Cardiac magnetic resonance in myocarditis

To the editor We read with interest the article by Tymińska et al[1] published in the previous issue of *Polish Archives of Internal Medicine* (*Pol Arch Intern Med*). The authors should be congratulated on this comprehensive review. In an elegant way, they presented the etiology, pathogenesis, clinical presentation, diagnostic process, and treatment of myocarditis. However, there is an issue that needs to be raised. The authors wrote that "Gadolinium accumulates in the extracellular space where it increases the T1 signal, allowing detection of edema in myocardial tissue." While both early enhancement (a sign of hyperemia and capillary leak) and late enhancement (a sign of necrosis and fibrosis) require administration of a gadolinium-based contrast agent, the detection of edema on T2-weighted images does not. T2-weigthed imaging as well as its quantitative counterpart, T2 mapping, are performed prior to intravenous administration of the contrast agent. It should be underlined that the presence of myocardial edema in the absence of late gadolinium enhancement represents reversible myocardial injury.[2] Conversely, increased signal on postcontrast T1-weighted late gadolinium enhancement sequences demonstrates irreversible processes.[3,4] Proper use of the capabilities of cardiac magnetic resonance (CMR) imaging enables the diagnosis of clinically suspected myocarditis with high sensitivity and specificity.[5] Emphasis should be put on the fact that CMR is a noninvasive assessment modality as opposed to the gold standard diagnostic method—endomyocardial biopsy. Cardiac magnetic resonance is widely used in clinical practice for differential diagnosis in patients with suspected myocarditis.[5] It is strongly recommended (European Society of Cardiology guidelines) for patients with myocardial infarction with nonobstructive coronary arteries. We believe that the new emerging techniques, including T1 and T2 mapping, together with extracellular volume measurements will increase the reliability of CMR in the diagnosis of myocarditis, allowing to obviate endomyocardial biopsy in the majority of clinically stable patients.

ARTICLE INFORMATION

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Authors' reply We would like to kindly thank Śpiewak and colleagues for their thorough analysis of our review[1] and their valuable commentary regarding the diagnostic utility of cardiac magnetic resonance (CMR) in myocarditis. Regardless of the etiology, the inflammatory cascade of myocarditis leads to pathological conditions in the myocardium, such as edema, hyperemic blood flow, necrosis, and eventual scar formation, which can be visualized by different CMR techniques.[2,3] The authors of the letter emphasized that myocardial edema, which is associated with myocyte swelling and fluid accumulation in the interstitial space, can be detected on T2-weighted sequences as increased signal intensity of the myocardium and does not require the administration of a gadolinium-based contrast agent.[3] On the other hand, assessment of other "indicators" of inflammation is possible after injection of gadolinium contrast that freely distributes and accumulates

into the extracellular space, shortening T1 values and leading to increased signal in affected myocardial areas on T1-weighted images.[2,3] Hyperemia, which refers to dilatation of the coronary microvasculature, is reflected by increased regional uptake of the gadolinium contrast agent by abnormal myocardium during the first minutes after injection (early gadolinium enhancement). Necrosis (at an acute stage) and fibrosis (at a chronic stage) are signs of irreversible injury and can be visualized using the late gadolinium enhancement (LGE) technique (≥10 min after contrast agent injection).[2,3] As emphasized by Śpiewak and colleagues, myocardial edema in the absence of necrosis or fibrosis represents reversible myocardial injury. It should be noted, however, that LGE does not necessarily imply irreversible myocardial injury, as it represents the extracellular space, which can be expanded also because of tissue swelling due to myocardial edema; therefore, LGE alone cannot reliably distinguish between recent and remote myocarditis.[3]

The advent of parametric T1 and T2 mapping is believed to increase the diagnostic accuracy of CMR in myocarditis by overcoming some of the limitations of standard tissue characterization sequences, such as the need for remote, normal myocardium, allowing the identification of diffuse pathologic processes. The combination of T1 and T2 mapping has also been shown to increase the accuracy of detection of acute, subacute, and chronic myocarditis, owing to its sensitivity to intracellular and extracellular free water content.[3] However, due to the lack of reference T1 and T2 values in myocarditis, further studies are required to implement the use of parametric mapping in clinical practice.[4]

Cardiac magnetic resonance has been established as the gold standard noninvasive method of the assessment of myocarditis in both acute and chronic settings and should be performed in standard clinical practice. Nevertheless, we would like to emphasize that CMR cannot replace endomyocardial biopsy (EMB) in the diagnosis of myocarditis in selected patients. The diagnostic accuracy of CMR varies according to clinical presentation, being higher for infarct-like presentation and lower for heart failure and arrhythmic presentation; moreover, CMR does not provide information on the underlying etiology of myocardial inflammation, thus limiting the potential treatment options (ie, starting immunosuppression in patients with immune-mediated virus-negative EMB-proven myocarditis).[4,5] Therefore, CMR and EMB are not alternative diagnostic tools in clinically suspected myocarditis but have complementary and incremental values.

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