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Magnetic Resonance Imaging of Old Myocardial Infarction in Young Patients with a History of Kawasaki Disease

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Summary

Background: On magnetic resonance imaging (MRI) using gadolinium diethylenetriamine pentaacetic acid (Gd-DTPA) as contrast medium, images of infarct regions are enhanced in acute myocardial infarction (AMI). In old myocardial infarction (OMI), thinning of the myocardial walls is present, but images are no longer enhanced by Gd-DTPA. On the other hand, MI in children with a history of Kawasaki disease (KD), several differences from adult MI are observed.

Hypothesis: The aim of this study was to evaluate the lesions that result from OMI in children with a history of KD using MRI with Gd-DTPA as a contrast medium.

Methods: The subjects were 16 young patients with a history of KD who were diagnosed as having OMI. Of these, both thinning of the myocardial wall and MRI enhancement by Gd-DTPA were observed in 4 cases, thinning alone was observed in 6 cases, Gd-DTPA image enhancement alone was observed in 3 cases, and neither thinning nor Gd-DTPA image enhancement was observed in 3 cases.

Results: The Gd-DTPA-image-enhanced, OMI-induced lesions observed in patients with KD were different from those observed in adults; this might be attributable to histologic differences.

Conclusion: Magnetic resonance imaging using Gd-DTPA can evaluate myocardial thinning and presence of circulation noninvasively at the same time and is considered to be useful for long-term follow up of the patients with KD and OMI.

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Introduction

The technique of gadolinium diethylenetriamine pentaacetic acid (Gd-DTPA)-enhanced magnetic resonance imaging (MRI) has been used to evaluate the acute stage of myocardial infarction (MI).[1–3] When Gd-DTPA is used as a contrast medium, images of infarct regions are enhanced in AMI and can be distinguished clearly from the normal myocardium. In the acute stage of MI, Gd-DTPA flows into the remaining vascular bed and interstitial edema in the infarct region. Since the wash-in and wash-out of Gd-DTPA at the site of the MI are delayed compared with normal tissue, image enhancement by Gd-DTPA is observed. In old myocardial infarction (OMI), an infarct lesion is cicatrized and thinning of the myocardial wall is present; however, the MRI image is no longer enhanced by Gd-DTPA in that tissue[4] because of the paucity of extracellular fluid.

Although MIs in children are seen rarely, 10–20% of children with Kawasaki disease (KD) have coronary artery lesions, and some develop MI.[5,6] It has been reported that MIs in KD are asymptomatic in 37% of patients, and reperfusion of the coronary artery and development of the collateral circulation are observed in many cases.[7,8]

To date, there are no reports that describe MRI findings of OMI in patients with KD. We used MRI with Gd-DTPA enhancement to evaluate the state of the myocardium of OMI in children with a history of KD.

Subjects

The study was undertaken at the National Cardiovascular Center in Osaka, Japan. The subjects were 16 children with a history of KD (10 males, 6 females, aged 7–28 years [mean 16.7 years]), who were also diagnosed as having OMI. Their clinical histories were obtained from the medical records at the

Center (Table I). The age of onset of KD was from 5 months to 11 years (mean 1.8 years). The age at which a diagnosis of MI was made ranged from 5 months to 17 years (mean 7.2 years), and the period between the diagnosis of MI and examination using MRI was from 9 months to 17 years (mean 10.3 years). The age at which MI was diagnosed in subjects who had no subjective symptoms was taken as the age when an abnormal Q wave was observed on the electrocardiogram (ECG).

As control subjects, we also studied four children with a history of KD-complicated coronary artery lesions without MI, who visited the National Cardiovascular Center for regular examinations.

