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4D flow CMR for diastolic function assessment in cardiac amyloidosis

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Topic(s):
Cardiac Magnetic Resonance: Flow Imaging

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Background. Cardiac involvement (CI) due to systemic light-chain amyloidosis leads to LV diastolic and systolic dysfunction, culminating with end-stage heart failure. Brain natriuretic peptides and echocardiography are commonly used for diagnosis and prognosis; CMR is becoming a standard to diagnose and track CI changes thanks to tissue characterization by late gadolinium enhancement (LGE) and T1 mapping. 4DFlow is a new technique that can quantitively assess LV 3D blood flow properties over the cardiac cycle with novel markers of diastolic dysfunction, superior to other techniques because of lack of restriction to imaging planes, high isotropic spatial resolution, low operator dependency. Specifically, quantification and timing of mechanical stimuli (wall shear stress, WSS) that blood flow exerts on LV wall and changes in blood momentum (hemodynamic forces, HF) during LV relaxation.

Aim. To compare LV diastolic function assessed by 4DFlow in pts with cardiac amyloidosis vs controls and to correlate 4DFlow markers with tissue characterization, echocardiographic and laboratory parameters.

Methods. A group of pts with cardiac amyloidosis defined by echocardiography underwent a comprehensive clinical, laboratory, CMR assessment with LGE, T1 mapping, and 4DFlow. Exclusion criteria were severe renal failure or other contraindications to CMR. 4DFlow was processed to quantify diastolic components of: i) WSS, ii) time-to-peak WSS (TTP); iii) HF along base-apex (HFba), septal-lateral (HFsl), posterior-anterior (HFpa) directions, the ratio between HFsl and HFba (HFsl-ba). Healthy subjects (n=15, 8 males, age 33±10) served as controls.

Results. Ten treatment naïve amyloid pts with CI (8 males, age 54±11 years) were analyzed (n=7/2/1 in stage III/II/I, Mayo cardiac staging 2004). Pts showed significantly increased LV mass (p<.01), native myocardial T1 (p<.0001), extracellular volume (ECV) and LGE extension (assessed by QALE score) (p<.0001) and decreased LV end-diastolic volume (LVEDV) (p<.01) and ejection fraction (LVEF) (p<0.01) vs controls. WSS was lower and TTP higher in pts vs controls (pNS), also with a significant reduction in all HF components (p<.01). In pts, WSS and HFsl were correlated with left atrial area (r=.88, p<.01; r=.73, p=.01, respectively), and HFsl-ba with EF (r=.73, p=.02). As to tissue characterization, HFsl-ba correlated with myocardial native T1 (r=.71, p=.02). For echo diastolic data, correlation was found between TTP and e/e’ (r=.77, p=.01), HFsl-ba with E/A (r=.80, p<.01) and PAPs (r=.73, p=.02).

Correlation was found between NT-proBNP, cTnI and LV native T1 (r=.82, p=.01; r=.62, p=.05 respectively) and between HFpa and serum free light chain (dFLC), r=.65, p NS.

Conclusions. 4DFlow CMR detected alterations in LV diastolic function in amyloid vs controls. Preliminary analyses highlighted good correlation with CMR tissue characterization, echocardiographic and laboratory markers of diastolic impairment and disease severity.
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Figure. a) CMR images of T1 mapping, pre- (left panel) and post-contrast (right panel). b) 4DFlow CMR data, encoding blood flow velocity (left panel) and allowing to visualize and quantify blood flow (right panel). c) correlations between 4DFlow variables and common CMR, echocardiography and bio-humoral markers of LV function and CI in amyloidosis.