



Editorial for "Quantitative Assessment of Myocardial Edema by MR T2 Mapping in Children With Kawasaki Disease"

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Cardiac magnetic resonance imaging (MRI) is a set of techniques designed to noninvasively evaluate the myocardial morphology, ventricular function, myocardial perfusion, tissue characterization, flow quantification and presence of coronary artery disease. In 2009, the Lake Louise Criteria (LLC) established imaging diagnostic criteria for myocarditis using cardiac MRI (1). Since then, evidence has been accumulated concerning myocardial injury assessments using T1/T2 mapping and extracellular fluid fractions, which indicate myocardial-specific signal values. Based on this, the LLC was revised in 2018 and broadly divided into imaging criteria based on T2, which captures myocardial edema, and those based on T1, which captures myocardial damage (2). T2 mapping captures an increase in water content due to edema as an increase in the T2 value, enabling a quantitative evaluation (3). On T2-weighted imaging, it is seen as a region of local or whole myocardial hyperintensity, which increases in the acute phase and slowly normalizes over months. For this reason, T2-weighted imaging and T2 mapping are also useful for monitoring the staging and recovery of myocarditis (4). In addition, the T2 value as determined by T2 mapping is a highly reliable quantitative index for myocardial edema (5), and fibrosis and scarring do not increase the signal value, so it is useful for distinguishing between acute and chronic scarring.

Kawasaki disease (KD) is a febrile disorder characterized by systemic vasculitis affecting infants and young children. Coronary artery lesions (CALs) are serious complications. Almost all patients with acute KD have myocarditis (6). In the present study, the authors prospectively quantified myocardial edema by comparing 31 controls and 90 patients with KD (including 40 with acute- and 50 with chronic-phase) using cardiac MRI and explored the independent predictors of T2 values (7).

To our knowledge, this is the first study to quantitatively evaluate myocardial edema in KD patients with T2 mapping. The authors concluded that global and regional myocardial edema were present in patients with KD and were more pronounced in patients with acute KD than in those with chronic KD. Myocarditis in KD occurs earlier than coronary arteritis, peaking around the 10th day of illness and gradually subsiding after the 20th day (8). In the present study, the authors defined that the acute phase as the period within 40 days after the onset of KD and the chronic phase as the subsequent period. The results of this study are consistent with this point.

The study concluded that myocardial edema occurs in patients with KD regardless of the presence or severity of CAL. Previous report has suggested that the severity of myocarditis in KD does not necessarily predict the severity of coronary artery injury. This report also suggested that only 15% of KD patients with symptoms of myocarditis, such as cardiogenic shock or a reduced left ventricular ejection fraction, had CALs (9). Early changes seen in KD myocarditis include myocardial interstitial edema, dilation of small blood vessels, and infiltration of neutrophils and macrophages. Degeneration and necrosis of myocardial cells are scarce, and a histological picture demonstrating so-called "interstitial myocarditis" is shown. The pathological change whereby the myocardial stroma is the main site of inflammation may be related to the mild myocardial damage and favorable prognosis in many patients with KD. Although the LLC are generally intended for diagnosing viral myocarditis (1), interestingly, a similar T2 elevation was detected in patients with KD in the present study.

The symptoms of myocarditis in the acute phase of KD often resolve without the need for treatment. According to a histopathological examination of myocardial lesions in autopsy cases,

inflammatory cell infiltration in the myocardium was observed in all cases, and inflammatory cell infiltration in the myocardial interstitium was the main cause, and cardiomyocyte injury was rare. The findings in this report are particularly interesting, as they may help clarify the natural history of myocardial inflammatory changes in KD.

Previous report has suggested that gadolinium used in contrast-enhanced MRI may be deposited in the body (brain, skin, etc.) (10). Therefore, careful judgment should be practiced, especially when performing this approach in young people. T2 mapping and native T1 without contrast media are useful even in children. These findings are meaningful, given that KD frequently occurs in infants and young children.

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