Int J Cardiovasc Imaging (2008) 24:609–611
DOI 10.1007/s10554-008-9312-6

EDITORIAL COMMENT

Late contrast enhancement by CMR: more than scar?

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Received: 7 April 2008 / Accepted: 7 April 2008 / Published online: 23 April 2008

Cardiac magnetic resonance imaging (CMR) has long been recognized as an accurate and reliable means of evaluating cardiac anatomy and ventricular function. Considerable progress has been made in the field of CMR, providing accurate evaluation of myocardial ischemia and infarction [1, 2]. Contrast-enhanced CMR can be used to visualize the transmural extent of myocardial infarction with high spatial resolution [3–5]. Infarcted myocardium appears hyperenhanced compared with normal myocardium when imaged by a delayed-enhancement MRI technique with the use of T1-weighted sequence after injection of gadolinium chelates. Late gadolinium-enhanced CMR can clearly delineate subendocardial infarction and the transmural extent of delayed enhancement potentially predicts functional outcome after revascularization in acute myocardial infarction and chronic ischemic heart disease. This indicates that the late enhancement technique can accurately discriminate between infarction and dysfunctional but viable myocardium. Experimental and clinical studies have shown that the extent of delayed enhancement is reproducible and closely correlates with the size of myocardial necrosis or infarct scar as determined by established in vitro and in vivo methods. Furthermore, CMR appears to be more sensitive than other imaging methods in detecting small subendocardial infarctions.

Stress first-pass contrast-enhanced myocardial perfusion CMR can be used to detect subendocardial ischemia [6, 7], and recent studies have demonstrated the high diagnostic accuracy of stress myocardial perfusion CMR for detecting significant coronary artery disease. Free-breathing, whole-heart coronary angiography (MRA) was recently introduced as a method that can provide visualization of all three major coronary arteries and coronary bypasses within a single three-dimensional acquisition [8]. With further improvements in CMR techniques and the establishment of a standardized study protocol, CMR will play a pivotal role in imaging patients with ischemic heart disease.

In recent years, late gadolinium enhancement CMR has also been used to visualize myocardial interstitial abnormalities. Studies by the groups of Pennell et al. and Sechtem et al. [9–12] have clearly shown late enhancement patterns in patients with different forms of cardiomyopathies, amyloidosis, myocarditis, storage diseases etc. Silva et al. [10] showed that late enhancement can be demonstrated in cardiomyopathy patients, with a mean signal intensity of 390 ± 220% compared with normal regions. The distribution pattern of late enhancement was unlike the subendocardial late enhancement related to coronary territories found in myocardial infarction. The affected areas included papillary muscles (sarcoid), the mid-myocardium (Anderson–Fabry disease, glycogen storage

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disease, myocarditis, Becker muscular dystrophy) and the global subendocardium (systemic sclerosis, Loeffler's endocarditis, amyloid, Churg-Strauss). Focal myocardial late gadolinium enhancement is found in the specific cardiomyopathies, and the pattern is distinct from that seen in myocardial infarction. Vogelsberg et al. [12] showed in patients with biopsy-proven cardiac amyloidosis, that late gadolinium enhancement frequently occurs in a peculiar pattern. Consequently, noninvasive CMR can be used to diagnose or rule out cardiac amyloidosis with good sensitivity and excellent specificity in a clinical routine setting. In a recent study Hartke et al. [13] demonstrated right ventricular late gadolinium enhancement in patients with congenital heart disease and right ventricular loading conditions. Clinical variables were associated with the presence of fibrosis but did not reliably predict severity. Myocardial preservation is likely a multifactorial process that may affect the right and left ventricles differently [14].

In an article in the present issue, Bohl et al. [15] aim to systematically categorize late enhancement patterns in a variety of non-ischemic heart diseases and to explore their relationship with left ventricular function. The authors evaluated 156 patients with NIHD who exhibited late enhancement on CMR. Late enhancement was correlated to left ventricular function. It was clearly shown that late enhancement spared the subendocardium. Consistent late enhancement patterns were observed in myocarditis, hypertrophic and dilated cardiomyopathy and systemic vasculitis. No conclusive late enhancement patterns were observed in patients with aortic stenosis, arterial hypertension, lupus erythematosus, sarcoidosis, ventricular arrhythmia and in a mixed subgroup of rare non-ischemic heart diseases. There was no significant relationship between late enhancement and ejection fraction. There was no correlation between end diastolic volume and late enhancement in either myocarditis or dilated cardiomyopathy. Late enhancement was unrelated to left ventricular mass in aortic stenosis and hypertrophic cardiomyopathy.

