Effects of Preinfarction Angina on Myocardial Injury in Patients with Acute Myocardial Infarction: A Study with Resting ^{123}I-BMIPP and ^{201}Tl Myocardial SPECT

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Methods: Recent studies have suggested that patients with preinfarction angina have smaller infarcts and a better in-hospital outcome than those without angina. The mechanisms responsible for limitation of infarct size in the presence of preinfarction angina are unclear. We examined the effects of preinfarction angina on myocardial injury in patients with the first acute myocardial infarction with resting ^{123}I-15-(*p*-iodophenyl)-3-(*R,S*)-methylpentadecanoic acid (BMIPP) ^{201}Tl myocardial scanning performed within 1 mo of infarction. **Results:** Of 136 patients tested, 48 (35%) had preinfarction angina within 72 h before infarction, whereas 88 (65%) did not. BMIPP and ^{201}Tl defects were scored in 9 segments of the left ventricle (0 = normal, 1 = mild defect, 2 = moderate defect, 3 = severe defect, and 4 = no uptake). The total defect score was defined as the sum of the defect scores. There was no significant difference in percentage diameters of stenoses of infarct-related arteries, collateral circulation, total defect scores for BMIPP, or ^{201}Tl between the groups with and without preinfarction angina. However, the ratio of total defect score for ^{201}Tl to that for BMIPP was significantly smaller for patients with than for those without preinfarction angina (0.64 ± 0.21 versus 0.74 ± 0.25, respectively; P = 0.007). **Conclusion:** Preinfarction angina did not affect the areas at risk in acute myocardial infarction, as shown by BMIPP defect, but decreased necrotic myocardium in the areas at risk, as shown by ^{201}Tl defect, and increased metabolically damaged but viable myocardium, as shown by BMIPP and ^{201}Tl mismatch through unidentified mechanisms other than collateral circulation (e.g., ischemic preconditioning).

Key Words: preconditioning; BMIPP; ^{201}Tl

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Several studies have suggested that patients with preinfarction angina have smaller infarcts (*1,2*), a better in-hospital outcome (*1,3,4*), a better preservation of left ventricular function (*5–8*), and a lower incidence of shock (*3*) than those without angina. The mechanisms responsible for limitation of infarct size in the presence of preinfarction angina are unclear, although ischemic preconditioning, described in experimental models (*9–13*), is presumed to be a possible mechanism. However, clinical evaluation of ischemic preconditioning is difficult because the area at risk for myocardial infarction (MI) is diminished by the presence of collateral circulation induced by prior ischemia (*14–19*). When assessing the effects of ischemic preconditioning, the influence of collateral circulation during coronary obstruction must be excluded. However, determination of the area at risk is difficult in the clinical setting. The degree of collateral flow has been reported to be higher in the presence than in the absence of preinfarction angina (*5–7,17,19*). However, all of these studies estimated the collateral circulation after the onset of MI, and therefore their findings did not reveal the condition of collateral flow at the time of abrupt occlusion of coronary arteries.

Fatty acids are the major fuel for the normal myocardium under normal aerobic conditions, and ^{123}I-15-(*p*-iodophenyl)-3-(*R,S*)-methylpentadecanoic acid (BMIPP) is used clinically as a probe to study the myocardial metabolism of fatty acids (*19–21*). Tamaki et al. (*22*) reported that mismatched segments with greater reduction of BMIPP uptake than of ^{201}Tl uptake were observed more often in acute than in chronic MI and more often in reperfused than in nonreperfused coronary arteries. Regions exhibiting mismatch of BMIPP and tracer for myocardial flow have been reported to reflect dysfunctional but viable myocardium, such as that with myocardial stunning or hibernation, which will subsequently exhibit improved regional function (*23–25*). Moreover, many investigators have reported that BMIPP imaging in the subacute phase of MI permits determination of the amount of myocardium at risk after MI (*24–27*). Therefore, resting myocardial dual-isotope SPECT using BMIPP and ^{201}Tl in the subacute phase can provide information on

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areas at risk and necrotic myocardium in patients with acute MI.

The aim of this study was to investigate the effects of preinfarction angina on myocardial injury in patients with acute MI using resting ^{123}I-BMIPP ^{201}Tl myocardial SPECT.

