ARCHIVES OF THE TURKISH SOCIETY OF CARDIOLOGY

Official journal of the TURKISH SOCIETY OF CARDIOLOGY

Hypereosinophilic Syndrome Complicated by Eosinophilic Myocarditis: Embolic Stroke or Eosinophilic Stroke? A Case Report

Eozinofilik Miyokardit ile Komplike Olan Hipereozinofilik Sendrom: Embolik İnme mi Eozinofilik İnme mi? Bir Olgu Sunumu

ABSTRACT

Hypereosinophilic syndrome (HES) is traditionally described as chronic peripheral eosinophilia with involvement of various organs and systems, including the heart and nervous system. In this report, we describe cardiac involvement and border zone stroke in a patient with idiopathic HES. A 37-year-old woman presented with sudden right-sided weakness and slurred speech, which began four days before admission, accompanied by palpitations, retrosternal exertional chest discomfort, dry cough, and progressive shortness of breath over approximately two months. Preliminary studies showed an increased number of white blood cells with eosinophilia. Further diagnostic investigation revealed apical thrombosis in both ventricles of the heart and moderate left ventricular systolic dysfunction. Magnetic resonance imaging of the brain indicated multifocal infarctions in the anterior and posterior border zones, as well as both cerebellar hemispheres, predominantly on the left side. Consequently, the patient was diagnosed with idiopathic HES and treated with corticosteroids, cyclophosphamide, anticoagulants, and medications for heart failure. She responded well both clinically and hematologically. Our case highlights the importance of multiple imaging modalities in diagnosing eosinophilic endomyocarditis and the impact of timely medical treatment to prevent disease progression.

Keywords: Eosinophilic myocarditis, eosinophilic stroke, hypereosinophilic syndrome

ÖZET

Hipereosinofil sendromu (HES), geleneksel olarak periferik eozinofili ve kalp, sinir sistemi gibi çeşitli organ ve sistemlerin tutulmasıyla karakterize bir hastalıktır. Bu raporda, idiopatik HES'li bir hastada kardiyak tutulum ve sınır bölgesi inme (border zone stroke) sunulmuştur. Otuz yedi yaşındaki kadın hasta, dört gün önce gelişen ani sağ taraf zayıflığı, konuşma bozukluğu ve yaklaşık iki aydır devam eden çarpıntı, eforda retrosternal göğüs ağrısı, kuru öksürük ve ilerleyici nefes darlığı yakınması ile başvurdu. Ön değerlendirmede, eozinofili ile birlikte beyaz kan hücrelerinin sayısında bir artış olduğu görüldü. İleri tanısal incelemede, kalbin her iki ventrikülünde apikal trombüs ve orta derecede sol ventrikül sistolik işlev bozukluğu görüldü. Beyin manyetik rezonans görüntülemede, anteriyor ve posteriyor sınır bölgelerinde ve sol tarafta daha belirgin olmak üzere her iki serebellar hemisferde çoklu enfarktlar saptandı. Sonuç olarak, hastaya idiopatik HES tanısı kondu ve kortikosteroid, siklofosfamid, antikoagulan ve kalp yetersizliği ilaçları başlandı. Hem klinik hem de hematolojik olarak olumlu yanıt alındı. Olgumuz, eozinofilik endokardit tanısında çoklu görüntüleme yöntemlerinin önemini ve hastalığın ilerlemesini önlemede zamanında tıbbi tedavinin etkisini vurgulamaktadır.

Anahtar Kelimeler: Eozinofilik myokarditis, eozinofilik inme, hipereosinofil sendromu

CASE REPORT
OLGU SUNUMU

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Received: July 18, 2023
Accepted: February 10, 2024

Cite this article as: Khajouei AS, Tajmirriahi M, Payandeh Z, Mashhadi MA, Shirani N, Mirbod SM. Hypereosinophilic Syndrome Complicated by Eosinophilic Myocarditis: Embolic Stroke or Eosinophilic Stroke? A Case Report. *Turk Kardiyol Dern Ars.* 2025;53(1):62-67.