Methods

Magnetic resonance imaging was performed on a 1.5-Tesla machine (Magnetom H15, Siemens AG, Munich, Berlin, Germany). An ECG-triggered, T1-weighted, spin-echo image with an echo time of 30 ms was obtained for transaxial and short axial sections. Four to five slices with a thickness of 10 mm and a slice interval of 15 mm covered the heart. The imaging was repeated prior to and 5–15 min after the intravenous administration of 0.1 mmol/kg of a contrast medium (Gd-DTPA). Wall thickness was evaluated visually on the precontrast image.

All patients were also subjected to coronary angiography[9,10] and resting or exercise-loading thallium-201 (^{201}Tl) myocardial planar imaging using conventional methods.[11]

Results

Three Typical Cases

Case 1 (Table I, No. 10): This was an 18-year-old girl who was found to be suffering from KD at the age of 8 years. Aneurysms developed in her right coronary artery (RCA) and left anterior descending coronary artery (LAD). During the observation period, localized stenosis of the LAD occurred. At the age of 10 years, since the stenosis progressed, she underwent coronary artery bypass grafting, which involved connecting the left internal thoracic artery with the LAD. However, at a later examination the bypass graft was found to be completely occluded. Seven years after the bypass surgery, this patient was admitted to hospital complaining of chest pain. At this point, she was diagnosed as having AMI. While

TABLE I Characteristics of patients with Kawasaki disease and magnetic resonance image findings

Patient No.	Sex	Age (years)	Years[a]	KD age (years)	MI age (years)	MI area	Coronary artery lesion LMT	LAD	LCx	RCA	Collateral	CABG	^{201}Tl myocardial imaging PD (rest)	PD (ex.)	MRI Thinning	Enhancement
1	M	7.6	(1.3)	1.9	6.3	Inf		LS	OC, AN	OC	LAD to RCA	ND	+	+	−	+
2	M	10.4	(10.0)	0.4	0.4	Inf		AN		SS		ND	−	−	−	−
3	F	11.9	(7.4)	4.2	4.5	Inf, IVS		LS		SS		OC	−	+	+	−
4	F	13.1	(12.1)	0.4	1.0	Ant		SS	Dil	LS, dil		ND	+	+	+	+
5	F	14.3	(6.3)	1.5	8.0	Ant, inf	LS	OC		SS	RCA to LAD	ND	−	+	−	+
6	M	14.5	(13.8)	0.4	0.7	Inf				SS		ND	+	+	+	−
7	M	15.7	(2.6)	11.6	13.1	Post			LS	OC	LAD to RCA	ND	−	−	−	+
8	M	16.3	(12.0)	3.8	4.3	Inf, ant		OC		SS		OC	−	+	+	−
9	M	17.0	(10.6)	0.6	6.4	Ant		LS		SS		ND	+	+	−	−
10	F	18.0	(0.8)	8.8	17.2	Ant, IVS		OC, LS		LS		OC	+	+	+	−
11	M	18.6	(2.5)	0.5	16.1	Inf				SS		ND	+	+	−	−
12	M	19.2	(8.2)	0.5	11.0	Ant		SS	SS	SS		ND	+	+	+	−
13	F	19.3	(17.3)	1.8	2.0	Inf		LS	SS	OC		ND	+	+	+	+
14	M	20.0	(10.5)	8.1	9.5	Inf		AN	AN	SS		ND	+	+	+	+
15	F	21.7	(13.7)	1.9	8.0	Ant	AN	AN	AN			ND	+	+	+	−
16	M	28.5	(13.6)	1.8	14.9	Ant		SS	OC	SS		OC	+	+	+	+
17	F	13.3		0.6	—	—		OC			RCA to LAD	ND	−	+	−	−
18	F	16.6		0.2	—	—		OC		SS	RCA to LAD	ND	−	+	−	−
19	F	20.0		2.2	—	—				LS, AN		ND	−	−	−	−
20	M	23.2		7.0	—	—	AN	LS, AN		LS, AN		ND	−	+	−	−

[a] Period between the onset of MI and MRI investigation.

