

Eosinophilic Heart Disease: A Case Report and Review of Literature (Poster).

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Eosinophilic Heart Disease: A Case Report and Review of Literature

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INTRODUCTION

Hypereosinophilic syndrome (HES) is a multisystem disorder diagnosed by the presence of hypereosinophilia (HE) defined as an absolute eosinophil count (AEC) $>1.5 \times 10^9/L$ (or >1500 cells/microL) and multi organ involvement attributable to HE. Myocardial dysfunction is a frequent finding and is a major cause of morbidity and mortality in HES. The diagnosis of hypereosinophilic heart disease (EHD) is made in the setting of hypereosinophilia and characteristic clinical features and imaging findings of damage to the heart. We describe those clinical features in this case of HES with EHD with review of literature.

CASE

A 63 year old active smoker male with no known past medical history was transferred to our hospital for hypotension, abnormal EKG and elevated troponin. The patient described nonspecific symptoms of cough, runny nose, malaise, muscle aches, nausea, diarrhea and low grade fever for 2-3 weeks. He also noted a skin rash which had resolved at the time of admission. He denied air travel outside the United States.

His examination revealed diminished lung sounds bilaterally, normal heart sounds with a soft 1/6 intensity holosystolic murmur without marked radiation of the murmur. There was mild lower extremity edema present.

LAB DATA AND IMAGING

Laboratory data revealed hyponatremia, normal creatinine and an elevated troponin I value of 2.89, WBC count of 15000, Hb of 9 gram/dl and platelet count of 40,000. His eosinophil count was 4800 (UNITS), LDH 719, 32% Eos.

CT contrast chest abdomen and pelvis revealed no PE or dissection, marked lymphadenopathy and small pleural effusions only.

His EKG was sinus rhythm with diffuse T wave inversions.

Transthoracic echocardiogram revealed hyperdynamic left ventricular (LV) systolic function and apical thickening concerning for an LV thrombus (Figure 1a & 1b).

MRI of the heart confirmed the diagnosis of apical thrombus along with fibrotic material seen in both LV and RV apex (Figure 2a & 2b).

