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Development of collateral circulation after acute myocardial infarction: Its role in preserving left ventricular function

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Summary

The present study evaluated the effects of coronary collateral circulation developing after acute myocardial infarction on global and regional left ventricular function during the chronic stage. The study group consisted of 16 patients with initial myocardial infarction having total occlusion of the proximal left anterior descending coronary artery. To eliminate the effects of collateral circulation existing at the onset of infarction, patients with pre-infarction angina were excluded from this study. The patients were categorized in two groups depending on the extent of their collateral circulation (collateral index: CI 0∼3): group A—patients with significant collateral circulation (CI=2 or 3) to the infarct-related coronary artery; group B—patients without significant collateral circulation (CI=0 or 1).

Their heart rate, left ventricular peak systolic and end-diastolic pressures and cardiac index were similar in the two groups.

The left ventricular end-systolic volume index in the group B was significantly greater than that in the group A (60±21 ml/m^2 vs 34±9 ml/m^2, $p<0.05$). Left ventricular ejection fraction in the group A was significantly greater than that of the group B (55±9% vs 39±15%, $p<0.05$), and a significant difference was observed in the percentage of segment shortening in the infarct area between the groups A and B (10.8±9.2% vs −0.2±5.4%, $p<0.01$).

It was concluded that coronary collateral circulation which develops after acute myocardial infarction exerts beneficial effects on global and regional left ventricular function during the chronic stage.

Key words

Acute myocardial infarction　Coronary collateral circulation　Left ventricular function

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Introduction

There have been numerous clinical investigations of the efficacy of existing collateral circulation at the onset of acute myocardial infarction in limiting infarct sizes[1~3], salvaging left ventricular function[4,5], and in reducing mortality[6]. However, the role of collateral perfusion developing after infarction in preserving myocardial function has yet to be clarified.

Experimental[7] and clinical studies[8] have revealed a significant development of collateral circulation following acute myocardial infarction. That pre-infarction angina causes formation of well-developed collateral circulation has been well documented[6,9,10]. In the present cardiac study, to eliminate the effects of pre-existing collateral circulation, patients with pre-infarction angina were excluded from analysis. For accurate evaluation of collateral perfusion to the jeopardized myocardium, the recipient coronary artery should be completely obstructed to establish a pressure gradient across a collateral network[11]. Accordingly, patients with recanalization of infarct-related coronary arteries were excluded from analysis.

The primary purpose of the present study was to evaluate the effects of collateral circulation developing after acute myocardial infarction on global and regional left ventricular function, as assessed by ventriculography during the chronic stages.

Patients and methods

From April 1979 to March 1988, 143 patients with recent transmural myocardial infarction were referred for angiographic evaluation. Diagnosis of myocardial infarction was made according to severe chest pain exceeding 30 min and the presence of abnormal Q waves on standard 12 lead electrocardiograms. Among the 143 patients, 119 had had initial myocardial infarction. Excluded were 37 patients with the recanalized infarct-related coronary artery, 48 with pre-infarction angina, and 18 with the total occlusions of either right coronary artery (n=13) or the left circumflex coronary artery (n=5). The remaining 16 patients with total occlusion of the proximal left anterior descending coronary artery composed the present study group (**Fig. 1**). There were 12 men and four women whose mean age was 55±9 years (range 32~68 years). Pre-infarction angina was defined as " typical " chest pain occurring more than one week prior to acute myocardial infarction.

