DIAGNOSTIC CHALLENGE PRESENTING AS NON RESOLVING PNEUMONIA IN A YOUNG FEMALE

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ABSTRACT

This case highlights the diagnostic challenge of a 35-year-old female initially managed as community-acquired pneumonia, who later presented with persistent respiratory symptoms, systemic complaints, and peripheral blood eosinophilia. Further investigations revealed positive peripheral antineutrophil cytoplasmic antibodies (p-ANCA)and pulmonary infiltrates, leading to a diagnosis of Eosinophilic Granulomatosis with Polyangiitis (EGPA), a rare form of ANCA-associated vasculitis. She was managed successfully with high-dose methylprednisolone, methotrexate, and supportive care. Our case illustrates the importance of suspecting rare causes in case of non resolving pneumonia.

Keywords: *Eosinophilic Granulomatosis with Polyangitis, Pneumonia, ANCA*

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INTRODUCTION

Eosinophilic Granulomatosis with Polyangiitis (EGPA), formerly known as Churg-Strauss syndrome, is a rare small-to-medium vessel vasculitis characterized by asthma, eosinophilia, and granulomatous inflammation. Its variable presentation usually causes a delay in its diagnosis. This report illustrates a classic presentation of a patient whose journey through persistent respiratory symptoms and vague systemic complaints eventually led to the diagnosis of this rare autoimmune condition. This case also underscores the importance of considering vasculitic syndromes in patients with unexplained eosinophilia and multi-systemic manifestations. Timely recognition and a multidisciplinary approach are important to prevent irreversible damage to the organs.

CAPSULE SUMMARY

A case is reported that emphasizes the need for high clinical suspicion and a multidisciplinary approach in cases of non-resolving pneumonia, especially when eosinophilia and systemic symptoms are present. Eosinophilic Granulomatosis with Polyangiitis, though rare, should be part of the differential in such cases.

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Case:

A 35-year-old housewife from Mandi Bahauddin presented in Medical out patient department on April 17th, 2024 with a one-week history of intermittent fever (up to 101°F) and productive cough with white mucoid sputum. She had a past medical history of hypothyroidism, for which she was taking Tablet Thyroxine 150 µg daily, and was experiencing asthma-like symptoms for the past one year. On examination, her temperature was 102°F with a pulse of 110 beats per minute. On auscultation, there were crepitations in the right lower chest posteriorly. Chest X-ray showed non-homogenous patchy opacities in the right lower zone. Liver function tests (LFT)and Renal function tests (RFT) were within normal limits. Initial diagnosis of community-acquired pneumonia was made, and she was managed with Inj Rocephin (Ceftriaxone) 1g I/V, 12 hourly, Tablet Leflox (Levofloxacin) 500mg 24 hourly, along with symptomatic treatment. Later her symptoms improved and she was discharged on Tablet Leflox (levofloxacin) 500mg per oral 24 hourly and follow-up advice.

After 02 months, on 16th June 2024, she came in OPD in deteriorated health. This time, she reported persistent cough with hemoptysis. She gave a history of weight loss of almost 3kg in the last 02 months with low-grade fever, numbness in both

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hands and generalized body aches. There were crepitation in the right lower chest and reduced pinprick sensations in her hands. The peripheral blood picture showed Hb 9.2g/dl ,TLC 8.1 ccm and a peripheral eosinophil count of more than 10%. HRCT showed ground glass attenuation in pulmonary parenchyma in mid and lower zones suggestive of an acute infective/allergic etiology. There was no evidence of bronchiectasis. There was no organomegaly, and the absence of any mass in HRCT made malignancy a least likely diagnosis. Sputum for gene expert was negative for tuberculosis. Liver function tests (LFT)and renal function tests (RFT) were within normal limits.

In the light of marked eosinophilia, the rare possibility of autoimmune vasculitis was considered and autoimmune profile was requested which revealed a positive peripheral antineutrophil cytoplasmic antibodies (p -ANCA) and negative anti nuclear antigen(ANA) serology. On the basis of X ray chest/ HRTC findings, peripheral neuropathy, peripheral eosinophila and positive p-ANCA serology she was diagnosed as a case of Eosinophilic Granulomatosis with Polyangiitis (EGPA). Skin biopsy couldn't be done because of the absence of any skin lesion, and the patient refused a lung biopsy.

She was managed with Injection Solu Medrol (Methylprednisolone) 1g/day, Tab Cytotrexate (Methotrexate) 15mg once weekly, supplemental oxygen and nebulization. Her symptoms improved. She was counselled in detail regarding her condition, importance of follow-up and compliance with medications.

DISCUSSION

Eosinophilic Granulomatosis with Polyangiitis (EGPA) is a rare systemic vasculitis that primarily affects small- to medium-sized vessels and is characterized by asthma, eosinophilia, and tissue infiltration by eosinophils, often accompanied by granuloma formation. The disease typically follows a triphasic clinical course: a prodromal phase with allergic features (e.g., asthma, allergic rhinitis), an eosinophilic phase, marked by peripheral eosinophilia and organ involvement (notably pulmonary and gastrointestinal), and a vasculitic phase, during which systemic manifestations and end-organ damage become evident due to necrotizing vasculitis.[1]

Presentation with stroke or cardiac events may also occur.[2,3] Although only 30–40% of EGPA patients are ANCA-positive, the presence of ANCA often correlates with more pronounced vasculitic manifestations such as glomerulonephritis, neuropathy and purpura, while ANCA-negative patients tend to have more eosinophilic tissue infiltration and cardiac involvement[4]. Our patient's clinical trajectory aligns with an ANCA-positive phenotype, with prominent neurologic and pulmonary findings.