MATERIALS AND METHODS

Patient Population

This study included 136 consecutive patients with acute MI (104 men, 32 women; mean age, 63 ± 11 y; range, 33–87 y) who were admitted to the coronary care unit of Osaka City University Medical School Hospital between March 1993 and February 1998. Each patient had undergone resting myocardial dual-isotope SPECT using BMIPP and ^{201}Tl in the subacute phase (within 1 mo after the onset of MI) and were selected retrospectively for this study. Acute MI was defined by the presence of typical chest pain, ST segment depression or elevation on the standard 12-lead electrocardiogram, and elevation of serum creatine kinase or the creatine kinase MB fraction to more than 3 times the upper limit of normal. Patients who did not undergo SPECT in the subacute phase because of uncontrolled heart failure, uncontrolled ischemia attack, or recurrent infarction or because they died in the acute phase were excluded from the study. Also excluded were patients who were unable to describe the details of anginal pain and patients with histories of previous MI or coronary artery bypass grafting. A total of 73 patients had anterior infarctions, whereas 63 had inferior infarctions. The mean interval between MI and resting SPECT was 13 ± 5 d.

Symptom status before infarction was elicited through questioning by the treating physician at the time of admission and was reviewed retrospectively in medical records. Preinfarction angina was defined as 1 or more episodes of typical chest pain lasting less than 30 min within 72 h before the onset of MI.

Radiopharmaceuticals

BMIPP, an ^{123}I-labeled β-methyl-branched fatty acid analog, was prepared and supplied by Nihon Medi-Physics Co., Ltd. (Hyogo, Japan). ^{201}Tl was supplied by Nihon Medi-Physics and Daiichi Radioisotope Labs, Ltd. (Tokyo, Japan).

Resting BMIPP and ^{201}Tl Dual-Isotope SPECT

After overnight fasting, each patient received an intravenous injection of BMIPP (111 MBq) and ^{201}Tl (111 MBq) at rest. Initial images were obtained 20 min after injection, and delayed images were obtained 4 h later. SPECT was performed using a single-head scintillation camera equipped with a low-energy, all-purpose, parallel-hole collimator. A total of 32 equidistant projections were acquired (30 s/projection) over 180° from right anterior oblique to left posterior oblique. The images from the 2 energy windows (159 keV ± 7.5% for ^{123}I and 70 keV ± 10% for ^{201}Tl) were collected in separate 64 × 64 matrices and then reconstructed using a Butterworth filter and a Shepp and Rogan filter along the short axis, horizontal long axis, and vertical long axis of the heart. Images were normalized to the maximal count in each image set and displayed as color-scale images by a computer system (Scintipac-7000; Shimadzu Corp., Kyoto, Japan). No downscatter correction was performed.

Data Analysis

SPECT images were examined visually. The left ventricular myocardium was divided into 9 segments (basal-anterior, midanterior, basal-septal, midseptal, basal-inferior, midinferior, basal-lateral, midlateral, and apical) on a vertical long-axis slice, a horizontal long-axis slice, and short-axis slices. Because the clinical significance of delayed images of BMIPP is not well understood, such images were not analyzed. Initial images of BMIPP and ^{201}Tl and delayed images of ^{201}Tl were analyzed individually by 2 experienced observers unaware of patients' clinical data. Disagreements in interpretation were resolved by consensus of the 2 observers. BMIPP and ^{201}Tl defects in each segment were scored using a 5-point grading system (0 = normal, 1 = mild defect, 2 = moderate defect, 3 = severe defect, and 4 = no uptake). The total defect score was defined as the sum of the defect scores for infarct-related segments. For the initial image, a decrease in the total defect score for the delayed image of ^{201}Tl was defined as redistribution, whereas an increase was defined as reverse redistribution. Fixed defect was defined as total defect score for the delayed image equal to that for the initial image. The total defect score in the 1 image of initial and delayed images of ^{201}Tl that showed a smaller total defect score was defined as the smaller total defect score of ^{201}Tl.

Peak Creatine Kinase and MB Fraction

Venous blood samples for estimation of peak serum creatine kinase were drawn every 3 h for the first 24 h and every 6 h for the next 24–48 h after the onset of infarction. All patients had a significant elevation of serum creatine kinase, but peak values could be determined for only 112 patients.

