Myocardial Flow Reserve in Long-Term Survivors of Repair of Anomalous Left Coronary Artery From Pulmonary Artery

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Objectives. This study sought to evaluate regional myocardial flow reserve in long-term survivors of repair of anomalous left coronary artery from pulmonary artery (ALCAPA) and to relate the flow abnormalities to the patients’ exercise performance.

Background. Patients with ALCAPA usually present during infancy with severe ischemic cardiomyopathy. The left ventricular function recovers after surgical repair. However, the extent of recovery of myocardial blood flow (MBF) and its potential physiologic significance in long-term survivors are unknown.

Methods. We evaluated MBF (ml/g per min) at baseline and during maximal coronary vasodilation by adenosine in 11 patients after ALCAPA repair (median age 17 years, range 7 to 22) using nitrogen-13 ammonia and dynamic positron emission tomographic imaging. Patients also underwent an incremental exercise test with metabolic monitoring. In each patient, MBF was quantified in the three major vascular territories: the left anterior descending and left circumflex coronary artery territories and the right coronary artery (control region) territory.

Results. Basal MBF was mildly reduced in the left coronary territories versus the control region (0.79 ± 0.14 vs. 0.85 ± 0.19, p = 0.05). During hyperemia, flow in the left coronary territories was significantly lower than that in the control region (2.1 ± 0.5 vs. 2.6 ± 0.5, p < 0.001). As a result, myocardial flow reserve was lower in the left coronary territories than in the control region (2.6 ± 0.7 vs. 3.2 ± 0.7, p < 0.001). Exercise performance was impaired in patients when compared with age-matched control subjects. Maximal oxygen consumption correlated linearly with maximal hyperemic flows in the left coronary artery territories (r = 0.73, p = 0.03).

Conclusions. Long-term survivors of ALCAPA repair demonstrate regional impairment of myocardial flow reserve. This may contribute to impaired exercise performance by limiting cardiac output reserve.

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ANOMALOUS CORONARY ARTERY

Anomalous origin of left coronary artery from pulmonary artery (ALCAPA) is a rare congenital anomaly. After birth, there is a physiologic decrease in pulmonary vascular resistance and pressure, which results in a critical reduction in perfusion of the left ventricular wall supplied by the anomalous left coronary artery. Patients usually present during infancy with congestive heart failure secondary to myocardial infarction and ischemic cardiomyopathy. Patient survival without operation is determined by the development of collateral channels from the right coronary artery. After surgical repair to establish blood flow to the left coronary artery from the aorta, dramatic improvement in left ventricular function occurs and normalizes within 2 to 3 years (1–3). However, the extent of recovery of myocardial blood flow (MBF) reserve and its potential physiologic significance in long-term survivors of surgical repair of ALCAPA are not known.

Positron emission tomography (PET) is an imaging modality that allows noninvasive quantification of regional MBF. Quantification of MBF using nitrogen-13 (N-13) ammonia and PET has been validated in animals and humans and has been shown to be both accurate and reproducible (4–7). Furthermore, flow measurements derived by PET correlate closely with the physiologic severity of coronary artery stenosis in patients with ischemic heart disease (8,9).

The purposes of this study were to evaluate regional MBF in long-term survivors of ALCAPA repair and to relate the flow abnormalities to the patients’ exercise performance. We used PET imaging with N-13 ammonia to assess MBF during baseline conditions and during maximal coronary vasodilation by adenosine. Patients also underwent a maximal exercise test with breath by breath analysis of oxygen consumption (\(V_{O2}\)) and carbon dioxide production during the test.

Methods

Study group. All patients who were ≥7 years and underwent repair of ALCAPA at our institution were invited to participate in the study. Of 16 eligible patients, 11 agreed to...
parasternal short-axis, parasternal long-axis and apical views by an expert reviewer (M.L.) who was unaware of the patients’ clinical, scintigraphic and exercise data. The left ventricle was divided into 16 segments, each of which was graded using the American Society of Echocardiography scoring system: 1 = normal; 2 = mild hypokinesia; 3 = severe hypokinesia; 4 = akinesia; and 5 = dyskinesia. A global score index was obtained by dividing the sum of the segment scores by the number of scored segments; thus, the more the index deviates from a score of 1, the worse the regional contraction abnormality.