IMAGES

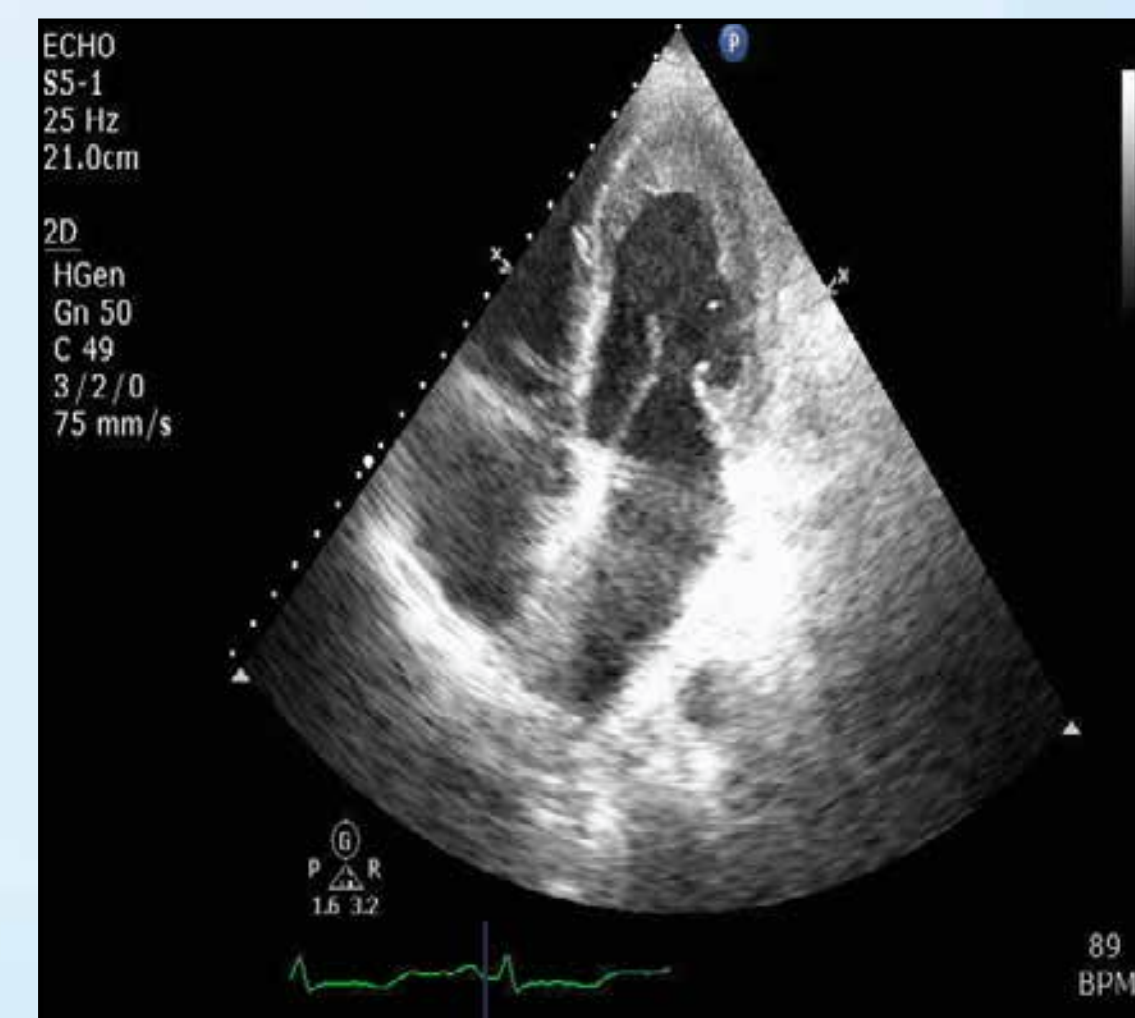


Figure 1:

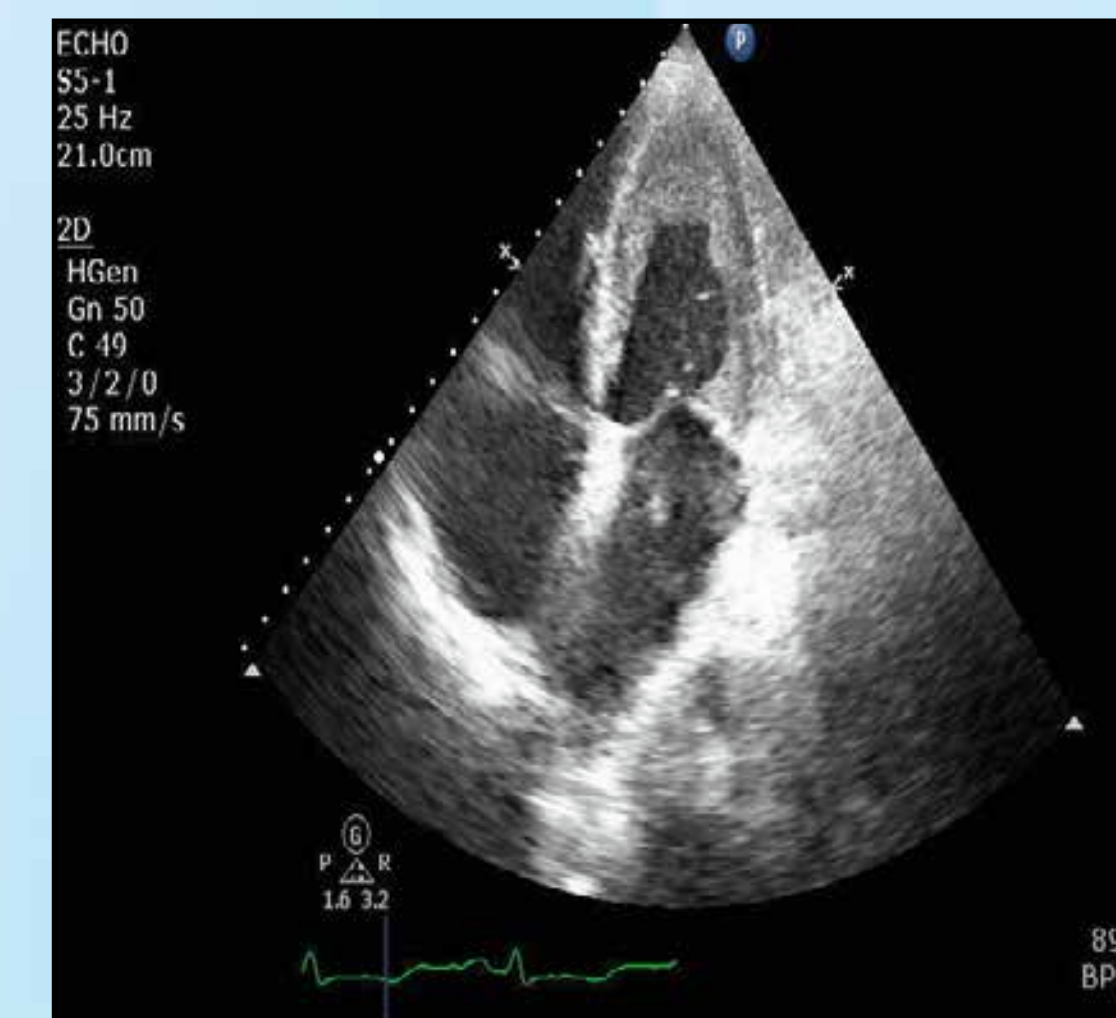


Figure 2:

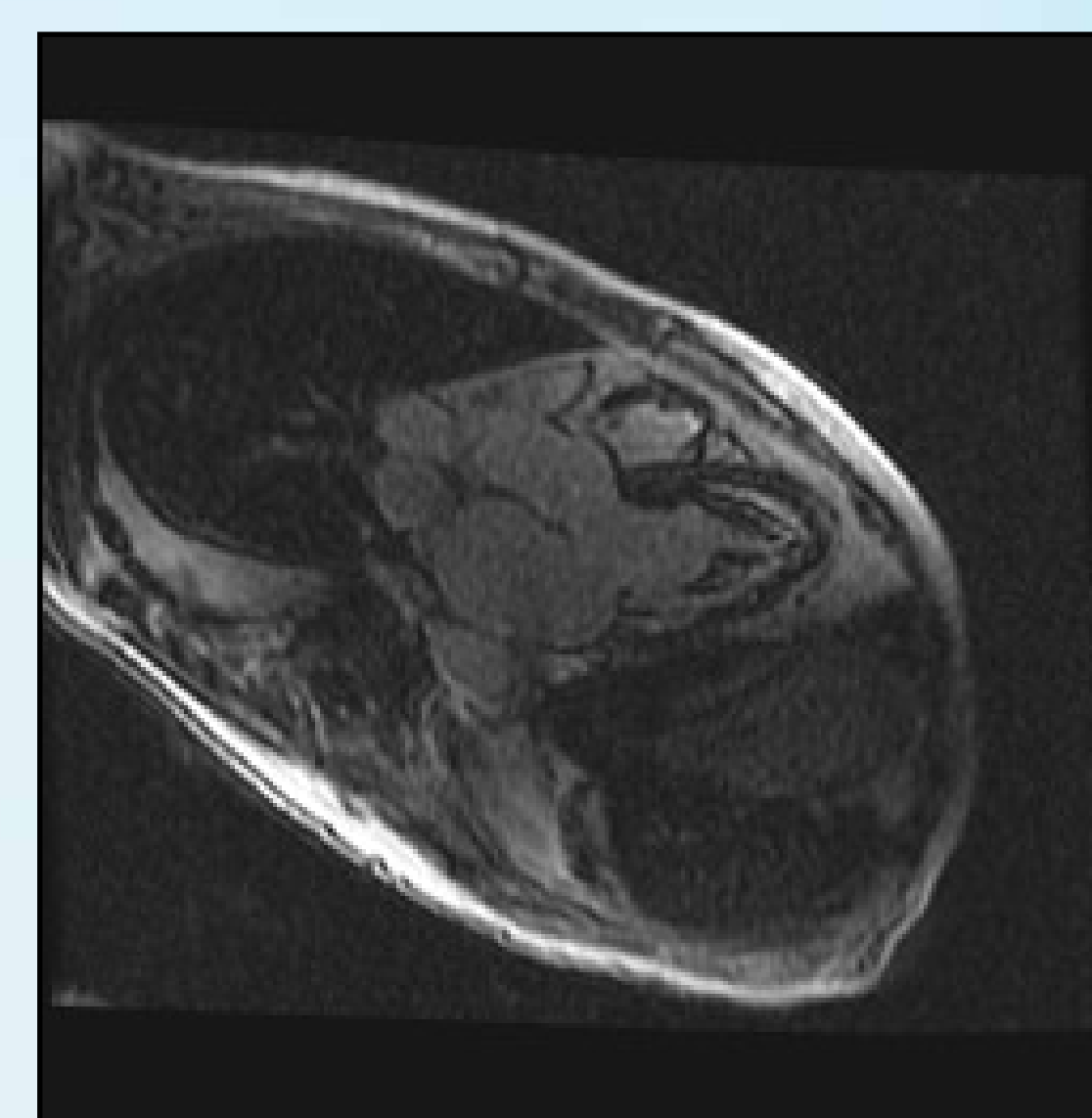
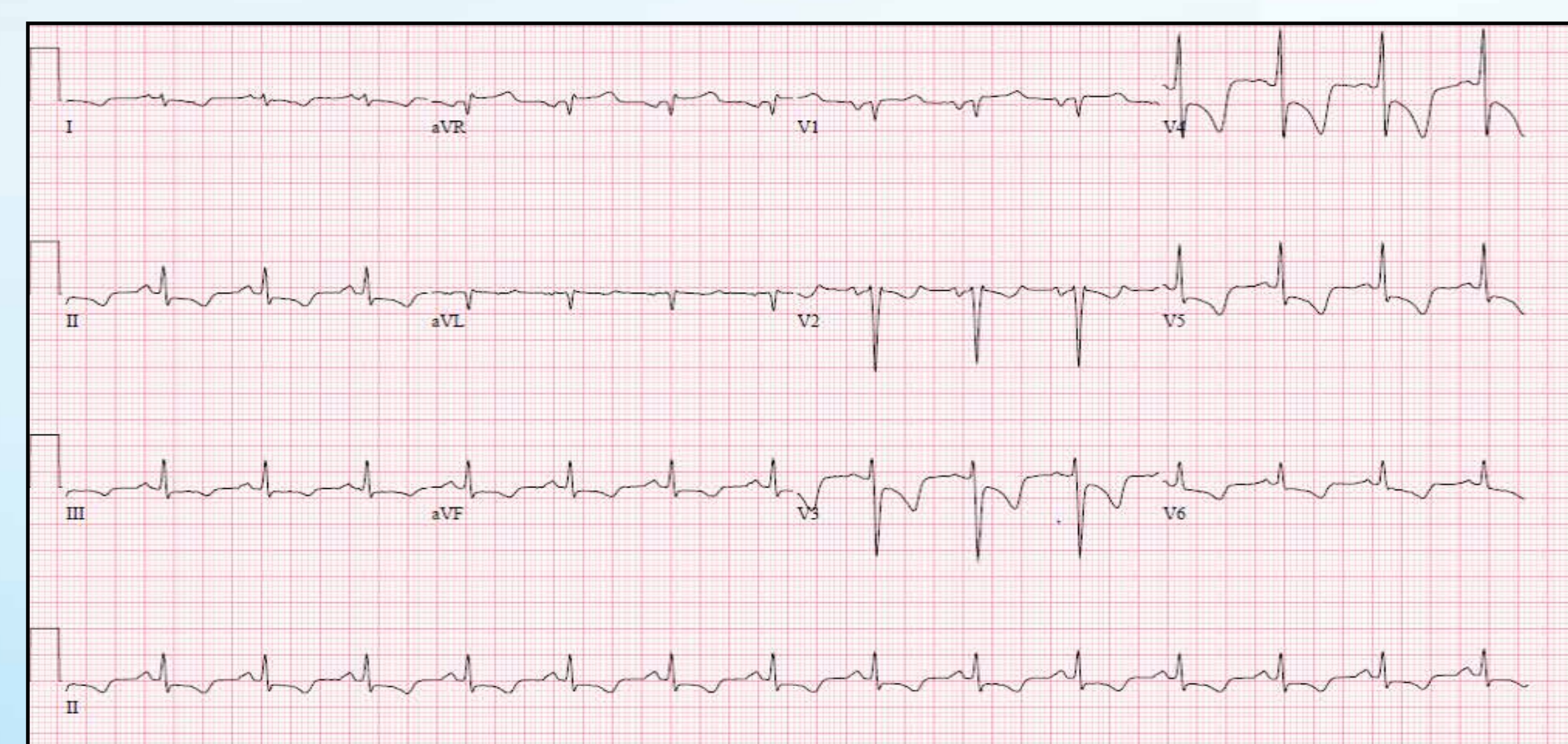