Diagnosis is largely clinical, supported by the American College of Rheumatology (ACR) criteria (Table 1), which include asthma, eosinophilia >10%, mononeuritis or polyneuropathy, pulmonary infiltrates, paranasal sinus abnormality, and biopsy showing extravascular eosinophils.[5] In this case, four of these criteria were met, reinforcing the diagnosis.

Table 1: American College of Rheumatology (ACR) criteria

Considerations when applying ACR criteria:

- These classification criteria should be applied to classify a patient as having eosinophilic granulomatosis with polyangiitis (EGPA) when a diagnosis of small- or medium-vessel vasculitis has been made.
- Alternative diagnoses mimicking vasculitis should be excluded prior to applying the criteria.

Criteria Type	Item	Score
Clinical Criteria	Obstructive airway disease	+3
	Nasal polyps	+3
	Mononeuritis multiplex	+1
Laboratory/ Biopsy Criteria	Blood eosinophil count ≥ 1×10^9/L	+5
	Extravascular eosinophilic-predominant inflammation on biopsy	+2
	Positive test for cytoplasmic ANCA or anti-PN3 ANCA antibodies	-3
	Hematuria	-1

Scoring Guideline:

- Sum the scores for the 7 items, if present.
- A score ≥ 6 is needed for classification of EGPA.

Management of EGPA (Table 2) hinges on disease severity. For non-severe disease, glucocorticoids 0.5mg-1mg/kg/day alone may suffice, but in severe or life-threatening cases—especially those with cardiac, renal, or neurologic involvement—immunosuppressive therapy (e.g., Cyclophosphamide, Rituximab) is warranted. Recent trials have shown promise with Mepolizumab, an anti-IL-5 monoclonal antibody, particularly in relapsing or refractory disease, reflecting a shift toward targeted biologic therapies.[6]

Treatment of EGPA relies on systemic glucocorticoids, in combination with DMARDs. Our patient responded favorably to high-dose corticosteroids and immunosuppressive therapy, highlighting the importance of early recognition and aggressive intervention in preventing irreversible organ damage. However, long-term monitoring remains essential given the potential for relapse and chronic complications, including steroid dependence and treatment-related adverse effects. EGPA has a good prognosis following timely detection and treatment, with a 5 years survival rate of 90%.The relapse rate is estimated to be approximately 20% to 30%, often presenting with fever, joint pain, and constitutional symptoms.[9]

A case involved a 72 years old male with a history of asthma, bronchitis and recurrent sinusitis who presented with shortness of breath and progressively worsening hypoxemic respiratory failure. Similar diagnostic markers p-ANCA positivity d

Table 2 -Management

Step	Category	Details
1	Assess Disease Severity	Non-Severe: No major organ involvement Severe: Cardiac, renal, CNS, or GI involvement[7]
2	Induction Therapy	Non-Severe Disease: • Systemic glucocorticoids (Prednisone 0.5–1 mg/kg/day, tapered slowly) • Consider mepolizumab for relapsing disease Severe Disease: • High-dose corticosteroids + immunosuppressive agent: 1. Cyclophosphamide (oral/IV) for 3–6 months 2. Rituximab (especially if ANCA-positive)
3	Maintenance Therapy	After remission: • Options: Azathioprine, Methotrexate, Mycophenolate mofetil • Consider mepolizumab for relapsing eosinophilic-predominant disease[8]
4	Supportive Measures	• TMP-SMX for Pneumocystis jirovecii prophylaxis (if on high-dose steroids or cyclophosphamide) • Bone protection: calcium, vitamin D, bisphosphonates • Monitor for infections • Regular surveillance for relapse, organ damage, treatment toxicity

eosinophilia supported the diagnosis of EGPA. Treatment with Methylprednisolone and Cyclophosphamide led to clinical remission[10].

Another case involved a 64 year old male with a history of asthma and multiple allergies. Over a long period of time he developed peripheral neuropathy and bronchiectasis. He was admitted with asthma exacerbation. His eosinophil count was 53% with similar diagnostic markers p-ANCA positivity peripheral neuropathy supported the diagnosis of EGPA. Treatment with Methylprednisolone and Methotrexate led to clinical remission but recurrent infections. Progressive respiratory failure developed and he succumbed to pneumonia[1].

Our case and these cases highlight the diagnostic challenge of EGPA due to overlapping symptoms with common respiratory illnesses. Early consideration of vasculitis in patients with asthma, systemic symptoms and eosinophilia is crucial. While treatment regimens varied slightly, both patients responded well to immunosuppressive therapy, underscoring the importance of timely diagnosis and intervention.

Our case also highlights the diagnostic challenge, EGPA poses due to its protean manifestations and overlapping features with other eosinophilic disorders, such as hypereosinophilic syndrome (HES), and vasculitides, including granulomatosis with polyangiitis (GPA) and microscopic polyangiitis (MPA). In this patient, the presence of longstanding asthma, marked peripheral eosinophilia, mononeuritis multiplex and pulmonary infiltrates raised the suspicion for EGPA, which was further supported by positive p-ANCA serology.

Infectious agents, allergens and certain drugs are known to precipitate EPGA. In our case it is likely that the initial episode of pneumonia trigged the onset of EPGA and presented as likely unresolved pneumonia.

CONCLUSION

This case contributes to the growing body of literature underscoring the heterogeneity of EGPA and emphasizes the need for high clinical suspicion and multidisciplinary approach in cases of non-resolving pneumonia, especially when eosinophilia and systemic symptoms are present. EGPA, though rare, should be part of the differential in such cases.

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