Coronary Arteriography and Left Ventriculography

Thirty-two patients underwent thrombolytic therapy with tissue-type plasminogen activator, and 35 patients underwent successful coronary angioplasty of the infarct-related artery within 24 h of the onset of MI. Ninety-five patients underwent diagnostic coronary arteriography during the acute or subacute phases. Coronary arteriography was performed in multiple projections using standard techniques. Coronary arterial narrowing was estimated visually and expressed as the maximal percentage of narrowing of luminal diameter. Estimates were determined by 2 experienced observers without access to the findings of scintigraphic studies. Coronary arterial narrowing exceeding 50% was considered significant. The extent of retrograde collateral flow to stenosed vessels was graded according to the classification of Cohen and Rentrop (*28*). Left ventriculography was performed in 106 patients. The mean left ventricular ejection fraction in these patients measured by a center-line method was 48% ± 15%.

Statistics

Values are presented as mean ± SD. Incidences of phenomena were compared by the χ^2 test. The significance of differences for continuous variables was determined using the Student *t* test. Differences for discrete variables were compared by the Wilcoxon rank sum test. $P < 0.05$ was considered significant.

RESULTS

Baseline Characteristics

Of the 136 patients studied, 48 (35%) experienced preinfarction angina, whereas 88 (65%) did not. Baseline characteristics of patients with and without preinfarction angina are given in Table 1. There were no significant differences in age, sex, coronary risk factors, preadmission medications, heart rate or systolic blood pressure on admis-

TABLE 1
Baseline Characteristics of 136 Patients with Acute MI With and Without Preinfarction Angina

Characteristic	Patients without angina (n = 88)	Patients with angina (n = 48)	*P*
Age (y)	63 ± 11	63 ± 10	NS
Sex (M/F)	70/18	34/14	NS
Coronary risk factors (n)*			
Diabetes mellitus	21 (24)	10 (21)	NS
Smoking	70 (80)	36 (75)	NS
Hypertension	40 (45)	16 (33)	NS
Hypercholesterolemia	20 (23)	15 (31)	NS
Hyperuricemia	5 (6)	4 (8)	NS
Family history	11 (13)	9 (19)	NS
Preadmission medications (n)*			
Aspirin	2 (2)	0 (0)	NS
Nitrates	8 (6)	5 (10)	NS
β-blockers	2 (2)	1 (2)	NS
Calcium-channel blockers	16 (18)	4 (8)	NS
Angiotensin-converting enzyme inhibitors	3 (3)	0 (0)	NS
Diuretics	2 (2)	1 (2)	NS
Heart rate on admission (bpm)	73 ± 18	77 ± 19	NS
Systolic blood pressure on admission (mm Hg)	124 ± 26	127 ± 24	NS
Killip class			NS
1	78 (89)	41 (85)	
2	5 (6)	3 (6)	
3	0	3 (6)	
4	5 (6)	1 (2)	
Location of infarction (anterior/inferior)	46/42	27/21	NS
Non–Q-wave infarction (n)*	14 (29)	10 (21)	NS
Peak creatine kinase (IU/L)	3518 ± 2550	2504 ± 1745	0.02
Peak creatine kinase MB fraction (IU/L)	380 ± 290	241 ± 211	0.009
Diseased vessels (n)*			NS
1	57 (65)	35 (73)	
2	24 (27)	10 (21)	
3	2 (2)	2 (4)	
Unknown	5 (6)	2 (4)	
Infarct-related artery (n)*			NS
Left anterior descending	47 (53)	28 (58)	
Right coronary	31 (35)	12 (25)	
Left circumflex	4 (5)	6 (13)	
Unknown	6 (7)	1 (2)	
% diameter stenosis of infarct-related artery (n)*			NS
100	17 (19)	8 (17)	
≤99	11 (13)	11 (23)	
≤90	3 (3)	5 (10)	
≤75	14 (16)	6 (13)	
≤50	37 (42)	17 (35)	
Unknown	6 (7)	1 (2)	
Collateral flow to infarct-related artery (n)*			NS
Grade 0	57 (65)	35 (73)	
Grade 1	4 (5)	4 (8)	
Grade 2	11 (13)	3 (6)	
Grade 3	7 (8)	5 (10)	
Unknown	9 (10)	1 (2)	
Thrombolysis (n)*	18 (20)	14 (29)	NS
Time to thrombolysis (h)	3.9 ± 2.7	3.0 ± 1.8	NS
Angioplasty in acute phase (n)*	23 (26)	12 (25)	NS
Ejection fraction (%)	49 ± 17	48 ± 13	NS

