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Exercise Capacity and Incidence of Myocardial Perfusion Defects After Kawasaki Disease in Children and Adolescents

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Objectives. **This study evaluated exercise performance and myocardial perfusion during exercise in patients with Kawasaki disease who had a broad spectrum of residual coronary abnormalities.**

Background. **Reports of exercise performance after Kawasaki disease have generally included a small number of patients evaluated by various protocols, frequently with incomplete data. Myocardial perfusion studies have usually been limited to those using pharmacologically induced coronary vasodilation. Therefore, to our knowledge there has not been a large study directly correlating exercise performance, electrocardiographic (ECG) changes and myocardial perfusion imaging.**

Methods. **Forty-six patients were classified into three groups on the basis of coronary artery status: group 1 (n = 27) had no objective evidence of coronary artery lesions; group 2 (n = 11) had resolved aneurysms; group 3 (n = 8) had persistent coronary aneurysms. All patients underwent exercise testing with monitoring of ECG changes and oxygen consumption. Single-photon emission computed tomographic imaging was performed at rest and during peak exercise using technetium-99m sestamibi.**

Results. **Maximal oxygen consumption was within normal limits and was similar for all three groups. Five patients had mild ST segment changes at peak exercise. Two of these patients had stress-induced perfusion defects. Myocardial perfusion defects were present in 37% of patients in group 1, 63% in group 2 and 100% in group 3. Perfusion defects corresponded to the coronary artery lesion site in all but three patients.**

Conclusions. **Maximal oxygen consumption is normal after Kawasaki disease regardless of coronary artery status. Stress-induced perfusion defects are frequent even in the absence of coronary abnormalities and are common in the absence of ST segment changes suggestive of ischemia.**

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Kawasaki disease is an acute febrile illness that results in demonstrable lesions of the coronary arteries in ~10% to 15% of the affected North American population. Reports of exercise performance in these patients have generally shown normal exercise capacity. However, these reports (1–3) have involved small numbers of patients evaluated by a variety of protocols and frequently with incomplete metabolic and cardiovascular data.

Studies of myocardial perfusion after Kawasaki disease (3–6) have largely been limited to nuclear imaging after pharmacologically induced coronary vasodilation. This method does not allow direct correlation between exercise performance, exercise-induced electrocardiographic (ECG) changes and myocardial perfusion imaging. Many of these studies were restricted to patients with either current or a history of previous coronary artery lesions. It is therefore difficult to assess the true incidence of coronary perfusion abnormalities in children who have had Kawasaki disease.

The purpose of this study was to evaluate myocardial perfusion during exercise using single-photon emission computed tomographic (SPECT) imaging in a large group of patients with a history of Kawasaki disease resulting in a broad spectrum of residual coronary artery abnormalities. These perfusion data were compared with the patient's simultaneous exercise performance and exercise-induced ECG changes.

Methods

Patients. Forty-six children and adolescents from the participating institutions with a previous history of Kawasaki disease formed the study group. Potential subjects were identified by review of the medical records of the participating institutions. All patients identified with the diagnosis of Kawasaki disease were eligible for inclusion in the study if they 1) met established diagnostic criteria for Kawasaki disease and were at least 6 months beyond the acute phase of their illness (7); and 2) were >6 years old at the time of testing. All patients contacted who agreed to participate were included in the study.

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The study patients were classified into three groups on the basis of coronary artery status: *group 1* = 27 patients with no previously demonstrated evidence of coronary artery lesions during any phase of the disease process; *group 2* = 11 patients with coronary artery aneurysms that had resolved completely before exercise testing; *group 3* = 8 patients with persistent coronary artery lesions at the time of exercise testing. Coronary artery status was established by either echocardiography or angiography within 6 months of the exercise studies in all patients. No patient had any evidence of left ventricular dysfunction or regional wall motion abnormalities at rest by echocardiography or angiography before exercise testing. Gender, age at diagnosis, age at exercise testing, coronary artery status and history of catheterization are summarized for each patient in Table 1. In group 2, resolved lesions had occurred primarily in the left coronary system (8 of 11), with the right coronary system involved in one patient and with bilateral involvement in two. Four patients in group 3 had diffuse bilateral lesions, and the remaining four had lesions confined to the left coronary system. One patient had persistent giant aneurysms (Table 1). No patient in any group had a stenotic lesion.

