No reflow-like pattern in intramyocardial coronary artery suggests myocardial ischemia in patients with hypertrophic cardiomyopathy

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Abstract
Background and purpose: To evaluate intramyocardial coronary flow velocity pattern by transthoracic Doppler echocardiography and its clinical significance in patients with hypertrophic cardiomyopathy (HCM).
Methods and results: In 48 patients with HCM who had angiographically normal coronary artery, coronary flow velocity in the left anterior descending coronary artery (LAD) and intramyocardial coronary artery (IMCA) derived from LAD were evaluated using transthoracic Doppler echocardiography. Two clearly different flow patterns in the IMCA were observed in patients with HCM. Twenty-seven HCM patients (group A) had slow deceleration slope in the IMCA flow (average diastolic deceleration time, 989 ± 338; range, 585—1680) and the remaining 21 patients (group B) had steep deceleration slope with diastolic deceleration time <300 ms, resulting in a no reflow-like pattern in the IMCA flow (average diastolic deceleration time, 166 ± 67; range, 55—280). There were no significant differences in the clinical characteristics and LAD flow velocity profiles between the two groups. The incidence of cardiovascular symptoms (chest pain or syncope) was significantly higher in group B than in group A (67% vs. 26%, p < 0.01). Additionally, exercise-induced ischemia as detected by thallium-201 scintigraphy was significantly more frequent in group B than in group A (6 of 9 (67%) vs. 0 of 9 (0%), p < 0.01).

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Conclusions: Two different intramyocardial coronary flow velocity patterns are observed in patients with HCM using transthoracic Doppler echocardiography. No reflow-like pattern in the IMCA is strongly related to myocardial ischemia in the absence of epicardial coronary artery stenosis, suggesting that coronary microvascular dysfunction may be a causative mechanism. © 2008 Published by Elsevier Ireland Ltd on behalf of Japanese College of Cardiology.

Introduction

Clinical signs and symptoms suggesting myocardial ischemia frequently develop in patients with hypertrophic cardiomyopathy (HCM) despite anatomically normal epicardial coronary artery [1,2]. Although there is growing evidence that abnormalities of the coronary microvasculature can result in myocardial ischemia in patients with HCM with normal epicardial coronary artery [2—4], clinical evaluation of the coronary microcirculation has not been fully investigated due to technical difficulties.

Characteristic phasic coronary flow velocity patterns have been reported in patients with HCM. However, there was no relationship between phasic flow abnormalities and those with symptoms [5—7]. Since the phasic blood velocity pattern in penetrating coronary arteries is different than that in large epicardial arteries, epicardial coronary flow velocity profiles may not accurately reflect intramyocardial coronary perfusion [8]. Recent advances in transthoracic Doppler echocardiography allow evaluation of flow velocity not only in the left anterior descending coronary artery (LAD) but also in the intramyocardial coronary artery (IMCA, 500—1000 μm) [9—12]. Therefore, we hypothesized that transthoracic Doppler echocardiography would potentially allow evaluation of an impaired flow velocity profile associated with myocardial ischemia in the absence of epicardial coronary stenosis. On this basis, the primary aim of this study was to evaluate the characteristics of the coronary flow velocity profiles in the IMCA using transthoracic Doppler echocardiography and to define the relationship between the coronary flow velocity pattern and clinical manifestations in patients with HCM. Furthermore, we compared transthoracic Doppler echocardiographic measurements of coronary flow velocity profiles with exercise thallium-201 scintigraphy results in patients with HCM.

Subjects and methods

Study patients

Sixty-eight consecutive patients with HCM who underwent evaluation of cardiac function and echocardiography at Kagoshima University Hospital or Nanpuh Hospital were enrolled in the study. The diagnosis of HCM was based on echocardiographic evidence of myocardial hypertrophy, defined as a maximal septal thickness of at least 13 mm, in the absence of any other cardiac or systemic cause of left ventricular hypertrophy [13].

Twenty patients were excluded from the analysis because of the presence of atrial fibrillation (n = 13) or an artificial pacemaker (n = 7). Consequently, the study population consisted of the remaining 48 patients (33 men, 15 women; mean age 53 years). Exercise thallium-201 scintigraphy was performed in 18 patients with HCM within 6 months before and after the echocardiographic examination as previously described [14]. Patient medications were not changed during the study. Nine subjects with normal echocardiograms and no known cardiovascular disease served as the control subjects (8 men and 1 woman, mean age 39 years). The study protocol was approved by the institutional committee of Kagoshima University, and each patient gave written informed consent prior to enrollment.

