Loeffler’s endocarditis

Dionne J.W. van Kessel¹, Anastasia Jerzewski¹, J. Joost Kardux², and Jesse Habets²,³*

¹Department of Cardiology, Gelre Hospitals, Apeldoorn, the Netherlands; ²Department of Radiology, Gelre Hospitals, Apeldoorn, The Netherlands; and ³Department of Radiology, University Medical Center Utrecht, Utrecht, The Netherlands

* Corresponding author. Heidelberglaan 100, PO BOX 85500, E01.132, 3508 GA Utrecht, The Netherlands. Tel: +31 887556689; Fax: +31 302581098.
E-mail: j.habets@umcutrecht.nl

A 73-year-old woman was admitted on the ICU with acute respiratory failure. Acute dyspnoea started several hours before presentation. Physical examination revealed a murmured, dyspnoeic patient with expiratory wheezing and peripheral oedema. Laboratory test showed very high BNP levels (2588 ng/L), decreased renal function (GFR 43 mL/min), serum creatinine (109 μmol), and a slightly increased C-reactive protein (44 mg/L). Her cardiac enzyme levels were within normal limits. Transthoracic echocardiography was performed and showed a reduced left ventricular systolic function, with apical akinesia and suspicion of an apical left ventricular thrombus (see Supplementary data online, Video S1 and Panel A).

SonoVue confirmed this suspicion (see Supplementary data online, Video S2 and Panel B).

To unravel the cause of the cardiomyopathy, cardiac magnetic resonance imaging (CMRI) was performed. We performed a standard cardiac MRI examination including standard left ventricle steady-state-free precession left ventricle cine imaging, first-pass contrast-enhanced rest perfusion, and delayed enhancement imaging after intravenous administration of gadolinium.

CMRI revealed a restrictive cardiomyopathy with a left ventricular ejection fraction of 40% (see Supplementary data online, Video S3). Rest perfusion images demonstrated a global subendocardial perfusion defect (Panel D—left arrow, and see Supplementary data online, Videos S4 and S5). Delayed enhancement imaging demonstrated global subendocardial uptake of gadolinium (Panels E and F, arrows). CMRI confirmed the presence of a left apical thrombus (Panels E and F). The CMRI findings nicely illustrate the typically findings seen in Loeffler’s endocarditis. The subendocardial delayed enhancement is caused by fibrosis of the endocard based on stacking of eosinophils. The fibrosis results in a restrictive cardiomyopathy with impaired left ventricular function and apical thrombus formation. In retrospect, an abnormal eosinophil count (2300/μL) was reported in her blood test since 2013. Additional CT images of the thorax and abdomen confirmed the CMRI findings (Panel C, arrows) and revealed no secondary manifestations of hypereosinophilic syndrome.

This case illustrates that CMRI helps to differentiate from other causes of restrictive cardiomyopathy because of the classic MRI findings of Loeffler’s endocarditis (diffuse global endocardial hypoperfusion on first-pass contrast-enhanced perfusion, diffuse subendocardial delayed enhancement, and thrombus formation). Furthermore, other common causes of restrictive cardiomyopathy often result in diffuse irregular hyperenhancement in non-coronary distributions (i.e. cardiac amyloidosis and sarcoidosis). Another case of restrictive cardiomyopathy, siderotic cardiomyopathy, can easily be differentiated from Loeffler’s endocarditis based on typical significant reduction in T₂* values in patients with siderotic cardiomyopathy.

Supplementary data are available at European Heart Journal — Cardiovascular Imaging online.

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