Exercise protocol. After written informed consent was obtained, all patients underwent exercise testing to maximal exertion. A bicycle protocol was used in all patients of sufficient body size. This consisted of 3 min of unloaded pedaling followed by a uniform increase in work rate each minute to peak exercise. Work rate increments were chosen on the basis of patient age (6 to 9 years old, 10 W/min; 10 to 13 years old, 15 W/min; ≥14 years old, 20 W/min). In patients too small for a cycle ergometer, a 2-min incremental treadmill protocol was used. Initial speed was 1.7 mph at 0% grade. Speed and grade were increased incrementally.

Depending on the institution, minute oxygen consumption ($\dot{V}O_2$), minute carbon dioxide production ($\dot{V}CO_2$), minute ventilation ($\dot{V}E$) and respiratory exchange ratio were monitored intermittently or continuously during the exercise test. A 12-lead ECG was obtained at rest in the supine position at 25- and 50-mm/s paper speed. A 12-lead ECG at both paper speeds was recorded at each stage of exercise testing and at maximal exercise.

Myocardial perfusion imaging. All patients underwent left ventricular myocardial perfusion imaging using SPECT with technetium-99m (Tc-99m) sestamibi as a radiopharmaceutical. All patients underwent a same-day rest–stress protocol. Rest Tc-99 sestamibi dosage was 7.4 to 11.1 MBq/kg, and the stress dose was 22.2 to 29.6 MBq/kg. The stress dose was injected at peak exercise 4 h after the rest image acquisitions. Both rest and stress images were reconstructed in standard horizontal long-axis, vertical long-axis and short-axis views. Regions of interest were defined as apical; septal; and anterior, lateral and inferior free wall. All defects were graded as either fixed (if present in both rest and stress images) or reversible (if present only with exercise, no partially reversible defects were observed in this study). Severity of perfusion defects was graded as follows: *normal* = no perfusion defects; *mild* = >50% perfusion of the surrounding normal myocardium; *moderate* perfusion <50% of the surrounding myocardium; *severe* = near or complete absence of regional perfusion.

Data analysis. All 12 lead ECGs at rest and at maximal exercise were evaluated by one reviewer who had no knowledge of the results of the exercise testing and myocardial perfusion imaging. Electrocardiographic ST segment changes were considered to show evidence of exercise induced ischemia if there was ST segment depression ≥1 mm that was either downsloping, flat or upsloping >80 ms after the J point.

All nuclear myocardial perfusion images were reviewed by the institutions that performed the imaging. Because of the incompatibility of various computer analyzing systems, no attempt was made to have all perfusion images read by a single reviewer. However, the imaging data from the first 20 patients were read by blinded reviewers from two separate participating institutions. There was a discordant reading for only one patient (read as normal at one institution and as a mild perfusion defect at another).

Maximal oxygen consumption data were compared with previously published data for healthy children of matched age and gender by Cooper et al. (8) using a similar bicycle protocol.

Statistical differences between the three patient groups were evaluated by one-way analysis of variance with correction for repeated measurements. A p value < 0.05 was considered significant.

Results

Exercise performance. Maximal heart rate, maximal $\dot{V}O_2$, and maximal respiratory exchange ratio are displayed for each patient in Table 1. There were no significant differences in any variables of exercise performance between three groups. Aerobic capacity as measured by maximal $\dot{V}O_2$ was generally normal for the study patients. Only seven patients (four from group 1, one from group 2 and two from group 3) had results <2 SD for maximal $\dot{V}O_2$ for age- and gender-matched healthy children, as reported by Cooper et al. (8). On the basis of low maximal respiratory exchange ratio values (<1.10), only the low $\dot{V}O_2$ values in group 3 patients can be attributed to a submaximal effort with exercise termination before exhaustion of aerobic capacity.

The remaining five patients in groups 1 and 2 had a maximal respiratory exchange ratio suggestive of significant metabolic acidosis at peak exercise and would support the conclusion that these patients achieved their maximal aerobic capacity despite their low maximal $\dot{V}O_2$ values. None of these patients had exercise-induced myocardial perfusion defects. One of these patients (Patient 9) had evidence of impaired chronotropic response with a decreased maximal $\dot{V}O_2$ and maximal heart rate of 162 beats/min. Patients 7 and 24 also had evidence of chronotropic impairment, although their maximal $\dot{V}O_2$ was within the normal range. All other patients with heart rates <175 beats/min appeared to represent submaximal tests rather than chronotropic impairment according to their maximal respiratory exchange ratio. None of the remaining subjects