Abbreviations: KD = Kawasaki disease, MRI = magnetic resonance imaging, MI = myocardial infarction, LMT = left coronary artery main trunk, LAD = left anterior descending coronary artery, LCx = left circumflex coronary artery, RCA = right coronary artery, CABG = coronary artery bypass graft, OD = perfusion defect, ex. = exercise loading, ^{201}Tl = thallium-201, Inf = inferior wall of left ventricule, IVS = interventricular septum, Ant = anterior wall of left ventricle, Post = posterior wall of left ventricle, LS = localized stenosis, SS = segmental stenosis, Dil = dilatation, OC = occlusion, AN = aneurysm, ND = not done.

performing an emergency catheterization it was found that the LAD was occluded; recanalization was achieved by percutaneous transluminal coronary recanalization, but within 1 day, the artery was once again occluded. The procedure was repeated, but this second attempt was unsuccessful.

One year and 3 months after the onset of AMI, it was no longer possible to use coronary angiography to image the LAD below the site of the aneurysm, indicating complete occlusion (Fig. 1A, B). No evident collateral circulation was found. One year and 4 months later, MRI revealed marked thinning of the myocardium from the ventricular septum to the left ventricular anterior wall (Fig. 1C). No enhancement by Gd-DTPA was observed (Fig. 1D).

Case 2 (Table I, No.1): A boy aged 7 years and 7 months was found to have developed KD at the age of 1 year and 10 months. Aneurysms had formed in both his left coronary artery (LCA) and RCA. At the age of 6 years, the patient consulted a nearby physician, complaining of chest pain. Examination of his ECG revealed an elevation of the ST wave in leads V_1–V_3, and echocardiography revealed a reduction in the movement of the left ventricular wall. This patient was diagnosed as having AMI, and tissue plasminogen activator was administered intravenously. Five months later, he again suffered chest pain and consulted the same physician. Since abnormal Q waves were found in leads II, III, and aVF on his ECG, he underwent emergency catheterization. Occlusion of the RCA and the left circumflex coronary artery (LCx) and localized stenosis of the LAD were found. Percutaneous transluminal coronary recanalization was performed; however, this was not successful.

Ten months after the second attack of AMI, coronary angiography revealed occlusion of the RCA and the LCx, as well as development of a collateral artery from LAD (Fig. 2A, B). Thinning of the myocardium was not evident, but MRI revealed the presence of an irregularly shaped area extending from the inferior wall to the posterior wall (Fig. 2C). Following the administration of Gd-DTPA, heterogeneous enhancement was observed in that lesion (Fig. 2D).

Case 3 (Table I, No. 4): This girl, aged 13 years and 1 month, was diagnosed with KD at the age of 5 months. Aneurysms developed in her LCA and RCA. At the age of 1 year, she was hospitalized for cardiac catheterization, and an ECG revealed abnormal Q waves in leads V_1–V_3. Coronary angiography demonstrated the presence of recanalization of so-called segmental stenosis and an aneurysm located in the LAD; aneurysms were also observed in the LCA and RCA. The ECG and coronary angiography findings led to the diagnosis of OMI.

