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Myocardial Bridging of the Left Anterior Descending Coronary Artery in Acute Inferior Wall Myocardial Infarction

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Summary

Background: We observed marked myocardial bridging of the left anterior descending coronary artery (LAD) in the acute stages of inferior wall myocardial infarction (MI) in a group of patients who developed shock despite successful reperfusion of the infarct-related lesion (IRL).

Hypothesis: The purpose of this study was to elucidate the clinical significance of myocardial bridging in patients with inferior wall MI and shock.

Methods: The study group consisted of 53 patients with single-vessel disease of the right coronary artery, who underwent coronary angiography for acute inferior wall MI. Clinical characteristics, coronary angiographic findings, and left ventricular function during the chronic phase were compared between the patients who developed shock (the shock group) and those who did not (the non-shock group). In addition, a multiple logistic analysis was performed to identify independent predictors of shock in patients with acute inferior wall MI.

Results: Reperfusion of the IRL was obtained in all 53 patients. The incidence of myocardial bridging of the LAD, the incidence of right ventricular MI, the peak creatine phosphokinase (CPK-MB), the pulmonary capillary wedge pressure, and the prevalence of pulmonary congestion seen on chest roentgenogram were significantly higher in the shock group than in the non-shock group. Myocardial bridging ($p = 0.0018$), right ventricular MI ($p = 0.0374$), and peak CPK-MB ($p = 0.0189$) were identified as independent predictors of shock in acute inferior wall MI.

Conclusion: This study suggests that myocardial bridging plays a role in left ventricular function in the acute stage of inferior wall MI.

Key words: myocardial bridging, inferior wall myocardial infarction, coronary angiography

Introduction

A myocardial bridge is defined anatomically as a band of cardiac muscle that crosses over a coronary artery passing over the surface of the heart.[1] The presence of myocardial bridging is indicated angiographically by narrowing of the coronary arteries during systole and normalization during diastole.[1] The incidence of myocardial bridging ranges from 5.4 to 85.7% as detected by autopsy studies,[1–4] and from 0.5 to 10% as detected by coronary angiography.[5–7] The clinical significance of myocardial bridging is unclear. Some authors have reported that myocardial bridging causes myocardial ischemia,[8–10] while others have found that bridging protects against coronary atherosclerosis[4] and that there is no specific relationship between myocardial bridging and myocardial ischemia.[1] We have observed marked myocardial bridging of the left anterior descending coronary artery (LAD) in patients with acute inferior wall myocardial infarction (MI) who developed shock despite successful reperfusion of the infarct-related lesion (IRL). The objective of this study was to elucidate the clinical significance of myocardial bridging in patients with inferior wall MI and shock.

Methods

Subjects

Of 230 patients admitted to this hospital within 24 h of onset of their first acute inferior wall MI between August 1990 and August 1997, 127 underwent coronary angiography immediately after presentation. Of these 127 patients, 74 with IRLs in the right coronary artery and significant stenosis of

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other coronary arteries (35 with right coronary artery + LAD lesions; 23 with right coronary artery + left circumflex coronary artery lesions; and 16 with right coronary artery + LAD + left circumflex coronary artery lesions) were excluded. The 53 patients (46 men, 7 women, mean age 60 ± 12 years; age range 27–83 years) included in the study had single-vessel disease of the right coronary artery. The criteria for the diagnosis of acute inferior wall MI were typical chest pain unrelieved by sublingual nitrates and continuing for at least 30 min; ST elevation of ≥ 0.1 mV in leads II, III, and aVF on standard 12-lead electrocardiograms (ECG); and a serum creatine phosphokinase (CPK) level more than twice the upper normal value. For all 53 patients, coronary angiography was performed within 6 h of onset, and reperfusion of the IRL was obtained using intracoronary thrombolysis or percutaneous transluminal coronary angioplasty.

The patients were divided into those who developed shock (the shock group) and those who did not (the non-shock group). Patients were considered to be in shock if their systolic blood pressure was ≤ 90 mmHg and ≥ 3 μg/kg/min of a vasopressor (dopamine and dobutamine preparations) was required to maintain a systolic blood pressure of 100 mmHg after hospitalization.

