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Evaluation of Coronary Circulation by ^{13}N-Ammonia Myocardial Perfusion Positron Emission Tomography in Patients with Right Coronary Artery Occlusion Due to Kawasaki Disease

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Background: Although occlusion of the right coronary artery (RCA) is common in the remote stages of Kawasaki disease, revascularization of the RCA is challenging in children and is usually managed by observation without intervention.

Methods: Using adenosine-stress ^{13}N-ammonia myocardial perfusion positron emission tomography, we evaluated coronary circulation in 14 patients (12 males) with RCA occlusion to identify ischemia (myocardial flow ratio < 2.0) in the RCA region and examined hemodynamics, cardiac function, and coronary aneurysm diameter. These variables were also compared in patients with/without RCA segmental stenosis (SS).

Results: There were five cases of ischemia in the RCA region. RCA myocardial blood flow (MBF) at rest was higher in patients with ischemia than in those without ischemia, but the difference was not significant (1.27 ± 0.21 vs. 0.82 ± 0.16 mL/min/g, p = 0.2053). Nine patients presented with RCA SS, and age at onset of Kawasaki disease tended to be lower in those with SS. The maximum aneurysm diameter of RCA was significantly smaller in patients with SS (10.0 ± 2.8 vs. 14.7 ± 1.6, p = 0.0239). No significant differences in other variables were observed between patients with/without ischemia and SS.

Conclusions: At rest, MBF in the RCA region was relatively well preserved, even in patients with RCA occlusion, and there was no progressive deterioration in cardiac function. Adenosine stress showed microcirculatory disturbances in only half of the patients, indicating that it is reversible in children with Kawasaki disease. (J Nippon Med Sch 2024; 91: 277–284)

Key words: Kawasaki disease, ^{13}N-ammonia positron emission tomography, coronary micro-circulation, segmental stenosis, right coronary artery occlusion

Introduction

Kawasaki disease, a vasculitis that predominantly affects infants and young children, causes severe coronary arteritis leading to dilated lesions and coronary artery aneurysms in the acute phase of the disease. Patients with coronary artery aneurysms require lifelong careful follow-up[1]. Thrombosis readily occurs within a coronary aneurysm, and significant intimal proliferation can lead to coronary artery stenosis or occlusion in the remote stage. Occlusion of the right coronary artery (RCA) can lead to acute myocardial infarction (AMI). However, asymptomatic occlusion is not uncommon[2,3], while AMI is common in adults. In adults, percutaneous catheter intervention (PCI) is the mainstay of treatment for AMI. However, PCI is often contraindicated for children, primarily owing to differences in their physique, and coronary artery bypass grafting is extremely difficult, especially for the RCA, because of underdevelopment of the internal mammary artery. Therefore, revascularization of RCA occlusion in childhood is challenging, and most pa-

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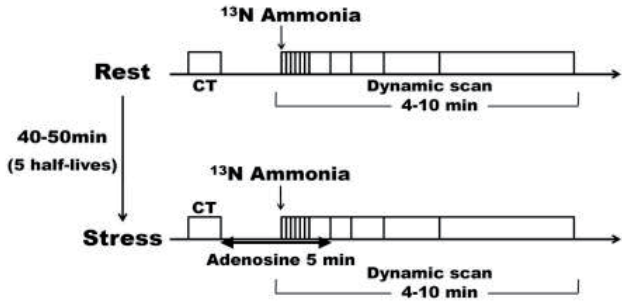

Fig. 1 ^{13}N-ammonia PET scan.
The same protocol was used for the resting and loading examinations, except for adenosine administration, and the interval between the resting and loading examinations was 4–5 half-lives.
CT: computed tomography.

tients are therefore followed up with medical therapy. In clinical reports, occlusion of the RCA caused problems related to myocardial ischemia and worsening of cardiac function because it is associated with segmental stenosis (SS) and development of collateral blood vessels[4,5].