DOI:10.5543/tkda.2024.46487

Hypereosinophilic syndrome (HES) is characterized by peripheral blood eosinophilia (>1.5 × 10^9/L) persisting for at least six months, associated with organ damage and without any identifiable underlying etiology.[1] The diagnosis of HES is established by excluding known causes of hypereosinophilia, such as infections, parasites, allergies, vasculitis, malignancies, and hematological disorders.[1] It is an uncommon, multisystem, heterogeneous syndrome with a high fatality rate.[2] Cardiac involvement, a rare but serious complication of HES, can lead to myocardial fibrosis, persistent heart failure, and death.[3-5] The myocardial injury in HES is caused by eosinophils, which

Table 1. Initial Laboratory Investigations of the Patient

Laboratory Parameter	Patient Result	Reference Range	Laboratory Parameter	Patient Result	Reference Range
White Blood Cell Count	16.9	4.8-10.8 × 10^9/L	CRP	43	0-10 mg/L
Red Blood Cell Count	3.6	4.70-6.10 × 10^9/L	ESR	40	0-25 mm/hr
Hemoglobin	11.7	14-18 g/dL			
Hematocrit	34.9	42.0-52.0%			
MCV	89.5	80-100 fL	AST	35	5-40 IU/L
MCH	30	26-34 pg	ALT	31	5-50 IU/L
MCHC	33.5	31-37 g/dL	Troponin	13.3	<3.5
Platelets	0.7	1.5-4.5 × 10^9/L	D-Dimer	3512	<500
FBS	95	--	Serum Albumin	3.5	3.5-5.0 g/dL
Blood Urea Nitrogen	6	7-20 mg/dL	Urinalysis	Positive pathological finding was not detected	
			Urine Culture	Negative	
Creatinine	1.2	0.4-1.2 mg/dL	Coagulation Profile (PT, PTT, INR)	Normal range	

ALT, Alanine Transaminase; AST, Aspartate Transaminase; CRP, C-Reactive Protein; ESR, Erythrocyte Sedimentation Rate; FBS, Fasting Blood Sugar; INR, International Normalized Ratio; MCH, Mean Cell Hemoglobin; MCHC, Mean Corpuscular Hemoglobin Concentration; MCV, Mean Corpuscular Volume; PT, Prothrombin Time; PTT, Partial Thromboplastin Time.

release cationic proteins that trigger necrosis and apoptosis.[6] Additionally, numerous studies have documented central nervous system involvement, such as stroke, demyelination, and meningoencephalitis.[2,7-17] There is ongoing debate concerning the pathophysiology of these central nervous system (CNS) symptoms. In this case report, we discuss a HES patient presenting with cardiac involvement and stroke.

Case Report

A 37-year-old right-handed woman was admitted to our hospital presenting with new-onset right-sided weakness and slurred speech that began four days prior to admission. She also reported a non-productive cough, palpitations, wheezing, retrosternal exertional chest discomfort, and progressive shortness of breath ranging from New York Heart Association (NYHA) class I to III over the preceding two months. Apart from a five-year history of asthma, she had no other comorbid diseases. Upon admission, the patient was confused. Her vital signs included a blood pressure of 110/70 mmHg, a heart rate of 80 beats per minute, an oral temperature of 37°C, and a respiratory rate of 20/min with an oxygen saturation of 93% on room air. Chest auscultation revealed scattered wheezing in both lungs, and cardiac examination noted regular-irregular heart sounds without any murmurs. Her abdominal examination was unremarkable. Peripheral pulses were full, and there was no limb edema or lymphadenopathy detected. Neurological examination showed dysarthria, right-sided facial weakness, and right extremity weakness. Initial laboratory investigations, summarized in Table 1, showed a white blood cell (WBC) count of 16.9 x 10^9/L, with 34.2% eosinophils.