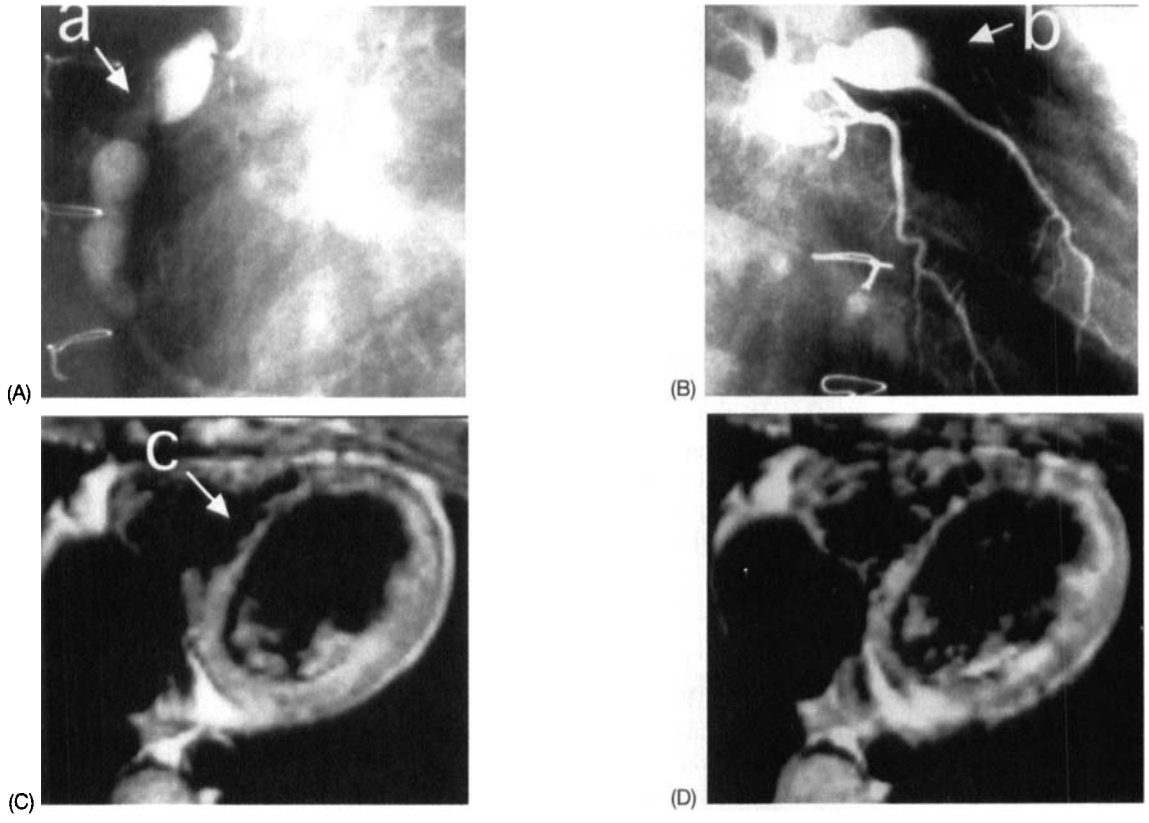

FIG. 1 Case No. 1 (18-year-old girl): Coronary angiography at 1 year and 3 months after onset of acute myocardial infarction (AMI) (A and B) and magnetic resonance imaging at 1 year and 4 months after onset of AMI (C and D). There is localized stenosis in the right coronary artery (arrow a). Complete occlusion is observed in left anterior descending artery (arrow b), and no evident collateral circulation is found (B). Marked thinning is observed in interventricular septum and anterior wall of the left ventricle (arrow c). After injection of gadolinium diethylentriamine pentaacetic acid, enhancement is not found (D).