^{13}N-ammonia myocardial perfusion positron emission tomography (ammonia PET) has facilitated evaluation of myocardial blood flow (MBF) in the territory of individual coronary arteries[6]. It enables a more detailed quantitative assessment of the coronary circulation, and ammonia PET is useful in diagnosing nonobstructive coronary arterial disease (INOCA) and evaluating myocardial microcirculatory disturbances caused by low attenuation plaque in adults[7,8]. Ammonia PET has been used to evaluate complex coronary circulation in Kawasaki disease[9,10].

To evaluate hemodynamics in the RCA region, we retrospectively reviewed data from patients with RCA occlusion due to Kawasaki disease coronary artery lesions who underwent adenosine-stress ammonia PET.

Materials and Methods

Participants

Medical records were collected for 51 patients with previous Kawasaki disease and cardiac complications who underwent adenosine-stress ammonia PET between July 2016 and December 2021 at our hospital for close examination of myocardial ischemia, and data from 14 patients (12 males) with RCA occlusion confirmed by coronary angiography or coronary computed tomography (CT) angiography before the examination were analyzed in this study.

This study was approved by the Ethics Committee of Nippon Medical School Hospital (approval number: 2023-930) and conducted in accordance with the Code of Ethics based on the Declaration of Helsinki.

Adenosine-Stress Ammonia PET Scan

Figure 1 shows the protocol for the adenosine-stress ammonia PET scan. After a chest CT scan for attenuation correction, 7.4 MBq/kg of ^{13}N-ammonia tracer was administered via the right elbow vein within 30 s, and data were collected for 10 min simultaneously with the administration. Data from 4 min after administration (6 s × 20, 30 s × 2, 60 s × 1) were used to quantify MBF by compartment model analysis. Absolute values of MBF were determined using a 1-issue (intravascular - intramyocardial), 2-compartment model. MBF was calculated from the time-activity curve of the counts of tracer inflow into the intramyocardial blood pool and the counts of tracer ingested into the myocardium. Electrocardiography-gated left ventricular function analysis was performed using the last 5 min of imaging, and left ventricular end-diastolic volume, left ventricular end-systolic volume, and left ventricular ejection fraction (LVEF) were calculated. Adenosine stress was started at 40-50 min after resting imaging (4-5 half-lives). Adenosine 144 μg/kg/min was administered through the left elbow vein over 5 min while monitoring the electrocardiogram, blood pressure, and oxygen saturation. Three minutes after adenosine administration, tracer administration and imaging were performed in the same manner as at rest. Left ventricular function analysis was also performed in the same manner as that at rest.

By quantitative MBF analysis, rest and stress MBF of the left anterior descending artery (LAD) territory, left coronary circumflex artery (LCX) territory, and RCA territory were calculated. Myocardial flow reserve (MFR) was also calculated as the ratio of stress to resting MBF. Microvascular resistance of the LAD and LCX was calculated from the diastolic blood pressure measured in the right upper extremity and the MBF (microvascular resistance = MBF/diastolic blood pressure).

In this study, myocardial ischemia was defined as an area with an MFR less than 2.0, as in a previous report[11]. The 14 cases were subdivided into 2 groups as those with and without myocardial ischemia in the RCA territory. The MBF and microvascular resistance at rest and during stress and the MFR of each coronary artery territory were compared between these groups. The results of blood flow scores by summed stress score (SSS), summed rest score (SRS), and summed difference score (SDS) were also examined between the groups. Additionally, we used the medical records to collect data on maximum

Table 1 The internal medication status

Aspirn	14
Candesartan	7
Warfarin	4
Carvedilol	2
Atorvastatin	1
Fluvastatin	1
Dipyridamole	1
Enarapril	1

aneurysm diameter of the LAD, LCX, and RCA; the presence of major adverse cardiac events (MACE); the number of remaining branches of the cardiac artery lesion (CAL) in the remote stage; the presence of SS of the RCA; and the presence or absence of a collateral artery in the left coronary artery (LCA), as determined by coronary angiography or coronary CT scan within 6 months of the PET scan. In addition, tricuspid annular plane excursion (TAPSE) was obtained by echocardiography and compared. SS in the RCA was classified into 3 types, in accordance with the method of Suzuki et al.[5], as follows: 1) braid-like lesions, 2) bridging lesions, and 3) pericoronary arterial communications. We compared and examined the above indicators in relation to the presence of SS.