The diagnosis of hypertension was based on the World Health Organization/International Society of Hypertension (WHO/ISH) classification system[11] using blood pressure readings obtained after hospitalization. Hypertension also was diagnosed if the patient was already being treated with antihypertensive agents. The criterion for diagnosis of hyperlipidemia was a total cholesterol level of ≥ 220 mg/dl on hematologic tests conducted after hospitalization. The criteria for diagnosis of diabetes mellitus were a fasting blood sugar level of ≥ 140 mg/dl on hematologic tests conducted after hospitalization, or a blood sugar level of ≥ 200 mg/dl at any time. The diagnosis of pulmonary congestion on admission chest roentgenogram was based on the existence of any of the following findings: bilateral hilar enlargement of the pulmonary arteries, vascular engorgement in the upper lung fields, Kerley B lines, or pleural effusion.

Serum CPK-MB was measured every 3 h after presentation, and the maximal value was defined as the peak CPK-MB. The criterion[12–14] for diagnosis of right ventricular MI was ST elevation of ≥ 0.1 mV in lead V_4R on standard 12-lead ECGs. Pulmonary capillary wedge pressure (PCWP) was measured using a Swan-Ganz catheter inserted through a femoral vein.

The study protocol was approved by the institutional review board of Kyorin University School of Medicine Hospital, and written informed consent was obtained from all subjects.

Coronary Angiography and Analysis of Angiographic Findings

Coronary angiography was performed within 6 h of the onset of chest pain using the Seldinger technique. Images were obtained at a rate of 25 frames/s using a Siemens cardiovascular imaging system (Siemens Medical Systems, Erlangen, Germany). Images were obtained in left anterior oblique 60° and right anterior oblique 30° projections, and the site of stenosis was clarified by obtaining images in several additional views. The lesion responsible for the inferior wall MI, the severity of stenosis, the residual stenosis, and the dominance of the right coronary artery after reperfusion were evaluated. The IRL and severity of stenosis were evaluated using the American Heart Association (AHA) classification system.[15]

The IRLs were divided into two groups: a proximal side group, in whom the IRL was located in AHA segment 1 between the ostium of the right coronary artery and the right ventricular branch; and a distal side group, in whom the IRL was located in AHA segment 2, segment 3, or its distal site. Dominance of the right coronary artery was used to classify the patients into two groups: super-right dominance, in which both the posterior descending coronary artery and all of the posterolateral coronary arteries were of right coronary artery origin; and non-super-right-dominance. The following three groups were included in the non-super-right-dominance group: left dominance, in which the posterior descending coronary artery was of left coronary artery origin; the balanced type, in which the posterior descending coronary artery was of right coronary artery origin and all of the posterolateral coronary arteries were of left coronary artery origin; and right dominance, in which the posterior descending coronary artery was of right coronary artery origin and some of the posterolateral coronary arteries were of right coronary artery origin. Development of collateral flow was studied by dividing the patients into two groups based on the presence or absence of collateral flow. Collateral flow was considered present if flow was grade I (only collateral flow was visualized; epicardial vessels were not) or higher on the Rentrop grading system.[16] Myocardial bridging was considered present if coronary artery narrowing of ≥ 90% was observed during systole.

Left Ventricular Function

Two-dimensional echocardiography was performed during the acute phase of MI and at 1 month (mean: 29 ± 8 days), using a Sonolayer SSH-160A (Toshiba Co., Ltd., Tokyo, Japan) or a Sonos 1000 (Hewlett-Packard Ltd., Andover, Mass., USA) system. To evaluate the wall motion abnormalities caused by MI, the left ventricle was divided into 11 segments. Left ventricular short-axis scans at the mitral and papillary muscle levels and a long-axis scan of the apical region were recorded with the transducer placed in the fourth intercostal space along the left sternal border. Left ventricular wall motion was assessed quantitatively in each segment by visual interpretation using a 4-point scale (normal wall motion, 0; hypokinesis, 1; akinesis, 2; or dyskinesis, 3). The sum of the scores was defined as the total wall motion index.

