



Case Report

Eosinophilic Granulomatosis with Polyangiitis Presenting with Cardiac and Neurological Involvement in the Absence of Late Gadolinium Enhancement

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Abstract

Eosinophilic Granulomatosis with Polyangiitis is a rare vasculitis characterised by asthma, eosinophilia and multisystem involvement. Cardiac manifestations are a major determinant of morbidity and mortality but may be diagnostically challenging, particularly in the absence of classical imaging findings.

We report a case of a 64-year-old man presenting with chest discomfort, marked eosinophilia and cutaneous and neurological features. Investigations demonstrated elevated cardiac biomarkers, left ventricular systolic dysfunction and non-obstructive coronary arteries. Notably, cardiac MRI demonstrated no late gadolinium enhancement despite biochemical and functional evidence of myocardial injury.

The patient fulfilled classification criteria for EGPA, with alternative diagnoses including hyper-eosinophilic syndrome, infection and haematological malignancy being excluded. Early initiation of corticosteroid therapy resulted in rapid clinical and biochemical improvement, with subsequent induction therapy using cyclophosphamide.

This case highlights that cardiac involvement in EGPA may occur in the absence of classical cardiac MRI findings, and emphasises the importance of early recognition and treatment in patients with eosinophilia and multisystem disease.

Introduction

Eosinophilic granulomatosis with polyangiitis (EGPA) is a rare autoimmune disorder, characterised by eosinophil-rich granulomatous inflammation of small-medium sized vessels and different organs, such as lungs, skin, upper respiratory tract and sometimes the heart. Whilst the exact pathogenesis of this condition is not yet fully understood, many possible underlying mechanisms have been described in the literature, with IL-5, being suggested as the main eosinophil-activating cytokine produced by Th2 lymphocytes to play an important role in the development of the disease [1,2].

The cardiovascular involvement in EGPA is multifaceted. Eosinophilic myocarditis (EM) is a well-known manifestation of the disease, where eosinophil granulocytes infiltrate the heart and often cause severe irreversible damage due to their cytotoxic activity. Systematic reviews found that around 20% of EM cases are caused by EGPA. Other signs of the disease may include chronic inflammatory cardiomyopathy, pericarditis, coronary vasculitis, arrhythmias. Timely diagnosis of cardiac involvement in EGPA is critical and is the most important factor affecting mortality in the disease.

History of Presentation

A 64-year-old Chinese male admitted to Khoo Teck Puat Hospital in Singapore who works in repairs and has a >20-pack-year smoking history presented to the Emergency Department with chest discomfort for 1 week, on a background of intermittent chest discomfort over several months. Physical examination revealed livid palpable purpura and nodules on the extensor surfaces of the upper extremities (Figure 1). On examination of the heart, regular rate and rhythm, no murmurs, rubs, or gallops were found. The lungs were clear to auscultation bilaterally, with no wheezes or crackles. Neurological examination showed left pronator drift. Sensory loss was noted to pinprick entire left upper limb till elbow. Proprioception was intact. No ataxia was noted. No toxocara lesions were noted bilaterally. The ECG on admission (Figure 2) showed anterolateral ST depression from leads V2 to V6. No other abnormalities were identified.



Figure 1: Upon physical examination, the depicted skin lesions were noted and can be described as palpable, tender nodules on the extensor surfaces of the upper extremity.

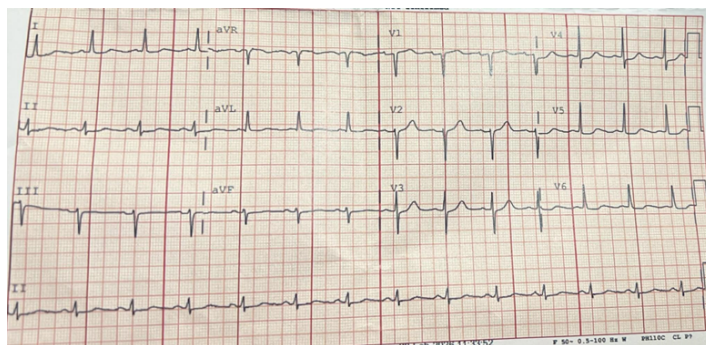


Figure 2: Electrocardiogram obtained upon admission shows sinus rhythm with a frequency of 79, borderline left axis deviation, and ST-segment depression in the anterolateral leads.