Statistical Analysis

The median (range) was used for age (in months) at Kawasaki disease onset and the number of months between onset of Kawasaki disease and the PET scan. Other continuous variables were expressed as mean ± SD, and comparisons were performed using the Mann-Whitney test. Fisher's two-tailed test was used to compare nominal scales. Statistical analysis was performed with JMP statistical software Ver. 16 (SAS Institute Inc., Cary, NC, USA).

Results

The median (range) age of the 14 patients (12 males) was 24 (3-70) months at the onset of Kawasaki disease, and the interval from onset of Kawasaki disease to the PET scan was 191.5 (50-516) months. The medication status of the 14 patients is shown in **Table 1**. Four patients with LAD, 2 with LCX, and 5 with RCA had an MFR < 2.0. The number of branches with CAL in the acute phase was 1 branch in 1, 2 branches in 2, and 3 branches in 11 patients, and the number of branches with CAL on PET scans was 1 branch in 3, 2 branches in 3, and 3 branches in 8: Lesions of the LAD and LCX were small aneurysms or dilated lesions, and no ischemia-causing aneurysms or stenotic lesions were observed. Eight patients had a history of MACE, including 2 with AMI and 7 who underwent coronary artery bypass surgery (CABG). CABG was performed with LITA-LAD in all cases.

Comparison of RCA Regions with and without Ischemia

Table 2 shows a comparison of indices in the classification of patients with and without ischemia (MFR < 2.0 or ≥ 2.0) in the RCA territory. Age at onset of Kawasaki disease was lower in patients with ischemia in the RCA territory, but the difference was not significant (19.4 ± 8.5 vs. 34.3 ± 7.5, p = 0.1601). A trend toward increased MBF in patients with ischemia in the RCA region at rest (1.27 ± 0.21 vs. 0.82 ± 0.16 mL/min/g, p = 0.2053) (**Fig. 2A**) and a similar trend for blood flow in the LAD territory (1.37 ± 0.66 vs. 0.90 ± 0.25 mL/min/g, p = 0.2053) (**Fig. 2B**) were observed. Stress MBF in the RCA territory tended to be lower in the ischemia group (1.51 ± 0.87 vs. 2.41 ± 1.49 mL/min/g, p = 0.1615) (**Fig. 2C**), indicating both a high MBF at rest and poor MBF increase during stress in MFR < 2.0. Furthermore, patients with ischemia in the RCA territory tended to have lower microvascular resistance at rest in the LAD and LCX (LAD: 44.5 ± 17.0 vs. 51.9 ± 14.3, p = 0.1615, LCX: 56.9 ± 16.1 vs. 64.4 ± 14.3, p = 0.2571) and lower resistance under adenosine stress (LAD: 17.0 ± 8.9 vs. 21.2 ± 7.5, p = 0.3645, LCX: 19.6 ± 7.6 vs. 22.3 ± 6.2, p = 0.3173). There was no difference in LVEF, TAPSE, or N-terminal pro-brain natriuretic peptide or in SSS, SRS, or SDS between patients with and without ischemia in the RCA region and no significant difference in collaterals from the LCA.