Left ventriculography was performed 1 month after hospitalization. The left ventricular ejection fraction was calculated by the Sheehan centerline method[17] using a system (Cathex, Tokyo, Japan) to analyze left ventriculograms obtained by simultaneous imaging with left and right anterior oblique angles of 60° and 30°, respectively. Coronary angiography was performed simultaneously using the methods described above.

Statistical Analysis

Statistical analysis was performed using StatView 4.11 (Abacus Concepts, Inc., Berkeley, Calif., USA). Logistic regression was performed using the procedure "Logistic Regression" of the Statistical Package for Social Sciences (SPSS Inc., Chicago, Ill., USA) for Macintosh, Version 6.1J, choosing the forward-stepping selection method and the default criteria offered by SPSS. Numerical data are expressed as the mean value ± standard deviation. For comparisons between two groups, the Student's unpaired *t*-test for continuous variables and the chi-square test with Yates' correction for categorical data were employed, and differences with a $p<0.05$ were considered significant. Multiple logistic analysis was used to identify predictors of shock in patients with acute inferior wall MI. The 95% confidence intervals (CIs) for each odds ratio (OR) were determined.

Results

Demographic and Other Baseline Patient Characteristics

Of the 53 subjects, 17 were classified in the shock group and 36 in the non-shock group. The mean duration of the shock, during which patients required ≥ 3 μg/kg/min of vasopressor support (dopamine and dobutamine preparations), was 39 ± 26 h. We initially treated patients with acute inferior MI whose systolic blood pressure after hospital admission fell to < 90 mmHg with intravenous fluid infusion so that the central venous pressure became 15 mmHg. When the systolic blood pressure still did not exceed 100 mmHg, we started simultaneous administration of dopamine and dobutamine at 3 μg/kg/min (total dose: 6 μg/kg/min). Then we increased the dose of both drugs by 2 to 3 μg/kg/min in order to maintain the systolic blood pressure at 100 mmHg. The mean maximum doses of dopamine and dobutamine given to the shock group were 13.1 ± 9.4 μg/kg/min each. There was no significant difference between the shock and non-shock groups with regard to gender, age, or incidence of hypertension, hyperlipidemia, diabetes mellitus, or smoking (Table I). There was no significant difference in the time from onset to reperfusion between the shock and non-shock groups (5.1 ± 0.9 h vs. 4.8 ± 1.2 h, not significant).

The peak CPK-MB was significantly higher in the shock group (345 ± 168 IU/l) than in the non-shock group (258 ± 133 IU/l) ($p = 0.0468$, Table I). Right ventricular myocardial infarction occurred as a complication in 11 (65%) of the 17 patients in the shock group, and 11 (31%) of the 36 patients in the non-shock group ($p = 0.0390$, Table I). Signs of pulmonary congestion on admission chest roentgenogram were observed more frequently in the shock group than in the non-shock group (11/17 = 65% vs. 8/36 = 22%, $p = 0.0067$, Table I). The PCWP was significantly higher in the shock group (14 ± 8 mmHg) than in the non-shock group (9 ± 3 mmHg) ($p = 0.0041$, Table I). However, there was no significant relationship between the incidence of myocardial bridging and the incidence of signs of pulmonary congestion on admission chest roentgenogram.

