A 34-year-old man with no past medical history presented to our emergency department with facial droop and dysarthria. He had a 1-day history of dyspnea and nonproductive cough, which he attributed to a viral illness. Examination was notable only for the neurological abnormalities in the chief complaint, which resolved during the emergency department evaluation. A 12-lead ECG showed sinus tachycardia, a prolonged corrected QT interval (530 ms), and T-wave inversion suggestive of ischemia (Figure 1). MRI of the brain was negative for acute stroke. Laboratory values were notable for a hemoglobin of 8.8 g/dL, white blood cell count of 75,400 cells/mL with absolute eosinophilia to 22,620 cells/mL, and a platelet count of 31,000/mL. Troponin T was elevated to 0.40 μg/mL (normal <0.03) with a normal creatine kinase of 22 U/L. An urgent transthoracic echocardiogram demonstrated a mildly reduced ejection fraction (45%) with apical akinesis. A large echogenic mass with a mobile component consistent with thrombus obliterated the apices of the left and right ventricles (online-only supplementary Movie I; Figure 2). The clinical findings were a transient ischemic attack, leukocytosis with eosinophilia, thrombocytopenia, anemia, and eosinophilic heart disease.

The patient was treated with leukapheresis, allopurinol, hydroxyurea, and intravenous methylprednisolone sodium succinate for 48 hours to reduce the white blood cell count. Heparin was initiated for the ventricular thrombus, as well as carvedilol for left ventricular dysfunction. Bone marrow biopsy revealed a myeloproliferative disorder (CHIC-2 deletion subtype) with secondary eosinophilia, and imatinib mesylate (Gleevec) was started. Hematologic derangements improved before the patient was discharged from the hospital, and warfarin was initiated for the apical thrombus. At 5 weeks after discharge, there was no evidence of ventricular dysfunction, apical thrombus, or eosinophilic infiltration (supplementary Movie II; Figure 3). There has been interval resolution of his peripheral eosinophilia and myeloproliferative disorder.

Eosinophilic infiltration of the heart was originally described in 1936 by Löffler1 in the postmortem examination of 2 patients seen in a 20-year period with afebrile leukocytosis and eosinophilia, progressive right-sided heart failure with hepatosplenomegaly, and ascites. Cardiac autopsy evaluation showed a layering of fibrosis that obliterated the ventricles but spared the valves.1 Eosinophilic heart disease is now recognized as a manifestation of the hypereosinophilic syndromes, in which up to 50% of patients have evidence of cardiac involvement.2,3 Typical cardiac findings include endocardial fibrosis with extensive mural thrombus occupying the apices of both ventricles (hence the term “obliterative cardiomyopathy”). Thrombus can also extend toward the base to involve the valvular apparatus (typically the posterior leaflet of the mitral valve), with resulting incompetence. Advanced forms include progressive myocardial damage, conduction system disease, and refractory heart failure.

The present case highlights the classic features of eosinophilic heart disease. The echo-dense mass was seen, contiguous with the adjacent myocardium, with a central area of echolucency. This is the echocardiographic appearance of large thrombus burden with central liquefaction necrosis.4 The thrombus overlies an area of normal wall motion, thus differentiating this entity from thrombus formation in association with dilated cardiomyopathy or ischemic ventricular dysfunction. The apical akinesis in our patient may have been a result of focal eosinophilic myocarditis or coronary artery thromboembolism. It is thought that the activated eosinophil that is admixed with thrombus secretes major basic protein and is responsible for the toxic damage to the heart.5 Regression of the intracavitary mass has been reported with treatment of anticoagulation plus interferon3 or with imatinib mesylate (Gleevec) as the sole agent.6 Two-dimensional echocardiography is the primary method for diagnosis of eosinophilic heart disease and should be performed in all patients with peripheral blood eosinophilia.

Disclosures

None.

References


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The online-only Data Supplement, consisting of Movies I and II, is available with this article at http://circ.ahajournals.org/cgi/content/full/115/23/e614/DC1.
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**Figure 1.** Twelve-lead ECG on admission. Note T-wave inversions in the inferior and precordial leads.
Figure 2. Still-frame echocardiographic image in diastole (A) and systole (B). Hyperechoic mass fills the left ventricular apex, which is consistent with thrombus. The apex is akinetic, yet there is still thrombus in areas of normokinesia.

Figure 3. Still-frame echocardiographic image in diastole (A) and systole (B). There has been interval resolution of the ventricular apical mass and thrombus and normalization of ejection fraction.
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