**Positron emission tomography.** Dynamic PET measurements were performed using a whole-body PET scanner (model Siemens EXACT HR), which acquires 47 contiguous transaxial planes with an image resolution of 3.6 ± 0.23 mm at full-width half-maximum (FWHM) in-plane and 3.5 ± 0.18 mm FWHM in the axial direction. The PET scanner has an interplane spacing of 3.125 mm and covers a 15-cm axial field of view. The images were reconstructed using a Hanning filter with a cutoff frequency of 1.10 cycles per centimeter, resulting in an effective resolution of ≈6 mm FWHM.

A 20-min transmission scan was acquired for correction of photon attenuation. Beginning with an intravenous bolus administration of N-13 ammonia (0.286 mCi/kg), serial images were acquired for 20 min (12 frames of 10 s each, 4 frames of 30 s, 1 frame of 60 s and 1 frame of 900 s). Thirty minutes later, after physical decay of N-13 ammonia, 140 µg/kg per min of intravenous adenosine was infused over 6 min. Three minutes after initiation of adenosine infusion (maximal hyperemia), a second dose of N-13 ammonia (0.286 mCi/kg) was injected and images recorded in the same acquisition sequence. In this study the radiation exposure to children was low and in accordance with federal regulations (less than one-tenth of the maximal radiation exposure allowed for research in adults) (12). Sedation was not required for any patient. Patients were instructed to lie still during image acquisition; motion was minimized by fastening a Velcro strap across the patient’s chest. Heart rate, cuff blood pressure (Dynamap, Criticon Inc.) and a 12-lead ECG were monitored continuously throughout the procedure and recorded at baseline, every minute during the infusion and for 5 min after termination of adenosine infusion.

**Exercise protocol.** Before exercise testing, each patient underwent rest 12-lead electrocardiography. The patients then performed a maximal exercise test using a 1-min incremental bicycle protocol. All patients were asked to initially pedal in an unloaded state (6 W). Work rate was then increased each minute until maximal exercise. Work rate increment was based on age and was 10 W/min for age 8 to 9 years, 15 W/min for 10 to 13 years and 20 W/min for >13 years (13). Pulmonary function and breath by breath analysis of VO\textsubscript{2} and carbon dioxide production were performed using a Medgraphics Cardio2 metabolic cart. Electrocardiograms were obtained every minute during exercise and for the first 5 min of the recovery. Blood pressure was obtained every 3 min, whereas ECG and pulse oximetry saturations were continuously monitored. Max-
imal $V_O^2$ and ventilatory anaerobic threshold were measured for each subject. Ventilatory anaerobic threshold was determined by the ventilatory equivalent method (14).

**Analysis of PET data. Visual analysis.** Semiquantitative visual analysis was performed as described previously (15). Briefly, transaxial images were reoriented into six short-axis and six vertical long-axis slices of the heart. The entire myocardium was divided into 20 segments. The left ventricular apex (consisting of two segments) was evaluated from vertical long-axis slices. The remaining portions of the left ventricle were analyzed from short-axis slices, which were divided into distal, mid and basal levels. At each level, the myocardium was scored semiquantitatively in all 20 segments by visual analysis.

Corresponding rest and stress N-13 ammonia images were scored semiquantitatively in all 20 segments by visual analysis of one expert observer (M.D.C.) who had no knowledge of the rest of the patients' data. The following 5-point scoring system was used to indicate segmental N-13 ammonia uptake: 0 = absence of uptake (equal to background); 1 = severe defect; 2 = moderate defect; 3 = mild defect; and 4 = normal uptake (15). Segments with a stress defect were classified as completely reversible when the rest N-13 ammonia score was normal, partially reversible when the rest N-13 ammonia score improved by at least 1 point but did not normalize or irreversible when the rest N-13 ammonia score did not change.

**Quantification of MBF.** In each study, the 47 tomographic slices were reoriented into 12 short-axis slices of the heart extending from the apex to the base of the left ventricle using a SUN SPARC IPX work station (SUN Microsystems Inc.).