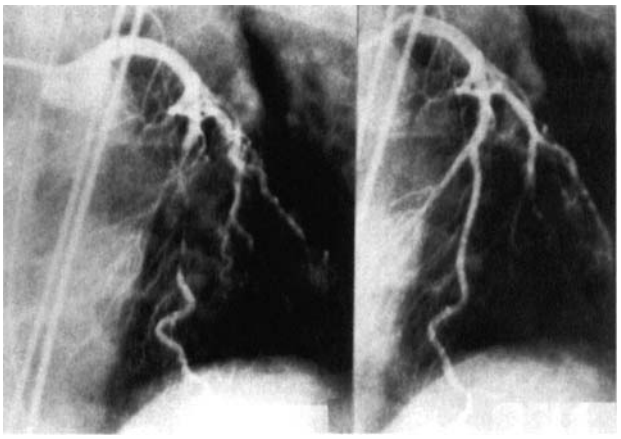
Coronary Angiographic Findings

The incidence of myocardial bridging, detected by coronary angiograms obtained in the acute stage, was significantly higher in the shock group than in the non-shock group (9/17 = 53% vs. 2/36 = 6%, $p<0.0001$, Table I). However, in all patients in whom myocardial bridging was observed in the acute stage (except in three who died and could therefore not be further assessed), myocardial bridging was no longer observed in the chronic stage (Figs. 1 and 2). Three patients in the shock group and two patients in the non-shock group had a myocardial bridging without right ventricular infarction.

TABLE I Baseline characteristics in patients with acute inferior wall myocardial infarction

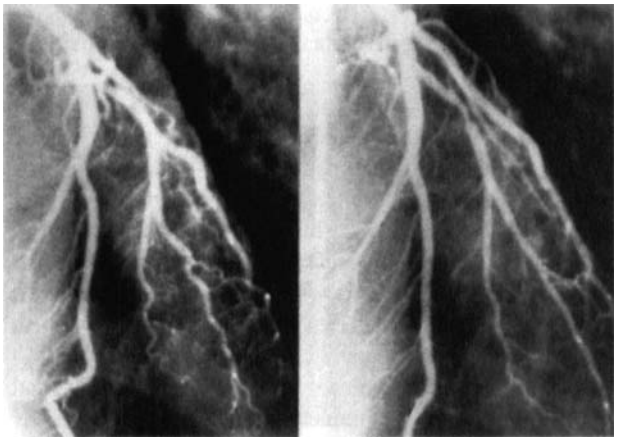
	Shock (n = 17)	Non-shock (n = 36)	p Value
Gender (M/F)	15/2	31/5	NS
Age, years	62 ± 11	59 ± 13	NS
Risk factors			
Hypertension, n (%)	10 (59)	16 (44)	NS
Hyperlipidemia, n (%)	4 (22)	13 (37)	NS
Diabetes mellitus, n (%)	4 (22)	7 (20)	NS
Smoking, n (%)	16 (94)	30 (83)	NS
Coronary artery findings			
Myocardial bridging, n (%)	9 (53)	2 (6)	< 0.0001
Location of IRL			
RCA proximal, n (%)	8 (47)	13 (36)	NS
Stenosis of IRL			
Before intervention, %	99 ± 3	97 ± 9	NS
After intervention, %	52 ± 23	64 ± 32	NS
Dominance of RCA			
Super-right, n (%)	8 (47)	10 (28)	NS
Collateral flow			
Absence, n (%)	10 (59)	18 (50)	NS
Time to reperfusion, h	5.1 ± 0.9	4.8 ± 1.2	NS
Peak CPK-MB, IU/l	345 ± 168	258 ± 133	0.0468
RV infarction, n (%)	11 (65)	11 (31)	0.0390
PCWP, mmHg	14 ± 8	9 ± 3	0.0041
Pulmonary congestion, n (%)	11 (65)	8 (22)	0.0067
Total wall motion index			
Acute phase	7.7 ± 2.7	6.4 ± 2.2	NS
Chronic phase	7.0 ± 2.6	5.1 ± 2.3	0.0173
LVEF during chronic phase, %	55 ± 12	63 ± 14	NS
In-hospital death, n (%)	3 (18)	1 (3)	NS

Abbreviations: M = male, F = female, CPK = creatine phosphokinase, IRL = infarct-related lesion, NS = not significant, PCWP = pulmonary capillary wedge pressure, RCA = right coronary artery, RV = right ventricle, LVEF = left ventricular ejection fraction.



(A) Systole (B) Diastole

FIG. 1 Coronary angiograms of the left coronary artery during the acute stage of myocardial infarction. The presence of myocardial bridging of the left anterior descending coronary artery is seen during systole (A).



