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Case Report

Acute Necrotizing Eosinophilic Myocarditis Presenting as ST-Elevation Myocardial Infarction: A Case Report

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ABSTRACT

Eosinophilic myocarditis (EM) is a rare disease. The spectrum of clinical presentation is wide and it is often fatal if left untreated. We report a case of eosinophilic myocarditis manifesting as ST-elevation myocardial infarction (STEMI) with cardiogenic shock treated successfully with steroids. This diagnosis should always be considered in patients with an acute coronary syndrome and normal coronary arteries, especially in the presence of a history of allergies, asthma or peripheral eosinophilia.

RÉSUMÉ

La myocardite à éosinophiles (MÉ) est une maladie rare. Le spectre de tableau clinique est large et il est souvent fatal s'il demeure sans traitement. Nous rapportons un cas de myocardite à éosinophiles se manifestant comme un infarctus du myocarde (IM) avec sus-décalage du segment ST et un choc cardiogène qui a été traitée avec succès à l'aide de stéroïdes. Ce diagnostic devrait toujours être considéré chez les patients ayant un syndrome coronarien aigu et des artères coronaires normales, spécialement en présence d'antécédents d'allergies, d'asthme ou d'éosinophilie périphérique.

A case of eosinophilic myocarditis manifesting as ST-elevation myocardial infarction (STEMI) with cardiogenic shock, successfully treated with steroids is presented. Etiology, clinical characteristics, diagnosis, pathology, and management are discussed.

Case Presentation

A 65-year-old man was admitted with a 1-month history of progressive dyspnea and intermittent retrosternal discomfort. His medical history was significant for hypertension, diabetes mellitus and asthma.

On admission his blood pressure was 90/68 mm Hg, pulse was 101 beats per minute, and temperature 36.6°C. The physical examination was remarkable for jugular distention, a fourth heart sound and peripheral edema. The electrocardiogram showed prolonged PR interval, right bundle branch block, left anterior hemi-

block, and ST elevation in leads V₁ to V₃ (Fig. 1). Blood analysis revealed troponin I of 12.9 ng/mL (upper limit 0.05) and total creatine kinase of 459 U/L (38-174 U/L), with a MB fraction of 77 U/L. The patient was admitted with diagnosis of STEMI.

He was started on aspirin, heparin, and inotropic drugs. An echocardiogram showed anteroapical hypokinesia with moderately depressed left ventricular systolic function (ejection fraction, 30%-35%) and a coronary angiogram revealed normal coronary arteries. A repeat electrocardiogram showed regression of ST elevation, with decline in serial creatine kinase measurements. A pulmonary artery catheter was placed, showing a cardiogenic pattern. His blood count showed leukocytosis (24,200 cells/uL) with 45% eosinophilia.

An eosinophilic myocarditis (EM) was suspected and the patient underwent cardiac magnetic resonance imaging (MRI), which showed late gadolinium septal hyperenhancement (LGE) with a midwall nonischemic pattern (Fig. 2, A). His endomyocardial biopsy (EMB) showed infiltration of the myocardial interstitium by collections of eosinophils associated with areas of myocyte destruction, consistent with EM (Fig. 2, B). There was no evidence of giant cells, granulomas, or vasculitis.

The patient was started on steroid therapy, with 3 intravenous boluses of methylprednisolone (0.5 g per day) followed by