An extensive inpatient diagnostic workup was performed under the guidance of a multidisciplinary team. The results included serum iron at 40 mcg/dl (normal range: 39-149 mcg/dl), total iron binding capacity (TIBC) at 246 mcg/dl (normal range: 220-450 mcg/dl), and ferritin at 180 ng/ml (normal range: 4.6-204 ng/ml). Thyroid-stimulating hormone (TSH) levels were 1.5 mIU/ml (normal range: 0.3-4.9 mIU/ml). Blood cultures were negative. The immunologic and rheumatologic workup, which included tests for antinuclear antibodies (ANA), antineutrophil cytoplasmic antibodies (cytoplasmic antineutrophil cytoplasmic antibodies [C-ANCA], perinuclear antineutrophil cytoplasmic antibodies [P-ANCA]), anti-double-stranded DNA (anti-dsDNA), anti-cardiolipin (Immunoglobulin M [IgM]), anti-cardiolipin (IgG),

ABBREVIATIONS

ANA	Antinuclear antibodies
BMB	Bone marrow biopsy
C-ANCA	Cytoplasmic antineutrophil cytoplasmic antibodies
CNS	Central nervous system
CTA	Computed tomography angiography
ECG	Electrocardiography
EMG-NCV	Electromyography and nerve conduction velocity
HES	Hypereosinophilic syndrome
HIV	Human immunodeficiency virus
INR	International normalized ratio
MRI	Magnetic resonance imaging
NYHA	New York Heart Association
P-ANCA	Perinuclear antineutrophil cytoplasmic antibodies
PT/INR	Prothrombin time/international normalized ratio
PTE	Pulmonary thromboembolism
PVCs	Premature ventricular contractions
TIBC	Total iron binding capacity
TSH	Thyroid-stimulating hormone
TTE	Transthoracic echocardiography

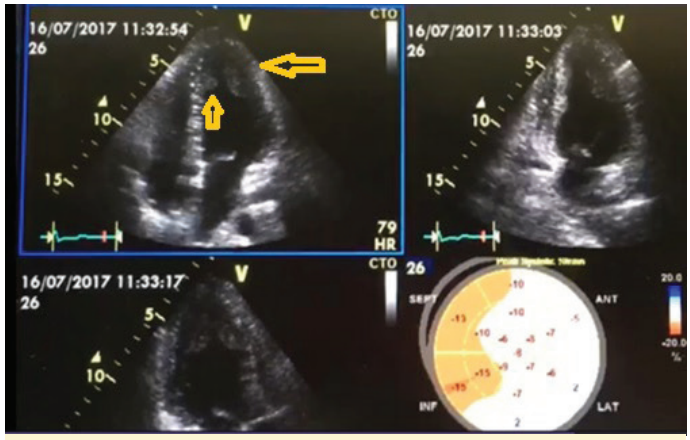

Figure 1. Cardiac echocardiography images showing thickening in the apical and apicolateral regions of the left ventricle.

B2 glycoprotein Ab (IgM), complement component 3 (C3), C4, factor V Leiden, protein C, and protein S, were all negative. Serological tests for hepatitis B and C viruses, and human immunodeficiency virus (HIV) were also negative.

Electrocardiography (ECG) showed sinus rhythm, a heart rate of 80 beat per minutes, nonspecific T-wave inversion in lateral leads (DI and AVL), and ST-segment depression in leads V2-V6 with frequent monomorphic premature ventricular contractions (PVCs). Chest radiography revealed hyperinflation of both lungs and flattening of the diaphragm.

Transthoracic two-dimensional echocardiography indicated a normal left ventricular size with moderate systolic dysfunction (left ventricular ejection fraction of 35%). The right ventricle was of normal size with mild systolic dysfunction. Thickened apical segments in both ventricles displayed significant trabeculation and hypokinesia. Diastolic dysfunction presented in a restrictive pattern, with no significant valvular involvement noted (Figure 1). Due to palpitations and recurrent PVCs, she was transferred to the cardiac care unit.

Subsequently, a chest computed tomography (CT) scan with contrast was performed, revealing no evidence of pulmonary thromboembolism (PTE).