Transthoracic Doppler echocardiography

Transthoracic Doppler echocardiography with a 2.5—3.5-MHz transducer was performed using a commercially available echocardiographic system (ATL HDI 5000CV, GE Logiq 500 MR, or Acuson Sequoia C256). Left ventricular end-diastolic and end-systolic dimension and left atrial dimension were measured in the parasternal long axis view. The peak instantaneous left ventricular outflow tract gradient was estimated under basal conditions with the use of continuous-wave Doppler echocardiography.
Recording of LAD and IMCA flow by transthoracic Doppler echocardiography

Using a 4–7-MHz transducer, the short axis of the anterior interventricular groove was visualized from the echo window between the left parasternal fourth or fifth intercostal space and the apex. Next, the transducer was rotated to visualize the long axis of the groove, and color flow mapping with a Nyquist limit $\leq 25$ cm/s was applied to visualize the mid-to-distal LAD flow with relatively slow flow velocities.

To image the IMCA flow, the left parasternal short axis and the classical or modified two-, three- and four-chamber views were used. First, through the left parasternal short axis view at the level of the mitral valve, chordae, and papillary muscle, we searched for IMCA flows in the anteroseptal left ventricular wall using color Doppler mapping with a low Nyquist limit. Next, using classical or modified two-, three- and four-chamber views, IMCA flow from the epicardium to the endocardium was also evaluated in the apical half of the left ventricular wall using color flow mapping with a low Nyquist limit. The flow velocity profiles in the LAD and IMCA were recorded by pulsed-wave Doppler echocardiography with angle correction.

The peak and mean diastolic velocity, diastolic velocity time integral, and deceleration time of diastolic flow were measured and averaged over three consecutive beats in each subject.

Thallium-201 scintigraphy

Each patient performed graded bicycle exercise starting at 25 W, with an increment of 25 W every 3 min. At peak exercise, 110 MBq (3 mCi) of thallium-201 chloride was injected and the exercise was continued for another 1 min. Stress thallium-201 perfusion scanning was begun within 10 min after the tracer injection. Delayed thallium-201 perfusion scanning was performed 4 h later.

Only anteroseptal and apical segments were evaluated by thallium-201 scintigraphy, because transthoracic Doppler echocardiographic evaluation of coronary flow abnormalities was limited to the territory of the LAD. Two experienced observers who had no knowledge of the clinical or echocardiographic data individually analyzed the images. The patients were considered to have myocardial ischemia when a perfusion defect was seen on the stress study but was absent in the redistribution images, or when a defect on the stress study was larger than that in the redistribution study. The images were considered negative when no perfusion defect was seen on either image, or when the defect on the stress study was the same as that in the redistribution.

Reproducibility of measurements

Interobserver variability of the measurements was assessed from 10 randomly selected recordings, with an observer blinded to previous echocardiographic data. For the assessment of intraobserver variability, 10 subjects were examined twice on different days by the same sonographer and cardiologist, who were blinded to prior Doppler echocardiographic data. All measurements were calculated as the standard deviation of the differences between the two measurements divided by the mean measurement and were expressed as the percentage of the average value.

Statistical analysis

Continuous variables are expressed as mean $\pm$ standard deviation, and categorical variables are given as absolute values, percentages, or both. Differences between continuous variables were assessed with the unpaired $t$-test. Proportions were compared by the chi-square or Fisher exact test. A $p$-value of $<0.05$ was considered statistically significant.

Results

Patients’ characteristics

Clinical characteristics and general echocardiographic findings are shown in Table 1. Left ventricular wall thickness, left atrial dimension, and fractional shortening were significantly greater in patients with HCM than in normal subjects, whereas left ventricular end-systolic dimension was significantly smaller. Twenty-one (44%) patients with HCM had clinical symptoms of chest pain or syncope with angiographically normal coronary arteries.

