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Clinical Implication of T2* Cardiac Magnetic Resonance Imaging in Cardiac Siderosis

To the Editor:

A 55-year-old woman with a history of myelodysplastic syndrome status after multiple blood transfusions presented with a 2-week history of shortness of breath, decreased exercise tolerance, weight gain, and bilateral leg edema. Transthoracic echocardiography 1 week before revealed a left ventricular ejection fraction of 35%. With the concern for iron-overload syndrome and new-onset heart failure, the patient was admitted for further evaluation. The patient had an irregularly irregular tachycardic rhythm with normal blood pressure and pulse oximetry on room air. Pertinent physical findings included jugular venous distention, bibasilar rales, and edematous lower extremities. Laboratory results revealed an anemia, mildly elevated transaminase levels, a ferritin level of 7300 ng/mL, and a negative troponin. Electrocardiogram showed atrial fibrillation with a ventricular rate of 130 beats/min without ST-T changes. Chest x-ray showed mild cardiomegaly with minimal perihilar vascular congestion. Transthoracic echocardiogram demonstrated moderate left atrial enlargement and left ventricular global hypokinesis with an ejection fraction of 20%. Cardiovascular magnetic resonance imaging revealed a decreased signal in both the heart and the liver with a myocardial T2* of 7.2 ms (**Figure 1**). Magnetic resonance imaging of the abdomen showed a decreased signal on T2-weighted imaging consistent with iron deposition in the liver, spleen, and pancreas. A diagnosis of iron overload syndrome with cardiac siderosis was made, and intravenous deferoxamine therapy was initiated. The hospital course was uneventful, and the patient was discharged with intravenous deferoxamine and oral deferiprone.

Cardiac siderosis resulting in cardiomyopathy is a serious complication of chronic transfusion therapy. Diagnosis of this condition can be challenging. Although history and physical examination can provide clinical clues, to quantify myocardial iron deposition requires further diagnostic evaluation. Serum ferritin level and hepatic iron concentration are useful as indirect estimates of total body iron; however, they correlate poorly to cardiac iron status and cardiac dysfunction.[1] Echocardiography is widely used as part of the initial and follow-up evaluations of ventricular size and function, but it does not accurately predict cardiac iron content. At an early stage of disease, myocardial iron deposition tends to be patchy and subepicardial in location; thus, an endomyocardial biopsy may provide a false-negative result. Furthermore, it is an invasive procedure that is not an ideal tool for screening asymptomatic patients or monitoring the response to chelation therapy.

Cardiovascular magnetic resonance indirectly measures myocardial iron concentration by detecting the paramagnetic influence of iron on the proton resonance behavior of tissue water.[2] The presence of iron creates abnormally rapid signal loss with increasing echo time that can be assessed as a reduction of the parameter T2*. Midventricular septal T2* has been shown to be highly representative of global myocardial T2* and iron content.[3] A T2*value of less than 20 ms, indicative of cardiac siderosis, is associated with a progressive and significant decline in left ventricular

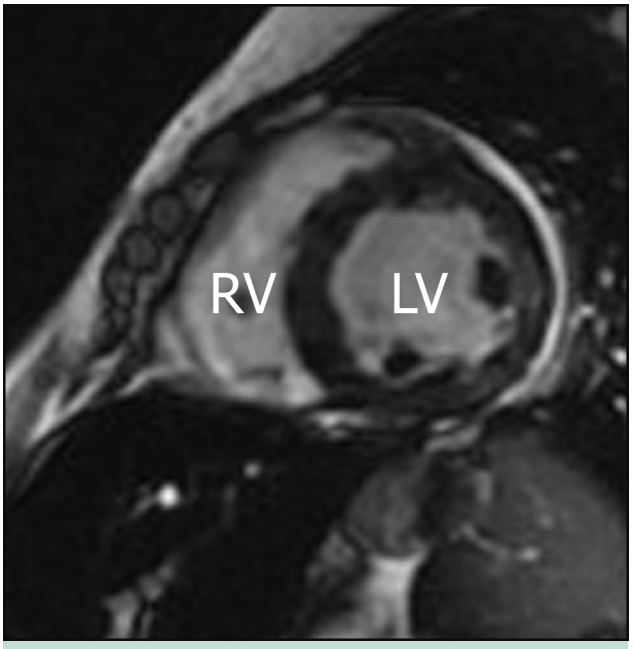

Figure 1 Short-axis cardiovascular magnetic resonance reveals decreased signal in both the heart and the liver compatible with severe cardiac and liver iron deposition with a myocardial T2* of 7.2 ms. LV = left ventricle; RV = right ventricle.

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ejection fraction[4] and a significant increased risk of arrhythmias.[5] Moreover, a T2* value of less than 10 ms provides high sensitivity and specificity in predicting heart failure within 1 year.[5] Myocardial iron clearance is exceptionally slow; thus, prolonged tailored treatment with iron chelation therapy guided by T2* value is usually required in patients with cardiac siderosis. In summary, cardiovascular magnetic resonance T2* provides diagnostic, prognostic, and therapeutic value in cardiac siderosis. This may lead to a reduction in mortality by early identification and treatment.

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