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Case Report

Hypereosinophilic Syndrome with COVID-19 Leading to Myocarditis and Stroke; A Case Report and Literature Review

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Abstract

A 63-year-old with no remarkable past medical history presented to the emergency room with left-side weakness. The initial lab was significant for eosinophilia and the nasal swab polymerase chain reaction (PCR) was positive for SARS-CoV-2. Brain magnetic resonance imaging (MRI) demonstrated multiple foci of infarction. Moreover, she started having chest pain accompanied by elevated troponin and new T-wave inversion in inferior and precordial leads. All infectious, autoimmune, and malignant causes were excluded. Cardiac MRI showed late gadolinium enhancement of the myocardium in the inferior and lateral walls. The patient was started on corticosteroids which resulted in improvement of her eosinophilia. Cardiac involvement in hypereosinophilia has been reported in 5% of patients. It can lead to severe arrhythmias and death. Steroid is the main treatment. However, newer medications have been reported to be effective.

Introduction

Hypereosinophilic syndrome (HES) is defined by persistently elevated eosinophils in blood and tissue with symptoms attributable to the elevated eosinophils regardless of the cause of elevated eosinophils [1]. It is nine times more common in men compared to women. Most patients are at the age of 20-50 years old [2]. The syndrome is categorized into 3 main subtypes. Primary, also known as neoplastic, is defined by clonal expansion of eosinophils in the setting of stem cell and myeloid malignancy. In the secondary or reactive subtype, the expansion is due to cytokines produced by other cells. This subtype is commonly associated with parasitic infections, new medications, vaccinations, solid tumors, and T-cell lymphoma. However, in the idiopathic subtype despite extensive workups, no specific cause can be identified [3]. It can affect various organs including the central nervous system, lungs, skin, gastrointestinal, and heart. Cardiac involvement was reported in 5% of patients. However, it is the major cause of mortality in these patients [1]. We are presenting an interesting case of hypereosinophilic syndrome with stroke and myocarditis after an asymptomatic COVID-19 infection.

Case Presentation

A 63-year-old right-handed female with no significant past medical history presented to the emergency room with the chief complaint of left-side weakness. Brain MRI demonstrated multiple scattered foci of infarction involving the cerebral hemispheres and the right cerebellar hemisphere. Lab was remarkable for white blood cells of 13.6 K/uL with 28.4 % eosinophil and platelet of 70 K/uL. Her nasal swab PCR was positive for SARS-CoV-2. While the patient was hospitalized she started having severe left-side chest pain. Her vital signs were stable. Her High-sensitivity

troponin was significantly elevated and uptrending. An electrocardiogram revealed new T-wave inversion in inferior and precordial leads (Figure 1).

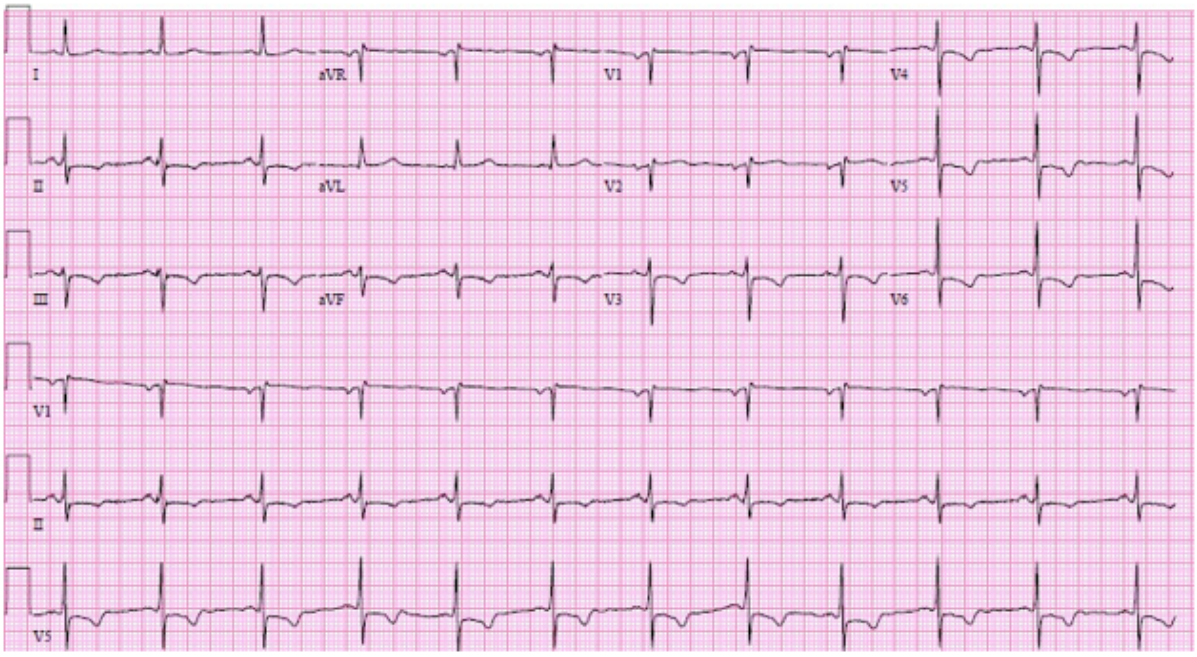

Figure 1: New T wave inversion in inferior and precordial leads

Regarding her thrombocytopenia and recent ischemic stroke, heparin drip was not started and cardiac catheterization was not performed. Transthoracic echo demonstrated an ejection fraction (EF) of 60% with grade two diastolic dysfunction without any wall motion abnormalities. Serum antibodies against other common viral causes of myocarditis were negative. Bone marrow biopsy revealed marked eosinophilia (more than 20%) and no evidence of malignancy. She had an extensive work-up including testing for autoimmune, infectious causes, and malignant processes, which were all negative. A cardiac MRI demonstrated late myocardial enhancement in the inferior and lateral walls (Figure 2).