This interesting study therefore proposed a methodological approach with which to describe and categorize late enhancement in patients with non-ischemic heart diseases. Distinct late enhancement patterns were demonstrated in four of the investigated subgroups with non-ischemic heart disease i.e. myocarditic type, dilated cardiomyopathy type, hypertrophic cardiomyopathy type and vasculitic type. A number of rare late enhancement phenotypes were elucidated that had not been described yet. Finally, it was shown that the extent of tissue injury in non-ischemic heart disease is, unlike in coronary artery disease, largely independent of systolic left ventricular function. Unlike in ischemic heart disease, the structure–function relationship did not appear to be strong. As the authors correctly state, a variety of myocardial tissue alterations occur, all of which may provide candidate substrates for positive late enhancement. Although late enhancement studies in non-ischemic heart disease are very encouraging, more sophisticated CMR techniques and prognostic clinical studies are warranted to understand the true underlying mechanisms and consequences of late myocardial contrast enhancement.

References

1. Sakuma H (2007) Magnetic resonance imaging for ischemic heart disease. J Magn Reson Imaging 26:3–13
2. Saraste A, Nekolla S, Schwaiger M (2008) Contrast-enhanced magnetic resonance imaging in the assessment of myocardial infarction and viability. J Nucl Cardiol 15:105–117
3. van der Wall EE, van Dijkman PR, de Roos A et al (1990) Diagnostic significance of gadolinium-DTPA (diethylenetriamine penta-acetic acid) enhanced magnetic resonance imaging in thrombolytic treatment for acute myocardial infarction: its potential in assessing reperfusion. Br Heart J 63:12–17
4. van Dijkman PR, van der Wall EE, de Roos A et al (1991) Acute, subacute, and chronic myocardial infarction: quantitative analysis of gadolinium-enhanced MR images. Radiology 180:147–151
5. Bax JJ, Lamb H, Dibbets P et al (2000) Comparison of gated single-photon emission computed tomography with magnetic resonance imaging for evaluation of left ventricular function in ischemic cardiomyopathy. Am J Cardiol 86:1299–1305
6. van Rugge FP, van der Wall EE, Bruschke AV (1992) New developments in pharmacologic stress imaging. Am Heart J 124:468–485
7. van Rugge FP, Holman ER, van der Wall EE, de Roos A, van der Laarse A, Bruschke AV (1993) Quantitation of global and regional left ventricular function by cine magnetic resonance imaging during dobutamine stress in normal human subjects. Eur Heart J 14:456–463

8. Langerak SE, Vliegen HW, de Roos A et al (2002) Detection of vein graft disease using high-resolution magnetic resonance angiography. Circulation 105:328–333
9. Westwood MA, Shah F, Anderson LJ et al (2007) Myocardial tissue characterization and the role of chronic anemia in sickle cell cardiomyopathy. J Magn Reson Imaging 26:564–568
10. Silva C, Moon JC, Elkington AG, John AS, Mohiaddin RH, Pennell DJ (2007) Myocardial late gadolinium enhancement in specific cardiomyopathies by cardiovascular magnetic resonance: a preliminary experience. J Cardiovasc Med 8:1076–1079
11. Mahrholdt H, Wagner A, Deluigi CC et al (2006) Presentation, patterns of myocardial damage, and clinical course of viral myocarditis. Circulation 114:1581–1590
12. Vogelsberg H, Mahrholdt H, Deluigi CC et al (2008) Cardiovascular magnetic resonance in clinically suspected cardiac amyloidosis: noninvasive imaging compared to endomyocardial biopsy. J Am Coll Cardiol 51:1022–1030
13. Hartke LP, Gilkeson RC, O'Riordan MA, Siwik ES (2006) Evaluation of right ventricular fibrosis in adult congenital heart disease using gadolinium-enhanced magnetic resonance imaging: initial experience in patients with right ventricular loading conditions. Congenit Heart Dis 1:192–201
14. Tulevski II, Hirsch A, Sanson BJ et al (2001) Increased brain natriuretic peptide as a marker for right ventricular dysfunction in acute pulmonary embolism. Thromb Haemost 86:1193–1196
15. Bohl S, Wasmuth R, Abdel-Aty H et al (2008) Delayed enhancement cardiac magnetic resonance imaging reveals typical patterns of myocardial injury in patients with various forms of non-ischemic heart disease. Int J Cardiovasc Imaging. doi:10.1007/s10554-008-9300-x