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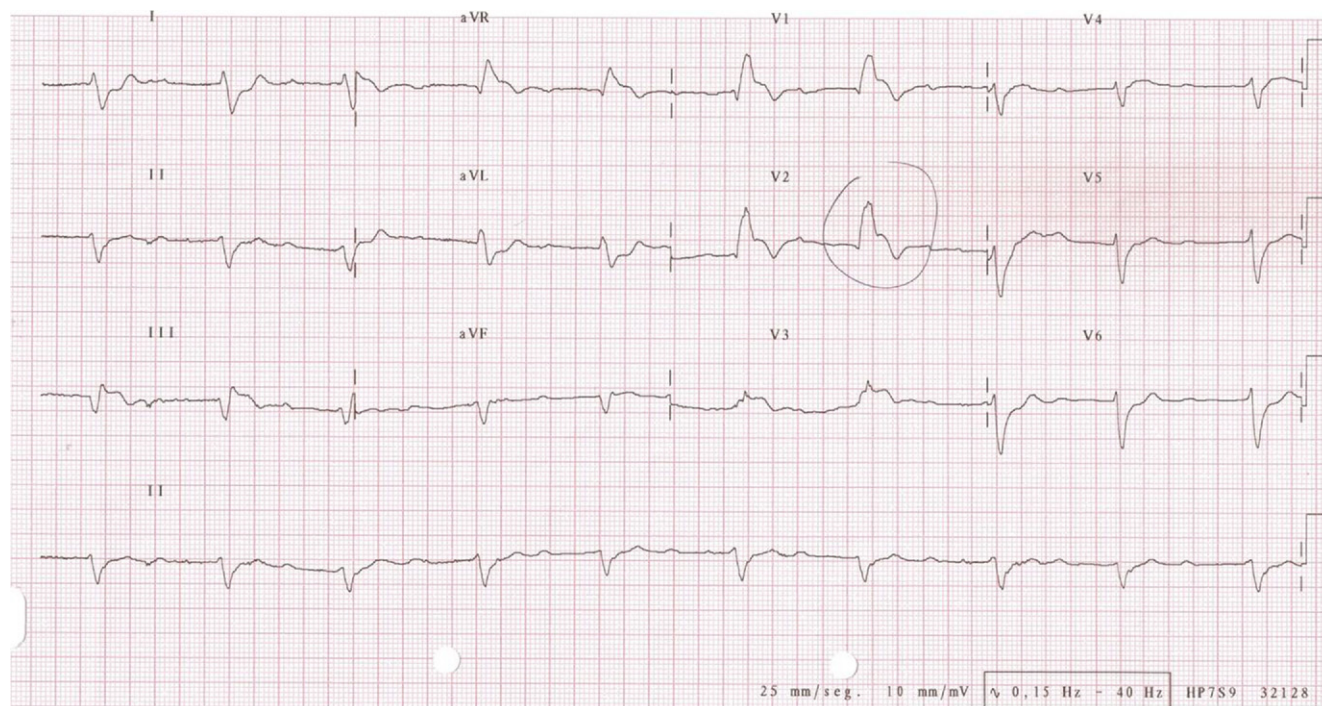


Figure 1. Electrocardiogram showing marked prolongation of the PR interval, right bundle branch block with left anterior hemiblock and ST elevation in leads V₁ to V₃, DIII, and aVR.

1 mg/kg per day oral prednisone at a tapering dose. He started to do better, with successful weaning of inotropic agents. The peripheral blood eosinophilia resolved quickly after the first dose of methylprednisolone. He was continued on loop diuretics, angiotensin converting enzyme inhibitors and spironolactone. Additional laboratory analyses were ordered to study the cause of his eosinophilia. Tests for HIV, antinuclear and anti-neutrophil cytoplasmic antibodies were negative. The stool studies and serologies for *Toxocara*, *Strongyloides*, and *Trichinella* were also negative. The marrow exam was negative for malignancy and showed a marked increase in the number of eosinophils.

The patient continued to improve and was discharged in good condition. He reported continued use of prednisone 5 mg daily at the 6-month follow-up visit. A repeat echocardiogram revealed significant improvement of left ventricular ejection fraction (50%).

Discussion

EM is a rare form of myocarditis characterized pathologically by diffuse or focal myocardial inflammation with eosinophilic infiltration, often in association with peripheral blood eosinophilia.¹

Different etiologies have been described. The most common cause reported is hypersensitivity myocarditis due to medications and numerous drugs have been implicated. Other etiologies include hypereosinophilic syndrome, Löeffler's endocarditis, tropical endomyocardial fibrosis, vasculitis such as Churg-Strauss, and some malignancies.