To quantify regional basal and hyperemic blood flows, sectorial regions of interest (ROI) encompassing each of the three major coronary territories (i.e., left anterior descending, left circumflex and right coronary artery territories) were automatically assigned to each of the four mid-ventricular short-axis slices of the N-13 ammonia images. Assignment of sectorial ROIs is based on radial activity profiles with a blood volume fraction of 50% to 60%, which allows incorporation of in-plane partial volume effects and blood to tissue cross-contamination into the model equation (16). To ascertain identical placement of these regions to the rest and hyperemic N-13 ammonia images, the same angle on the circumferential profile served as the starting point for the sectorial ROI on each of the two image sets that were analyzed in each subject. An additional small circular ROI was manually placed in the center of the left ventricular blood pool of the two most basilar left ventricular planes to obtain the arterial input function. The ROIs were then copied to the entire serially acquired N-13 ammonia image sequence, and regional myocardial tissue and blood pool time–activity curves were obtained. In each vascular territory, a single time–activity curve was obtained by averaging the corresponding N-13 ammonia data in adjacent ventricular planes. The time–activity curves were then fitted with a previously validated three-compartment tracer kinetic model (16). Because MBF at rest is related to the rate–pressure product, an index of cardiac work (17), basal flow values were normalized to the corresponding rate–pressure product in each patient by dividing the rest flow value by the rate–pressure product and multiplying by a linear factor of 8,000 in each individual patient. The myocardial vasodilator reserve was defined as the ratio of peak MBF during the administration of adenosine to the MBF under basal conditions.

**Statistical analysis.** The change in cardiac hemodynamic data from rest to maximal hyperemia (i.e., heart rate, arterial blood pressure and rate–pressure product) was assessed using the paired Student t test. In each subject, blood flows in the left coronary artery territories (the site of previous surgical repair) were compared with those measured in the right coronary artery territory (control region) using repeated measures analysis of variance. Estimates of $V_O^2_{max}$ and anaerobic threshold in the patients with ALCAPA were compared with those obtained in control subjects using the unpaired Student t test. The relation between $V_O^2_{max}$ and hyperemic blood flow was evaluated using linear regression analysis performed by least-squares fitting. All values are expressed as the mean value ± SD. A p value <0.05 was used to define statistical significance.

**Results**

**Left ventricular function.** The position of the interventricular septum during diastole was normal in all patients. None of the patients had ventricular aneurysm, and the ventricular shape was normal in all patients. The rest left ventricular ejection fraction was 63 ± 6% (range 54% to 73%). Regional wall motion, as determined by the global score index, was 1.07 ± 0.08 (range 1.0 to 1.2). Seven of the 11 patients had septal hypokinesia.

Two patients had mild mitral regurgitation by color Doppler echocardiography. Although mild turbulence in the supravalvular area was common, the estimated peak gradient was <20 mm Hg in all patients. None of the patients had baffle leaks in the intrapulmonary grafts.

**Hemodynamic findings.** Table 1 summarizes the hemodynamic findings at rest and during maximal hyperemia by adenosine in the patients with ALCAPA. During adenosine-induced hyperemia, there was a significant increase in heart rate and rate–pressure product. No significant changes were observed in systolic and mean arterial blood pressure.

**Regional MBF. Visual analysis.** Ten of the 11 patients with ALCAPA had adenosine-stress perfusion defects, typically located in the anterior and anterolateral ventricular walls (Fig. 439JACC Vol. 31, No. 2 SINGH ET AL. MYOCARDIAL FLOW RESERVE IN ALCAPA

| Table 1. Hemodynamic Changes With Adenosine |
|-----------------|-----------------|-----------------|
|                | Rest            | Adenosine       | p Value |
|-----------------|-----------------|-----------------|
| HR (beats/min)  | 69 ± 9          | 109 ± 15        | <0.01   |
| Systolic BP (mm Hg) | 108 ± 17      | 123 ± 28        | NS      |
| Mean BP (mm Hg) | 73 ± 9          | 76 ± 10         | NS      |
| Rate–pressure product (mm Hg × beats/min) | 7,452 ± 1,600 | 13,580 ± 4,424 | <0.01   |

Data presented are mean value ± SD. BP = blood pressure; HR = heart rate.
Stress Myocardial Perfusion

