Reversible left ventricular dysfunction with coronary stenotic or obstructive lesions in Kawasaki disease

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Background: Few studies have been reported on the prognosis or reversibility of ischemic myocardial lesions in Kawasaki disease. In this study, the pathophysiologic causes of reversible left ventricular dysfunction were evaluated in seven children with severe stenotic coronary lesions caused by Kawasaki disease.

Methods: The clinical process and the changes of coronary lesions in the repeated angiography were retrospectively evaluated in seven children. The ages of the seven patients (six boys and one girl) ranged from 7 to 15 years (mean, 10.4±2.6 y); the duration from the onset of Kawasaki disease to the last angiographic study ranged from 6 to 15 years (mean, 9.6±3.0 y).

Results: During the acute stage, an acute myocardial infarction was detected clinically in two patients, transient ST depression in two, and deep Q waves without any symptoms in the remaining three patients. In the first angiographic study (2 mo to 6 y after the onset of Kawasaki disease), significant left ventricular dysfunction was observed in the posterior or inferior wall in all patients. In the right coronary arteries, giant aneurysms (≥8 mm in diameter) were angiographically detected in two patients, localized stenosis (≥95%) in one, and recanalization in four. In a recent angiographic study, the left ventricular dysfunction had normalized in all seven patients. Recanalization was observed, even in two patients with giant aneurysms and in one with localized stenosis. Collateral circulation was detected in five patients in this study. Abnormal deep Q waves on ECG were detected in only one patient, and no abnormal findings on ECG were seen in the remaining six. Upon ^{201}Tl myocardial scintigraphy, no reduction of ^{201}Tl uptake was observed in any of the seven patients at the site of the abnormal wall motion on the first angiography.

Conclusions: The regional left ventricular dysfunction in the seven patients may have been induced by severe acute ischemia due to a transient right coronary obstruction or by severe chronic ischemia due to coronary lesions. Eventually, left ventricular wall motion normalized after an improvement in the blood flow supply as a result of early recanalization and development of collateral circulation.

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Myocardial ischemic changes have been considered reversible with quick recovery, when the ischemia is mild and of short duration, but are considered irreversible when abnormal left ventricular contraction is observed at rest. Recently, it has been reported that an abnormal regional contraction of the left ventricle persisted in some patients with a viable myocardium. Such clinical phenomena have been conceptualized as stunned [1] or hibernating myocardium [2–4]. However, much remains unknown about the ischemia myocardial lesions in Kawasaki disease; for example, how the coronary lesions change of whether the left ventricular dysfunction is reversible.

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Table 1. Angiographic changes in the grade and extent of left ventricular dysfunction and coronary lesions

		First angiographic study				Latest angiographic study			
				Coronary lesions†				Coronary lesions†	
Patient	Age, y	ECG results	LV wall motion*	RCA	LCA	ECG results	LV wall motion	RCA	LCA
1	10	Deep Q in II, III, aVF	Severe hypokinesis (4–5,7)	REC (1–2)	AN (5–7,11)	Normalized	Normalized	REC (1–2)	AN (5–6)
2	10	ST depression in II, III, aVF	Akinesis (7)	LS (2)	AN (6)	Normalized	Normalized	REC (2)	LS (6)
3	11	Deep Q in II, III, aVF	Dyskinesis (4–5)	G-AN (1–3)	—	Normalized	Normalized	REC (1–2)	—
4	8	Deep Q in II, III, aVF	Akinesis (5)	G-AN (1–3)	AN (5–6,11)	Normalized	Normalized	REC (1–3)	AN (5–6)
5	12	AMI (anteroseptal & inferior)	Akinesis (4–5)	REC (1–3)	AN (5–6,11)	Normalized	Normalized	REC (1–3)	REC (6)
6	15	ST depression in II, III, aVF	Severe hypokinesis (4)	REC (1–2)	AN (5–6,11)	Normalized	Normalized	REC (1–2)	AN (5–6,11)
7	7	AMI (inferior)	Akinesis (4–5)	REC (1–2)	AN (6)	Deep Q in II, III, aVF	Normalized	REC (1–2)	AN (6)

*Numbers in parentheses indicate the left ventricular area by the reporting system of the American Heart Association Committee Report [8].
†Numbers in parentheses indicate the area of coronary lesions by the reporting system of the American Heart Association Committee Report [8].
AMI—acute myocardial infarction; AN—aneurysm (<8 mm in diameter); G-AN—giant aneurysm (≥8 mm in diameter); LCA—left coronary artery; LS—localized stenosis (≥95%); LV—left ventricle; RCA—right coronary artery; REC—recanalization.

Methods

Between January 1989 and December 1991, 170 children received coronary angiography and left ventriculography to evaluate coronary sequelae.

Angiographic normalization of the previously abnormal contraction of the left ventricular wall was detected in seven patients who had severe stenotic (≥95%) or obstructive lesions in the main coronary branches caused by Kawasaki disease. The ages of these seven patients (six boys and one girl) ranged from 7 to 15 years (mean, 10.4 ±2.6 y), the duration from the onset of Kawasaki disease to the last angiographic study ranged from 6 to 15 years (mean, 9.6 ±3.0 y), and the interval from the first to the last angiographic study ranged from 3 to 11 years (mean, 7.7 ±2.8 y). Selective coronary angiography was performed in multiple projections by Judkin's method. The regional wall