(A) Systole (B) Diastole

FIG. 2 Coronary angiograms of left coronary artery in the chronic stage. Myocardial bridging of the left anterior descending coronary artery is not observed during systole, and (B) indicates normal coronary angiogram during diastole.

The IRL was on the proximal side of the right coronary artery in 47% of the shock group and in 36% of the non-shock group. This difference was not significant (Table I). Stenosis of the IRL was present before reperfusion in 99 ± 3% of the shock group and in 97 ± 9% of the non-shock group, and after reperfusion in 52 ± 23% of the shock group and in 64 ± 32% of the non-shock group. None of these differences were significant (Table I). Super-right-dominance was observed in 47% of the shock group and in 28% of the non-shock group, with no significant difference between the two groups (Table I). Collateral flow was absent in 59% of the shock group and in 50% of the non-shock group. This difference was not significant (Table I).

Left Ventricular Function

The total wall motion index during the acute phase of infarction did not differ between the groups (7.7 ± 2.7 in the shock group and 6.4 ± 2.2 in the non-shock group), but the total wall motion index 1 month after infarction was higher in the shock group than in the non-shock group (7.0 ± 2.6 and 5.1 ± 2.3, respectively, p = 0.0173). There was no significant difference between the patients with and without myocardial bridging with regard to total wall motion index during acute and chronic phase, or left ventricular ejection fraction 1 month after infarction (total wall motion index during acute phase, 6.9 ± 2.6 vs. 6.8 ± 2.3; total wall motion index during chronic phase, 5.5 ± 2.6 vs. 6.1 ± 1.7; chronic left ventricular ejection fraction, 54 ± 14 vs. 62 ± 14%). Chronic left ventricular ejection fraction did not differ significantly between the groups (55 ± 12% in the shock group and 63 ± 14% in the non-shock group, Table I). Left ventriculography could not be performed in 13 patients; of these, four died (three in the shock group and one in the non-shock group), renal function was impaired in three, and six refused.

Multivariate Analysis of Shock Predictors

A multiple logistic analysis was conducted to identify predictors of shock in acute inferior wall MI, using acute coronary angiographic findings (IRL, dominance of the right coronary artery, development of collateral flow, and incidence of myocardial bridging), peak CPK-MB, and right ventricular MI as independent variables. Myocardial bridging (OR: 78.0, 95% CI = 5.10–1191, p = 0.0018), right ventricular MI (OR: 6.70, 95% CI = 1.13–39.9, p = 0.0374), and peak CPK-MB (OR: 1.00, 95% CI = 1.00–1.01, p = 0.0189) were identified as predictors (Table II).

Discussion

Myocardial Bridging of the Left Anterior Descending Artery during Acute Inferior Wall Myocardial Infarction

In this study, myocardial bridging of the LAD, right ventricular MI, and peak CPK-MB were identified as predictors of shock in patients with acute inferior wall MI. Creatine

TABLE II Multiple logistic regression analysis of factors associated with shock after acute inferior wall myocardial infarction

Factor	Odds ratio	95% CI	p Value
Myocardial bridging	78.0	5.10–1191	0.0018
RV infarction	6.70	1.13–39.9	0.0374
Peak CPK-MB	1.00	1.00–1.01	0.0189

Abbreviations: CI = confidence interval, RV = right ventricle, CPK = creatine phosphokinase.

phosphokinase-MB reflects the size of the infarct and has already been shown to be related to left ventricular function and prognosis.[18–20] However, the results of multivariate analysis indicate that CPK-MB is not as closely related to shock as the other two factors. Since the significant difference and standard deviation were small for the CPK values, the onset of shock cannot be explained solely by the size of the myocardial infarct. Shock associated with inferior wall MI has previously been identified as a major complication of right ventricular MI.[12] However, the relationship between myocardial bridging of the LAD observed during acute inferior wall MI and the hemodynamic deterioration during inferior wall MI remains unclear.