Basal Myocardial Perfusion

Figure 1. Positron emission tomographic N-13 ammonia images of the heart in short-axis views obtained in corresponded mid-ventricular levels at rest (bottom row) and during adenosine stress (top row) in a 16-year-old patient who underwent repair of ALCAPA during infancy. The images were oriented with the anterior wall on the top, the inferior wall on the bottom, the lateral wall to the right and the interventricular septum to the left. The stress images demonstrate a large blood flow defect of moderate severity throughout the anterior, anterolateral and lateral ventricular walls (left coronary territory). The rest images show near complete reversibility in all hyperperfused regions. The quantitative myocardial flow reserve by PET was reduced in the left coronary artery territories compared with control subjects (Table 2). As expected, this difference persisted after correction of basal flow for the rate–pressure product, an index of cardiac work (0.79 ± 0.19 vs. 0.85 ± 0.19 ml/g per min, p = 0.05) (Table 2).

Myocardial blood flow and flow reserve. The exercise test was maximal in 9 of 11 patients who underwent repair of ALCAPA during infancy. The images were oriented with the anterior wall on the top, the inferior wall on the bottom, the lateral wall to the right and the interventricular septum to the left. The stress images demonstrate a large blood flow defect of moderate severity throughout the anterior, anterolateral and lateral ventricular walls (left coronary territory). The rest images show near complete reversibility in all hyperperfused regions. The quantitative myocardial flow reserve by PET was reduced in the left coronary artery territories compared with control subjects (Table 2). As expected, this difference persisted after correction of basal flow for the rate–pressure product, an index of cardiac work (0.79 ± 0.19 vs. 0.85 ± 0.19 ml/g per min, p = 0.05) (Table 2).

Quantitative analysis. Basal flow. Basal MBF was mildly reduced in the left coronary artery territories compared with the right coronary territory (0.75 ± 0.19 vs. 0.81 ± 0.23 ml/g per min, p = 0.05) (Table 2). As expected, this difference persisted after correction of basal flow for the rate–pressure product, an index of cardiac work (0.79 ± 0.14 vs. 0.85 ± 0.19 ml/g per min, p = 0.05) (Table 2).

Hyperemic flow. During adenosine-induced hyperemia, blood flow increased significantly in both the left coronary artery territories (0.79 ± 0.14 to 2.07 ± 0.49 ml/g per min, p < 0.001) and the control region (0.85 ± 0.19 to 2.62 ± 0.50 ml/g per min, p < 0.001). Hyperemic blood flow was significantly lower, however, in the left coronary artery territories as compared with the control region (2.07 ± 0.49 vs. 2.62 ± 0.50 ml/g per min, p < 0.001) (Fig. 2). As a result, myocardial flow reserve (ratio of hyperemic to basal flow) in the left coronary territories was lower than that in the control region (2.6 ± 0.7 vs. 3.2 ± 0.7, p < 0.001). As expected, hyperemic flows were lower in the three patients with occluded grafts than in those with patent grafts (1.57 ± 0.47 vs. 2.23 ± 0.38 ml/g per min, p < 0.05). Importantly, hyperemic flows in myocardial regions served by the left coronary artery were lower than those in regions served by the right coronary artery in patients with occluded grafts (1.57 ± 0.47 vs. 2.34 ± 0.25 ml/g per min, p < 0.05), as well as in those with patent grafts (2.23 ± 0.38 vs. 2.78 ± 0.51 ml/g per min, p < 0.001).

Exercise performance. The exercise test was maximal in 9 of 11 patients with ALCAPA based on patient effort during the test and metabolic data (i.e., respiratory exchange ratio and VO₂ max). The exercise test was submaximal in the other two patients owing to leg fatigue, and so they were excluded from the analysis. Pulmonary reserve was normal in all patients. Despite a maximal test, three patients had chronotropic impairment (<85% of maximal predicted heart rate). Four patients had a blunted blood pressure response during exercise (<30 mm Hg increase in systolic blood pressure). Two patients had ST segment depression suggestive of myocardial ischemia. No rhythm abnormalities were observed in any patient. Variables of maximal (VO₂ max: 23.9 ± 5.3 vs. 33.5 ± 3.5 ml/kg per min, p < 0.01) and submaximal exercise performance (ventilatory anaerobic threshold: 14.0 ± 2.6 vs. 18.4 ± 2.2 ml/kg per min, p < 0.01) were significantly lower in patients compared with control subjects (Table 3).