Figure 3:



Figure 4:



His axillary lymph node biopsy showed angio-immunoblastic T cell lymphoma. He was readmitted again for VT and chemotherapy induced pancytopenia. Required high doses of steroids while inpatient.

His Eosinophil count on discharge was 0% compared to 32% maximum on first admission.

DISCUSSION

HES are a heterogeneous group of disorders. Typically symptoms are very nonspecific as in our case. A rash, GI disturbances, cough and dyspnea are the common symptoms. Pathophysiology of EHD includes three overlapping stages: 1) an acute necrosis, 2) thrombosis and 3) fibrosis. The clinical presentation of EHD is variable and may range from an asymptomatic myocardial involvement to heart failure syndrome, conduction abnormality, fatal arrhythmia, sudden cardiac death or an acute coronary like syndrome. It is important to note that EHD can be seen in any cause of eosinophilia or hypereosinophilia such as drugs and hypersensitivity reactions, parasitic or protozoal infections, malignancies, idiopathic hypereosinophilic syndromes or vasculitis syndromes.

Endomyocardial biopsy is the reference standard test for the definite diagnosis of EHD. Eosinophilia is present in the majority of the patients. Electrocardiographic changes are common but nonspecific and may include criteria for left ventricular hypertrophy, arrhythmias or AV block.

CONCLUSIONS

Echocardiography plays a vital role in suspecting and identifying EHD. Cardiac MRI has higher sensitivity and specificity than any other imaging modalities. It is important to find the etiology of hypereosinophilia and treat it accordingly. Offending drug needs to be discontinued.

Malignancies and Vasculitis treated accordingly. Steroids have remained the mainstay of therapy in acute settings

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