Table 1. Clinical Characteristics of 46 Study Patients

Pt No./ Gender	Age (yr) At DX	Age (yr) At Test	CA Site	Ergo	Max HR (beats/min)	Max RER	Max $\dot{V}O_2$ (ml/ kg per min)	ECG	Tc-99m
Group 1									
1/M	5	13		1	184	1.17	45	−	Lateral (3)
2/F	9	12		1	188	1.27	29	−	Normal
3/F	6	9		2	207	1.24	27	−	Inferior (2)
4/M	4	8		1	198	1.27	32	−	Normal
5/M	5	6		2	192	1.21	47	−	Normal
6/F	4	14		1	185	1.30	26	−	Anterior (1)
7/M	9	10		1	166*	1.18	40	+	Normal
8/M	5	6		2	184	1.18	37	−	Normal
9/F	3	6		2	162*	1.28	25†	−	Normal
10/M	5	8		2	192	1.15	30	−	Normal
11/M	3	9		1	185	1.24	48	+	Inferoapical (2)
12/M	1	9		2	198	1.28	54	−	Inferolateral (2)
13/M	3	6		2	190	1.14	26†	+	Normal
14/F	3	7		2	185	1.17	44†	−	Normal
15/F	8	9		1	195	1.22	22†	−	Normal
16/M	2	7	‡	1	189	1.26	33	−	Septolateral (3, 3)
17/M	3	7		1	190			+	Normal
18/F	0.5	9		2	173*	1.04§	34	−	Inferior (1)
19/M	5	8		2	153*	1.01§	34	−	Apical (1)
20/M	4	9		2	165*	1.00§	41	−	Normal
21/M	3	16		1	160*	1.04§	41	−	Normal
22/M	2	13		1	110*	0.97§	32	−	Anterior (2)
23/M	7	17		1	170*	1.04§	40	−	Normal
24/M	1.5	15		1	157*	1.44	28	−	Inferior (2)
25/F	6	7		2	197	1.11	37	−	Normal
26/M	1	9		1	177	1.42	31	−	Normal
27/F	5	13		1	195	1.39	28†	−	Normal
Mean ± SD	4 ± 2	10 ± 3			180 ± 20	1.19 ± 0.13	35 ± 8		
Group 2									
28/F	2	7	LMCA	1	179	1.22	29	−	Lateral (2)
29/F	5	8	LMCA	1	193	1.29	28	−	Inferior (3)
30/F	5	8	LMCA‡	2	204	1.11	33	−	Septoinferior (2, 2)
31/M	1.5	9	LMCA/proximal RCA	1	204	1.44	35	−	Inferior (1)
32/M	7	10	LMCA	1	177	1.32	28†	−	Normal
33/F	4	14	LMCA	1	197	1.48	34	−	Anteroseptal (2, 2)
34/M	3	8	LMCA	2	175	1.07§	43	−	Normal
35/M	8	9	Diffuse RCA	1	188	1.15	38	+	Inferolateral (2)
36/M	4	8	LMCA	2	193	1.09§	27	−	Inferolateral (2)
37/F	5	8	Diffuse LMCA/proximal RCA	2	205	0.94	43	−	Normal
38/M	2	8	LMCA	1	176	1.37	32	−	Normal
Mean ± SD	4 ± 2	9 ± 2			190 ± 12	1.23 ± 0.17	34 ± 6		
Group 3									
39/M	0.5	10	Diffuse bilateral	1	193	1.37	47	−	Inferior (2)
40/M	7	18	Diffuse bilateral	1	193			−	Anteroinferior (3, 3)
41/M	3	13	LMCA, LAD‡	1	189	1.27	42	−	Anterior (fixed) (1)
42/M	4	10	Diffuse bilateral‡	2	177	1.12	42	−	Inferoapical (fixed) (3, 3)
43/M	5	13	Giant LMCA‡	1	192	0.98§	15.5†	−	Anteroseptal (2)
44/M	1	9	LMCA‡	2	180	0.92§	22†	−	Inferoanterior (1, 2)
45/M	7	10	Diffuse bilateral‡	1	179	1.02§	34	−	Anterior (1)
46/M	3	8	LMCA‡	2	190	0.95§	51	−	Inferior (1)
Mean ± SD	4 ± 2	11 ± 3			187 ± 7	1.09 ± 0.17	36 ± 13		

*Heart rate (HR) <175 beats/min. †<2 SD of normal, from Cooper et al. (5). ‡Diagnosis (Dx) confirmed by angiography. §Maximal (Max) respiratory exchange ratio (RER) suggestive of submaximal effort. CA = coronary aneurysm; ECG = ischemic changes on electrocardiogram (+ = positive; − = negative); Ergo = Ergometer (1 = bike; 2 = treadmill); F = female; LAD = left anterior descending coronary artery; LMCA = left main coronary artery; M = male; RCA = right coronary artery; Pt = patient; Tc-99m = site of perfusion defect by technetium 99-m (1 = mild; 2 = moderate; 3 = severe); $\dot{V}O_2$ = minute oxygen consumption.