Comparison in RCA with and without SS

SS was present in 9 of the 14 patients and was classified as braid-like lesions in 2 patients, bridging lesions in 4 patients, and pericoronary arterial communications in 3 patients. There was no significant difference in ischemia in the RCA region between patients with and without SS (**Table 3**). Similarly, there was no significant difference in MBF across each branch. Age (in months) at onset of Kawasaki disease tended to be lower in SS cases (22.1 ± 17.5 vs. 41.4 ± 25.4, p = 0.1239) (**Fig. 3A**). The maximum aneurysm diameter of RCA was significantly smaller in SS (10.0 ± 2.8 vs. 14.7 ± 1.6, p = 0.0239) (**Fig. 3B**). There was no significant difference in LVEF, TAPSE, or N-terminal pro-brain natriuretic peptide or in SSS, SRS, or SDS between patients with and without SS, and there was no significant difference in relation to the presence of collaterals from the LCA.

Table 2 Examination based on the presence or absence of ischemia in the RCA region

	Without RCA ischemia (n = 9)	With RCA ischemia (n = 5)	p
Age at onset of Kawasaki disease (months)	34.3 ± 7.5	19.4 ± 8.5	0.1601
Kawasaki disease course in months (months)	223.4 ± 121.3	219.6 ± 147.2	0.8938
Age at time of inspection (months)	257.9 ± 120.9	239.2 ± 137.4	0.5934
LAD resting MBF (mL/min/g)	0.90 ± 0.25	1.37 ± 0.66	0.2053
LCX resting MBF (mL/min/g)	0.84 ± 0.28	0.98 ± 0.29	0.2571
RCA resting MBF (mL/min/g)	0.82 ± 0.16	1.27 ± 0.21	0.2053
LAD stress MBF (mL/min/g)	2.52 ± 1.26	2.80 ± 1.60	0.5485
LCX stress MBF (mL/min/g)	2.26 ± 0.62	2.10 ± 0.68	0.4634
RCA stress MBF (mL/min/g)	2.41 ± 1.49	1.51 ± 0.87	0.1615
LAD resting vascular resistance	59.1 ± 14.3	44.5 ± 17.0	0.1615
LCX resting vascular resistance	64.4 ± 14.3	56.9 ± 16.1	0.2571
LAD stress vascular resistance	21.2 ± 7.5	17.0 ± 8.9	0.3645
LCX stress vascular resistance	22.3 ± 6.2	19.6 ± 7.6	0.3173
LAD MFR	2.87 ± 1.33	2.02 ± 0.48	0.1615
LCX MFR	2.78 ± 0.72	2.16 ± 0.35	0.1251
RCA MFR	2.95 ± 1.49	1.22 ± 0.34	0.0056
SSS	11.67 ± 10.32	14.00 ± 2.61	0.2800
SRS	4.89 ± 6.67	3.20 ± 3.06	0.2797
SDS	6.78 ± 8.20	10.8 ± 2.93	0.1205
CABG Yes/No	5/4	2/3	1.0000
MACE with/without	5/4	3/2	1.0000
LAD aneurysm max diameter (mm)	10.0 ± 2.4	10.0 ± 4.4	0.9244
LCX aneurysm max diameter (mm)	8.6 ± 2.4	7.1 ± 5.4	0.2558
RCA aneurysm max diameter (mm)	12.0 ± 4.1	11.7 ± 2.0	1.0000
Number of remote CAL branches	2.8 ± 0.4	1.6 ± 0.9	0.0169
LVEF	0.70 ± 0.11	0.68 ± 0.10	0.7386
TAPSE (mm)	19.9 ± 4.5	17.2 ± 5.1	0.4404
NT-ProBNP (pg/mL)	45.3 ± 32.7	48.8 ± 34.1	0.8415
Collateral from LCA Yes/No (Number of cases diagnosed by cardiac catheterization)	5/4 (3/1)	3/2 (3/1)	1.0000
RCA SS	5/4	4/1	0.5804

MBF, myocardial blood flow; RCA, right coronary artery; LAD, left anterior descending artery; LCX, left circumflex artery; MFR, myocardial flow ratio; CABG, coronary artery bypass graft; MACE, major adverse cardiovascular event; CAL, coronary artery lesion; LVEF, left ventricular ejection fraction; LCA, left coronary artery; SS, segmental stenosis.