We carried out cardiac computed tomography angiography (CTA) for the patient as she appeared too unstable to be transferred to a different hospital for magnetic resonance imaging (MRI) during the initial days of hospitalization. The CTA results indicated biventricular apical thickening and delayed acquisitions of low attenuation laminar densities due to thrombosis (Figure 2). Notably, there was no evidence of coronary artery disease in the coronary CTA. A few weeks later, a cardiac MRI was performed, revealing apical laminar thrombosis and secondary restrictive cardiomyopathy due to endomyocardial fibrosis with biventricular involvement (fibrothrombotic phase of hypereosinophilic syndrome) and a subendocardial inflammatory process in T2 images (Figure 3).

The patient also underwent a brain MRI which identified multiple areas of diffuse significant infarctions in the anterior and posterior border zones, as well as both cerebellar hemispheres, with a

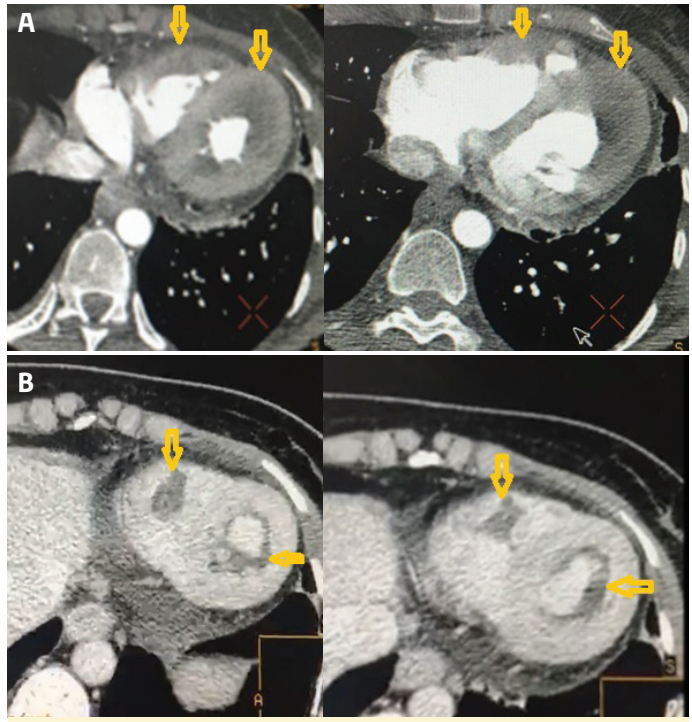

Figure 2. (A) Axial cardiac computed tomography images at the mid-cardiac level, illustrating increased thickness in the left and right ventricular wall, particularly at the apex. (B) Subsequent images at the same level display a low attenuation area, indicative of mural thrombosis.

greater concentration on the left side. Magnetic resonance angiography (MRA) of the head and neck yielded unremarkable findings. Additionally, electromyography and nerve conduction velocity (EMG-NCV) tests were performed, indicating acute axonal motor ulnar and median neuropathy in the left upper limb, consistent with mononeuritis multiplex.

A peripheral blood smear revealed normal red-cell morphology, low platelets, marked eosinophilia, and no blast cells. Subsequently, an aspiration and bone marrow biopsy (BMB) were performed. Histopathological analysis of the BMB revealed an increase in eosinophils with no evidence of malignancy. Flow cytometry results were negative. A multidisciplinary team meeting involving cardiologists, neurologists, hematologists, and internal medicine specialists discussed the patient's findings. After a thorough discussion, a provisional diagnosis of eosinophilic myocarditis resulting from hypereosinophilic syndrome was made, and she received a one-day intravenous infusion of 1 gram of cyclophosphamide followed by a three-day intravenous infusion of methylprednisolone at 1 g/kg, then switched to a two-day intravenous infusion of prednisolone at 1 mg/kg/day. Additionally, during her hospitalization, she was treated with budesonide and seretide (fluticasone propionate and salmeterol) sprays twice daily, and intravenous heparin. Due to the patient's unstable clinical condition, intravenous heparin was initiated in the first days and then switched to enoxaparin at 1 mg/kg twice a day, followed by oral warfarin. Warfarin treatment has continued up to the present for the prevention of cardioembolism, with the therapeutic dose