Comparison of LAD flow velocity profiles between HCM and normal subjects

Adequate spectral Doppler recordings of diastolic coronary flow in the LAD were obtained in all study populations. The peak and mean diastolic velocity and velocity time integral of LAD flow were significantly increased and diastolic deceleration time was significantly prolonged in patients with HCM compared to those in normal subjects (Table 1).
Table 1  Baseline characteristics of the study population

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Controls (n = 9)</th>
<th>Patients with HCM (n = 48)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>39 ± 13</td>
<td>53 ± 16</td>
<td>0.015</td>
</tr>
<tr>
<td>Men/women (n)</td>
<td>8/1</td>
<td>33/15</td>
<td>NS</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>68 ± 8</td>
<td>61 ± 11</td>
<td>NS</td>
</tr>
</tbody>
</table>

Echocardiographic measurements

- Maximal wall thickness (mm): 10 ± 2 vs. 24 ± 5, p < 0.001
- IVS thickness (mm): 10 ± 2 vs. 21 ± 7, p < 0.001
- PW thickness (mm): 9 ± 2 vs. 12 ± 3, p = 0.004
- LV end-diastolic dimension (mm): 48 ± 4 vs. 44 ± 7, NS
- LV end-systolic dimension (mm): 32 ± 5 vs. 26 ± 5, p = 0.006
- Fractional shortening (%): 35 ± 5 vs. 42 ± 8, p = 0.024
- LA dimension (mm): 31 ± 4 vs. 39 ± 6, p < 0.001
- LVOT pressure gradient ≥ 30 mmHg: 0 (0%) vs. 3 (17%), NS
- Incidence of moderate to severe MR: 0 (0%) vs. 0 (0%), NS

LAD flow

- Peak diastolic velocity (cm/s): 26 ± 6 vs. 49 ± 23, p < 0.001
- Mean diastolic velocity (cm/s): 18 ± 4 vs. 32 ± 15, p < 0.001
- Diastolic velocity time integral (cm): 10 ± 3 vs. 23 ± 11, p < 0.001
- Diastolic deceleration time (ms): 840 ± 144 vs. 1008 ± 353, p = 0.028

IMCA flow

- Peak diastolic velocity (cm/s): 31 ± 10 vs. 95 ± 43, p < 0.001
- Mean diastolic velocity (cm/s): 24 ± 8 vs. 61 ± 31, p < 0.001
- Diastolic velocity time integral (cm): 12 ± 4 vs. 34 ± 17, p < 0.001
- Diastolic deceleration time (ms): 657 ± 100 vs. 613 ± 485, NS

Data are presented as number (%) or mean ± 1 S.D. HCM = hypertrophic cardiomyopathy; IVS = interventricular septum; PW = posterior wall; LV = left ventricular; LA = left atrial; LVOT = LV outflow tract; MR = mitral regurgitation; LAD = left anterior descending coronary artery; IMCA = intramyocardial small coronary artery.

Comparison of IMCA flow velocity profiles between HCM and normal subjects

Adequate spectral Doppler recording of the diastolic flow in the IMCA were obtained in all study populations. The peak and mean diastolic velocity and velocity time integral of IMCA flow were significantly increased in patients with HCM compared to those in normal subjects. However, there was no significant difference in diastolic deceleration time for IMCA flow between patients with HCM and normal subjects (Table 1).

Two different IMCA flow velocity patterns

Two characteristic flow patterns in the IMCA were observed in patients with HCM (Figs. 1B, 2B and 3B). One flow pattern in the IMCA showed peak velocity is observed in early diastole and then gradually decreased during the remainder of diastole (Fig. 1B). The other flow pattern was characterized by steep decelerated diastolic flow with deceleration time <300 ms. Overall, the latter flow velocity pattern has a no reflow-like appearance (Figs. 2B and 3B). Therefore, the 48 study patients were divided into two groups according to their IMCA flow pattern. Group A consisted of 27 patients with HCM who showed slow deceleration slope in the IMCA (average deceleration time, 989 ± 338; range, 585—1680). Group B consisted of the remaining 21 patients with HCM who showed a no reflow-like pattern in the IMCA (average deceleration time, 166 ± 67; range, 55—280).

Comparison of LAD and IMCA flow velocity profiles between two groups in HCM patients

LAD and IMCA flow velocity profiles are summarized in Table 2. Measurements of LAD flow velocity profiles were not significantly different between the two groups. However, the mean diastolic velocity and velocity time integral in the IMCA were decreased in group B compared to group A (p < 0.05), and the diastolic deceleration time of IMCA flow was significantly shortened in group B by definition (p < 0.001).