(Patients 18 to 23) had a maximal respiratory exchange ratio >1.04. Despite their submaximal efforts, all these patients had a maximal $\dot{V}O_2$ within the normal range. Of those patients with maximal heart rates >175 beats/min, Patients 34 and 43 to 46 appear to have a submaximal test on the basis of a RER <1.10. However, maximal $\dot{V}O_2$ values are within the normal range for Patients 34, 45 and 46.

Electrocardiographic data. All patients had normal rest ECG results. There were no ST segment abnormalities or evidence of previous infarction. At maximal exercise, five patients had ST segment changes suggestive of possible myocardial ischemia in the left lateral precordial leads at maximal exercise. In all five patients, these changes consisted of 1 mm upward-sloping ST segment depression resolving within 3 min of recovery. Two of these patients had evidence of stress-induced regional myocardial perfusion defects (Table 1).

Nuclear perfusion imaging. Ten (37%) of the 27 patients in group 1 had evidence of at least one stress-induced perfusion defect. Patient 16 had two separate lesions in the lateral free wall and septum. All lesions in this group were reversible, and none were associated with any ECG changes (Table 1).

Seven (63%) of 11 patients in group 2 had evidence of at least one stress-induced perfusion defect. Patients 30 and 33 had two separate lesions. All lesions were reversible. Patient 35 had associated ST segment changes with exercise. All lesions except for that in Patient 29 were at least partially located in the regions of the myocardium that were perfused by a previously aneurysmal coronary artery (Table 1).

All eight patients in group 3 had perfusion defects that were reversible in 6. All lesions in Patient 41 and one in Patient 42 were nonreversible. Two separate defects were present in Patients 40, 42 and 44. All perfusion defects except those in Patient 46 and one in Patient 44 were in the region of the myocardium supplied by the persistently aneurysmal coronary arteries (Table 1).

Discussion

Exercise performance. There has been limited evaluation of exercise performance and aerobic capacity in patients after Kawasaki disease. In 1992 Allen et al. (1) reported the results of a retrospective study in 47 patients. Expired gases were measured in only 23 of their patients. They noted that, as a group, work rate, heart rate response and maximal $\dot{V}O_2$ were within the normal range for healthy children. No data for individual patients were presented.

Data from our study would confirm that, taken as a group, exercise performance and aerobic capacity are within the range of normal healthy age- and gender-matched patients. This finding appears to be true regardless of coronary artery status. However, there does appear to be wide variation in the aerobic capacity of our study patients because five (11%) performed maximal studies that were below the range of normal for aerobic capacity. The reason for this low aerobic capacity in these five patients is unclear. None of them had evidence of myocardial perfusion defects on nuclear imaging. One patient (Patient 13) did have mild ECG ST segment changes. Patient 9 had chronotropic impairment, with a maximal heart rate of 162 beats/min. There is no obvious reason for the impaired aerobic capacity of the remaining three patients, although it may be a result of poor cardiovascular conditioning due to a sedentary life-style. However, we do not have physical activity data to support or reject such a possibility.

No patient with a myocardial perfusion defect had an aerobic capacity below the normal range. It would appear that such perfusion defects do not result in sufficient impairment of cardiac function to cause below normal aerobic capacity. In fact, group 3 patients had a mean maximal $\dot{V}O_2$ similar to that in the other two groups despite the presence of myocardial perfusion abnormalities in all of these patients. However, the range of normal maximal $\dot{V}O_2$ for the population in the age range that we studied is quite broad (~28 to 55 ml/kg per min) (8). It is certainly possible that some of the patients in the present study who were within the low normal range of maximal $\dot{V}O_2$ values might have had higher $\dot{V}O_2$ values if they had normal myocardial perfusion. Serial testing of these patients would be necessary to confirm such speculations.

Electrocardiography. The major ECG finding of this study is the lack of exercise-induced ST segment changes in the study patients despite a high incidence of exercise-induced myocardial perfusion defects. This finding is in agreement with previously reported studies. Tatara et al. (3) examined the effects of exercise on ST segment changes in 30 patients with angiographically demonstrated stenosis or occlusion. They found very good correlation between exercise-induced ST segment changes and myocardial perfusion defects imaged during dipyridamole vasodilation for lesions in the left main and left anterior descending coronary arteries. However, there was no correlation for lesions in the circumflex and right coronary artery systems. Other reports (2,5,6) have shown that the presence of aneurysms without overt stenosis correlate very poorly with exercise-induced ST segment changes regardless of lesion site.