Porstmann and Iwig[21] used coronary angiography to demonstrate that some coronary arteries are embedded in the myocardium and narrow during systole. The myocardium that covers these coronary arteries is called a myocardial bridge. Whether narrowing during systole due to bridging actually causes myocardial ischemia has been debated, since most blood flow occurs during diastole in the coronary circulation. However, it is now accepted that myocardial bridging does cause myocardial ischemia[8–10] and may even result in MI and sudden death.[22–24]

The results of this study support these previous findings. We were unable to evaluate left ventricular wall motion in the patients with shock, since left ventriculography was not performed during the acute stage. However, Chambers *et al.*[25] have reported a transient reduction in wall motion due to myocardial bridging. In the present study, although there was no difference in left ventricular ejection fraction in the chronic stage between the shock and non-shock groups, pulmonary congestion was observed more frequently, and the PCWP in the acute stage was significantly higher in the shock group than in the non-shock group. It is possible that a transient reduction in myocardial function in the perfusion area of the LAD resulting from myocardial bridging may have been a cause of shock.

At the clinical level, Raizner *et al.*[26] have reported improvement in angina pectoris after cardiomyotomy for myocardial bridging. The results of other studies also have shown improvement in angina pectoris following surgical revascularization for myocardial bridging.[27–29] Noble *et al.*[30] have noted that the incidence of chest pain, ECG ST changes, and elevated lactic acid production increases as myocardial bridging becomes more severe. Endo *et al.*[31] have reported MI in patients with myocardial bridging accompanied by tachyarrhythmia. It has previously been suggested that a reduction of diastolic filling time associated with tachycardia may be responsible for the myocardial ischemia caused by myocardial bridging. In a study of LAD blood flow in patients with myocardial bridging using ultrasound, Hill *et al.*[32] have found that there was a lag between mechanical dilation of the ventricles and the initial phase of diastolic flow in the coronary arteries, and that this lag was greater when tachycardia was more severe. This time lag may lead to marked impairment of diastolic flow. In their study, the time lag resolved and diastolic coronary blood flow increased when the myocardial bridging was removed surgically. Mouratidis *et al.*[33] have reported a transient reduction of blood flow in the LAD region on exercise thallium myocardial scintigraphy in patients with angina pectoris with myocardial bridging of the LAD but no intracoronary lesions. Studies by Sueda *et al.*[34] and Ahmad *et al.*[35] showed similar findings.

We observed that myocardial bridging occurred only in the acute phase of MI and not in the recovery stage. The myocardial bridge is an anatomical feature that is present in both the acute and chronic phases. In the acute phase of inferior wall infarction, compensatory hypercontraction of the anterior wall is assumed to take place in response to the sudden decrease in the movement of the inferior wall. Other factors of hypercontraction of the anterior wall myocardium are intrinsic catecholamines released by the onset of shock and the use of dopamine and dobutamine. As a result, severe coronary compression occurs at the site of the myocardial bridge due to far stronger contraction of the myocardium around the coronary arteries when compared with normal. The reason for the absence of coronary compression in the chronic phase appears to be disappearance of the compensatory anterior wall hypercontraction so that the myocardial bridge no longer has a compressive effect.

Myocardial Bridging in Acute Inferior Wall Myocardial Infarction

The presence of myocardial bridging in a state of increased myocardial oxygen consumption can lead to ischemia.[36] Oxygen consumption by the anterior wall myocardium likely increases during compensatory hypercontraction of the anterior wall in response to reduced inferior wall motion caused by MI of the inferior wall. It is believed that the major cause of shock in the patients described in this study was an inability to meet the increased oxygen demands of the anterior wall myocardium subsequent to inferior wall MI, due to severe myocardial ischemia resulting from reduction of blood flow caused by myocardial bridging of the LAD.