Comparison of clinical symptoms between two groups in HCM patients

There were no significant differences in age, sex, type of HCM, and echocardiographic data between...
Figure 1 Fifty-two-year-old female without symptoms. Transthoracic Doppler echocardiographic measurement of left anterior descending coronary artery (A) and intramyocardial coronary artery (B). Diastolic peak velocity of intramyocardial coronary flow decreases gradually, so we classified this patient into group A. (C) Thallium-201 scintigraphy showing stress and redistribution images in the short-axis (SA) views. It demonstrates no ischemia.

the two groups (Table 2). Syncope was observed in 1 patient (4%) in Group A and 8 patients (36%) in Group B, and chest pain was observed in 6 patients (22%) in Group A and 10 patients (48%) in Group B. Thus, the incidence of cardiovascular symptoms (chest pain or syncope) was significantly higher in group B than in group A ($p=0.008$) (Table 2).

Comparison of thallium-201 scintigraphy between two groups in HCM patients

Exercise thallium-201 scintigraphy was performed in 18 of the 48 patients with HCM. Exercise-induced myocardial ischemia at the anteroseptal or apical segment was found in 6 of the 9 patients (67%) in group B, while none of the 9 patients (0%) in group A showed ischemia, a statistically significant difference in incidence ($p=0.009$) (Table 2). Figures show representative images of coronary flow velocity patterns in the LAD and IMCA as seen by transthoracic Doppler echocardiography, both without and with exercise-induced apical ischemia as detected by thallium-201 scintigraphy.

Observer variability

Interobserver variabilities for peak diastolic velocity, time velocity integral and deceleration time of diastolic flow were 4.2%, 4.9%, and 3.9%, respectively. Interobserver variabilities for peak diastolic velocity, time velocity integral and deceleration time of diastolic flow were 3.8%, 4.1%, and 3.9%, respectively.

Discussion

In the present study, we evaluated the value of intramyocardial coronary flow velocity measurements using transthoracic Doppler echocardiography in patients with HCM. We have found that there were two different IMCA flow patterns in patients with HCM, despite no difference of LAD flow velocity pattern between these patients. One flow pattern obtained from IMCA showed anterograde diastolic flow velocity, reaching peak velocity in early diastole with subsequent gradual deceleration. The other flow pattern showed no reflow-like pattern (sharp acceleration of coro-
Figure 2  Fifty-three-year-old female with a history of syncope. Transthoracic Doppler echocardiographic measurement of left anterior descending coronary artery (A) and intramyocardial coronary artery (B). The pulsed Doppler echocardiography in the intramyocardial coronary artery demonstrating characteristic no reflow-like pattern, so we classified this patient into group B. (C) Thallium-201 scintigraphy showing abnormal perfusion with reversible ischemia in the anteroseptal segments.

Figure 3  Thirty-year-old male with a history of syncope and chest discomfort. Transthoracic Doppler echocardiographic measurement of left anterior descending coronary artery (A) and intramyocardial coronary artery (B). The pulsed Doppler echocardiography in the intramyocardial coronary artery demonstrating characteristic no reflow-like pattern, so we classified this patient into group B. (C) Thallium-201 scintigraphy showing abnormal perfusion with reversible ischemia in the anteroseptal segments.
in patients with HCM in the absence of epicardial coronary stenosis.

Clinical findings suggestive of myocardial ischemia in patients with HCM are found to have normal coronary arteries at angiography despite having objective evidence of reversible exercise induced defects in myocardial perfusion by thallium-201 imaging [15]. Abnormalities in coronary flow dynamics have been considered as a possible mechanism for myocardial ischemia in HCM patients with normal coronary arteries [2—4]. Previously, alterations of the phasic coronary flow velocity profile in the LAD in patients with HCM characterized by flow reversal in early systole, a reduced systolic component, and preserved or even increased diastolic flow velocity have been demonstrated in several studies using invasive or noninvasive Doppler methods. However, these studies demonstrated no relationship between LAD flow velocity profiles and those with symptoms and myocardial ischemia [5—7].

In this study, we have demonstrated that no reflow-like pattern in the IMCA was related to clinical symptoms in addition to exercise-induced ischemia by thallium-201 scintigraphy. The normal value of the diastolic deceleration time is a matter of dispute, but it has been reported that the decay in the diastolic deceleration slope correlated with the severity of microvascular dysfunction [16,17]. The mechanism of this diastolic change is speculative, but rapid deceleration of diastolic flow would be expected in patients with increased microvascular resistance and impedance [18—21]. Morphologic studies have revealed that HCM is characterized by many abnormal intramural coronary arteries and subendocardial arterioles characterized by thickened walls and narrowed lumens [4,22—24]. This may result in an increase in minimal coronary resistance.

