SCIENTIFIC LETTER

Intramyocardial coronary flow characteristics in patients with hypertrophic cardiomyopathy: non-invasive assessment by transthoracic Doppler echocardiography

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Inadequate coronary vasodilator reserve,¹ luminal narrowing of coronary capillaries,² and systolic compression of the intramyocardial small arteries³ are considered to contribute towards myocardial ischaemia in patients with hypertrophic cardiomyopathy (HCM). Previously, abnormal phasic coronary flow velocity pattern in the left anterior descending coronary artery (LAD) in patients with HCM, which was characterised by flow reversal in systole and slow acceleration and rapid deceleration in diastole, has been observed by Doppler guide wire method.⁴ However, intramyocardial coronary flow characteristics in patients with HCM have not been reported. We aimed to evaluate phasic intramyocardial coronary flow velocity pattern in patients with HCM by transthoracic Doppler echocardiography.

METHODS

The study population comprised 16 consecutive patients with non-obstructive HCM (12 men, mean (SD) age 63 (16) years, range 16–79 years). The diagnosis of HCM was based on the echocardiographic demonstration of an asymmetrically hypertrophied and non-dilated left ventricle. All patients were in sinus rhythm. Patients with coronary artery disease, congestive heart failure, and diabetes mellitus were not enrolled in this study. The control subjects were 10 healthy volunteers (eight men, mean age 56 (13) years, range 35–67 years). All participants gave informed consent to the protocol approved by the committee for the protection of human subjects in research at Kawasaki Medical School.

Echocardiographic examinations were performed with an HDI-5000 (Philips Medical Ultrasound, Bothell, Washington, USA) and a Sequoia 512 (Acuson Corp, Mountain View, California, USA) digital ultrasound system. The end diastolic thickness of interventricular septum and left ventricular posterior wall were measured in parasternal long axis views. Ejection fraction was calculated by using the modified Simpson's bi-plane formula.

The spectral Doppler signals were recorded in the mid portion of the LAD and the septal artery by transthoracic Doppler echocardiography. Colour Doppler and pulsed Doppler echocardiography were performed with the HDI-5000 with 4–7 MHz transducer and the Sequoia digital ultrasound system with 3.5–7 MHz transducer. In colour Doppler flow mapping, the velocity range was set at ± 28.8 to ± 8.0 cm/s. The transducer was positioned in the fourth or fifth intercostal spaces in the left side of the sternum. The left ventricle was imaged in the short axis section, and then the transducer was rotated counterclockwise, inclined laterally toward the interventricular sulcus. Coronary flow in the LAD and the septal artery were identified in colour Doppler flow mapping. The

Abbreviations: DPV, diastolic peak velocity; HCM, hypertrophic cardiomyopathy; LAD, left anterior descending coronary artery; SPV, systolic peak velocity; TPVd, time from the beginning of diastole to diastolic peak velocity; VHTd, diastolic velocity half time

Figure 1 (Left) Left anterior descending and septal arteries by colour Doppler echocardiography. IVS, interventricular septum; LAD, left anterior descending coronary artery; LV, left ventricle. (Right upper panel) Measurements of variables of coronary flow velocity recording. DPV, diastolic peak velocity; SPV, systolic peak velocity; TPVd, time from the beginning of diastole to diastolic peak velocity; VHTd, diastolic velocity half time. (Right lower panel) Intramyocardial septal artery flow velocity profile recorded by using pulsed Doppler echocardiography in patients with hypertrophic cardiomyopathy.

sample volume was positioned on the colour Doppler flow signal. Phasic Doppler spectral tracings of each flow velocity were assessed utilising pulsed Doppler technique (fig 1). All studies were stored digitally and recorded on half inch super-VHS videotape for off-line analysis. The following measurements were performed from the phasic coronary flow velocity recordings, using the computer incorporated in the ultrasound system: (1) systolic peak velocity (SPV); (2) diastolic peak velocity (DPV); (3) time from the end of T wave in ECG to DPV (time to peak velocity, TPVd) corrected by $\sqrt{\text{RR}}$ interval; and (4) deceleration of diastolic velocity from DPV (corrected by $\sqrt{\text{RR}}$ interval) indicated by velocity half time (VHTd).