The reason for the lack of exercise-induced ST segment changes in the setting of a high incidence of exercise-induced myocardial perfusion defects is unclear. In previous studies (1–6), except for Paridon et al. (2), the exercise testing was performed separately from nuclear imaging, which was performed after pharmacologic coronary vasodilation. Because no data from these previous studies are available regarding the amount of stress achieved during exercise, ST segment–myocardial perfusion discordance might be explained on the basis of an inadequate effort during exercise. However, the present study clearly shows this not to be the case because myocardial imaging was performed using exercise testing to achieve coronary vasodilation. Therefore, direct comparison of ST segment changes and myocardial perfusion can be made while the coronary arteries are in the same state of vasodilation. The present study clearly shows a large number of exercise-induced perfusion defects that do not result in ST segment changes.

Myocardial perfusion imaging. The most striking aspect of the data from our current study is the large number of patients

with normal coronary arteries by imaging who have evidence of perfusion defects on stress nuclear imaging. The finding of perfusion defects in 37% of those patients with no previous history of coronary abnormalities is particularly disconcerting. Previous studies of Kawasaki disease have generally focused on those patients with persistent coronary abnormalities or resolved lesions. Little data are available on those patients who never had gross anatomic coronary artery lesions. Fukazawa et al. (5) recently reported on a series of 16 patients with anatomically normal coronary arteries who underwent nuclear perfusion imaging after pharmacologic coronary vasodilation. Of these, six had normal echocardiographic findings in the acute phase of illness. Two of these six patients had perfusion defects, a proportion similar to that seen in group 1 of our current study.

Certainly there have been varying degrees of false positive perfusion defects noted in adult populations with coronary artery disease (9). This finding has lead to the use of control values that are gender specific for correction of attenuation and tissue overlap in adult populations. Unfortunately, no such data are available for children. Kondo et al. (6) reported the only attempt to establish normal SPECT perfusion patterns in children. However, their normal population consisted in part of children with a history of Kawasaki disease and no known coronary artery involvement (the same criteria as our group 1). These children are not a suitable "normal" population, as the data from our study suggest. It is therefore difficult to know with any certainty how the presence of any false positive perfusion defects might be reflected in our data. This remains a potential limitation of our study.

The mechanism responsible for any true perfusion defects seen in these patients must presumably be related to either distal stenotic lesions that were missed by echocardiography or perhaps to an abnormal vasodilatory response to exercise or to pharmacologic vasodilation. The lack of angiography to assess distal coronary artery anatomy in group 1 and 2 patients makes the former possibility difficult to exclude, although echocardiography has generally been considered reliable in identifying coronary lesions compared with angiography. Abnormal intimal thickening has been shown to be involved in the process by which aneurysmal coronary arteries remodel after Kawasaki disease. Sugimura et al. (10) have shown angiographically that coronary arteries that have been previously aneurysmal have an abnormal response to vasodilators. Patients with no previous history of aneurysms had a vasodilator response similar to a control population without Kawasaki disease. A more recent study by the same group (11) using intravascular ultrasound imaging of the coronary arteries demonstrated increased intimal thickening in many regions of resolved aneurysms but normal intimal thickness in those patients with Kawasaki disease but no previous history of a coronary artery lesion.

The findings of Sugimura et al. (11) would predict normal myocardial perfusion for patients such as those in group 1 of our study. A possible explanation for this discrepancy may be suggested by the pathologic findings at the acute stage of the illness. Fujiwara et al. (12) reported that a global coronary vasculitis is present in those children who die during the acute phase of Kawasaki disease. We speculate that in some patients this vaculitis might result in microvascular disease but not subsequent aneurysm formation in the large epicardial vessels. The microvasculature then might have limited ability to vasodilate in response to exercise or to pharmacologically induced stress and could produce regional perfusion defects even in patients such as those evaluated by Sugimura et al. (10) who had normal dilation of the larger epicardial vessels. Although nothing in our current data support this speculation, we believe that more research into the state of the microvasculature in patients with Kawasaki disease is warranted.

It is difficult to assess the clinical implications of these large numbers of perfusion defects in patients with no objective coronary artery lesions. This is particularly true for those patients in group 1 who have previously been considered to be at low risk for ischemic cardiac disease, at least in the childhood period (13). Certainly, the findings of the present study would support continued surveillance of all children with a history of Kawasaki disease regardless of coronary artery status.

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