### Table 2 Baseline characteristics and echocardiographic measurements of HCM patients

<table>
<thead>
<tr>
<th></th>
<th>Group A (n = 27)</th>
<th>Group B (n = 21)</th>
<th>p value</th>
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<tr>
<td>Age (years)</td>
<td>55 ± 13</td>
<td>51 ± 19</td>
<td>NS</td>
</tr>
<tr>
<td>Men/women (n)</td>
<td>20/7</td>
<td>13/8</td>
<td>NS</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>62 ± 11</td>
<td>60 ± 10</td>
<td>NS</td>
</tr>
<tr>
<td>Type of HCM</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>ASH</td>
<td>17 (63%)</td>
<td>15 (71%)</td>
<td>NS</td>
</tr>
<tr>
<td>Apical</td>
<td>10 (37%)</td>
<td>6 (29%)</td>
<td>NS</td>
</tr>
<tr>
<td>Symptom</td>
<td></td>
<td></td>
<td>0.008</td>
</tr>
<tr>
<td>No symptoms</td>
<td>20 (74%)</td>
<td>7 (33%)</td>
<td></td>
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<tr>
<td>Chest pain or syncope</td>
<td>7 (26%)</td>
<td>14 (67%)</td>
<td></td>
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<tr>
<td>Echocardiographic measures</td>
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<tr>
<td>Maximal wall thickness (mm)</td>
<td>23 ± 5</td>
<td>25 ± 6</td>
<td>NS</td>
</tr>
<tr>
<td>IVS thickness (mm)</td>
<td>19 ± 6</td>
<td>23 ± 8</td>
<td>NS</td>
</tr>
<tr>
<td>PW thickness (mm)</td>
<td>12 ± 3</td>
<td>12 ± 3</td>
<td>NS</td>
</tr>
<tr>
<td>LV end-diastolic dimension (mm)</td>
<td>45 ± 6</td>
<td>44 ± 7</td>
<td>NS</td>
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<tr>
<td>LV end-systolic dimension (mm)</td>
<td>27 ± 5</td>
<td>26 ± 6</td>
<td>NS</td>
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<tr>
<td>Fractional shortening (%)</td>
<td>40 ± 5</td>
<td>44 ± 10</td>
<td>NS</td>
</tr>
<tr>
<td>LA dimension (mm)</td>
<td>38 ± 5</td>
<td>40 ± 6</td>
<td>NS</td>
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<tr>
<td>Gradient ≥ 30 mmHg</td>
<td>4 (15%)</td>
<td>5 (24%)</td>
<td>NS</td>
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<tr>
<td>LAD flow</td>
<td></td>
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<tr>
<td>Peak diastolic velocity (cm/s)</td>
<td>52 ± 28</td>
<td>45 ± 14</td>
<td>NS</td>
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<tr>
<td>Mean diastolic velocity (cm/s)</td>
<td>34 ± 18</td>
<td>29 ± 9</td>
<td>NS</td>
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<tr>
<td>Diastolic velocity time integral (cm)</td>
<td>24 ± 14</td>
<td>21 ± 7</td>
<td>NS</td>
</tr>
<tr>
<td>Diastolic deceleration time (ms)</td>
<td>975 ± 349</td>
<td>1045 ± 362</td>
<td>NS</td>
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<tr>
<td>IMCA flow</td>
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<td>Peak diastolic velocity (cm/s)</td>
<td>90 ± 44</td>
<td>101 ± 43</td>
<td>NS</td>
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<tr>
<td>Mean diastolic velocity (cm/s)</td>
<td>70 ± 35</td>
<td>51 ± 22</td>
<td>0.036</td>
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<tr>
<td>Diastolic velocity time integral (cm)</td>
<td>38 ± 19</td>
<td>29 ± 11</td>
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<tr>
<td>Diastolic deceleration time (ms)</td>
<td>989 ± 338</td>
<td>166 ± 67</td>
<td>&lt; 0.001</td>
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<td>201-thallium scintigraphy (n = 18)</td>
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<tr>
<td>Ischemia</td>
<td>0 (0%)</td>
<td>6 (67%)</td>
<td>0.009</td>
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</table>

ASH = asymmetric septal hypertrophy. Abbreviations as in Table 1.
and might be a cause of no reflow-like pattern in the IMCA.

