Right Ventricular Late Enhancement – A Rare Correlate of Different Pathologies in Contrast-Enhanced Cardiac MRI

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Introduction:

Myocardial late enhancement (LE) in contrast-enhanced MRI (CE-MRI) has proven reliable in the detection and characterization of different ischemic and non-ischemic diseases affecting the left ventricular myocardium [1-3]. Particularly in inflammation and cardiomyopathy, LE facilitates the differentiatial diagnosis of the underlying pathology. Whereas LE imaging of the left ventricle is easy, the right ventricle (RV) is difficult to assess due to its thin wall and heavy trabeculation. MRI has been referred to as the reference technique in RV function assessment. However, reliable imaging techniques are requested to approach and differentiate RV myocardial disease. First studies have been reported in this respect [4-6].

Aim of this study was to review cases of RV free wall LE in a large collective of consecutive cardiac MRI examinations and to elucidate the possible diagnostic benefit of the LE technique.

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. In case of RV pathology / indication, the uniformly utilized MRI protocol consisted of a functional study in standard RV long axis and contiguous short axis orientations of the entire RV using a segmented TrueFISP sequence. LE images 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, 5mm). All cases of non-ischemic RV LE were retrospectively collected and reviewed. The presence and 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 of the left or right ventricular myocardium. In 10 of 1905 cases (0.5%) any kind of LE was detected in the RV free wall: In 5 pts., the underlying pathology was arrhythmogenic RV cardiomyopathy (ARVC/D, Fig. 1), most probably representing fibrous tissue replacement. RV myocarditis was the cause in 2 pts. Endomyocardial fibrosis was found in another 2 pts. In one patient, RV involvement in acute cardiac sarcoidosis was responsible for RV LE (Fig. 2). In many more cases, RV LE might have been suggested, was, however, denied because of possible artifacts. The major difficulty in detecting RV LE and differentiating it from artifacts corresponds to the thin wall and the heavy trabeculation.

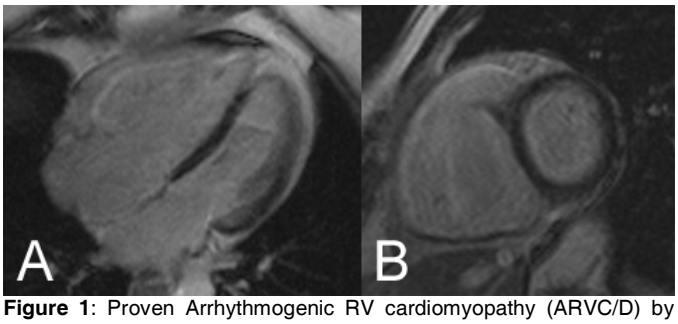

Figure 1: Proven Arrhythmogenic RV cardiomyopathy (ARVC/D) by means of ESC criteria. TurboFLASH images in 4-chamber view (A) and short axis (B) show a transmural, strongly hyperintense LE in largel parts of the RV free wall (anterior and lateral). This pattern suggests fibrous / fibro-fatty replacement of the RV myocardium.

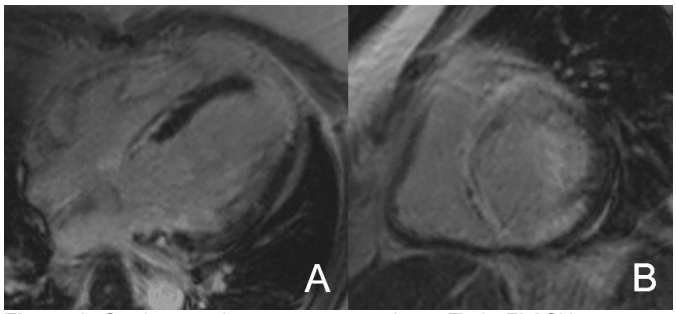

Figure 2: Cardiac involvement in sarcoidosis. TurboFLASH images in horizontal long (A) and short axis (B) orientation. Intermediate hyperintense, transmural or subepicardial LE in the anterior and lateral parts of the RV free wall. Notice the strong LE in left ventricular myocardium, particularly within the septum.

References:

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Discussion:

In left myocardial pathologies, LE has been established as a valuable tool in the differential diagnosis of ischemic vs. non-ischemic disease during recent years. Generally, the characterization of RV pathologies by means of imaging is more difficult due to the thin wall. Whereas MR functional assessment of the RV works well, LE has not been widely evaluated. This study suggests an additional diagnostic value of LE also in a variety of RV diseases.