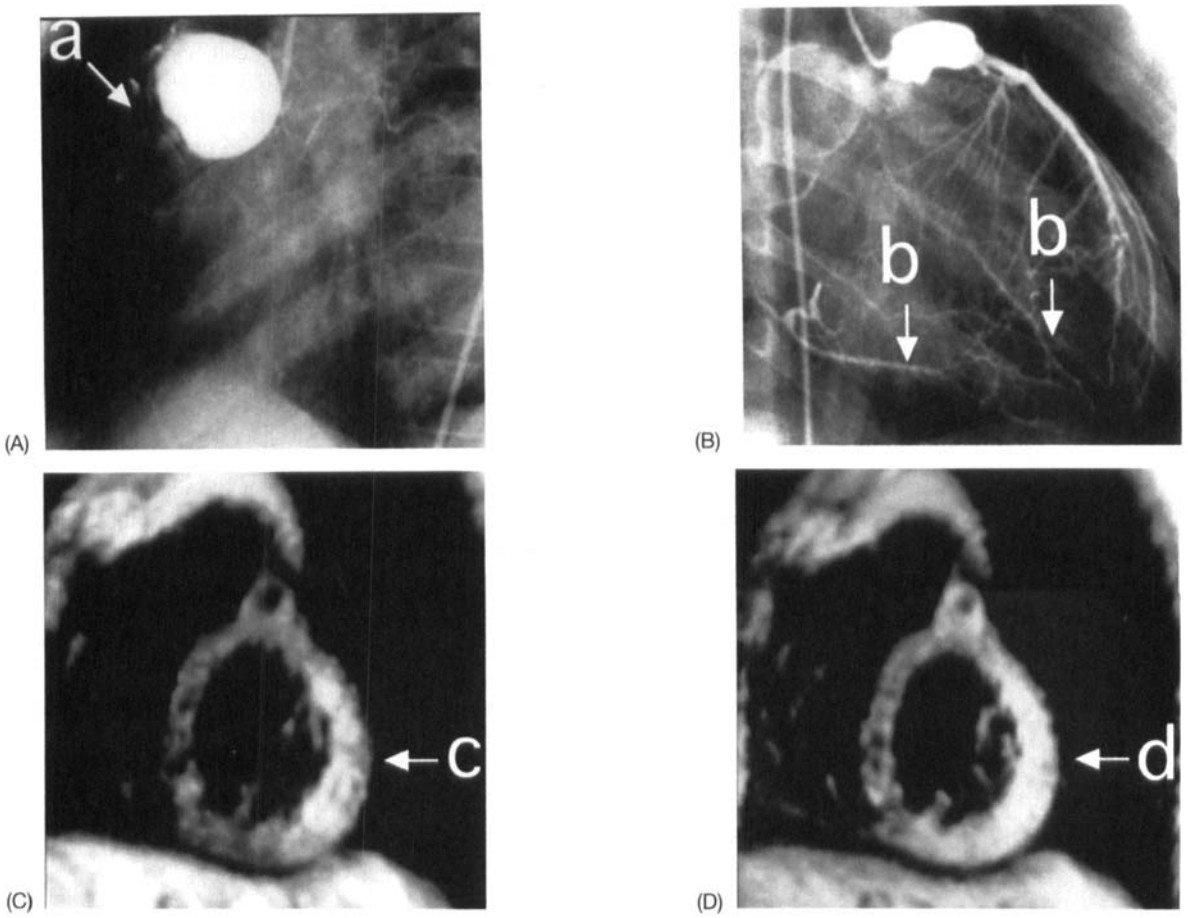

FIG. 2 Case No. 2 (7.6-year-old boy): Coronary angiography (A and B) and magnetic resonance imaging (C and D) at 10 months after the second acute myocardial infarction. Complete occlusion is observed in the right coronary artery (RCA) (arrow a). The left circumflex artery (LCx) is also occluded (B). Development of collateral circulations from the left anterior descending artery to the LCx and RCA are seen (arrow b). Thinning of the myocardial wall is not evident, an irregular shape of the wall is seen from the inferior wall to the posterior wall of the left ventricle (arrow c). After injection of gadolinium diethylentriamine pentaacetic acid, heterogeneous enhancement is seen in the same region (arrow d).

Ten years after the diagnosis, a further coronary angiography examination revealed the presence of segmental stenosis in the LAD and localized stenosis in the RCA (Fig. 3A, B). Two years after coronary angiography, slight thinning of the anterior wall of the left ventricle was observed on an axial MRI image (Fig. 3C). The administration of Gd-DTPA resulted in enhancement of the image of the anterior wall (Fig. 3D).