Thallium scintigraphy could potentially provide a means of obtaining the cause of myocardial ischemia, which is reported in 50–80% of patients with HCM in the absence of epicardial coronary disease [25]. Previously, thallium perfusion defects during exercise have been attributed to exercise-induced ischemia due to microcirculation abnormalities [26,27]. Using transthoracic Doppler echocardiography in the present study, the presence of myocardial ischemia was found to be closely related to the flow velocity pattern in the IMCA. Thus, the characteristic intramyocardial coronary flow velocity pattern demonstrated in the present study might be related to exercise-induced ischemia due to microcirculation abnormalities.

Angina and myocardial ischemia are predominant features of HCM and ischemia may contribute to the development of syncope and sudden cardiac arrest in such patients [26]. Previous reports indicate that abnormalities of intramural coronary artery may be the cause of myocardial ischemia. It is thus possible that ischemia caused by these vascular abnormalities may eventually result in myocardial scarring and diminished left ventricular function [28]. In the present study, basal left ventricular thickness and coronary velocity profiles in the LAD were similar in the two groups, but cardiac events were frequently developed in patients with no reflow-like pattern in the IMCA, reflecting disturbed coronary microcirculations in such patients.

In this study, the characteristic pattern observed in the IMCA had no relationship to the pattern seen in the epicardial LAD flow. This discrepancy between IMCA and LAD flow suggests that the intramyocardial coronary flow pattern may express abnormalities of the microcirculation in relatively small areas. Thus, the intramyocardial coronary flow velocity measurement appears to be more sensitive than epicardial coronary flow analysis for detecting abnormal coronary microcirculation in patients with HCM.

**Clinical implications**

Visualization of small arteries by coronary arteriography is limited by the physical properties of radiographic systems. Although other imaging modalities (such as magnetic resonance imaging and positron emission tomography) may provide significant information regarding the functional and metabolic consequences of small coronary artery disease [29,30], transthoracic Doppler echocardiography is the only available method for directly detecting the flow velocity of the IMCA (500–1000 μm). Thus, this noninvasive technique has clinical merit for investigating and understanding of the role of IMCA physiology in coronary circulation, hemodynamics, and natural history of disease with left ventricular hypertrophy. Additionally, early detection of no reflow-like pattern in the IMCA by transthoracic Doppler echocardiography may provide a useful prediction of cardiac events in patients with HCM.

**Limitations**

There are several limitations in this study. The echocardiographic technique described here allows noninvasive assessment of phasic coronary flow velocity profiles in the LAD as well as IMCA. The methods have satisfactory interobserver and intraobserver variability. However, assessment of coronary flow velocity profiles in the IMCA using transthoracic Doppler echocardiography is restricted to the hypertrophic septal wall. Therefore, information about the rest of the wall cannot be used for comparison. We analyzed IMCA flow velocity from diastolic flow velocities alone and not from velocities throughout the entire cardiac cycle, because cyclic cardiac motion makes it difficult to obtain complete Doppler spectral envelopes especially in systoles. We did not measure coronary flow velocity reserve with intravenous infusion of adenosine, although we believe this measurement might be useful to find out the effect of microvessel mechanism on myocardial ischemia. However, the purpose of the present study was to define the relationship between IMCA flow velocity patterns and clinical manifestations, and we found a characteristic flow velocity pattern specific to microvascular damage even without intravenous infusion of adenosine. The thallium-201 scintigraphic evaluations could not be performed entirely at our institution. Although there were no clinical differences in the patients who underwent thallium-201 scintigraphy compared with those who did not undergo 201-thallium scintigraphy, the number of study patients was limited in the present study.

**Conclusion**

We evaluated coronary flow velocity profiles in the LAD and IMCA by transthoracic Doppler echocardiography in patients with HCM. Two different intramyocardial flow velocity patterns are noted.
No reflow-like pattern in the IMCA was related to cardiovascular symptoms and exercised-induced myocardial ischemia. Thus, measurements of IMCA flow using transthoracic Doppler echocardiography are useful for evaluating intramyocardial perfusion abnormality and prediction of cardiac events in patients with HCM.

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References

