**Value of phase-sensitive inversion recovery sequence to perform and analyse late gadolinium enhancement in cardiac amyloidosis**

A 62-year-old man was referred with new onset right heart failure and polyneuropathy. Echocardiography demonstrated severe left ventricular (LV) hypertrophy, with an ejection fraction of 76%. The interventricular septal thickness was 20 mm.

On cardiovascular magnetic resonance (CMR), short-axis dark-blood T2-weighted (Fig. 1A) and four chamber steady-state free precession (SSFP) cine (Fig. 1B) sequences showed concentric thickening of the left ventricle and bilateral pleural effusions (Supplemental material). Multi-TI inversion recovery sequence performed 8 min after gadolinium (Gd) injection showed nulling of the myocardium at TI = 160 ms, whereas nulling of the blood pool was observed at TI = 240 ms. Delayed enhancement CMR (DE-CMR) using a single breath-hold three-dimensional inversion recovery gradient-echo (turbo-FLASH) sequence demonstrated widespread enhancement of the left ventricle with subendocardial predominance in favour of cardiac amyloidosis (Fig. 1C). To eliminate any doubt between genuine widespread LV enhancement or misinterpretation due to incorrect determination of the inversion time, a subsequent three-dimensional phase-sensitive inversion recovery sequence was performed with a TI = 220 ms, which confirmed the widespread enhancement of the myocardium.
Figure 1. (A): cardiovascular magnetic resonance image of short-axis dark-blood T2-weighted and (B): four chamber steady-state free precession cine sequences showing concentric thickening of the left ventricle. (C): delayed enhancement CMR sequence showing widespread enhancement of the left ventricle with subendocardial predominance. (D): three-dimensional phase-sensitive inversion recovery confirmed the same pathologic pattern. (E): Congo red-stained and (F): haematoxylin-eosin stained sections showing amyloid deposits with a predominant interstitial pattern.

(PSIR) sequence was performed and revealed the same pathological pattern (Fig. 1D). Myocardial biopsy and molecular biology confirmed the diagnosis of transthyretin amyloid. Congo red-stained (Fig. 1E) and haematoxylin-eosin stained (Fig. 1F) sections showed amyloid deposits with a predominant interstitial pattern.

Amyloid deposition is generally homogeneous in the myocardium, leading to a diffuse hyperintense appearance on DE-CMR, which may be misdiagnosed as a technical failure of myocardial signal suppression. PSIR sequences provide improved image quality due to constant nulling of normal myocardial signal, independent of the choice of inversion time. Furthermore, using a multi-TI inversion recovery sequence, early myocardial nulling relative to blood has high sensitivity and specificity in diagnosing amyloid. In cardiac amyloidosis, PSIR sequences should be performed as they remove any doubt of the reality of diffuse delayed enhancement.

Conflicts of interest

None.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.acvd.2009.06.006.