Catheterization and data acquisition

At fifty-seven days (range 26~185 days) after acute myocardial infarction, all patients undergoing conventional diagnostic right- and left cardiac catheterization together with left ventriculography and selective coronary angiography signed appropriate consent forms. All medications were withheld for at least 24 hours prior to the procedure. Cardiac output was determined by the thermodilution technique. Pressures were measured under the basal conditions with standard water-filled catheters. Selective coronary arteriography was performed by the brachial or femoral approach using Renografin-76® contrast medium. All angiograms were recorded at 60 frames/sec on 35 mm Kodak

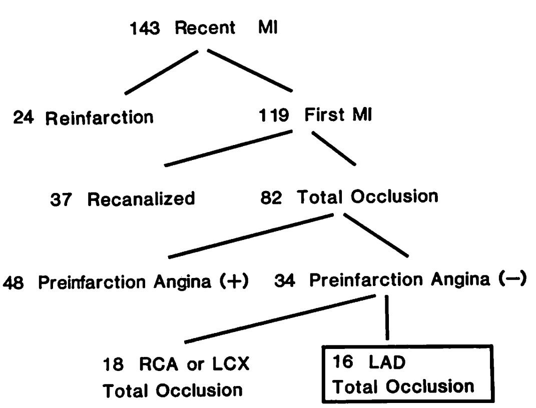

Fig. 1. Selection of the study population.

There were 119 patients with a first myocardial infarction. Thirty-seven patients with a subtotal occlusion of the infarct-related artery in coronary cineangiograms, 48 with pre-infarction angina and 18 with a total occlusion of either the right coronary artery or the left circumflex coronary artery were excluded.

CFS film using a six-inch Toshiba image intensifier system. Contrast medium was injected with sufficient force and quantity to achieve good visualization of the coronary arteries. Multiple projections of the right and left coronary arteries were routinely obtained. After left ventricular pressures had returned to the baseline level, left ventricular cineangiography was performed in a 30° right anterior oblique projection using the 9-inch image intensifier system. Left ventricular opacification was achieved after the injection of 30～40 ml radiopaque contrast medium (80% Angioconray®) at a rate of 10～14 ml/s. Two lead markers were used as fixed references for the superimposition of images. After completion of this study, a 1 cm cross-hatched grid was filmed at the same distance from the X-ray tube and an image intensifier, as the fixed reference.

Analysis of data

A consensus of impressions of three observers who were unaware of the detailed data concerning left ventriculography was used for visual assessments of the coronary cineangiograms, including collateral artery opacifications. The extent of opacification of the collateral and epicardial arteries distal to the completely occluded coronary artery was scored from 0 to 3 (collateral index: CI): grade 0=no collateral opacification; grade 1=filling of branches of the artery to be perfused by collateral vessels without visualization of the epicardial segments; grade 2=partial filling of the epicardial segments by collateral vessels; and grade 3=complete filling of the epicardial segments by collateral vessels. Using a sonic digitizing device one observer who was unaware of the data of coronary angiography traced two left ventricular silhouettes in end diastole and end systole. The end-diastolic frame was determined by ECG simultaneously recorded on the cinefilm as the frame nearest the peak of an R wave. The frame showing the smallest ventricular volume designated as the end-systolic frame. Left ventricular volumes were calculated using a modified formula of Kennedy et al[12]. The ventricular silhouette at end-systole was superimposed on the end-diastolic frame using two external reference markers[13]. Thirty-six radial grids were drawn from the center of gravity of the end-diastolic silhouette to the endocardial margin.

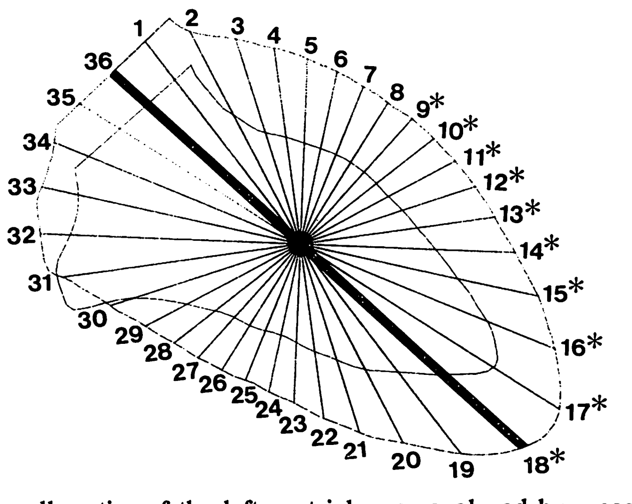

Fig. 2. Regional wall motion of the left ventricle was analyzed by measuring the length of radial grids drawn from the geometric center of gravity of the end-diastolic silhouette to the endocardial margin. Changes in the lengths of grid lines (#9～#18) represent segmental motion of the anterior wall.