Results

The results of the 16 patients who underwent MRI using Gd-DTPA medium are summarized in Table I. Thinning of the myocardial wall and enhancement by Gd-DTPA was observed in 10 (62.5%) and 7 cases (43.8%), respectively. Both thinning of the myocardial wall and enhancement by Gd-DTPA were observed in four cases (25.0%), thinning alone was found in six cases (37.5%), and enhancement alone in three cases (18.8%). Three cases (18.8%) exhibited neither thinning of the myocardial wall nor enhancement by Gd-DTPA. The four patients who had a history of KD but had never been diagnosed as having MI showed neither thinning of the myocardial wall nor image enhancement by Gd-DTPA (controls: Table I, No. 17–20). No correlation was found between the presence of abnormal findings on MRI, such as thinning of the myocardial wall and enhancement by Gd-DTPA, and the age of the patients or the time at which the diagnosis of MI was made.

We examined resting or exercise-loading ^{201}Tl myocardial planar imaging (Table I). A perfusion defect was observed on resting ^{201}Tl myocardial imaging in 8 of the 10 patients who exhibited thinning of the myocardial wall, and 3 of the 6 patients who did not. Perfusion defects were observed on exercise-loading ^{201}Tl myocardial imaging for all 10 patients with and for 4 of the 6 patients without thinning of the myocardial wall. A perfusion defect was observed on resting ^{201}Tl myocardial imaging in five of the seven patients who exhibited Gd-DTPA-induced MRI enhancement, and in six of the nine patients who exhibited no such enhancement. A perfusion defect was observed on exercise-loading ^{201}Tl myocardial imaging in six of the seven patients who exhibited Gd-DTPA-induced