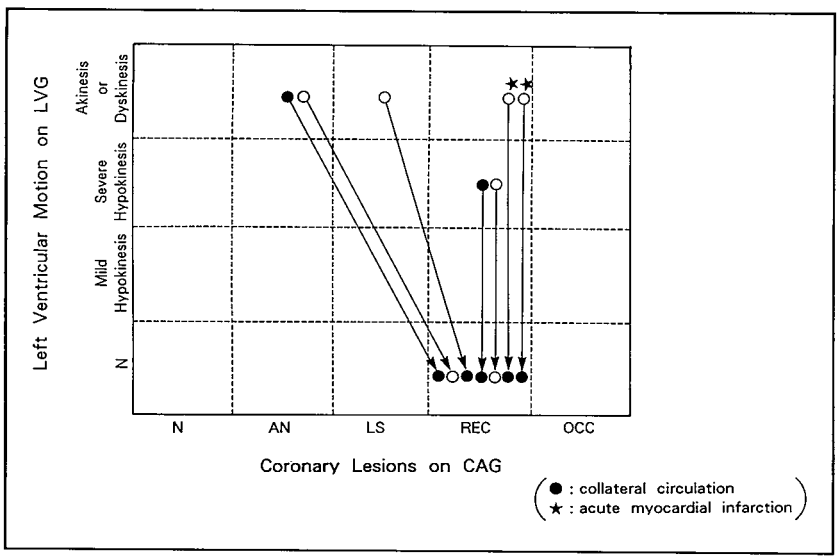

Fig. 1. Angiographic changes in coronary lesions and left ventricular wall motion. AN—aneurysm; CAG—coronary arteriography; LS—localized stenosis; LVG—left ventriculography; N—normal; OCC—occlusion; REC—recanalization.

motion of the left ventricle was evaluated in the right anterior oblique and left anterior oblique views, and graded as normal, mild hypokinesis, severe hypokinesis, akinesis, or dyskinesis. In patients with various angiographically proven coronary lesions, the most serious coronary lesions were expressed in this study as a stenotic lesion combined with an aneurysm, termed a "stenotic lesion;" recanalization combined with a localized stenosis, labeled as "recanalization;" and an occlusion combined with recanalization or localized stenosis, defined as an "occlusion."

Results

The seven patients underwent their first angiography between 2 months to 6 years (mean, 1.9 ± 2.2 y) after the onset of Kawasaki disease. During the acute stage, an acute myocardial infarction was detected clinically in two patients (Table 1), transient cardiomegaly and a gallop rhythm with ST depression in two other patients, and deep Q waves on their ECGs without any subjective symptoms in the remaining three patients. An acute myocardial infarction was confirmed clinically by ECG changes and by an increase in the serum levels of myocardial enzymes with chest pain. In the first angiographic study (Table 1), abnormal regional wall motion of the left ventricle (severe hypokinesis, akinesis, or dyskinesis) was observed in the posterior or inferior wall in all patients. In the right coronary arteries, giant aneurysms (≥8 mm in diameter) were angiographically detected in two patients, localized stenosis (≥95%) in one, and recanalization in four. After their first angiographic study, all seven patients received anticoagulation therapy and had no subsequent clinical episodes of myocardial ischemia.

The latest angiographic study was performed to reevaluate the coronary lesion and left ventricular wall motion. In this angiographic study, the abnormal regional wall motion of the left ventricle had normalized in all seven patients (Fig. 1). There was detectable change angiographically in the recanalization of the right coronary artery in four patients, whereas recanalization changes were observed in two patients with a giant aneurysm and in one with localized stenosis (Table 1). Collateral circulation, which was observed in only two patients on the first angiographic study, was detected in five patients in this study (Fig. 1). In two patients, recanalization or localized stenosis (≥95%) was also observed in the left anterior descending artery. Deep Q waves on the resting ECG were detected in only one patient, and no abnormal findings on ECG were seen in the remaining six. Treadmill exercise ECG revealed mild ST-segment depression in two patients in leads II, III, and aVF. Upon ^{201}Tl myocardial scintigraphy performed within 2 weeks prior to their most recent angiographic study, no reduction of ^{201}Tl uptake was observed in the seven patients at the site of the abnormal wall motion on the first angiography.

Discussion

Children, particularly neonates and infants, have been well known clinically and experimentally to be more resistant to ischemia than adults [5,6]. As we reported earlier [7], clinical findings, including ECG changes that indicate previous myocardial ischemia in children with Kawasaki disease, normalize or improve with time; therefore, the evaluation of whether ischemic myocardial lesions in children are reversible to is often difficult.

The abnormal regional wall motion of the left ventricle initially observed in the seven patients was induced by transient obstruction, localized stenosis, or a giant aneurysm with poor run-off of the right coronary artery. The ECG and ^{201}Tl myocardial scintigraphy findings and the subsequent improvement in the left ventricular wall motion suggest that these ischemic myocardial lesions were reversible. Possible etiologic factors in this reversibility include the early development of recanalization after an occlusion at the coronary lesion or of collateral circulation, which maintains the coronary blood flow supply to that myocardial area. The reversible regional left ventricular dysfunction in the seven patients may have been induced by severe acute ischemia due to a transient right coronary obstruction (stunned myocardium) or by severe chronic ischemia due to a reduction in the coronary blood flow supply (hibernating myocardium). Eventually, the left ventricular wall motion normalized after an improvement in the blood flow supply as a result of recanalization and the development of collateral circulation.

Conclusions

The fact that seven of our 170 patients exhibited reversible left ventricular dysfunction should be considered when evaluating the treatment and long-term prognosis of children with severe coronary sequelae due to Kawasaki disease, in which the changes and progression of the coronary lesions have not yet been clarified. Elucidation of the pathology and the development of methods to directly evaluate the reversibility of myocardial lesions are needed. Sufficient clinical follow-up is necessary for the evaluation of the reversibility of the ischemic myocardial lesions, as well as for the evaluation of the clinical course and the coronary lesions in individual cases.

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