Quantification of Energy Loss in Hypertrophic Cardiomyopathy using 4D Flow MRI

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Introduction: Hypertrophic cardiomyopathy (HCM) is a primary myocardial disease characterized by unexplained asymmetric left ventricular hypertrophy and dynamic obstruction of the left ventricular outflow tract (LVOT). HCM is caused by mutations in sarcomeric contractile proteins and it is characterized by structural alterations, such as cardiomyocyte disarray, scarring and interstitial fibrosis, despite clinically hyperdynamic systolic function. Current diagnosis of obstructive HCM is based on the peak pressure gradient (∆P≥30 mmHg at rest) over the LVOT measured by Doppler echocardiography. However, this technique relies on the single-direction measurement of local peak velocities and may lead to misclassification due to assumptions associated with the simplified Bernoulli equation. Recently, a technique based on 4D flow MRI that estimates irrevocable energy loss (E’L) due to viscous dissipation, showed elevated energy loss in aortic dilatation and aortic valve disease. In this study, we hypothesized that obstructive HCM severity can be characterized by the amount of peak systolic E’L as calculated from 4D Flow MRI velocity fields covering the entire LVOT. Furthermore, as elevated E’L may result in increased ventricular loading and lead to worsened myocardial fibrosis, we evaluated myocardial extracellular volume fraction (ECV) with T1 mapping MRI to explore the correlation between E’L and myocardial ECV.

Methods: Navigator and prospectively cardiac gated 4D Flow measurements in the 3-Chamber view were performed in 19 HCM patients (mean age 51±15 years) and 11 healthy controls (mean age 43±14 years) at 1.5T and 3T (Avanto, Aera and Skyra, Siemens, AG, Germany). Pulse sequence parameters were as follows: T/TE/TR/FA: 2.2-2.5 ms/4.6-4.9 ms/15º, spatial resolution: 2.1-3.8x2.1-4.1x2.4-3.4 mm, temporal resolution: 37-40 ms. Number of cardiac phases: 11-27, FOV: 255-340x255-360x65-132 mm, velocity encoding: 1.5-2.5 m/s. 4D flow velocity data were corrected for Maxwell terms, eddy currents and velocity aliasing and filtered with a median filter (3x3x3 voxels). For all subjects, time-averaged 3D PC-MR angiogram (MRA) data were derived from the 4D flow data. 3D segmentation (MIMICS, Materialise, Leuven, Belgium) based on the 3D-PC-MRA was performed to extract the 3D LVOT geometry. Peak systole was defined as from the 4D flow data. T1-mapping was performed in 12 of the 19 HCM patients (mean age 50±15 years) using a modified look-locker inversion recovery (MOLLI) technique as described previously. Data for each slice (base, mid, apex) were acquired pre (T1pre) and 10-25 minutes post (T1post) contrast agent administration (gadopentetate dimeglumine, 0.1 mmol/Kg) using breath holding. Imaging parameters were as follows: spatial resolution = 1.7-2.1 x1.7-2.1x8 mm, slice thickness = 8mm, temporal resolution: 4.6-4.9 ms, number of cardiac phases: 11-27, slice thickness: 8-12 mm. T1 was calculated by the difference of T1pre and T1post (T1 = T1pre - T1post).

Results: T1-mapping was performed in 12 of the 19 HCM patients (mean age 50±15 years) using a modified look-locker inversion recovery (MOLLI) technique as described previously. Data for each slice (base, mid, apex) were acquired pre (T1pre) and 10-25 minutes post (T1post) contrast agent administration (gadopentetate dimeglumine, 0.1 mmol/Kg) using breath holding. Imaging parameters were as follows: spatial resolution = 1.7-2.1 x1.7-2.1x8 mm, slice thickness = 8mm, temporal resolution: 4.6-4.9 ms, number of cardiac phases: 11-27, slice thickness: 8-12 mm. T1 was calculated by the difference of T1pre and T1post (T1 = T1pre - T1post).

Figure 1: Maximum intensity projections of velocity (left) and energy loss (right) in the control and a HCM patient.

Figure 2: Energy Loss at peak systole compared with myocardial ECV.

Discussion: A strong correlation was found between E’L and myocardial ECV suggesting that increased interstitial tissue is associated with viscous energy loss in the LVOT. Energy loss may help drive myocardial fibrosis in these patients, and longitudinal studies are required to better understand this relationship. 4D Flow MRI analysis of the LVOT and E’L calculated from this non-invasive technique may be useful in HCM severity assessment and risk stratification without the limitations associated with gradient estimation including the confounding effect of pressure recovery and may aid in improved understanding of HCM pathophysiology.

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