Among these 36 radial grids, 30 encompassed the outline of the left ventricular cavity, excluding the regions of the aortic and mitral valves. In the present study, changes in the means of lengths of ten grids (#9~#18) in the anterior segment were considered to denote wall motion of the anterior infarct region (**Fig. 2**). The percentage of systolic segment shortening was calculated using the following formula: (end-diastolic length − end-systolic length) × 100/end-diastolic length.

Subgroups

The patients were categorized in two groups depending on the extent of their collateral circulation: group A-patients who had significant collateral circulation (CI=2 or 3) to the infarct-related coronary artery, and group B-patients who had no significant collateral perfusion (CI=0 or 1).

Statistical analysis

All values are expressed as means±SD. Differences in means were compared using the unpaired Student's t-test. A probability (p) value of less than 0.05 was considered significant.

Results

Coronary collateral index

Among the six patients in the group A, five had fairly good collateral development (CI=2)

Table 1. Summary of 16 patients each with a completely occluded LAD

Patient No.	Age (yrs) & sex	Segment* and extent of coronary stenosis (%)			Collateral index	Interval**
		RCA	LAD	LCX		
Group A (good collaterals)						
1	65 M	2: 75	6: 100	11: 90	2	4 W
2	54 M	—	6: 100	—	2	10 W
3	52 M	—	7: 100	—	2	5 W
4	57 M	—	7: 100	—	3	5 W
5	68 F	—	7: 100	—	2	4 W
6	55 M	2: 75 3: 90	7: 100	14: 75	2	8 W
Group B (poor collaterals)						
1	55 F	—	6: 100	—	0	9 W
2	57 M	—	7: 100	—	1	6 W
3	47 M	—	7: 100	—	1	6 W
4	58 F	—	6: 100	11: 75	1	6 W
5	65 M	—	7: 100	—	0	5 W
6	59 M	—	7: 100	—	0	13 W
7	32 M	—	7: 100	—	0	5 W
8	56 M	—	6: 100	—	0	5 W
9	62 F	2: 75	7: 100	13: 90	0	8 W
10	41 M	—	6: 100	—	0	9 W

* Coronary artery segments were identified and categorized according to a reporting system proposed by the American Heart Association[16].

** Time interval from the onset of myocardial infarction to angiographic evaluation.

Abbreviations: LAD=left anterior descending coronary artery; RCA=right coronary artery; LCX=left circumflex coronary artery; W=weeks.

Table 2. Hemodynamics and global left ventricular function

	HR (beats/min)	LVPSP (mmHg)	LVEDP (mmHg)	CI (L/min·m²)	LVEDVI (ml/m²)	LVESVI (ml/m²)	EF (%)
Group A	70±11	135±12	12±6	3.3±0.4	80±15	34±9	55±9
Group B	76±14	122±13	17±9	3.1±1.0	99±23	60±21	39±15
p value	NS	NS	NS	NS	NS	<0.05	<0.05

HR=heart rate; LVPSP=left ventricular peak systolic pressure; LVEDP=left ventricular end-diastolic pressure; CI=cardiac index; LVEDVI=left ventricular end-diastolic volume index; LVESVI=left ventricular end-systolic volume index; EF=ejection fraction; NS=not significant.

and one had a well-developed collateral circulation (CI=3). In seven of the 10 patients in the group B, collateral vessels were not visualized (CI=0); and three had poor collateral development (CI=1) (**Table 1**).

