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Eosinophilic Myocarditis Presenting as ST-segment Elevation Myocardial Infarction Diagnosed with Cardiac Magnetic Resonance Imaging

To the Editor:

Acute ST-segment elevation myocardial infarction is a medical emergency requiring prompt recognition of the syndrome and initiation of reperfusion therapy. The usual pathophysiologic mechanism of transmural myocardial injury is acute thrombotic occlusion of an epicardial coronary artery. Other conditions can masquerade as ST-elevation myocardial infarction with different mechanisms of myocardial injury. We report a case of eosinophilic myocarditis manifesting as acute ST-segment elevation myocardial infarction in the setting of chronic idiopathic hypereosinophilia diagnosed using cardiac magnetic resonance imaging.

CASE REPORT

A 60-year-old gentleman with a history of chronic obstructive pulmonary disease and hypertension was referred to the Emergency Department by his hematologist for further work-up of new-onset arthralgias, leukocytosis (23,000 cells/μL) with eosinophilia (41%), and elevated erythrocyte sedimentation rate (51 mm/h). He had been referred to a hematologist after a recent hospitalization with splenic infarcts, leukocytosis, and eosinophilia of unclear etiology. Further review of his records demonstrated eosinophilia as far back as 18 months before admission. During his previous admission, an extensive work-up including anticardiolipin antibody, beta-2 glycoprotein, antinuclear antibody, JAK2 mutation, FIP1L-PDGfRA, lupus anticoagulant, tryptase and bcr/abl, anti scl-70, antineutrophil cytoplasmic antibodies, anticyclic citrullinated peptide antibody, rheumatoid factor, flow cytometry, karyotyping, stool ova and parasites, and strongyloides, was negative. A bone marrow biopsy demonstrated significant eosinophilia, suggesting a hypereosinophilic state.

He was admitted to our hospital for further evaluation and management. On day 1 of his hospitalization, he developed sudden-onset retrosternal chest pain while lying in bed. Electrocardiogram showed 3-mm ST-segment elevation in leads II, III, and AVF (**Figure 1**). Troponin I was elevated to 0.536 ng/mL. He was administered aspirin, clopidogrel, metoprolol, and atorvastatin, and started on an intravenous heparin infusion. A bedside transthoracic echocardiogram showed hypokinesis of the inferolateral and anterolateral segments, which were new compared with a previous echocardiogram (**Figure 2**). Urgent coronary angiography demonstrated mild nonobstructive coronary disease (**Figure 3**). He subsequently underwent cardiac magnetic resonance imaging using a Philips 3T scanner (Philips Healthcare, Andover, MA) with gadolinium contrast that was administered (0.2 mmol/kg) with images taken after a delay of 10 minutes. This demonstrated multiple noncontiguous focal areas of late gadolinium enhancement, including transmural and epicardial areas, with corresponding wall motion abnormalities, suggestive of infiltrative myocarditis (**Figure 4**).

DISCUSSION

Hypereosinophilia is a syndrome whose hallmark feature is overproduction of eosinophils, defined as having an absolute eosinophil count of $>1.5 \times 10^9$/L (1500/μL) on 2 separate occasions.[1] This is often noted incidentally on routine blood work, and the cause is not immediately obvious. A hypereosinophilic syndrome is diagnosed when there is evidence of end-organ damage from eosinophilic tissue infiltration with resultant oxidative and enzymatic injury.[2,3] Myocardial involvement is common, occurring in upwards of 60% of patients with hypereosinophilic syndrome, and is now a major cause of morbidity and mortality if not treated appropriately.[4-6] The association between eosinophilia and cardiac injury was first described by Löffler in 1936,[7] initially as *fibroplastic endocarditis* or *Loeffler's endocarditis*. He proposed that eosinophilic infiltration of the endocardium with resultant fibrosis was the mechanism of injury resulting in constrictive physiology and severe heart failure.

Myocardial injury as a result of eosinophilic infiltration involves recruitment of eosinophils into the myocardial tissue with toxic degranulation.[3] The current understanding of eosinophilic myocarditis describes a 3-stage process of myocardial injury.[4,5,8-10] The first stage is the acute necrotic phase, which involves myocardial infiltration with eosinophils

