MR Imaging of Nonischemic Cardiomyopathy

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KEYWORDS

- Cardiac MR imaging • Nonischemic cardiomyopathy
- Coronary artery disease

Cardiomyopathy, or failing heart, is broadly divided into ischemic and nonischemic cardiomyopathy. The term ischemic technically includes processes at the macrocirculatory and microcirculatory level. However, ischemic cardiomyopathy for the purposes of this review refers to myocardial dysfunction due to coronary artery disease (CAD), and does not include entities that cause primary or secondary dysfunction solely of the microcirculation. The most common cause of CAD is atherosclerosis.[1]

Nonischemic cardiomyopathy represents an assortment of disorders whose common primary defining character is the absence of a causative link with CAD. While broad-based, this distinction is important because it affects management. Ischemic cardiomyopathy is potentially remediable by revascularization (coronary artery bypass graft and/or percutaneous coronary interventions). The treatment of nonischemic cardiomyopathy focuses on the cause, and often the only treatment is heart transplantation.

Cardiac magnetic resonance imaging (CMR) interrogates the heart with a high spatial, temporal, and soft-tissue resolution, and multiplanar capability. It is able to quantify flow and function in a manner at least comparable to echocardiography. CMR is not plagued by technical factors that afflict echo such as the degree of operator dependency, patient body habitus, and coexisting pathology such as chronic airflow limitation[2]; this means that CMR, at least in terms of quantification, is more reproducible than echo.[3] Due to the complex geometry of the right ventricle, CMR is able to interrogate this structure with far greater clarity than echocardiography.[4] However, the uniqueness of CMR lies not in its ability to surpass echo in quantification, but to provide distinction of tissue types, the most important being myocardium that has sustained injury. This appearance is referred to as scar imaging or late gadolinium enhancement (LGE).[5]

LATE GADOLINIUM ENHANCEMENT

LGE is a feature of injured myocardium. The injury may be acute or chronic.[5] The pattern of scar facilitates the distinction of ischemic from nonischemic cardiomyopathy (**Fig. 1**).[6]

Injured myocardium is detected by the principles of inversion recovery in magnetic resonance (MR) imaging. In brief, application of an inversion pulse flips the longitudinal magnetization from its +Mz resting state to −Mz state, or a similar magnitude but negative polarity. Thereafter, the longitudinal magnetization begins to recover. The rate of recovery is proportional to the T1 recovery time of the tissue, an inherent property of the tissue. Tissues with shorter T1 recover their longitudinal magnetization sooner than tissue with longer T1. Regardless of the recovery time, each tissue crosses a so-called null point. The null point

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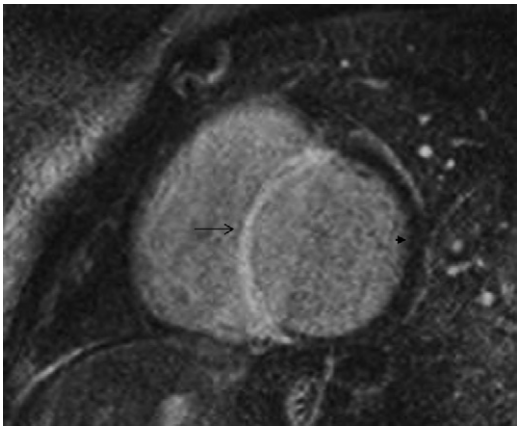

Fig. 1. Inversion recovery obtained 15 minutes after the injection of gadolinium (scar imaging) in the short-axis plane shows septal wall displaying late gadolinium enhancement (LGE) representing scar (*large arrow*), whereas the lateral wall is dark or nulled normal myocardium (*small arrow*). The LGE is transmural, that is, it involves the entire thickness of the myocardium and is territorial. Territorial means that the distribution of the LGE corresponds to coronary artery territory.

is the time point after the inversion pulse when the tissue has no net longitudinal magnetization.[7]

The time taken for the tissue to cross the null point is its unique inversion time (TI). If a pulse is applied at the TI, the nulled tissue fails to emanate signal and is said to be suppressed. The TI is, of course, dependent on the tissue's T1. Fat has a shorter TI than water because of its shorter T1. Thus, inversion recovery is a method of tissue suppression. Pulse sequences employing inversion recovery include STIR (short-tau inversion recovery) and FLAIR (fluid-attenuation inversion recovery) for the suppression of fat and water, respectively.

In scar imaging, inversion recovery suppresses the normal myocardium (**Fig. 2**). The scar appears bright, giving rise to the oft-quoted phrase in the cardiac MR imaging community "bright is dead." Normal myocardium takes up gadolinium sooner than scar and releases the gadolinium before scar. Stated differently, gadolinium is retained by the scar tissue longer than normal myocardium. At a certain time after the injection of gadolinium the difference in the quantity of retained gadolinium between normal myocardium and scar is maximal, and, if the normal myocardium is nulled the contrast between scar and normal myocardium is optimal.

MYOCARDIAL EDEMA

Myocardial edema indicates an acute process and does not per se distinguish between ischemic and nonischemic entities (**Fig. 3**). However, the pattern of edema may point to a nonischemic entity if it is patchy and nonterritorial in distribution. Myocardial edema is detected by fluid-sensitive sequences such as triple inversion recovery, in which both blood and fat are suppressed.[8]

PATTERN OF LATE GADOLINIUM ENHANCEMENT

The pattern of LGE distinguishes between ischemic cardiomyopathy and nonischemic entities.[9] The pattern of LGE depends on its distribution, mural extent, and morphology. The mural extent may be subendocardial, midmyocardial, subepicardial, or transmural, depending on the portion of the myocardium involved. The distribution may be diffuse, territorial, or nonterritorial. The territorial distribution corresponds to an area subtended by a coronary artery with sharp demarcation. A nonterritorial distribution may be focal or multifocal, without appreciation of anatomic borders defined by blood supply. The morphology of the LGE may be linear or patchy.

Ischemic cardiomyopathy tends to have LGE with a territorial distribution (see **Fig. 1**; **Fig. 4**). The LGE may be subendocardial or transmural. Because the subendocardial layer of the myocardium is most prone to ischemia, sparing of the subendocardium suggests a nonischemic cause.

The LGE pattern caused by nonischemic insults may be subepicardial or midmyocardial. Less likely nonischemic entities may cause a transmural or subendocardial pattern. Diffuse symmetric LGE is more likely to be due to a nonischemic cause. Multifocal LGE not obeying vascular territories also points to a nonischemic cause.

CARDIAC MR IMAGING AND DECISION MAKING

CMR through LGE is both diagnostic and prognostic, with the following applications:

a. Distinction between ischemic and nonischemic cardiomyopathy
b. Search for a specific nonischemic etiology
c. Search for a potential arrhythmogenic focus
d. Prognostication patients
e. Guiding biopsy.

The distinction between ischemic and nonischemic cardiomyopathy is, of course, important for the subsequent management (**Fig. 5**). If the cardiac MR imaging is incorporated early enough in the diagnostic algorithm of a patient with heart failure of unknown etiology, and the pattern of LGE suggests a nonischemic cause, then a diagnostic catheter angiogram may be forgone. Such a candidate for