Statistical analysis was carried out with unpaired two tailed *t* testing and Mann-Whitney U testing where appropriate. A probability value of $p < 0.05$ was considered significant.

RESULTS

The average septal thickness was 17.2 (1.7) mm and the average septal/posterior wall thickness ratio was 1.4 (0.2) in patients with HCM. This ratio was significantly higher than in the control subjects (1.0 (0.2), $p < 0.0001$). Average ejection fraction was significantly higher in patients with HCM than controls (74.8 (8.8)% *v* 67.1 (7.0)%, $p = 0.04$).

Adequate biphasic flow velocity profile in the LAD was recorded in 13 (81%) of 16 patients and 7 (70%) of 10 volunteers. In all 13 patients with HCM, systolic flow reversal in the LAD was obtained (SPV -19.0 (3.7) cm/s). In all seven control subjects, normal antegrade systolic flow was obtained (SPV 18.5 (3.8) cm/s). DPV was not significantly higher in patients with HCM than in normal subjects (41.7 (13.8) *v* 32.3 (10.2) cm/s, $p = \text{NS}$). TPVd corrected by $\sqrt{\text{RR}}$ was prolonged in patients with HCM compared with control subjects (4.9 (1.2) *v* 2.9 (0.9) ms, $p = 0.0003$). VHTd corrected by $\sqrt{\text{RR}}$ in patients with HCM was shorter than in the control subjects (5.8 (1.9) *v* 15.8 (4.3) ms, $p < 0.0001$).

Adequate biphasic flow velocity profile in the septal artery was recorded in 12 of 16 (75.0%) patients and 6 of 10 (60.0%) volunteers utilising pulsed Doppler echocardiography. In all 12 patients with HCM, systolic flow reversals were obtained (SPV -48.8 (11.0) cm/s), while normal antegrade flow was obtained in all six control subjects (SPV 11.4 (4.8) cm/s). DPV of septal artery flow in patients with HCM were significantly higher than in normal subjects (50.9 (23.6) *v* 22.7 (8.2) cm/s, $p = 0.0015$). TPVd corrected by $\sqrt{\text{RR}}$ was prolonged in the patients with HCM compared with control subjects (3.6 (0.4) *v* 2.8 (0.7) ms, $p = 0.04$). VHTd corrected by $\sqrt{\text{RR}}$ in patients with HCM was shorter than in control subjects (10.9 (6.5) *v* 17.8 (5.9) ms, $p = 0.01$). Systolic flow reversal, slow acceleration, and rapid deceleration of the diastolic flow were characteristic in the intramyocardial septal arteries as well as in the LAD.

DISCUSSION

In this study, abnormal phasic flow pattern in the septal artery was obtained. This result suggests that the abnormal systolic flow reversals in the LAD, which have been previously reported,⁴ simply reflect the systolic flow reversal in the intramyocardial coronary arteries. Systolic compression or narrowing of the intramyocardial coronary arteries in patients with HCM has been reported²; the asynchronous contraction of the left ventricular wall and increased intramyocardial pressure would contribute to systolic narrowing of the intramyocardial coronary arteries, which participate in myocardial perfusion abnormalities in patients with HCM. Presently, transthoracic Doppler echocardiography is the only method available to evaluate intramyocardial flow characteristics. These results may therefore prove helpful for the further clinical investigation of the coronary microvascular circulation in patients with HCM.

In summary, systolic flow reversal, slow acceleration, and rapid deceleration of the diastolic flow velocity are characteristic findings in the intramyocardial coronary artery in patients with HCM.

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