# Emerging nuclear medicine modalities to improve diagnostic accuracy in myocarditis

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Nuclear medicine modalities are not included in the routine diagnostic work-up of clinically suspected myocarditis due to limited evidence.[1-3] A 35-year-old woman with a history of a recent respiratory infection was admitted to the hospital with dyspnea, palpitations, increased abdominal girth. Her electrocardiogram showed atrial flutter (with ventricular rate, 140 bpm) and left bundle branch block. Echocardiography showed globally decreased contractility of both ventricles with left ventricular (LV) ejection fraction of 28% and tricuspid annular plane systolic excursion of 5 mm; severe tricuspid regurgitation, mild pericardial, pleural, and abdominal effusions, and 2 hyperechogenic thrombi fixed to the LV apex. Invasive coronary angiography showed no changes. Laboratory investigations showed the following abnormalities: a N-terminal pro-B-type natriuretic peptide concentration of 5785 pg/ml (reference range <125 pg/ml); a troponin I concentration of 4.213 ng/ml (reference range <0.05 ng/ml); a D-dimer concentration of 5597 ng/ml. Cardiac magnetic resonance (CMR) findings confirmed a depression of the biventricular systolic function and a nondilated LV. Notably, no myocardial edema was observed but subepicardial and mid-wall (nonischemic distribution) diffused areas of late gadolinium enhancement (LGE) in the anterior, lateral, and postero-lateral walls, interventricular septum, and apex were observed (FIGURE 1A and 1B). The patient was discharged in a stable general condition after 6 weeks of hospitalization. She was prescribed standard guideline-recommended treatment for heart failure as well as warfarin and restriction of physical activity for 3 to 6 months.

A follow-up visit after 3 months showed: clinical improvement; normal resting electrocardiogram; normalization of left ventricular ejection fraction (54%) and persistent right ventricular systolic dysfunction (tricuspid annular plane systolic excursion of 5 mm); persistent LGE areas on CMR. The patient was included in the ongoing, prospective clinical trial STREAM (ClinicalTrials.gov identifier: NCT04 085 718),[4] and referred for resting single-photon emission computed tomography (to assess possible myocardial perfusion defects) and 18F-2-fluoro-2-deoxy-D-glucose positron emission tomography (FDG-PET) (performed within 2 days). To suppress physiological glucose uptake in the myocardium, 2 days of a low-carbohydrate-high-fat diet, followed by prolonged fasting, and intravenous heparin (50 IU per kilogram) just before FDG-PET imaging were administered. FDG-PET revealed extensive areas of high FDG uptake in the apex, interventricular septum, lateral and postero-lateral LV walls (FIGURE 1C). High FDG uptake corresponded with the resting single-photon emission computed tomography perfusion defect after a 99mTc-MIBI injection (total extent of perfusion defect was 47%), as well as with LGE at CMR (Supplementary material, *Figure S1*), suggesting active myocardial inflammation. Based on these results, the patient was referred for a right ventricular endomyocardial biopsy which revealed virus-negative, chronic-active myocarditis and myocardial fibrosis; other diseases with cardiac involvement were excluded. Immunosuppressive therapy for inflammatory cardiomyopathy was not started because of the breast cancer accidentally detected by FDG-PET. The standard

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**Received:** September 7, 2020.
**Revision accepted:** October 8, 2020.
**Published online:** October 15, 2020.
Kardiol Pol. 2020; 78 (12): 1297-1298
doi:10.33963/KP.15647

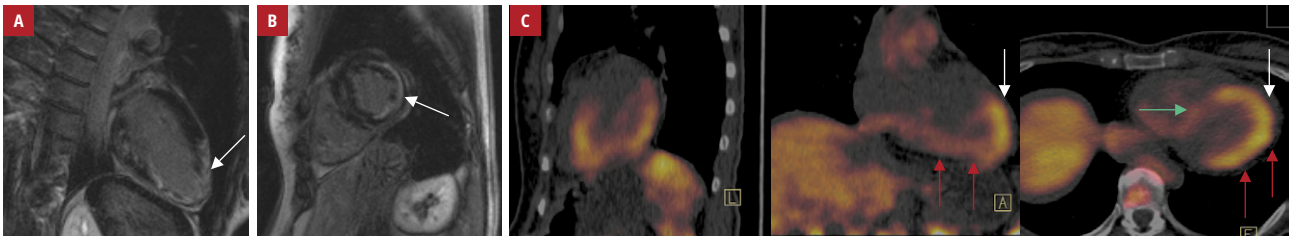


**FIGURE 1** **A**, **B** – cardiac magnetic resonance images with late enhancement techniques; **A** – the long-axis 2-chamber view demonstrating subepicardial and mid-wall late gadolinium enhancement in the left ventricular wall (arrow); **B** – the short-axis demonstrating diffuse areas of late gadolinium enhancement representing bands of increased signal running in the short axis plane (arrow); **C** – fusion positron emission tomography images (sagittal, coronal, and axial presentation, left, middle, and right, respectively) of myocardial 18F-2-fluoro-2-deoxy-D-glucose uptake showing the area of high preferential glucose uptake in the apex (white arrows), lateral and postero-lateral walls (red arrows). High 18F-2-fluoro-2-deoxy-D-glucose uptake area corresponds with the decreased single-photon emission computed tomography perfusion area (Supplementary material, *Figure S1*). Green arrow shows low septal 18F-2-fluoro-2-deoxy-D-glucose uptake, corresponding with normal single-photon emission computed tomography perfusion.

treatment for heart failure and scheduled follow-up were continued, and the patient was referred to an oncologist.

In our case, baseline and follow-up CMR showed LGE areas, but in keeping with the present state of knowledge, failed to show active inflammation, confirming that it has limited value in the diagnosis of chronic-active myocarditis.[1-3] We would like to highlight the potential role of the application of a new noninvasive diagnostic method, FDG-PET, in order to facilitate an appropriate selection of patients with low-intermediate probability of ongoing myocarditis to endomyocardial biopsy.[5] According to the European Society of Cardiology recommendations, endomyocardial biopsy is the gold standard for diagnosis of myocarditis,[1] which confirmed the suspicion made with FDG-PET.

## SUPPLEMENTARY MATERIAL

Supplementary material is available at www.mp.pl/kardiologiapolska.

## ARTICLE INFORMATION

**CONFLICT OF INTEREST** None declared.



**HOW TO CITE** Tymińska A, Ozierański K, Caforio ALP, et al. Emerging nuclear medicine modalities to improve diagnostic accuracy in myocarditis. Kardiol Pol. 2020; 78: 1297-1298. doi:10.33963/KP.15647

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