Relation between exercise performance and maximal blood flow. In normal subjects, VO₂ max, a measure of maximal oxygen delivery to tissues during exercise, is known to be linearly related to cardiac output. As myocardial blood supply during exercise may determine left ventricular function and cardiac output, we correlated VO₂ max and peak flow in the left coronary artery territories (average of the left anterior descending and left circumflex coronary territories) in the nine patients with a maximal exercise test. A significant and linear correlation was observed between VO₂ max and hyperemic flow (r = 0.73, p = 0.03) (Fig. 3).

Discussion

The results of our study demonstrate, for the first time, that vasodilator reserve is reduced in myocardium supplied by the left coronary artery (site of previous surgical repair) in long-term survivors of surgical repair of ALCAPA. This is demonstrable in patients with both patent and occluded grafts. Furthermore, the linear correlation between peak flow and VO₂ max suggests that the reduced myocardial flow reserve may contribute to impaired exercise performance in these patients. These findings may be important because patients with severely reduced flow reserve, especially those with occluded grafts, may be at increased risk for adverse ischemic events.

Impaired myocardial flow reserve in the left coronary territories of long-term survivors of ALCAPA repair may have

Table 2. Myocardial Blood Flow and Flow Reserve

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<th>RCA (n = 11)</th>
<th>LCA (n = 22)</th>
<th>p Value</th>
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<tr>
<td>Myocardial blood flow (ml/g per min)</td>
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<tr>
<td>Basal</td>
<td>0.81 ± 0.23</td>
<td>0.75 ± 0.19</td>
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<tr>
<td>Basal, corrected*</td>
<td>0.85 ± 0.19</td>
<td>0.79 ± 0.14</td>
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<tr>
<td>Adenosine</td>
<td>2.62 ± 0.50</td>
<td>2.07 ± 0.49</td>
<td>&lt;0.001</td>
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<tr>
<td>Myocardial flow reserve</td>
<td>3.2 ± 0.7</td>
<td>2.6 ± 0.7</td>
<td>&lt;0.001</td>
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*Basal, corrected = corrected for rate–pressure product. Data presented are mean value ± SD. LCA = left coronary artery territories; RCA = right coronary artery territory.
different underlying mechanisms. In patients with occluded grafts, the marked impairment of vasodilator reserve is due, at least in part, to the fact that blood flow supply to the left coronary territory is dependent on collateral flow from the right coronary artery. This is supported by experimental and clinical studies demonstrating a diminished vascular reserve in collateral-dependent myocardial territories (18). In patients with patent grafts, the reduced hyperemic flow may be due to impaired vasodilation of the coronary graft, as reported recently in adults with aortocoronary bypass grafts (19). Alternatively, the impaired flow reserve may be due to the presence of patchy fibrosis in the interstitial tissue surrounding viable myocytes. Biopsy specimens taken from ischemic myocardium of patients with ALCAPA at the time of operation have shown increased collagen tissue surrounding viable myocytes (20). It may be speculated that the persistence of this perivascular fibrous tissue may diminish the vasodilator response of the microcirculation, even after normalization of myocyte structure and function. Further support for this mechanism is provided by clinical studies demonstrating variable degrees of reversible perfusion defects, as assessed by thallium scintigraphy, in the left coronary artery territories of patients undergoing ALCAPA repair (3,21–23). Importantly, in the study by Seguchi et al. (21), involving patients who had undergone direct aortic implantation of the left coronary artery, none of the patients had coronary stenosis to account for the observed perfusion abnormalities.

Could the right coronary region have “supernormal” flow reserve to account for this regional heterogeneity? Patients with ALCAPA develop collateral channels from their right coronary artery to jeopardized territories after reduction in perfusion pressure in their left coronary system. Although this results in an increase in the total amount of blood flowing through the epicardial right coronary artery, its effect on tissue perfusion in regions normally served by the right coronary artery is unknown. Evidence from experimental and clinical studies suggests that if a single normal coronary vessel provides perfusion to a large collateral-dependent zone, the vasodilator reserve in myocardium served by the “donor” artery is impaired (24,25). The pathophysiology of this impaired vasodilator reserve is not well understood (24). Although maximal flow to regions supplied by the normal vessel improves with time, it does not become supernormal (24). These experimental findings would support the use of the right coronary artery territory as the internal control region in our patients with ALCAPA. Furthermore, hyperemic blood flows and myocardial flow reserve have been shown to be regionally homogeneous and similar in both the right and left coronary artery territories in normal adults of all ages studied (18 to 80 years) (17). Therefore, in our patients the presence of hyperemic blood flow abnormalities, which were limited to the left coronary system, is a significant finding.