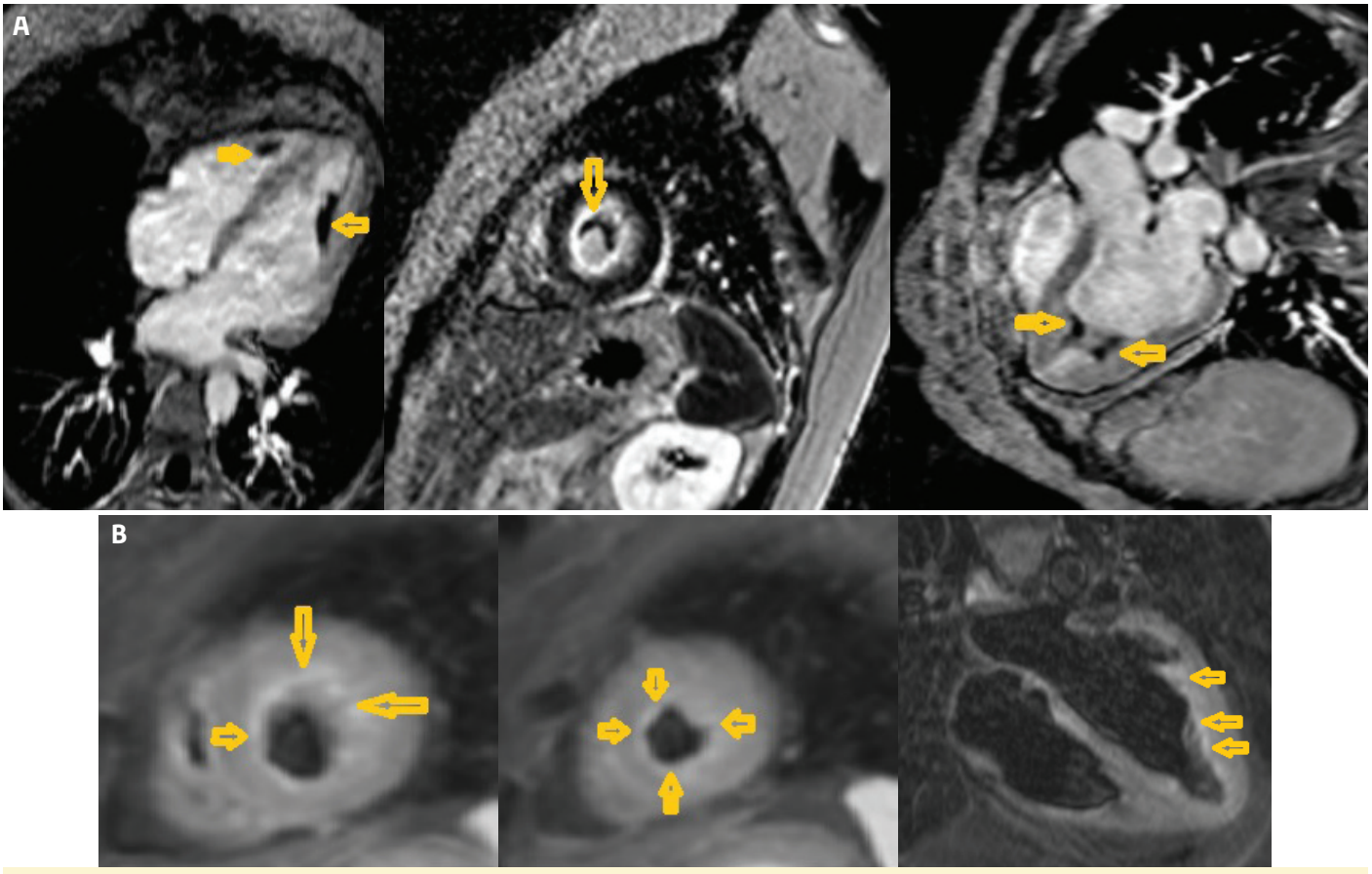

Figure 3. (A) Cardiac MRI images in short and long axis views showing late gadolinium hyperenhancement due to fibrotic changes in the apices of the left and right ventricles. (B) Cardiac MRI T2-weighted images reveal a hypersignal in the left ventricle's (LV) subendocardium, suggestive of edema from an inflammatory process.

