Acute myocardial necrosis in newly diagnosed hypereosinophilic syndrome – a case report of early detection and management

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Introduction: Hypereosinophilic syndromes (HES) are defined as marked peripheral blood eosinophilia (> 1.5 × 10^9/L) associated with evidence of eosinophil-induced organ damage when secondary causes of hypereosinophilia are excluded[1]. Cardiac involvement occurs in approximately 50% of the patients and has three stages: acute necrosis, thrombosis, and fibrosis. If left untreated, progression of the stages can be expected.

Case report: 57-year-old female patient, with no known chronic illnesses was admitted to the hospital with abdominal pain and severe eosinophilia. During clinical evaluation, a computed tomography (CT) scan revealed thickening of the distal third of the esophagus, the pyloric part of the stomach, and the duodenum. An incidental finding was a mild pericardial effusion. Electrocardiography (ECG) demonstrated newly diagnosed paroxysmal atrial fibrillation (EHRA class I). This prompted cardiac evaluation and sampling of NT-proBNP and cardiac troponins, both significantly elevated. Mandatory echocardiography and coronary angiography were performed. Echocardiography showed infiltrated, hypoechogenic and hypocontractile lateral and inferoseptal wall (**Figure 1**) with preserved systolic function but reduced global longitudinal strain (**Figure 2),** and impaired diastolic function with hemodynamically nonsignificant pericardial effusion. Coronary angiography ruled out coronary stenosis while cardiac MRI confirmed the echocardiographic findings. A subsequent hematological assessment was conducted which excluded lymphoproliferative disease. Additionally, multiple endoscopic biopsies showed no eosinophilic infiltration of the digestive tract. After serologic exclusion of parasitic cause for eosinophilia, treatment with systemic corticosteroids, guideline-directed medical therapy, and low-molecular-weight heparin was initiated. Over the following days, normalization of eosinophil blood count and a significant decline of cardiac enzymes was recorded that allowed discharge of the patient with multidisciplinary follow-up.

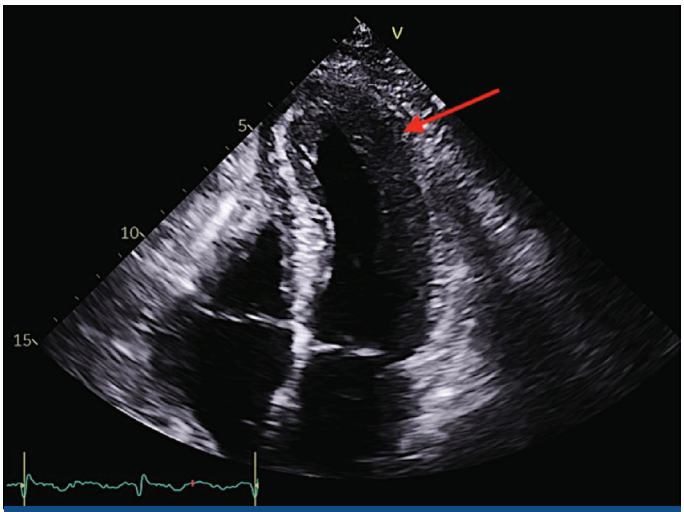

FIGURE 1. Red arrow pointing at the infiltrated, hypoechogenic and hypocontractile lateral wall.

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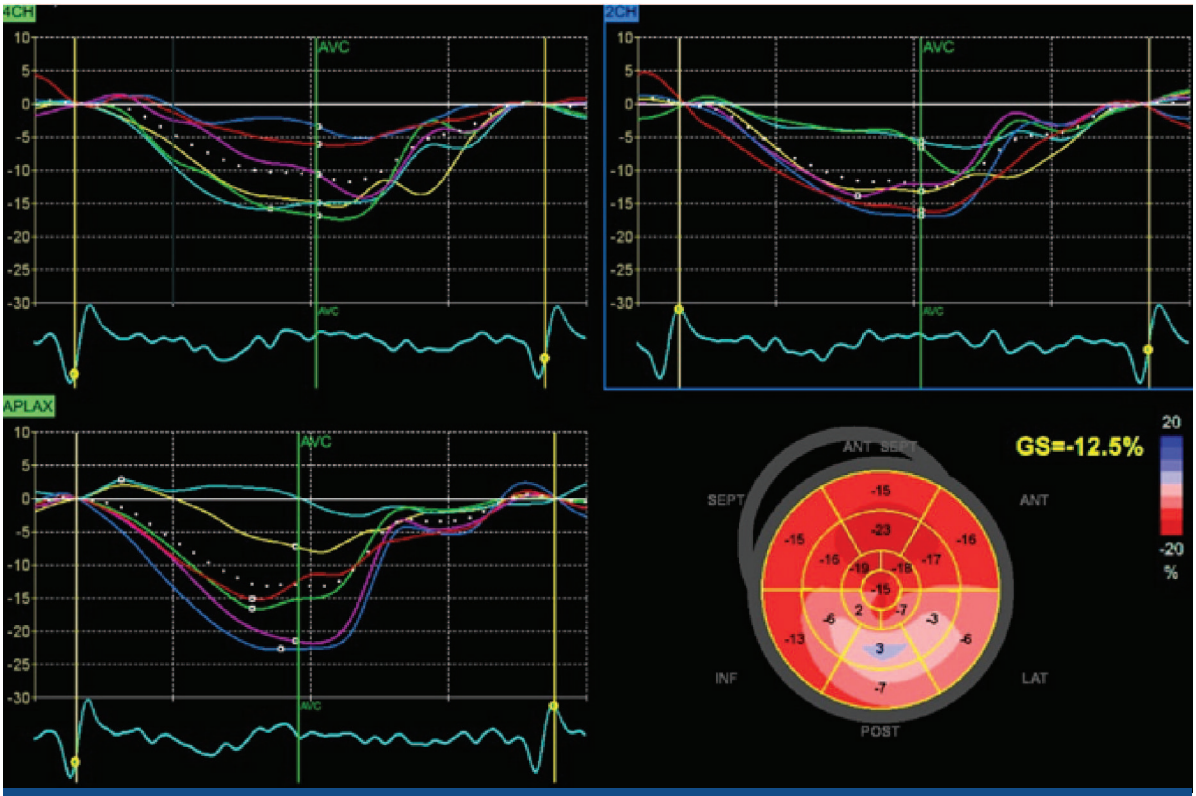

FIGURE 2. Speckle tracking of the left ventricle, showing reduced global longitudinal strain -12.5%, with hypocontractile lateral segments.

Conclusion: Hypereosinophilic syndrome causes acute cardiac involvement within 5 weeks and leads to a severe increase in cardiac enzymes[2]. Clinical symptoms may be absent at this stage, but detection is possible with imaging and pathohistological methods. Early recognition is possible, and therapy should aim to reduce the eosinophil count, in our case with systemic corticosteroids, and prevention of thrombotic complications and heart failure.

LITERATURE

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