The spectrum of presentations is wide. Patients may present with fever, skin rash, sinus tachycardia, chest pain, shortness of breath, symptoms of heart failure, conduction delays, and

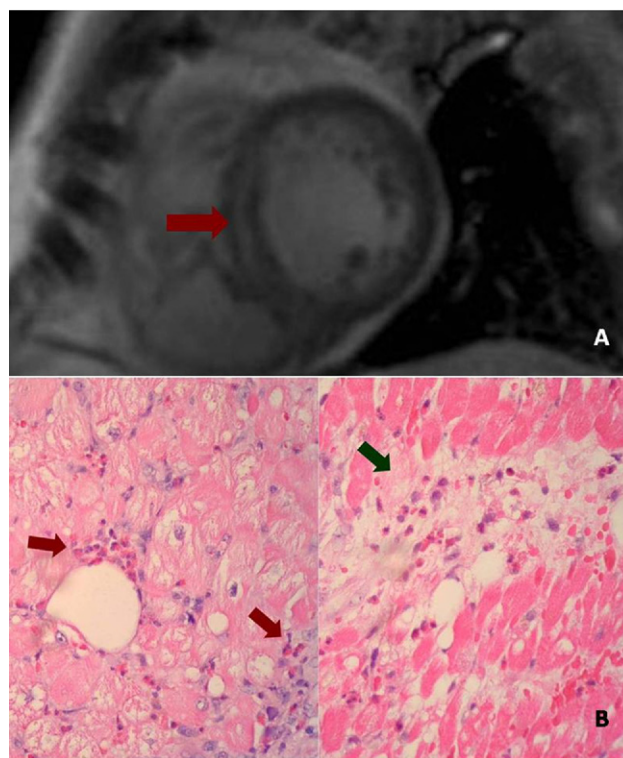


Figure 2. (A) Cardiac magnetic resonance image showing late gadolinium septal hyperenhancement with midwall distribution (red arrow) compatible with fibrosis. (B) Endomyocardial biopsy (magnification 100 $\times$, hematoxylin and eosin stain). Interstitial and perivascular infiltrates of eosinophils are seen (red arrows), in association with areas of loss of myocytes (green arrow).

ST-T abnormalities. Cardiac arrhythmias and sudden death are possible presentations as well.² The mechanism of transient ST elevation in this case is unknown, but the absence of angiographic coronary stenosis suggests the possibility of vasospasm. Some cases of coronary artery spasm associated with eosinophilia have been described and it is likely that eosinophilic basic proteins or vasoactive cytokines directly stimulate constriction of nearby vascular smooth muscle.³

If EM is clinically suspected an EMB should be considered, however biopsy is not very sensitive (50%) because the infiltrates are often focal. The presence of peripheral blood eosinophilia, although not present in all cases, supports the diagnosis. A midwall or subepicardial LGE pattern on cardiac MRI is also supportive of a nonischemic etiology.⁴

Treatment should include withdrawal of the possible offending agents and standard treatment for heart failure. Many patients exhibit dramatic response to steroid therapy, including those with a history of severe ventricular dysfunction and aborted sudden death.⁵ In 3 cases presented by Wong et al., all the patients demonstrated complete recovery and normalization of cardiac contractility after treatment with high-dose oral steroids with gradual tapering.³ The role of immunosuppressive therapy is controversial and immunosuppressive drugs may prevent recurrences.

Our patient presented several clinical features suggestive of an acute coronary syndrome (ACS). Marked peripheral blood eosinophilia and negative coronary angiogram aroused the sus-

picion of EM, which was supported by EMB and cardiac MRI. The diagnosis of idiopathic hypereosinophilic syndrome was postulated.

EM should be considered in the differential diagnosis of acute coronary syndrome in patients with normal coronary arteries, especially in the presence of a history of allergies, asthma or peripheral eosinophilia.

Disclosures

The authors have no conflicts of interest to disclose.

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