*Values in parentheses are percentages.
NS = not significant.

sion, Killip class, location of infarction, occurrence of non–Q-wave infarction, number of diseased vessels, infarct-related artery, percentage diameter stenosis of infarct-related artery, grade of collateral flow to infarct-related artery, thrombolysis, time to thrombolysis, angioplasty in acute phase, or left ventricular ejection fraction between the 2 groups. Peak creatine kinase and peak MB fraction were significantly smaller in patients with than in those without preinfarction angina.

BMIPP and ^{201}Tl SPECT Findings

In 2 patients without preinfarction angina and 1 with preinfarction angina, significant defects of BMIPP or ^{201}Tl (or both) were observed in remote areas. BMIPP and ^{201}Tl SPECT findings are shown in Table 2 and Figure 1.

No significant difference was noted in the interval between the onset of infarction and SPECT study or total defect scores for BMIPP between the groups with and without preinfarction angina. Total defect scores for ^{201}Tl in initial and delayed images and the smaller total defect score of ^{201}Tl tended to be smaller for patients with preinfarction angina than for those without but not to a statistically significant extent. There was no difference in the changing pattern of ^{201}Tl from initial to delayed image between the 2 groups.

The ratio of the total defect score for the initial image of ^{201}Tl to that for the image of BMIPP was significantly smaller for patients with preinfarction angina than for those without (0.66 ± 0.23 versus 0.76 ± 0.25; $P = 0.011$). The ratio of the smaller total defect score of ^{201}Tl to that for the image of BMIPP was significantly smaller for patients with preinfarction angina than for those without (0.64 ± 0.21 versus 0.74 ± 0.25; $P = 0.007$) (Fig. 1).

TABLE 2
BMIPP and ^{201}Tl SPECT Findings

Parameter	Patients without angina (n = 88)	Patients with angina (n = 48)	P
Interval between onset of infarction and SPECT (d)	13 ± 6	12 ± 4	NS
Total defect score for BMIPP	9.2 ± 5.1	8.6 ± 4.4	NS
Total defect score for ^{201}Tl*			
Initial image	7.4 ± 5.2	6.0 ± 4.3	NS
Delayed image	8.0 ± 5.0	6.9 ± 4.1	NS
Smaller total defect score	7.2 ± 5.2	5.7 ± 4.0	NS
Changing pattern from initial to delayed image (n)†			NS
Fixed defect	41 (47)	17 (35)	
Redistribution	10 (11)	8 (17)	
Reverse redistribution	37 (42)	23 (48)	

*Smaller total defect score of ^{201}Tl is total defect score in 1 image of initial and delayed images of ^{201}Tl that showed smaller total defect score.
†Values in parentheses are percentages.
NS = not significant.

Relationship Between Timing of Angina and BMIPP and ^{201}Tl SPECT Findings

The ratio of the smaller total defect score of ^{201}Tl to that for the BMIPP image was significantly smaller for patients with angina than for those without within 24 h before infarction (0.65 ± 0.22 versus 0.73 ± 0.25; $P = 0.03$). The ratio of the smaller total defect score of ^{201}Tl to that for the BMIPP image tended to be smaller for patients with angina than for those without 24–72 h before infarction (0.64 ± 0.23 versus 0.72 ± 0.24; $P = 0.1$) but not to a significant extent. There was no difference in the ratio of the smaller total defect score of ^{201}Tl to that for the BMIPP image between patients with angina and those without 72 h to 1 wk before infarction (0.70 ± 0.26 versus 0.70 ± 0.24; P = not significant).

Representative scintigraphic images from patients with anterior MI with and without preinfarction angina are shown in Figure 2.

DISCUSSION

In this study, preinfarction angina did not affect the areas at risk in acute MI, as shown by BMIPP defects, but decreased necrotic myocardium in the areas at risk, as shown by ^{201}Tl defects, and increased metabolically damaged but viable myocardium, as shown by BMIPP and ^{201}Tl mismatch.

