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CASE REPORT

Eosinophilia: Rare cause of arterial thrombosis and cardioembolic stroke in childhood

Sanjeev Kumar Sharma, Suman Kumar, Tulika Seth, Pravas Mishra, Manoranjan Mahapatra

Sanjeev Kumar Sharma, Suman Kumar, Tulika Seth, Pravas Mishra, Manoranjan Mahapatra, Department of Hematology, All India Institute of Medical Sciences, New Delhi 110029, India

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Correspondence to: Dr. Tulika Seth, Department of Hematology, All India Institute of Medical Sciences, New Delhi 110029, India. tuliseth@yahoo.com

Telephone: +91-98-68397236 Fax: +91-11-26593363

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Abstract

Eosinophilia has been reported as a very rare cause of stroke in children. The thrombotic event may be either due to cardiac damage induced by eosinophils and their granular protein, that is, the major basic protein, or the systemic hypercoagulable state induced by eosinophilia. We report here a case of eosinophilia whose initial presentation was recurrent strokes and cardiac and arterial thrombosis.

Key words: Eosinophilia; Cardioembolic stroke; Thrombosis; Low molecular weight heparin; Warfarin

Peer reviewers: Gergely Feher, MD, PhD, Department of Neurology, Medical School, University of Pecs, 2 Ret str., Pecs, H-7623 Baranya, Hungary; Moses S Elisaf, Professor of Medicine, Department of Internal Medicine, Medical School, University of Ioannina, 45110 Ioannina, Greece

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INTRODUCTION

There are diverse causes and risk factors for stroke in childhood. The most common arterial ischemic causes are congenital or acquired heart diseases and sickle cell disease. The other hematological causes associated with stroke include paroxysmal nocturnal hemoglobinuria, polycythemia vera, essential thrombocythemia and various hereditary hypercoagulable states. Eosinophilia is common in childhood but has rarely been reported as a cause of stroke in children. Eosinophilia can lead to cardiac and arterial thrombosis, leading to stroke, which can be the lone initial manifestation of eosinophilia.

CASE REPORT

An 11-year-old boy was admitted with recurrent right sided hemiparesis. He gave a history of right lower limb monoparesis 2 wk before, from which he had complete spontaneous resolution. There was no history of fever, chest pain, palpitations, breathlessness, diarrhea, skin rash, joint pains, bleeding diathesis or viral exanthema. There was no evidence suggestive of congenital or rheumatic heart disease. There was no history suggestive of allergic rhinitis or asthma. The family history was noncontributory. The clinical examination was unremarkable except for right hemiparesis with power of 2/5 in the right upper and lower limbs with right extensor plantar response. Hemography revealed hemoglobin of 12.2 g/dL, total leukocyte count 7.9 × 10^9/L, platelet count 156 × 10^9/ L, the differential count showed polymorphs 26%, lymphocytes 25%, monocytes 1% and eosinophils 48% [absolute eosinophilic count (AEC), 3792/mm^3]. Renal and liver function tests were normal. The contrast-enhanced computed tomography (CECT) of the brain

revealed left lenticular nucleus infarct. High-resolution CT and CECT of the chest were normal. Echocardiography revealed mildly dilated left ventricle, with 60% ejection fraction and multiple friable clots. Color Doppler ultrasound of bilateral lower limb vessels revealed mild occlusion of the right external iliac and right common femoral arteries. Doppler ultrasound of the bilateral carotid vessels was normal. Stool examination for ova and parasites was negative. Autoimmune markers for antineutrophil antibody, antineutrophil cytoplasmic antibody, rheumatoid factor, and anticardiolipin antibodies (immunoglobulin G and immunoglobulin M) were negative. The lipid profile and serum homocysteine levels were normal. Other laboratory parameters including erythrocyte sedimentation rate, high-sensitivity C-reactive protein and troponin were within normal limits. *Wuchereria* microfilariae were not seen on peripheral smeared blood films. Bone marrow examination revealed increased marrow cellularity with increased eosinophilic component. The endomyocardial biopsy was unremarkable and there was no evidence of granulomatosis or vasculitis. FIP1L1-PDGFRA and FIP1L1-PDGFRB gene rearrangements by nested reverse-transcriptase polymerase chain reaction were not detected. The patient was started on low-molecular-weight heparin (LMWH; enoxaparin 1 mg/kg twice daily) and warfarin 4 mg daily. LMWH was stopped after 5 d when international normalized ratio of 2.5 was achieved with warfarin. Hydroxyurea 500 mg daily was also started. The patient had complete clinical recovery within 10 d. Three months following stroke, the patient is clinically stable with AEC 600/mm^3 and is on warfarin and hydroxyurea, with normal echocardiography and color Doppler ultrasound of the lower limbs.

DISCUSSION

The common causes for eosinophilia are parasitic infestations, allergic diseases, drugs, neoplasia and connective tissue diseases[1]. Patients in whom the underlying cause of eosinophilia is not found and who have AEC > 1500/mm^2 for > 6 mo with organ dysfunction are labeled as idiopathic hypereosinophilic syndrome (HES). Eosinophilia has rarely been reported as a cause of stroke in children[2]. Stroke is the most devastating neurological consequence of eosinophilia in adults with an incidence in HES patients of around 17%[3,4]. Other than cardiac emboli and direct eosinophil toxicity, there is a hypercoagulable state in eosinophilia which can contribute to strokes in HES[5]. The characteristic features of stroke in eosinophilia are the occurrence of multiple strokes, in different vascular territories, at different times. The etiology may be due to the direct eosinophilic damage to the endocardium and myocardium or by the release of eosinophilic basic proteins which initiate endomyocardial necrosis. This makes the heart a potential source of these recurrent emboli[6]. Causes of thrombogenicity are multifactorial including the release of tissue factor from specific granules, inactivation of thrombomodulin by binding to the major basic protein, endothelial damage or by elevation of fibrinogen levels[5,7]. Anticoagulation and antiplatelet agents are used in the management of stroke but simultaneously lowering eosinophil count with hydroxyurea or steroids results in a better outcome[8]. Distinction of HES into myeloproliferative and lymphoproliferative variants helps with further characterization of the disease and has therapeutic and prognostic implications[9]. Our patient had involvement of the heart and major vessels, with thrombus induced by eosinophilia. Reduction of eosinophils with hydroxyurea and simultaneous anticoagulation led to the resolution of thrombus and complete clinical recovery of the patient.

The American Heart Association Stroke Council and the Council on Cardiovascular Disease in the Young does not include eosinophilia as a cause or risk factor for stroke in children[10]. This case highlights the fact that eosinophilia can be a risk factor for both hypercoagulable state and stroke in children and should be considered as a risk factor for stroke in childhood.

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