A CASE OF ALLERGIC BRONCHOPULMONARY ASPERGILLOSIS WITH INFILTRATING EOSINOPHILIC MYOCARDITIS

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INTRODUCTION: Eosinophilic myocarditis (EM) is associated with myriad clinical presentations. With both chronic and acute forms, there exists a wide range of cardiac dysfunction and etiologies, including hyper-eosinophilia, hypersensitivity reaction, and parasitic or fungal infections. Chronic EM is associated with congestive heart failure, a lower mortality rate when compared to acute EM, and clinical response to corticosteroids. Here we present a case of a 57-year-old male with suspected long standing undiagnosed allergic bronchopulmonary aspergillosis, presenting as asthma, leading to infiltrative eosinophilic myocarditis.

CASE PRESENTATION: Our patient was diagnosed with asthma at age 45 and he had pansinusitis with recurrent polypectomies. He presented with 10 days of dyspnea, chest tightness, palpitations, and orthopnea. He had no history of cardiac disease. CXR on admission showed bilateral patchy opacities and pleural effusions. CT of the chest demonstrated upper lobe bronchiectasis and pulmonary edema. His EKG was significant for a right axis deviation and new Q waves in V1-V4, consistent with myocardial scarring. An ejection fraction of 30-34% with global hypokinesis was seen on transthoracic echocardiogram. Based on the clinical presentation, he was thought to have allergic bronchopulmonary aspergillosis. When his previous laboratory was reviewed, he had significant eosinophilia that had been persistent over time. Since he had persistent eosinophilia and new cardiomyopathy with possible myocarditis, the diagnosis of eosinophilic myocarditis was suspected. He had no evidence of coronary artery disease on left heart catheterization and cardiac MRI showed scarring of the septal and inferoseptal walls, and bilateral atria, consistent with chronic EM. Prednisone therapy was begun and his eosinophil count returned to normal in 5 days and improved to discharge with prednisone therapy. Two weeks later, he had an endomyocardial biopsy (EMB), which showed fibrosis, but no eosinophilic infiltration. He was asymptomatic at his last clinic appointment.

DISCUSSION: EM can result in irreversible and fatal myocardial dysfunction. For diagnosis, EMB is considered the gold standard, for which serum eosinophil concentration, echocardiography, and cardiac MRI may assist. Though biopsy in this case showed fibrosis without eosinophilic infiltration, this can be attributed to previous corticosteroid treatment, in addition to variation with tissue sampling.

CONCLUSIONS: Our patient had a misdiagnosis of asthma, associated with recurrent nasal polyps, persistent eosinophilia, positive aspergillus IgE and bronchiectasis, consistent with allergic bronchopulmonary aspergillosis. In this case, corticosteroid administration resolved his respiratory and cardiac symptoms. To date, there is no gold standard for treatment; however, regimens typically include corticosteroids, cytotoxic agents, and immunosuppressive therapy.


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