Incidence of Preinfarction Angina in Patients with Acute MI

In our study, preinfarction angina within 72 h before the onset of acute MI was found for 35% of patients with the first acute MI. The reported incidence of angina before infarction is from 27% to 65% (*1–8*) and depends on the study population and definition of preinfarction angina.

Mechanisms of Effects of Preinfarction Angina on Myocardial Injury

The presence of collateral circulation is more common in patients with angina than in those without (*5–7,17,19*). The recruitability of these collateral channels during transient coronary occlusion in coronary angioplasty appears to be related to preexisting critical coronary artery stenosis (*18*). This collateral circulation protects against myocardial damage in the setting of abrupt coronary occlusion in patients with acute MI (*14–16,18*). Therefore, collateral circulation is thought to be one of the possible mechanisms responsible for limitation of infarct size in patients with preinfarction angina. However, all previous studies estimated the collateral circulation after the onset of MI, and the findings therefore did not reveal the condition of collateral flow at the time of abrupt occlusion of coronary arteries. In this study, collateral flow to the infarct-related artery did not differ between patients with and without preinfarction angina. Moreover, although the area at risk was the same in patients with and without preinfarction angina, as shown by BMIPP defects, preinfarction angina decreased necrotic myocardium in the areas at risk, as shown by ^{201}Tl defects. Andreotti

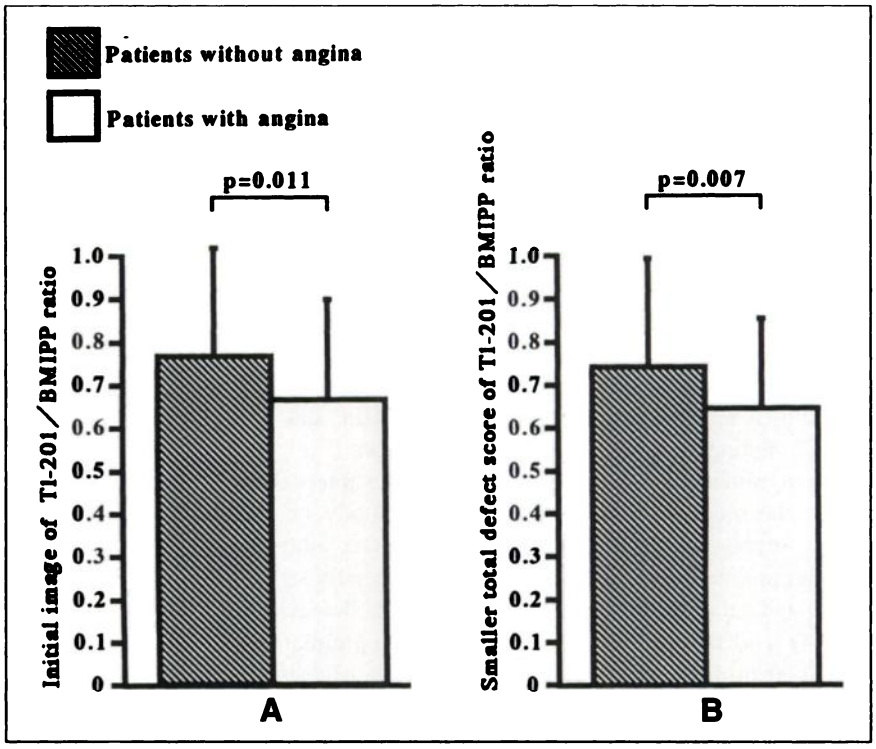

FIGURE 1. Ratio of total defect score for initial ^{201}Tl image to that for BMIPP image was significantly smaller for patients with than for those without preinfarction angina (A) (0.66 ± 0.23 versus 0.76 ± 0.25; P = 0.011). Ratio of smaller total defect score of ^{201}Tl image to that for BMIPP image was significantly smaller for patients with than for those without preinfarction angina (B) (0.64 ± 0.21 versus 0.74 ± 0.25; P = 0.007).