Another important finding in our study was that $V_{O2,max}$
during exercise correlated linearly and significantly with peak MBF in the left coronary territory during adenosine-induced hyperemia. In normal subjects and in patients with mild congestive heart failure, the ability of the left ventricle to increase cardiac output during exercise (cardiac output reserve) is the major determinant of VO$_2$max (26). It is possible that the reduced coronary flow reserve in the patients with ALCAPA resulted in impaired cardiac output increase, which contributed to their reduced exercise performance. The limited cardiac output reserve may have been mediated by chronotropic impairment or by limited ability of the left ventricle to increase stroke volume during exercise. Limited cardiac output reserve as the underlying mechanism for reduced VO$_2$ is further supported by ST segment depression during exercise in two patients and blunted blood pressure response in four of nine patients. Peripheral mechanisms are also important determinants of VO$_2$ during exercise in patients with moderate to severe heart failure and in those with metabolic abnormalities of skeletal muscle (26). Of note, all patients in this study had normal rest left ventricular function and all were asymptomatic. Although a relatively deconditioned status in our patients cannot be completely ruled out and remains a limitation of the study, a normal ratio of anaerobic threshold and VO$_2$max in this cohort suggests it to be a less likely mechanism of reduced exercise performance.

**Study limitations.** This study is limited by a relatively small patient group because of the rare incidence of this congenital anomaly. The heterogeneity in age at clinical presentation of these patients results in a variable degree of collateral development and myocyte loss before surgical repair. These may have been further modified by the duration of medical management before surgical repair, intraoperative myocardial protection and long-term graft patency after revascularization. Because of the small patient group, it is not possible to differentiate myocardial flow abnormalities between patient groups based on age at presentation, age at operation and graft type (arterial vs. venous). Second, although MBF is regionally homogeneous in normal adults, data in normal children <18 years old is not available because of ethical constraints concerning radiation exposure. Furthermore, the correlation between hyperemic MBF and VO$_2$max observed in this study may have resulted from a variable degree of conditioning. This relation may be more convincingly considered causal if there was direct evidence of exercise-induced ischemia, such as with dobutamine and exercise echocardiographic studies, which were not a part of this study. Finally, because cardiac catheterization was not part of our study protocol, we reported the angiographic status of the grafts as observed during the last cardiac catheterization. Therefore, we cannot exclude the possibility that some of the patients with previously patent grafts may have had graft stenosis or occlusion at the time of the PET study. However, our data show that myocardial flow abnormalities were present in almost all patients irrespective of the reported graft status. In fact, two patients showed moderate and near completely reversible stress defects in spite of the presence of a patent graft on selective coronary arteriography performed at the time of the PET study, suggesting a discrepancy between structure and function.

**Study implications.** Our results demonstrate that myocardial flow reserve is reduced in the left coronary artery territories of long-term survivors of ALCAPA regardless of the angiographic status of the graft. This may result in myocardial ischemia during periods of increased oxygen demand such as exercise and contribute to impaired exercise performance. Although the extent and severity of stress perfusion defects in adults with coronary artery disease have been shown to have prognostic significance and relate to adverse cardiovascular events (27), it is unknown whether these prognostic indicators are applicable to long-term survivors of ALCAPA repair. Long-term follow-up in a larger patient cohort will be
needed to answer this question. Based on our study, we would argue that these patients mandate a close clinical follow-up. Myocardial perfusion imaging or stress echocardiography should be used to identify patients with severely reduced flow reserve and exercise-induced ischemia. Advice on physical activities should be based on the result of these imaging studies. Anti-ischemic medications (e.g., beta-blockers, recommended to two patients in our study group) should be considered in patients with evidence of exercise-induced myocardial ischemia or severely reduced myocardial flow reserve. In addition, education of these patients regarding known risk factors for coronary artery disease is important.

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