Discussion

In this study, we used ammonia PET to quantitatively evaluate hemodynamics of each coronary artery territory in the remote stage of Kawasaki disease with RCA occlusion. Among the 14 patients, 5 exhibited ischemia in the RCA but no significant differences in relation to CABG, cardiac function, collateral blood flow from the LCA, or MACE. Patients with ischemia in the RCA territory showed a trend toward higher resting MBF, not only in the RCA but also in the LAD and LCX territories. This result may reflect the physiological response of microvasculature to epicardial stenotic lesions to prevent myocardial ischemia. This finding is consistent with a study by Murthy et al.[11], which reported that MBF at rest is relatively well preserved in coronary artery stenosis because of autonomic regulation of vasodilation. The present study also demonstrated that resting MBF in the occluded RCA territory was not reduced.

As a rule, decreased MFR is correlated with the severity of coronary artery stenosis[12]. However, 9 of the present 14 cases of RCA occlusion did not show decreased MFR. Myocardial ischemia was not related to angiographic collateral blood flow from the LCA, suggesting that in cases without ischemia, the increase in collateral blood flow at the microvasculature level compensates for the increased blood volume in the RCA region. In this study, patients with ischemia tended to develop Kawasaki disease at a younger age, which suggests that vasculitis at a younger age inhibits peripheral vascular bed development. Nevertheless, whether this increase in peripheral vascular beds is due to spontaneous development at a young age or induced by myocardial ischemia

