A curious case of cardiac fat deposition in a patient with hypereosinophilic syndrome

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A 60-year-old woman with a history of eosinophilia, asthma, allergic rhinitis, chronic obstructive pulmonary disease, hypertension, coronary artery disease, and embolic stroke, presented with hemorrhagic stroke and persistent hypoxemia. The patient was noted to have eosinophilia and elevated immunoglobulin E (IgE) in peripheral blood with a positive radioallergosorbent test for anti-Aspergillus IgE antibodies. The sputum culture contained Aspergillus, whereas bronchoalveolar lavage was hypocellular with negative polymerase chain reaction for Aspergillus antigen. Computed tomography (CT) of the chest revealed enlarged lymph nodes, as well as patchy upper lobe ground glass opacity with centrilobular nodules throughout the lungs. Based on initial findings, the patient was diagnosed with allergic bronchopulmonary aspergillosis and started on steroid therapy with resolution of eosinophilia and improvement in lung infiltration.

Given elevated troponins on admission, a cardiac workup was simultaneously performed. Transthoracic echocardiography revealed global left ventricular (LV) hypokinesis with ejection fraction (LVEF) of 45%–50%. Single-photon emission CT showed no evidence of ischemia. Cardiovascular magnetic resonance (CMR), after 2 months of steroid therapy, showed thinning and hypokinesis of the inferolateral walls, prominent LV trabeculae, and moderately decreased systolic function (LVEF, 35%). Given the atypical dense appearance of the trabeculated LV myocardium on long-axis cine imaging, additional fat water cine imaging was requested by the supervising physician. Fat water cine imaging demonstrated prominent fatty infiltration of the subendocardium and trabeculae (Figure 1A, B). Late gadolinium enhancement (LGE) imaging demonstrated fibrosis involving the subendocardium and trabeculae in the inferolateral and anterior walls, as well as apical segments (Supplementary material, *Figure S1*). Fat water LGE images were utilized to demonstrate both fatty infiltration and fibrosis in the LV (Figure 1C). In addition, the patient exhibited elevated native T1 signal (1129 ms; site specific normal <1084 ms), and mildly elevated T2 signal (56 ms, site specific normal <52 ms) (Supplementary material, *Figure S1*). Given the clinical scenario, findings were concerning for hypereosinophilic syndrome (HES) with Loeffler's endocarditis (LE). In light of this discovery, the initial hypoxemia and pulmonary parenchymal opacities may have been signs of HES lung involvement.

HES is a group of rare diseases characterized by unexplained peripheral blood hypereosinophilia with evidence of end-organ damage [1, 2]. Cardiac involvement is present in about 20%–60% of cases [1, 2]. LE is defined as inflammation of both myocardium and endocardium with eosinophilic infiltration [1, 2]. LE has heterogeneous clinical manifestations ranging from an asymptomatic form to restrictive cardiomyopathy (RCM) [1]. Patients present with heart failure, intracardiac thrombosis, arrhythmias, myocardial ischemia, and pericarditis [1]. Three stages of the disease may overlap [1]. The necrotic stage with eosinophilic infiltration and myocardial inflammation is followed by a thrombotic stage with common distal embolization, and a fibrotic stage [1, 3].

A comprehensive CMR with tissue characterization can aid in diagnosis and guide response to therapy in HES with LE [4]. CMR often reveals T2 signal elevation in the acute phase and patchy or diffuse subendocardial LGE in a noncoronary distribution in the

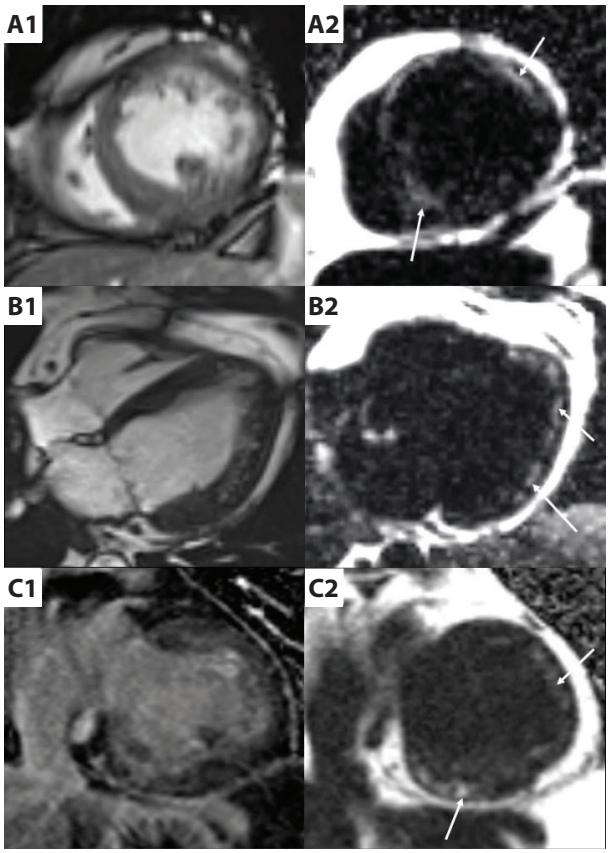

Figure 1. Midmyocardial and subendocardial fatty infiltration (the arrows) in the left ventricle on steady-state free-precession cine and fat water short axis and horizontal long-axis cine images (**A1** and **B1**: SSFP images; **A2** and **B2**: fat images). Fatty infiltration on fat water late gadolinium enhancement images best seen in the outflow tract view (**C**: phase sensitive inversion recovery water image in **C1** and fat image in **C2**)

chronic phase [1]. The case is unique in the presence of myocardial fat deposition, previously described only once, and may represent an atypical feature of the disease [5]. The pathophysiologic mechanism of fat deposition is unknown in HES [5]. The images confirming fatty infiltration are generally not included in standard myocarditis or cardiomyopathy protocols in CMR laboratories. Therefore, fatty infiltration would not have been detected were it not for the alertness of the supervising physician.

Supplementary material

Supplementary material is available at https://journals.viamedica.pl/kardiologia_polska.

Article information

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