et al. (2) reported that thrombolytic therapy results in more rapid reperfusion in patients with preinfarction angina than in those without preinfarction angina. This phenomenon might contribute to the limitation of infarct size in patients with preinfarction angina. However, this phenomenon of rapid reperfusion in patients with preinfarction angina can only explain in part the limitation of infarct size in patients' preinfarction angina and acute MI because only 32 (24%) of our patients underwent thrombolytic therapy. These findings suggest that neither collateral circulation nor rapid reperfusion is the major mechanism responsible for limitation of infarct size in the presence of preinfarction angina in

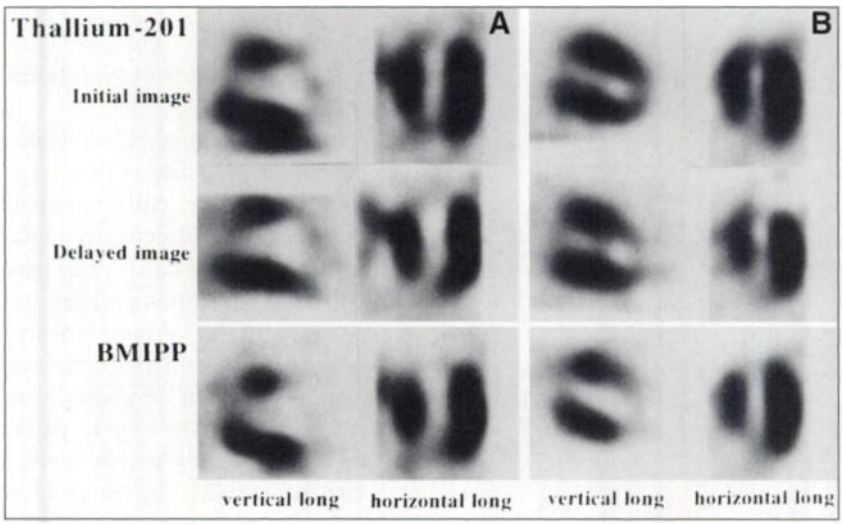

FIGURE 2. Representative scintigraphic images from cases of anterior MI without preinfarction angina (A) and with preinfarction angina (B). (A) A 60-y-old man without preinfarction angina underwent thrombolytic therapy in acute phase of infarction. Coronary arteriography in subacute phase revealed 90% stenosis of left anterior descending artery and no uptake of ^{201}Tl or BMIPP in anteroapical wall. Defects of ^{201}Tl and BMIPP were matched. (B) A 66-y-old man with preinfarction angina underwent thrombolytic therapy in acute phase of infarction. Coronary arteriography in subacute phase revealed 75% stenosis of left anterior descending artery. In ^{201}Tl study, mild defects are observed in anteroapical wall on initial images, and moderate defects are observed in anteroapical wall on delayed images. ^{201}Tl study shows reverse redistribution. Severe defects are observed in anteroapical wall in BMIPP study. Defects of ^{201}Tl and BMIPP are mismatched.

patients with acute MI but that ischemic preconditioning is possibly the major mechanism.

Medical treatment before acute MI may also reduce infarct size. In particular, administration of a β-adrenergic blocking agent as pretreatment before coronary occlusion is reported to limit the infarct size (*29*). However, in this study there were no significant differences in preadmission medications between patients with and those without preinfarction angina.

Type of Angina Pectoris and Effect on Myocardial Protection

Many investigators have reported the effects of preinfarction angina in patients with acute MI but have used differing definitions of preinfarction angina. In a canine experiment, Murry et al. (*9*) showed that much of the protective effect of brief ischemia was lost if the time between the preconditioning episodes of brief ischemia and the later sustained ischemia was extended to several hours. In contrast, Kuzuya et al. (*13*) showed that after the initial preconditioning effect was lost, the delayed preconditioning effect appeared 24 h after a brief episode of ischemia and that at that time the synthesis of manganese superoxide dismutase was augmented. Nakagawa et al. (*8*) reported that preinfarction angina within 24 h before the onset of infarction preserved myocardial contractile function in patients with reperfused anterior wall MI. Kloner et al. (*3*) reported that for patients who experienced angina pectoris within 48 h before infarction, there were trends toward a lower rate of in-hospital death, a lower incidence of severe heart failure or shock, a smaller infarct size, and fewer Q-wave infarcts in patients with thrombolytic therapy. Shiraki et al. (*4*) reported that angina 24–72 h before infarction was most strongly associated with reductions in the rates of right ventricular infarction in patients with acute inferior wall MI. In this study, angina within 72 h before infarction had a significant effect on BMIPP and ^{201}Tl SPECT findings, but angina 72 h to 1 wk before infarction did not. These findings are consistent with those of previous reports (*3,4,8*).