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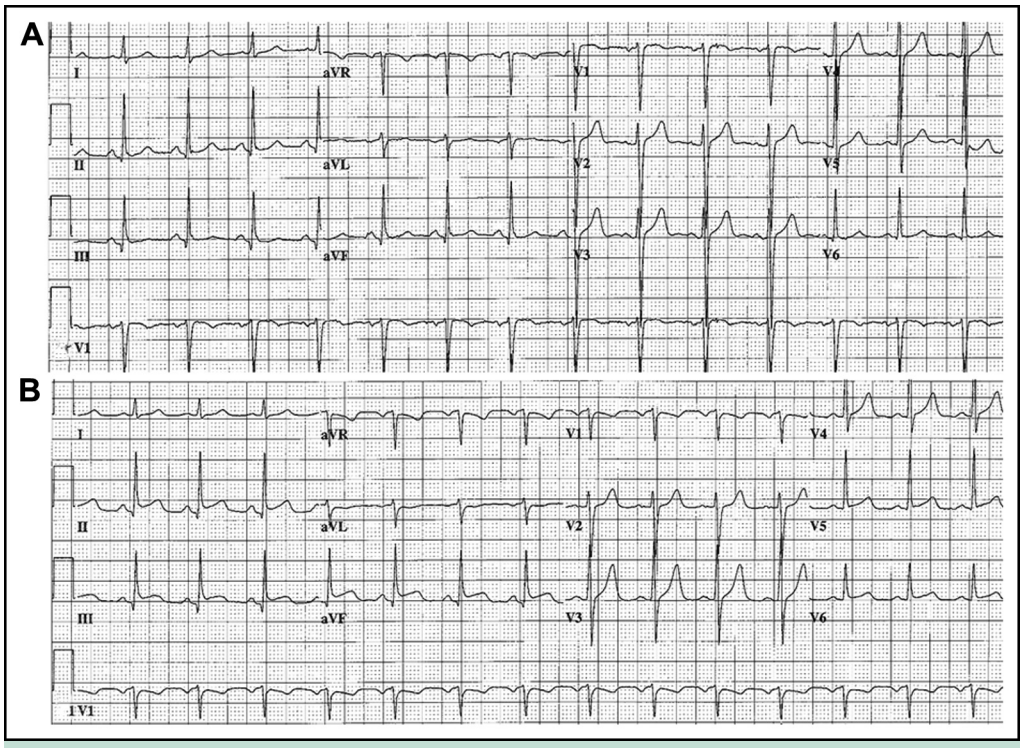

Figure 1 (**A**) Baseline 12-lead electrocardiogram (ECG) on admission. (**B**) ECG during chest pain showing inferior ST-segment elevation.

and toxic degranulation, resulting in myocardial injury. This stage is typically subclinical and patients are often asymptomatic. The second stage is the thrombotic phase, involving thrombus formation on the endocardial surface. Symptoms often first appear during this stage due to thromboembolic events. This was the case with our patient, who had initially presented with splenic infarcts. The final stage is a fibrotic phase, where scar replaces the injured myocardium, resulting in endomyocardial fibrosis, which may lead to a restrictive cardiomyopathy and congestive heart failure. Combinations of fibrosis and inflammation can be seen across a continuum in the latter 2 stages.

There are few reported cases of ST-segment elevation myocardial infarction as the presenting feature of patients with eosinophilic myocarditis.[11-13] Endomyocardial biopsy (EMB) has historically been the gold standard for diagnosis of cardiac eosinophilic infiltration. However, due to the noncontiguous and patchy nature of eosinophilic infiltration, EMB may miss active areas of infiltration, resulting in a false negative and delaying

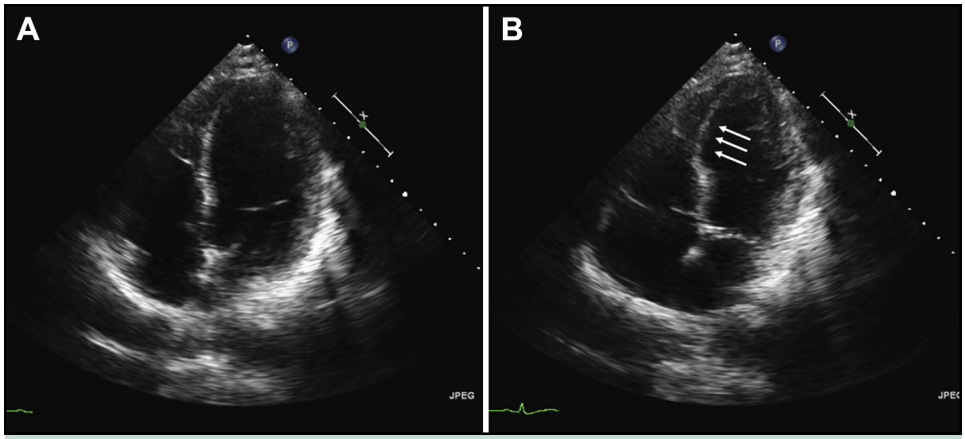

Figure 2 Transthoracic echocardiogram apical 4-chamber views in diastole (**A**) and systole (**B**) showing hypokinesis of the mid-inferoseptum (arrows).