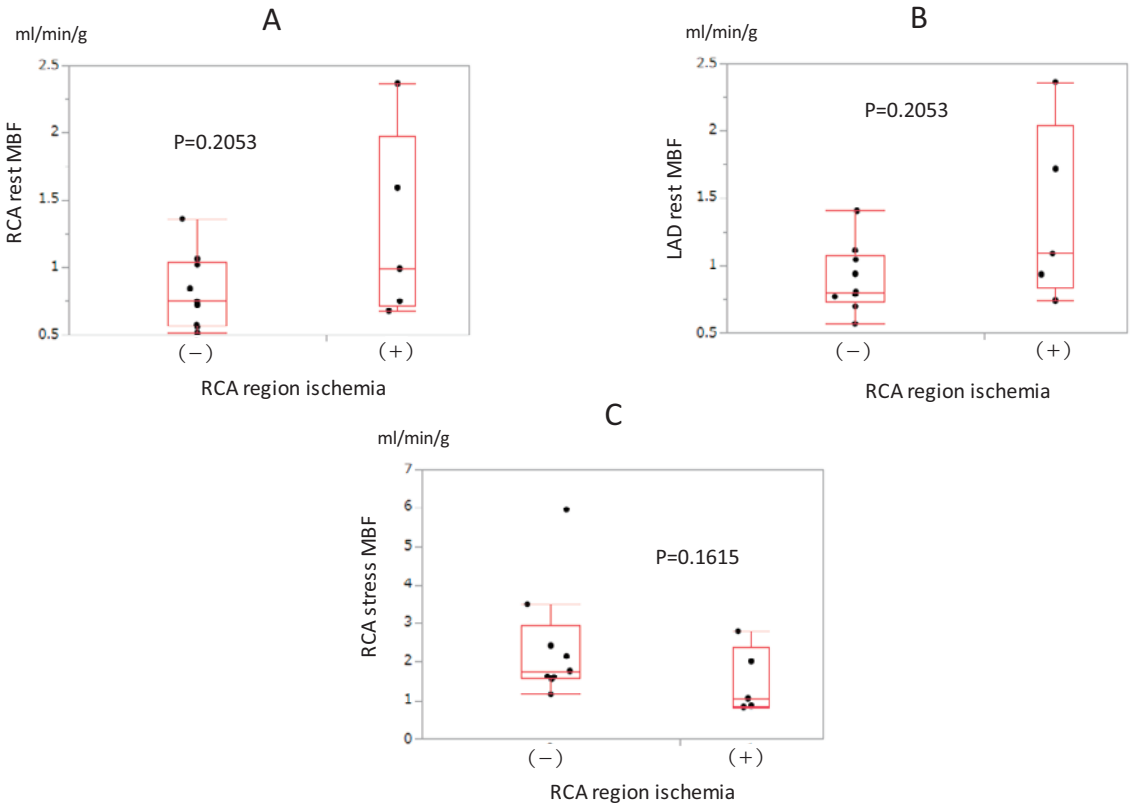

Fig. 2 Resting myocardial blood flow (MBF) and vascular resistance ratio in the right coronary artery (RCA) territory in patients with and without ischemia.
A: Patients with ischemia in the RCA territory tended to have increased MBF in the RCA region at rest (1.27 ± 0.21 vs. 0.82 ± 0.16 mL/min/g, p = 0.2053). B: Patients with ischemia in the RCA territory tended to have increased MBF in the region at rest (1.37 ± 0.66 vs. 0.90 ± 0.25 mL/min/g, p = 0.2053). C: Adenosine stress tended to cause a lower increase in blood flow in the group with ischemia in the RCA (1.51 ± 0.87 vs. 2.41 ± 1.49 mL/min/g, p = 0.1615).

remains unknown. Furthermore, RCA occlusion is relatively common in Kawasaki disease, and expansion of the peripheral vascular bed via collaterals at this microvascular level may improve peripheral circulatory disturbances. Microvascular resistance in the LAD and LCX regions tended to be lower in patients with RCA ischemia than in those without ischemia, not only at rest but also during stress. This suggests that development of collaterals at the microvascular level decreases microvascular resistance in the LAD and LCX territories to compensate for the lack of blood flow in the RCA.

Right coronary artery occlusion attributable to atherosclerosis is mainly a thrombotic occlusion due to plaque rupture, which almost always results in AMI. In contrast, thrombotic occlusion due to Kawasaki disease coronary artery aneurysm may cause AMI within 2 years of onset. After that, however, occlusion is mostly asymptomatic, probably because of slow occlusion[13].

A further important mechanism of vascular stenosis and occlusion in Kawasaki disease is that of intimal thickening. Active vascular remodeling has been reported to continue in Kawasaki disease CAL decades after onset, with extensive expression of vascular growth factors (GFs) in and around smooth muscle cells in the thickened intima at the site of CAL[14], all of which are highly expressed in the newly formed microvasculature within the intima. In contrast, in adult atherosclerosis GF is almost never expressed in the intima, and, if it is, it is localized and weakly expressed. These histological differences suggest that the frequency of collateral vascularization and the rate of increased blood flow is also greater in Kawasaki disease than in atherosclerosis.

MFR is considered clinically equivalent to coronary flow reserve (CFR)[11]. As an evaluation of coronary blood flow, fractional flow reserve (FFR) is a useful index for evaluating stenosis of the epicardial artery, whereas CFR is believed to reflect both stenosis of the epicardial artery and peripheral circulation and is related to the prognosis of coronary artery disease[15]. Multiple reports have demonstrated the benefits of FFR and CFR as indicators of