maintaining an international normalized ratio (INR) of 2-3. Additionally, during hospitalization, the patient received an intravenous infusion of amiodarone and standard medical treatments for heart failure, including carvedilol 6.25 mg every 12 hours, enalapril 10 mg every 12 hours, spironolactone 25 mg daily, and 20 mg intravenous furosemide every 12 hours. The patient showed significant clinical improvement a few days after starting corticosteroids. She was discharged with a prescription for oral therapy consisting of 60 mg/day of prednisolone, 6.25 mg of carvedilol every 12 hours, 10 mg of enalapril every 12 hours, 25 mg of spironolactone daily, and 20 mg of furosemide every 12 hours. Over the next three months, the corticosteroid dose was tapered to 30 mg/day, followed by a reduction of prednisolone by 5 mg per week. At subsequent follow-ups, she remained asymptomatic and continues to do well to this day. Follow-up echocardiography performed six months after discharge showed a Left Ventricular Ejection Fraction (LVEF) of 55%, resolved apical thrombosis with just a strand of fibrotic material remaining, and normal systolic function. Consequently, spironolactone and enalapril were tapered off six months after discharge and discontinued, but carvedilol at 6.25 mg every 12 hours is still being continued. White blood cell counts, eosinophil counts, and platelet levels also remained within normal limits.

Discussion

Hypereosinophilic syndrome is a rare systemic disease that causes multi-organ failure. HES is defined by persistently elevated blood eosinophil levels (>1500/μL) and is associated with evidence of organ damage.[18] Cardiac involvement is observed in more than 50% of patients with HES, typically presenting with uni- or biventricular, pericardial, or global myocardial involvement.[19] It is associated with poor clinical outcomes, with the majority of deaths related to cardiac complications and thromboembolic events.[20] In this study, we reported a case of idiopathic HES with myocardial involvement, biventricular apical thrombus, and stroke. The patient was initially diagnosed with heart failure; thus, we needed to determine the etiology of heart failure for this patient. Primary myocardial damage, including ischemic heart disease, cardiomyopathy, myocarditis, myocardial toxicity, metabolic disorders, and immune damage, is usually the main cause of heart failure.[21,22] The patient denied having hypertension, diabetes, thyroid disease, or a history of drug abuse. Based on clinical manifestations, elevated troponin levels, ECG changes, and echocardiographic findings, the patient was considered to have suffered from acute coronary syndrome. However, no evidence of atherosclerosis was found in the coronary CTA. Additionally, we performed a pulmonary CTA to rule out PTE. The markedly increased eosinophils in routine blood tests drew our attention and