Past Medical History

The patient's relevant past medical history is notable for asthma-COPD overlap, hypertension, and likely type 2 chronic rhinosinusitis.

Differential Diagnosis

The differential diagnosis for this patient's presentation includes hypereosinophilic syndrome, eosinophilic granulomatosis with polyangiitis, haematological malignancy, and sarcoidosis. Infective causes of myocarditis not typically associated with eosinophilia include HIV, COVID-19, adenovirus, and enterovirus. Infective differentials potentially associated with eosinophilia include helminthic infections such as *Strongyloides*, *Schistosoma*, and *Trichinella* species. The patient fulfilled ≥ 4 ACR criteria for EGPA (asthma, eosinophilia, sinus disease, biopsy-proven vasculitis), supporting the diagnosis.

Investigations

High-sensitivity troponin-T levels were elevated ($3356 \rightarrow 509 \rightarrow 962 \rightarrow 552$ ng/L), consistent with myocardial injury. Coronary angiography revealed minor coronary artery disease with no flow-limiting stenosis. Transthoracic echocardiography showed an LVEF of 45% with mid-apical hypokinesis. Angiogram showed minor CAD with proximal 10% stenosis noted in LAD and RCA. Cardiac MRI demonstrated mild global systolic dysfunction. Notably, the absence of late gadolinium enhancement despite biochemical and functional evidence of myocardial injury suggests early-stage eosinophilic myocarditis.

Laboratory testing showed marked eosinophilia (peak $40 \times 10^9/L$), elevated LDH and inflammatory markers (CRP 39.6 mg/L, ESR 58 mm/h), and otherwise unremarkable morphology on peripheral smear and bone marrow biopsy apart from eosinophilic proliferation. Infectious work-up including syphilis serology, hepatitis B surface antigen, and HIV screening was negative. Autoimmune testing (ANA, ANCA, RF, anti-CCP) was also unremarkable. Histology from right foot sole and right nasal polyp showed leukocytoclastic vasculitis and inflammatory polyp respectively.

CT brain and CT carotid arteries showed right cerebellar haematoma, possible subarachnoid haemorrhage. MRI brain demonstrated extensive scattered cortical-cortical areas, raising suspicion for vasculitis-related embolic phenomena.

Management

Given the rapid rise in eosinophil count and concern for eosinophilic myocarditis within a multisystem process, empiric therapy was initiated. Ivermectin 200 $\mu\text{g/kg}$ was administered for 2 days to exclude occult helminthiasis, in parallel with corticosteroid therapy. Prednisolone 0.5 mg/kg daily was then commenced for 4 days, resulting in a prompt and substantial decline in eosinophil count ($40 \rightarrow 7.79 \times 10^9/L$ within 48 hours; Figure 3). Dermatology assessment confirmed prurigo-type nodular lesions without indication for biopsy. Neurology reviewed the

incidental cerebellar haemorrhage; MRI patterns were consistent with vasculitic or septic-embolic phenomena, but without clinical evidence of active infection, so CT angiogram was deferred. Supportive therapy included management of vitamin D deficiency and symptomatic care for cutaneous manifestations. The overall clinical and biochemical improvement with corticosteroids supported the working diagnosis of newly diagnosed eosinophilic granulomatosis with polyangiitis with major organ involvement.

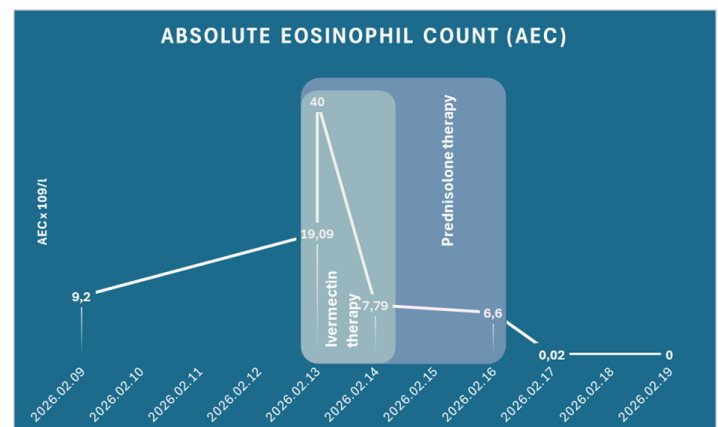


Figure 3: Eosinophil count (y-axis) as a function of time (x-axis). The rapid increase in eosinophil count prompted initiation of therapy: ivermectin 200 $\mu\text{g/kg}$ for two days (2026.02.13–14) for potential helminthiasis, and prednisolone 0.5 mg/kg for four consecutive days (2026.02.13–16). A rapid drop in eosinophil count was observed during the same period.

