Late Enhancement in Non-Ischemic Myocardial Disease – Specific Patterns of Late Enhancement in Contrast-Enhanced Cardiac MRI Help to Characterize Different Etiologies

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Introduction:

Late enhancement (LE) in contrast-enhanced MRI (CE-MRI) has been proven to reliably identify and quantify scar after myocardial infarction in coronary artery disease (CAD) [1,2]. However, LE is not specific for ischemic damage since different fibrotic and inflammatory diseases with enlarged interstitial volume are accompanied by LE [3,4]. During recent years, many studies have been performed showing the clinical usefulness of the additional information given by LE imaging. Different myocardial diseases seem to present with specific patterns of LE enabling to differentiate different etiologies.
Aim of this study was to review cases of LE certainly not caused by myocardial infarction in a large collective of consecutive cardiac MRI examinations and to characterize specific patterns of LE in different groups of non-ischemic heart disease.

Methods:

Within 50 months, a total of 5676 contrast-enhanced cardiac MRI studies were performed on different 1.5T scanners (Magnetom Sonata (2) and Avanto (1), Siemens, Germany) in two affiliated institutions for different clinical indications. The uniformly utilized MRI protocol consisted of a functional study in standard long axis and contiguous short axis orientations of the entire left ventricle using a segmented TrueFISP sequence. Data sets for LE detection were acquired 8-15 min after administration of 0.2mmol/kg BW of Gd-based, extracellular contrast agents in the same orientations using a segmented inversion-recovery TurboFLASH sequence (TR, 8ms; TE, 4ms; TI, 200-260ms; slice thickness, 8mm). All cases of non-ischemic LE were retrospectively collected and reviewed. The different patterns of LE were related to the underlying pathology as stated by means of clinical and other diagnostic imaging features.

Results:

A total of 1905 (34%) patients presented with LE. All cases of confirmed ischemic infarction (1644, 28%) presented with typical subendocardial or transmural, strongly hyperintense LE with sharp demarcation. 261 (4.6%) patients yielded different patterns of non-ischemic LE: A) Mid-myocardial / intramural LE, 130/261 (50%): e.g. acute or chronic myocarditis hypertrophic cardiomyopathy, dilative cardiomyopathy, Fabry´s disease etc. B) Subepicardial LE, 48/261 (18%): acute or chronic myocarditis, etc. C) Transmural LE, 18/261 (7%): Chronic myocarditishypertrophic CMP, Fabry´s disease etc. D) Other kind of LE, 58/261 (22%): Fibrosis in aortic valve stenosis, hypertrophic CMP, microembolization after PCI, vasculitis etc. Within the different LE pattern groups, remarkable differences in pattern, intensity, and localization existed to define the underlying pathology. In general, non-infarction LE could well be distinguished from ischemic damage because of characteristic LE patterns.

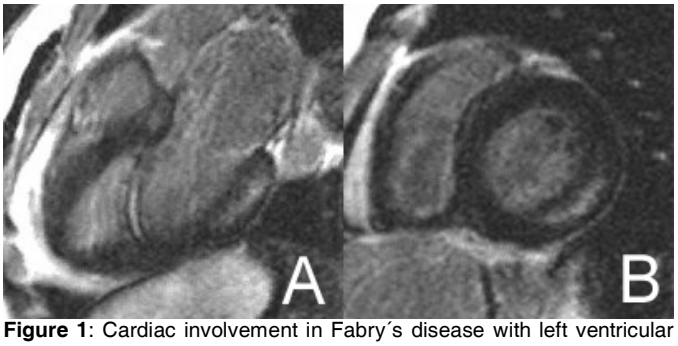

Figure 1: Cardiac involvement in Fabry´s disease with left ventricular hypertrophy. TurboFLASH images in 3-chamber view (A) and short axis (B) show a mid-myocardial, not sharply delineated, little hyperintense LE in the basal parts of the inferolateral wall (segment 5). This pattern seems characteristic for Fabry´s disease.

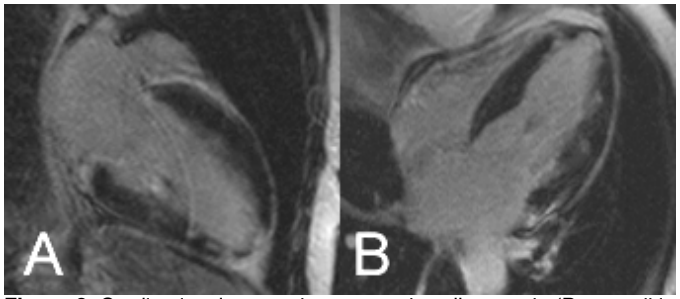

Figure 2: Cardiac involvement in a systemic collagenosis (Panarteriitis nodosa, Livedo racemosa). TurboFLASH images in vertical (A) and horizontal (B) long axis orientation. Patchy, less clearly defined, mid-myocardial LE in the inferior and lateral wall of the LV myocardium.

Discussion:

After establishing the Late enhancement technique in myocardial viability imaging, multiple applications have recently been arising in non-ischemic disease. Many non-ischemic myocardial diseases are accompanied by LE because of higher contrast material concentration in fibrosis or edema [5]. Different groups of myocardial diseases seem to present with specific LE patterns and localizations allowing for both, differentiation from ischemic myocardial damage and differential diagnosis of the underlying pathology. It is now time to define and catalog these specific appearances.

References:

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