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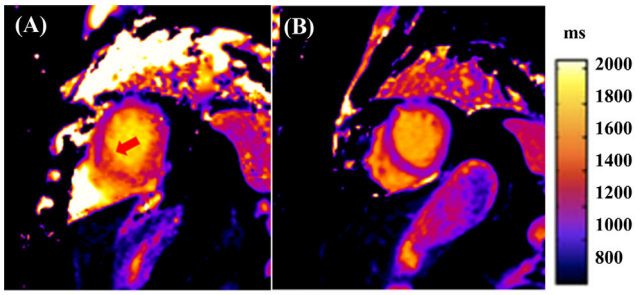
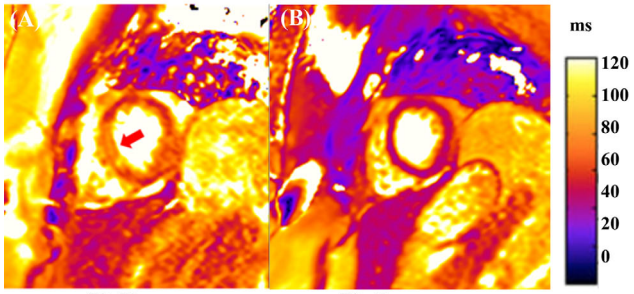
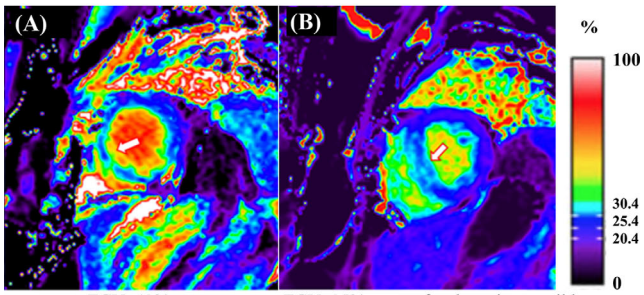
Clinical Images: Cardiovascular magnetic resonance to detect and monitor inflammatory myocarditis in systemic lupus erythematosus



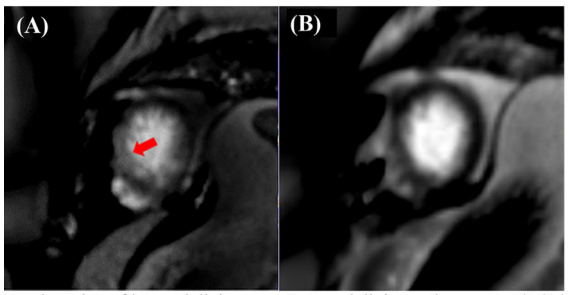
T1~1300 ms T1~ 1030 ms
Normal T1 at 1.5T 950-1050 ms



T2~73 ms T2~42 ms
Normal T2 at 1.5T 42-54 ms



ECV~43% ECV~25% except focal persistent mild elevation in septum (~42%)
Normal 25% ±2-3



Focal patches of late gadolinium enhancement (LGE) Late gadolinium enhancement (LGE)

The patient, a 37-year-old woman with systemic lupus erythematosus (SLE), presented to the emergency department with exertional pressure-like chest discomfort, dyspnea, and palpitations. On the acute presentation, results of the physical examination, chest x-ray, and electrocardiogram were unremarkable, except for sinus tachycardia. Her troponin I level was 2.24 ng/ml (normal, <0.04 ng/ml).

Coronary computed tomography angiography performed within the prior year showed normal epicardial coronary arteries. An echocardiogram demonstrated preserved left ventricular ejection fraction without wall motion abnormality. Cardiovascular magnetic resonance (CMR) revealed normal global and regional left ventricular systolic function and focal areas of myocardial edema, as demonstrated by parametric T1, T2, and extracellular volume fraction (ECV) mapping, as well as focal patches of late gadolinium enhancement (arrows in **A**). Given the patient's underlying SLE, the CMR findings were considered consistent with inflammatory myocarditis. The patient received intravenous steroids, then oral steroids (1 mg/kg) and rituximab. After treatment, her chest pain improved, and her troponin I level returned to a normal value. The repeat CMR performed 2 months later showed a reduction in myocardial edema (**B**). Eighteen months after treatment, the patient was doing well, without any cardiovascular symptoms. Inflammatory lupus myocarditis is rarely recognized clinically, and even less so in young women from ethnic minority groups, who are the most likely demographic to present with unexplained chest pain. CMR has been shown to clarify the diagnosis of patients presenting with an acute chest pain syndrome in the setting of nonobstructive epicardial coronary arteries (1). Recent CMR studies revealed a high prevalence of subclinical myocarditis in patients with SLE (2,3). One study showed that reduction in myocardial edema in CMR may be helpful to monitor SLE myocardial injury (3). CMR illustrated acute myocarditis in our case, and CMR findings were used to monitor improvement and response to treatment.

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Omer N. Pamuk, MD
National Institute of Arthritis and Musculoskeletal and Skin Diseases, NIH
W. Patricia Bandettini, MD
Jalal Vargha, MD
Sujata M. Shanbhag, MD
National Heart, Lung, and Blood Institute, NIH
Sarfaraz Hasni, MD
National Institute of Arthritis and Musculoskeletal and Skin Diseases, NIH
Bethesda, MD