Outcome and Follow-Up

The patient was started on cyclophosphamide injections once a month and tapering dose of prednisolone (70mg OM for 2 weeks, followed by 60mg OM for 2 weeks and then 50mg OM for 2 weeks). His eosinophil counts decreased from 40 to $0.15 \times 10^9/L$. No neurological deficits were noted during his review 1 month post discharge. Moving forward, this patient was planned for 1 monthly cyclophosphamide injections. Subsequent CT brain showed interval improvement and evolution of right sided cerebellar haematoma and sulcal/grey-white haemorrhage in the right frontoparietal and bilateral occipital regions. He is planned for a repeat TTE in 2 months' time and is still working as a renovation contractor.

Discussion

Eosinophilic granulomatosis with polyangiitis (EGPA) is a rare condition characterised by asthma, eosinophilia and multisystem involvement. Cardiac manifestations are a major determinant of prognosis and may present with a wide spectrum of clinical features. This case highlights an atypical presentation with cardiac

and neurological involvement in the absence of classical cardiac MRI findings.

The diagnosis was supported by the presence of asthma, marked eosinophilia, chronic rhinosinusitis and biopsy-proven vasculitis, fulfilling established classification criteria for EGPA. Alternative diagnoses including hyper-eosinophilic syndrome, infection and haematological malignancy were considered but were less likely given the pattern of multisystem involvement and the rapid response to corticosteroid therapy.

Cardiac involvement is reported in up to 50% of EGPA cases and is a major predictor of mortality. Cardiac involvement in EGPA is commonly associated with eosinophilic myocarditis and typically demonstrates late gadolinium enhancement (LGE) on cardiac MRI. In this case, however, no LGE was observed despite clear biochemical and functional evidence of myocardial injury. This likely reflects early-stage eosinophilic myocardial involvement prior to the development of fibrosis prior to the development of myocardial fibrosis, emphasising that a normal CMR does not exclude cardiac involvement. Clinicians should therefore maintain a high index of suspicion in patients with eosinophilia and unexplained cardiac dysfunction.

Early recognition and treatment are essential, as prompt initiation of corticosteroids can lead to rapid improvement and prevent irreversible organ damage. In cases with major organ involvement, additional immunosuppression such as cyclophosphamide may be required. This case underscores the importance of early empirical therapy in suspected EGPA, even in the setting of diagnostic uncertainty.

Conclusion

This case highlights the importance of recognising EGPA as a potential cause of acute cardiac injury, particularly when accompanied by marked eosinophilia and multisystem involvement. Early initiation of corticosteroids led to rapid clinical and biochemical improvement. Prompt identification and treatment of eosinophilic myocarditis can prevent irreversible organ damage and improve outcomes.

Learning Points

Eosinophilic Granulomatosis with Polyangiitis should be suspected in patients with eosinophilia and unexplained cardiac injury, particularly when there is coexisting asthma or chronic rhinosinusitis.

Cardiac involvement may occur without late gadolinium enhancement on cardiac MRI, and a normal CMR does not exclude eosinophilic myocarditis.

Multisystem involvement and a rapid response to corticosteroids are key diagnostic and therapeutic clues, and early treatment is essential to prevent irreversible organ damage.

References

1. Furuta S, Iwamoto T, Nakajima H. (2019). Update on eosinophilic granulomatosis with polyangiitis. *Allergology international: official journal of the Japanese Society of Allergology*. 68: 430-436.
2. Grayson PC, Ponte C, Suppiah R, Robson JC, Craven A, et al. (2022). 2022 American College of Rheumatology/European Alliance of Associations for Rheumatology Classification Criteria for Eosinophilic Granulomatosis with Polyangiitis. *Annals of the rheumatic diseases*, 81: 309-314.