Assessment of Areas at Risk in Acute MI with BMIPP Scan in Subacute Phase

The areas at risk and salvaged myocardium can be estimated accurately by myocardial perfusion imaging with ^{99m}Tc-sestamibi performed before and after revascularization therapy (*30*). However, this method requires tracer administration on admission and imaging shortly after revascularization and is frequently difficult in patients with serious clinical conditions. However, BMIPP imaging in the subacute phase of MI has been reported to permit determination of the amount of myocardium at risk after MI (*24–27*). Kawai et al. (*26*) indicated that the ability of BMIPP imaging at 1 wk after infarction to identify areas at risk is similar to that of ^{99m}Tc-tetrofosmin perfusion imaging performed in the acute phase.

The presence of ^{201}Tl activity at rest represents restored myocardial perfusion and cell viability in an infarct-related segment, and reduced BMIPP uptake reflects metabolic alterations in both viable and nonviable myocardium. Therefore, resting myocardial dual-isotope single-acquisition SPECT using BMIPP and ^{201}Tl in the subacute phase can provide information both on areas at risk and necrotic myocardium in patients with acute MI.

However, Kawai et al. (*26*) reported that the defect score for ^{99m}Tc-tetrofosmin in the acute phase was slightly greater than that for BMIPP in the subacute phase, indicating that the BMIPP defect score was smaller that the actual area at risk. Therefore, the total defect score for BMIPP in this study might have been slightly smaller than the actual area at risk; we believe, however, that this would not influence the conclusions of this study.

Assessment of Myocardial Viability with ^{201}Tl Scan

In previous studies of the relationship between the BMIPP–^{201}Tl mismatch and dysfunctional but viable myocardium, initial images of resting ^{201}Tl SPECT were used to assess myocardial viability (*22–25*). However, many investigators have reported that redistribution on rest–redistribution ^{201}Tl imaging was observed in some patients with chronic coronary artery disease and that delayed images on resting ^{201}Tl scanning were more accurate for detecting myocardial viability than were initial images in patients with old MI (*31,32*). On the other hand, reverse redistribution of ^{201}Tl has commonly been observed in patients with acute MI, and almost all myocardial segments exhibiting reverse redistribution retain glucose metabolism, indicating that initial images more accurately reflect viability than delayed images in patients with reverse redistribution on resting ^{201}Tl scanning (*33,34*). In this study, 18 (13%) patients showed redistribution of ^{201}Tl, and 60 (44%) showed reverse redistribution. Therefore, we used both the initial images and the 1 image of initial and delayed images that showed the smaller total defect score of ^{201}Tl to assess myocardial viability, and the same tendency was observed for both.

Study Limitations

In this study, objective markers of preinfarction angina were obtained for few patients because most patients were admitted to our hospital only after the onset of MI. Accordingly, we could not separate typical from atypical angina. It is well known that myocardial ischemia is not always symptomatic. Ambulatory electrocardiographic monitoring has shown that myocardial ischemia is symptomatic in 10%–40% of events (*35,36*). Moreover, MI in itself is reported to be asymptomatic in 30% of patients (*37*). Because this study was based only on symptoms, we were unable to determine whether symptoms were those of true angina induced by myocardial ischemia, and the significance of silent myocardial ischemia could not be evaluated.

The difference in tissue attenuation between ^{123}I and ^{201}Tl was a potential limitation of this study, and methods for downscatter correction have not been established. Because quantitative analyses often reveal less ^{201}Tl than BMIPP uptake in septal and inferior regions as a result of the

difference in photon attenuation between these 2 tracers, we used a visual semiquantitative scoring system for evaluation of BMIPP and ^{201}Tl uptake. Each tracer uptake was carefully scored considering the effects of photon attenuation.

CONCLUSION

Our findings indicate that preinfarction angina does not affect the areas at risk in acute MI but that it decreases necrotic myocardium in the areas at risk and increases jeopardized but viable myocardium through unidentified mechanisms other than collateral circulation (e.g., ischemic preconditioning).

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