The fact that ischemic symptoms appear from middle age onward[5] in patients with myocardial bridging, believed to be essentially congenital in nature,[4] suggests that various factors play a role in the appearance of ischemic symptoms associated with myocardial bridging. Ge *et al.*[37] have proposed that myocardial ischemia associated with myocardial bridging may result from reduction of coronary blood flow reserves. In this study, coronary angiography performed in the chronic stage of infarction showed an absence of myocardial bridging in the same patients in whom myocardial bridging was observed in the acute stage, and left ventriculography performed at this time showed that anterior wall motion had already returned to normal. Echocardiography conducted in the chronic stage showed improvement in the motion of the inferior wall compared with the acute stage. This was attributed to the disappearance of myocardial bridging in the chronic stage following the resolution of compensatory hypercontraction of the anterior wall. Disappearance of compensatory anterior wall hypercontraction in the chronic phase appeared to be related to coronary revascularization of the responsible coronary artery

and resolution of stunning of the residual inferior wall myocardium. This decreased the load on the anterior wall. Also, the effect of catecholamines administered during the acute phase to treat shock was no longer present.

Pulmonary congestion and the high PCWP suggest reduced left ventricular function. If myocardial bridging causes ischemia of the left ventricular myocardium and reduces left ventricular function, there should be a correlation between the presence of myocardial bridging and the frequency of pulmonary congestion or high PCWP. However, our results showed a correlation between shock and myocardial bridging, but no correlation between myocardial bridging and the frequency of pulmonary congestion or a high PCWP during the acute phase. The PCWP tended to be higher in the myocardial bridge group than in that with no bridge, although the difference was not statistically significant (13.3 ± 9.5 vs. 10.0 ± 3.2 mmHg, $p = 0.0884$). The high frequency of pulmonary congestion and high PCWP in the shock group suggested a relationship between shock and reduced left ventricular function. It is possible that in a state of shock, left ventricular function is reduced even in patients without myocardial bridging because of secondary myocardial ischemia in the subendocardial region. In patients who are in shock, it is difficult to study the correlation of myocardial bridging alone with pulmonary congestion or a high PCWP. Therefore, we used multivariate analysis to identify the independent factors for cardiogenic shock during acute inferior MI. In the present study, results of multivariate analysis revealed that myocardial bridging is one of the independent factors that was significantly related to shock. However, there is no evidence that the myocardial bridging seen in these patients was a direct cause of myocardial ischemia, since left ventricular function could unfortunately not be evaluated by left ventriculography during the acute stage.

Study Limitations

The results of this study suggest that myocardial bridging of the LAD plays a role in the induction of shock in acute inferior wall MI. However, it is unclear whether left ventricular contraction was actually impaired in the acute stage, since left ventricular function could not be assessed by left ventriculography at this time; nor were we able to confirm a cause–effect relationship between myocardial bridging and myocardial ischemia, despite being able to predict the phenomenon clinically, since dobutamine stress echocardiography and exercise myocardial scintigraphy were not performed in the chronic stage in patients with myocardial bridging of the LAD. Further prospective study would be needed to confirm the influence of myocardial bridging on left ventricular function in the acute phase of infarction.

We tried to elucidate the clinical significance of myocardial bridging in patients with inferior wall MI and shock, but we could not determine the cause and effect. Although we observed an association between the occurrence of myocardial bridging and cardiogenic shock complicating acute inferior MI, we are still unsure of the exact clinical significance of the myocardial bridge.

Clinical Implications

In the present study, myocardial bridging of the LAD was observed in some patients with acute inferior wall MI who developed shock despite reperfusion of the IRL. Myocardial bridging does not generally require treatment and has a good prognosis, but it can lead to shock if myocardial oxygen consumption increases as a result of compensatory hypercontraction of the anterior wall, such as occurs in acute inferior wall MI. Caution should therefore be exercised even when the IRL is successfully reperfused. Myocardial bridging should also be considered as a possible cause of myocardial ischemia without intracoronary stenosis.

Conclusion

Marked myocardial bridging of LAD was observed in some patients with acute inferior wall myocardial infarction who developed shock despite reperfusion of the IRL. Myocardial bridging, right ventricular myocardial infarction, and peak CPK-MB were identified as predictors of shock in acute inferior wall myocardial infarction, suggesting that myocardial bridging plays a role in left ventricular function in the acute stage of inferior wall myocardial infarction.

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