Quantitative assessment of subendocardial perfusion abnormality in hypertrophic cardiomyopathy: Correlation with myocardial scar.

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Introduction:

Previous studies demonstrated that myocardial ischemia occurs in patients with hypertrophic cardiomyopathy (HCM), despite of angiographically normal coronary arteries (1). A study employing ^{15}O-water PET indicated that subendocardial hypoperfusion could be an important mechanism of myocardial ischemia in patients with HCM (2). However, PET assessment of endocardial/epicardial (endo/epi) flow ratio was only feasible in the markedly hypertrophied septum due to limited spatial resolution. Patlak plot analysis of arterial input and myocardial output functions on myocardial perfusion MRI can provide quantitative assessment of transmural distribution of myocardial blood flow (MBF) for the entire LV myocardium (3). The purposes of this study were to determine the endo/epi flow ratio in HCM patients with MRI, and to clarify the relationship between transmural perfusion abnormality and myocardial scar.

Methods:

Nine patients (6 men, mean age of 56 +/- 17 years) with HCM were evaluated with a 1.5 T MR system (Signa CV/i, GE Healthcare). First-pass myocardial perfusion MR images were acquired by using a saturation recovery steady-state MR sequence (TR 3.0ms; TE 1.2ms; TI 180ms; flip-angle 45 degrees). To obtain blood time-intensity curve without signal saturation, we initially acquired first-pass contrast enhanced MR images by injecting low-concentration (0.005mmol/kg) Gd-DTPA. Then first-pass contrast enhanced myocardial perfusion MR images were obtained during ATP stress and in the resting state with intravenous bolus injections of Gd-DTPA (0.05mmol/kg, 4ml/sec). After correcting saturation of the blood signal and coil sensitivity profile, arterial input and myocardial output time-intensity curves were analyzed with a Patlak plot method to quantify tissue K1, which represents product of tissue plasma flow and extraction fraction of Gd-DTPA from capillary to the extracellular space in the myocardium. Steady-state cine MRI (TR 3.9ms; TE 1.7ms) and delayed-enhancement MRI (inversion recovery 2D fast GRE, TR 4.8ms, TE 1.3ms, TI adjusted to null myocardial signal) were acquired on the same imaging planes. Image analysis was performed by using a 16 segment model.

Results:

In HCM patients, the endo/epi flow ratio was 0.99 +/- 0.13 in the resting state, and significantly decreased to 0.92 +/- 0.14 during stress ($p<0.001$), which showed a good agreement with the results reported by previous radio-water PET study (1). No significant correlation was observed between the endo/epi flow ratio and end-diastolic wall thickness of the left ventricle. In the 17 segments exhibiting delayed hyperenhancement, the endo/epi ratio was significantly reduced in comparison with the segments without delayed hyperenhancement both in the resting state and during vasodilator stress (0.91 +/- 0.10 vs 1.00 +/- 0.13 at rest; $p=0.005$, 0.82 +/- 0.12 vs 0.94 +/- 0.13 during stress; $p=0.001$).

Conclusion:

While no transmural gradient of myocardial perfusion was observed in the resting state, subendocardial myocardial perfusion was significantly reduced during vasodilator stress in patients with HCM. In the segments showing scar, however, the endo/epi flow ratio was significantly deteriorated both in the resting state and during stress, indicating the close relation between subendocardial perfusion abnormality during stress and scar formation in HCM..

References:

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