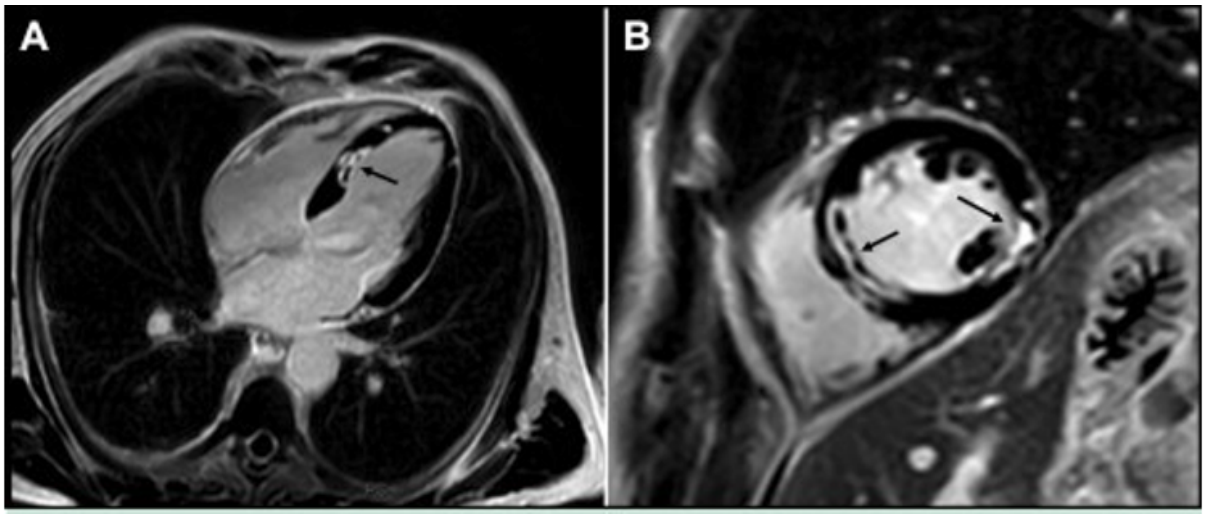

Figure 2: Cardiac MRI A) Four chamber view and B) Short axis view demonstrating late gadolinium enhancement in the septal and inferolateral wall.

Fluorescence in situ hybridization (FISH) and flow cytometry were negative for platelet-derived growth factor receptor alpha (PDGFRA) rearrangement or T-cell abnormalities. B12 tryptase was normal and IgE was elevated. The patient was started on 1 mg/kg steroids which led to improvement of her eosinophil and platelet count. She was discharged on a tapering dose of steroid, Coreg, atorvastatin, prednisone, and aspirin.

Discussion

Cardiac involvement in hypereosinophilia is the major cause of death as it can lead to malignant arrhythmias or acute heart failure [4]. It is unpredictable and might not happen in some patients with prolonged eosinophilia. There is no correlation between the severity of eosinophilia and cardiac involvement. However, patients with FIP1L1 PDGFR are more prone to have cardiac involvement [5]. Cardiac involvement is divided into 3 stages: acute necrotic phase, intermediate phase, and fibrotic stage. Sometimes these stages can overlap. Although most patients are asymptomatic in the acute necrotic phase, some patients might have evidence of microemboli such as splinter hemorrhage [6]. The duration can vary from one day to three months [7]. The intermediate phase is characterized by damaged endocardium and thrombus formation. The pathology underlying intracardiac thrombosis is believed to involve hypothiocyanous acid formation through eosinophilic peroxidase, leading to the induction of tissue factor expression in cells and impaired thrombomodulin function caused by eosinophils cationic protein. In our case, given the multiple foci of stroke, the source could be microemboli originating from the damaged heart. The fibrotic stage is associated with fibroinflammatory remodeling of cardiac tissue and valves. Evaluation may demonstrate cardiomegaly, restrictive cardiomyopathy, mitral or tricuspid regurgitation, and T-wave inversion [8].

Transthoracic echo is usually the first modality to use. Restrictive patterns, valvar abnormalities, and intracardiac thrombosis are the most common findings [9]. However, dilated cardiomyopathy has been reported in a few cases [10]. Cardiac MRI has higher specificity and sensitivity for detecting thrombosis. Moreover, MRI is very helpful in the diagnosis and treatment follow-up of cardiac involvement as it can characterize tissue features [11]. Late gadolinium enhancement due to endomyocardial fibrosis and inflammation is a typical MRI finding in these patients. Despite the accuracy of cardiac MRI, endomyocardial biopsy remains the gold standard method for diagnosis. However, it is reserved for patients with uncertain diagnoses [12] Given the typical presentation and MRI finding in our patient, no endocardial biopsy was performed.

The target of the treatment is decreasing the number of eosinophils. Corticosteroid is the cornerstone treatment of cardiac involvement in hypereosinophilic syndrome. Imatinib in patients with FIP1L1–PDGFRA rearrangement can be advantageous and can be used as the first agent [13]. In the case of corticosteroid resistance HES, immunomodulatory, immunosuppressives, and biologics including Mepolizumab have been used [14].

Conclusion

Our knowledge regarding COVID-19 complications is limited. This case emphasized the complications of hypereosinophilic syndrome secondary to COVID-19.

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