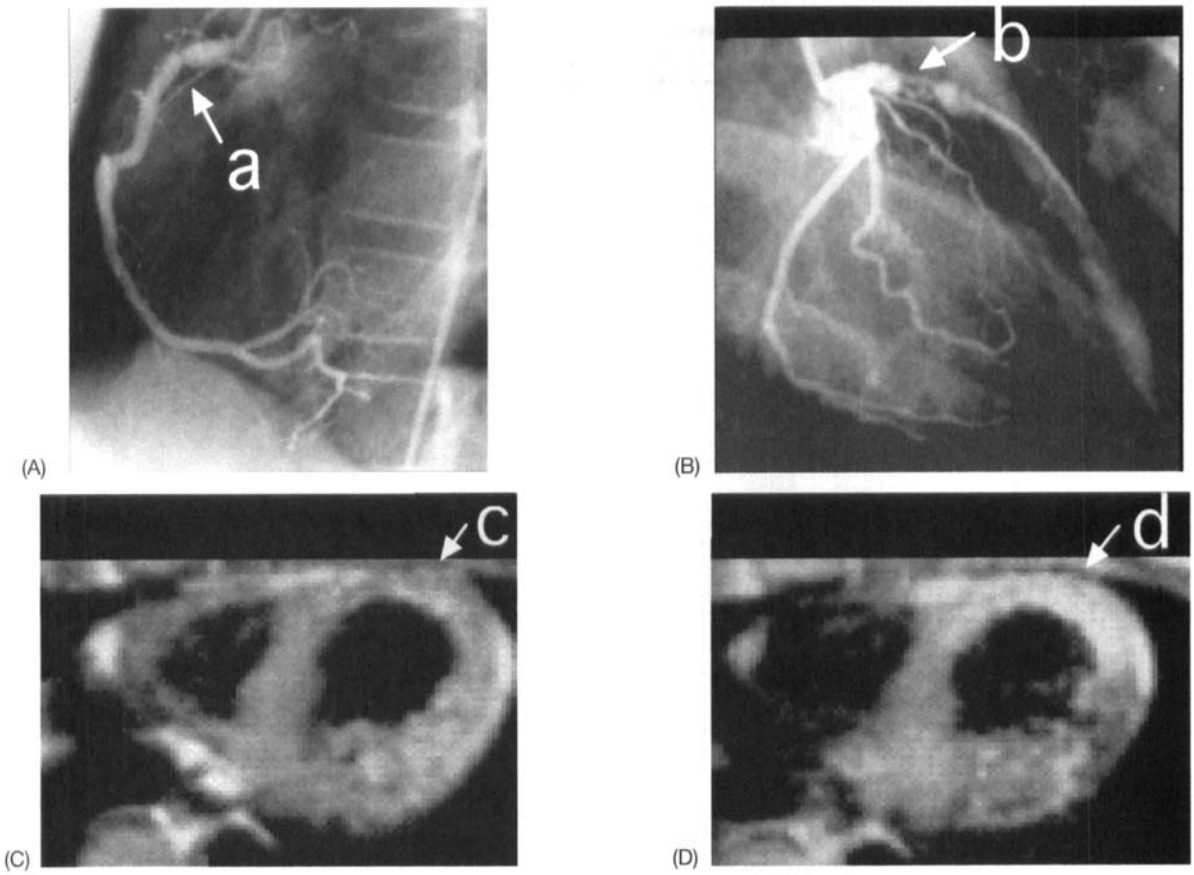

FIG. 3 Case No. 3 (13-year-old girl): Coronary angiography at 10 years after diagnosis as old myocardial infarction (OMI) (A and B) and magnetic resonance imaging at 12 years after diagnosis of OMI (C and D). Localized stenosis is seen in the right coronary artery (arrow a), segmental stenosis is observed in the left anterior descending artery (arrow b). Thinning of the anterior wall is found (arrow c). After injection of gadolinium diethylentriamine pentaacetic acid, enhancement is observed in the same region (arrow d).

MRI enhancement, and in eight of the nine patients who exhibited no enhancement. We found no correlation between abnormal MRI findings and the existence of perfusion defect on ^{201}Tl myocardial planar imaging.

Discussion

Kawasaki disease is an acute systemic vasculitis of unknown cause first described in Japan in 1967. It affects children younger than 10 years, predominantly those younger than 3 years. It causes aneurysm formation in medium-sized arteries, particularly the coronary arteries. Complications of KD include MI, which may occur during acute illness or later, as a result of coronary artery lesions.[12]

We examined MRI images of patients with KD and OMI. In six patients who showed only thinning in MRI, myocardium was considered to be cicatrized completely, similarly to OMI in adults. In three patients with neither thinning nor enhancement, it was highly conceivable that abnormality was not detected because the infarct foci were extremely small. This finding does not contradict similar results obtained for four controls who were diagnosed to have no MI. However, it was suggested that myocardium of the infarcted region in seven patients with enhancement by Gd-DTPA was apparently abnormal, but the region was not completely cicatrized despite the fact that 1.3 to 17.3 years had passed since the episode of infarction. In the present study, a heterogeneous pattern of thinning of the myocardium was observed in OMI of patients with KD. It resembled the findings in dilated cardiomyopathy rather than those in OMI in adults. It is our speculation that the MRI enhancement by Gd-DTPA observed in patients with KD was, to some extent, attributable to the development of myocardial fibrosis and to the retention of the blood circulation by means of reperfusion or development of a collateral circulation. The heterogeneity of MRI findings observed in patients with KD may reflect different histologic patterns.[13]

Conclusion

The reperfusion of the coronary artery and development of the collateral circulation are observed in many cases of MI in patients with KD. It is thought that the subjects with enhance-

ment on MRI have reperfusion of blood in the infarcted regions.[14] Although blood reperfusion in the infarcted region can be evaluated to some extent by resting ^{201}Tl myocardial planar imaging, the evaluation of myocardial thinning and the presence of circulation at the same time is impossible and diagnosis of the inferior wall is sometimes difficult. Cardiac catheterization is an essential examination for evaluation of coronary lesions, but it is highly invasive. Magnetic resonance imaging using Gd-DTPA can evaluate myocardial thinning and presence of circulation noninvasively at the same time; thus, we thought this could be considered to be useful for long-term follow up of patients with KD and OMI.

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