Table 3 Examination with and without RCA SS

	SS None (N = 5)	SS Yes (N = 9)	p-value
Age at onset of Kawasaki disease (months)	41.4 ± 25.4	22.1 ± 17.5	0.1239
Kawasaki disease course in months (months)	161.0 ± 67.4	256.0 ± 140.2	0.2296
LAD resting MBF (mL/min/g)	1.12 ± 0.72	1.04 ± 0.33	0.7389
LCX resting MBF (mL/min/g)	0.78 ± 0.22	0.94 ± 0.31	0.3861
RCA resting MBF (mL/min/g)	1.04 ± 0.77	0.95 ± 0.34	0.4634
LAD stress MBF (mL/min/g)	2.59 ± 1.32	2.64 ± 1.43	0.9468
LCX stress MBF (mL/min/g)	2.08 ± 0.28	2.24 ± 0.21	0.8415
RCA stress MBF (mL/min/g)	1.87 ± 0.36	2.21 ± 0.46	0.8415
LAD resting vascular resistance	48.2 ± 16.2	57.1 ± 16.5	0.3861
LCX resting vascular resistance	59.3 ± 6.8	63.1 ± 18.0	0.9865
LAD stress vascular resistance	18.3 ± 6.3	20.6 ± 9.0	0.8415
LCX stress vascular resistance	20.2 ± 4.0	22.0 ± 2.3	0.6407
LAD MFR	2.47 ± 0.56	2.61 ± 1.41	0.8415
LCX MFR	2.73 ± 0.53	2.47 ± 0.75	0.2571
RCA MFR	2.32 ± 0.87	2.34 ± 1.84	0.3173
RCA MFR<2.0	1/4	4/5	0.5804
SSS	13.00 ± 13.14	12.22 ± 3.99	0.4565
SRS	2.80 ± 3.66	5.11 ± 6.44	0.2220
SDS	10.20 ± 9.56	7.11 ± 4.86	0.2847
CABG Yes/No	3/2	4/5	1.0000
MACE with/without	3/2	5/4	1.0000
LAD aneurysm max diameter (mm)	12.1 ± 1.3	8.8 ± 1.0	0.0576
LCX aneurysm max diameter (mm)	7.8 ± 3.0	8.2 ± 4.1	1.0000
RCA aneurysm max diameter (mm)	14.7 ± 1.6	10.0 ± 2.8	0.0239
Number of remote CAL branches	3.0 ± 0.0	2.0 ± 0.9	0.0251
LVEF	0.70 ± 0.08	0.68 ± 0.12	0.8938
TAPSE (mm)	19.5 ± 7.1	18.9 ± 2.9	0.6605
NT-ProBNP (pg/mL)	46.7 ± 35.7	45.4 ± 31.9	0.8415
Collateral from LCA Yes/No (Number of cases diagnosed by cardiac catheterization)	4/1 (4/0)	4/5 (2/2)	0.3007

RCA, right coronary artery; LAD, left anterior descending artery; LCX, left circumflex artery; MFR, myocardial flow ratio; CABG, coronary artery bypass graft; MACE, major adverse cardiovascular event; CAL, coronary artery lesion; LVEF, left ventricular ejection fraction; LCA, left coronary artery; SS, segmental stenosis.

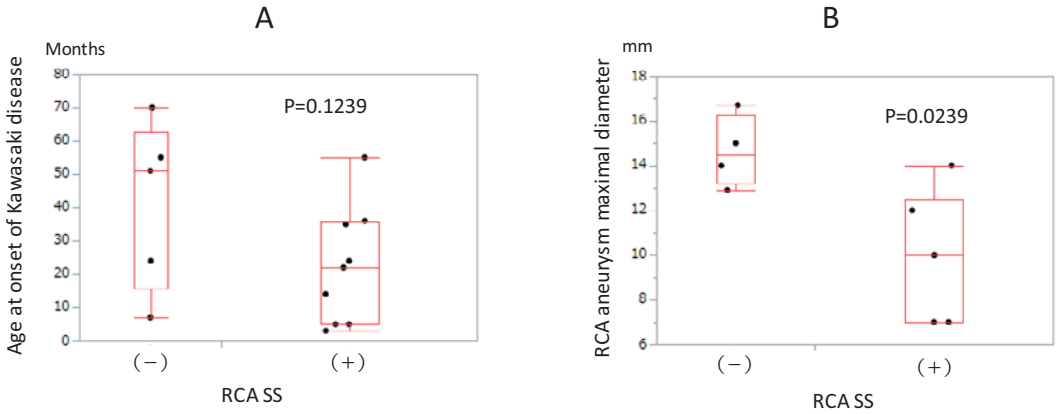

Fig. 3 Age (in months) at Kawasaki disease onset and maximum right coronary artery aneurysm diameter with and without segmental stenosis (SS) of the right coronary artery (RCA).
A: Age at Kawasaki disease onset tended to be lower in SS cases (22.1 ± 17.5 vs. 41.4 ± 25.4, p = 0.1239). B: Maximum RCA mass diameter was significantly smaller in SS cases (10.0 ± 2.8 vs. 14.7 ± 1.6, p = 0.0239).