led us to propose that this patient might suffer from eosinophilic myocarditis, which in turn induced heart failure. Secondary causes of hypereosinophilia were ruled out, and the patient was finally diagnosed with idiopathic HES and idiopathic HES-mediated heart failure. Consequently, the patient was started on steroids and cyclophosphamide, along with medical treatment for left ventricular systolic dysfunction and anticoagulation for apical thrombi. Warfarin treatment was maintained until now, ensuring that the prothrombin time/international normalized ratio (PT/INR) was kept within the range of 2-3. Abdul Baqi et al.[3] described a 48-year-old man experiencing worsening dyspnea and low-grade fever over a year. Laboratory tests revealed an elevated white blood cell count with eosinophilia, and further investigations confirmed a diagnosis of HES. Transthoracic echocardiography (TTE) and cardiac MRI revealed biventricular thrombi. Consequently, he was prescribed oral steroids, hydroxyurea, imatinib mesylate, and oral anticoagulation. The patient responded well to the treatment, leading to the resolution of symptoms and the elimination of the biventricular thrombi. Similarly, in our case, oral anticoagulation with warfarin was initiated, targeting an INR of 2-3.[3] Kian-Guan Lee et al.[5] reported on a sixty-year-old man with progressive heart failure symptoms and eosinophilia, indicative of HES. Despite thorough assessments, no underlying cause was identified. TTE detected a large thrombus in the left ventricle, supporting the diagnosis of hypereosinophilic cardiac involvement. The patient began steroid therapy and experienced a positive clinical and hematological response, with his eosinophil count normalizing within a week. He also received warfarin to prevent cardioembolism. The symptoms of heart failure improved, and follow-up TTEs over the first and second months showed a reduction in the size of the thrombus.[5] Moreover, Ji-won Hwang et al.[23] reported a case of cerebral embolic infarction and intracardiac atypical linear-shaped thrombus in a 55-year-old man with idiopathic HES. The patient was treated with corticosteroids, hydroxyurea and warfarin. One week after starting anticoagulant therapy, a massive intracranial hemorrhage was detected on brain computed tomography. At that time, the INR was confirmed to be 2.18. After discontinuing the anticoagulant, the thrombotic linear intracardiac lesion completely resolved on follow-up TTE one month after corticosteroid and hydroxyurea treatment. In this case, an atypical-shaped thrombus accompanied by eosinophilia was entirely resolved with corticosteroid and hydroxyurea alone; however, warfarin was associated with a poor prognosis due to excessive bleeding.[23] To the best of our knowledge, evidence regarding the definitive duration of anticoagulation after improvement of cardiac thrombosis in patients with idiopathic HES remains limited.

HES may be complicated by cerebral thromboemboli, encephalopathy, peripheral neuropathy, or longitudinal and/or transverse sinus thrombosis.[10] Cerebrovascular disease (60%) was the most prevalent CNS involvement in HES, followed by border zone infarct (45%) and disturbed mental state (40%).[24] Although our patient experienced heart failure and a cardiac thrombus on a cardiac magnetic resonance imaging scan, the CNS involvement was a border zone infarct, not a typical embolic infarct. There is ongoing debate about the mechanism of HES-related border zone infarcts. The border zone regions of the deep and superficial middle cerebral artery perforators show signs of stroke. The border zone infarcts may result from localized thrombus development rather than thromboembolism from endomyocardial fibrosis, vascular endothelial toxic effects of eosinophilic cells, or a combination of these factors.[25] Another possibility is reversible vasculopathy, related to reversible posterior encephalopathy and caused by the neurotoxic effects of eosinophilia. These patients' border zones exhibit lower cortical blood flow than normal. Therefore, if an embolic stroke pattern is present, we should focus on anticoagulant treatment; if a border zone infarct pattern is evident, both anticoagulant therapy and immunomodulation should be considered. With 15% of eosinophils and bone marrow involvement in our case, hypereosinophilia, another common finding in individuals with cardiac involvement, was also present.

Conclusion

Cardiac involvement in HES is common and can be associated with unfavorable clinical outcomes and thromboembolic events. Border zone infarct is a frequent CNS complication in HES and may result from thromboembolism, vascular endothelial toxic effects of eosinophilic cells (similar to reversible posterior encephalopathy), or, in some cases, local thrombus formation rather than thromboembolic causes.

Informed Consent: Written informed consent was obtained from the patient.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – A.S., M.T.; Design – A.S., S.M.M.; Supervision – A.S., S.M.M.; Resource – A.S.; Data Collection and/or Processing – A.S., Z.P., M.A.; Analysis and/or Interpretation – S.M.M.; Writing – A.S.K., M.T., Z.P., M.A.M., N.S., S.M.M.

Use of AI for Writing Assistance: Artificial intelligence-assisted technologies were not used in this article.

Conflict of Interest: The authors have no conflicts of interest to declare.

Funding: The authors declared that this study received no financial support.

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