therapeutic intervention for coronary artery stenotic lesions in adults[15–17] and in children with Kawasaki disease[18]. In Kawasaki disease, deterioration of coronary circulation has been observed even in coronary artery branches without CAL[9]. This suggests the importance of inflammation severity in microcirculation, not just in epicardial coronary vessels, at the onset of Kawasaki disease. Similarly, in adults, coronary artery disease without obstruction has recently been termed INOCA, and treatment trials for managing circulatory disturbances have been conducted[19]. The WARRIOR Trial[20] is currently underway to evaluate the efficacy of a combination of a potent statin and angiotensin-converting enzyme inhibitor, at the maximum tolerated dose, for improving circulatory disturbances. This regimen is expected to be adopted for patients with Kawasaki disease in the future.

SS is a finding of recanalization through collateral vessels around the occluded area after coronary artery occlusion and is more frequently observed in the RCA in patients with Kawasaki disease. Neovascularization is a hallmark of vascular remodeling in Kawasaki disease. Overexpression of vascular endothelial growth factor was observed in well-developed neovessels of occluded aneurysms, in thick intimal microvessels, and in numerous vascular cords in the adventitia[14,21,22]. In other words, active remodeling of Kawasaki disease occurs after the acute phase of the disease, and SS development may be stimulated to compensate for ischemia in the distal perfusion zone of occluded aneurysms. Suzuki et al.[5] reported that the SS was observed in 68 branches in 62 of 1,392 CAG cases, of which 56 (82.3%) were in the RCA and 12 (17.7%) were in the LCA. They classified SS into 3 categories: braid-like lesions, bridging lesions, and pericoronary arterial communications. Braid-like lesions occurred less than 2 years after Kawasaki disease onset and bridging lesions within 4 years. Pericoronary arterial communications are recognized later than braid-like lesions. However, no such trend was observed in the present study (data not shown), possibly because of the small number of cases. Moreover, ischemia in braid-like lesions often resolves over time, whereas recovery from ischemia in other lesions is less frequent[5]; however, this was not the case in our study (data not shown). Our findings suggest that SS is associated with earlier Kawasaki disease onset and that aneurysms in SS cases are significantly smaller. Nevertheless, formation of SS does not affect hemodynamics or contribute to ischemic improvement. Therefore, careful follow-up should be continued.

This study had some limitations. Only 14 patients were examined. Although the findings suggested disturbance of peripheral coronary blood flow, the results were not statistically significant. Studies of additional cases are thus warranted. In the evaluation of collaterals, coronary CT may not be sufficient to show intricate collaterals of peripheral vessels; hence, the evaluation was considered insufficient. Additionally, coronary vascular bed development is difficult to demonstrate morphologically.

The present study suggests that ischemia can recover over time in children with right coronary artery occlusion. Even when ischemia is present, aggressive therapeutic intervention is unnecessary when no deterioration of cardiac function is observed. In addition, when ischemia in the RCA region due to right coronary artery occlusion is observed, it is important to maintain good coronary circulation in the LAD and LCX by confirming changes in MBF over time with ammonia PET, as the deficiency is compensated by blood flow in the LAD and LCX.

In conclusion, the present study assessed coronary circulation in patients with Kawasaki disease with RCA occlusion. The findings suggest that blood flow in the RCA territory is relatively well preserved at rest and that there is no progressive deterioration in cardiac function. About half of the present patients escaped myocardial ischemia, suggesting that collateral vessels may have developed at the microvascular level and that the peripheral vascular bed of the RCA may have increased. Establishing treatment methods that improve coronary microvascular circulation in Kawasaki disease is therefore desirable.

Conflict of Interest: None declared.

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