Hemodynamics and left ventricular function

The hemodynamic data for each group are shown in **Table 2**. There were no significant differences in heart rate, left ventricular peak systolic and end-diastolic pressures and the cardiac index between the two groups. The left ventricular end-diastolic volume index tended to be greater in the group B, and the end-systolic volume index was significantly greater in the group B as compared to the group A (60±21 ml/m² vs 34±9 ml/m², $p<0.05$). Ejection fraction was significantly greater in the group A as compared to the group B (55±9% vs 39±15%, $p<0.05$) (**Fig. 3**). Regional wall motion in the infarct region was also significantly greater in the group A than in the group B (10.8±9.2% vs −0.2±5.4%, $p<0.01$) (**Fig. 4**).

Discussion

Role of collaterals in the preservation of left ventricular function

In the present study angiographically demonstrable collaterals were associated with better preservation of regional wall motion in the

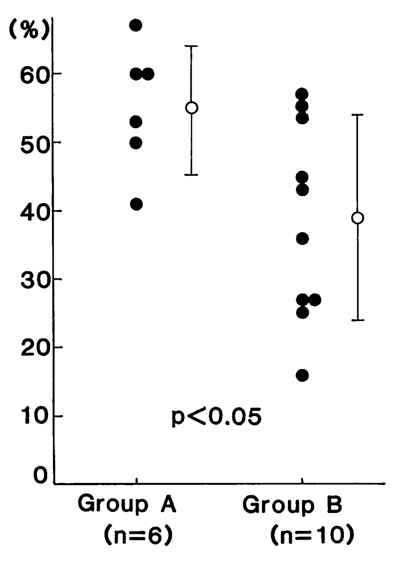

Fig. 3. Left ventricular ejection fraction.

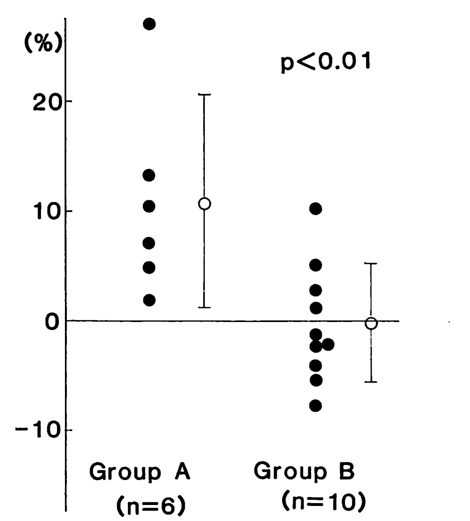

Fig. 4. Left ventricular regional wall motion in the infarct area.

region perfused by the infarct-related coronary artery. Previous studies have emphasized the importance of collateral blood flow to the infarct zone upon acute coronary occlusion for salvaging the myocardium to be rendered ischemic[1,14,15], for protection against cardiogenic shock[6] and preserving regional myocardial function[1,4]. Our data demonstrated that collateral circulation developing after infarction also has a crucial role in preserving regional function of the jeopardized myocardium.

Collateral development after infarction

Although this retrospective study could not provide any insight into the presence of collateral vessels at the onset of infarction, it seems unlikely that patients without pre-infarction angina have good collateral circulation before infarction. We have recently reported that collateral circulation was observed in only two of 19 patients without pre-infarction angina who had coronary angiography within six hours of the onset of infarction[9]. Thus, it is reasonable to assume that collateral vessels in our patients developed after infarction, although there is no information as to when or how they were formed. In view of these acknowledged beneficial effects of collateral perfusion, attempts could be made to stimulate collateral growth after infarction.

Conclusion

Coronary collateral circulation developing after acute myocardial infarction exerts beneficial effects on global and regional left ventricular function during the chronic stage.

要　約

梗塞後に発達した冠側副血行循環の慢性期左心機能に及ぼす影響

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急性心筋梗塞後に発達した冠側副血行循環の心筋梗塞慢性期左室機能に及ぼす影響を検討した．梗塞前狭心症を有さない初回梗塞患者で，慢性期における冠動脈造影にて，左前下行枝近位部に完全閉塞を認めた前壁梗塞患者 16 例を対象とした．冠側副血行循環の発達の程度を 4 段階に分け（CI＝0～3)，発達良好群（A 群；CI＝2,3）と発達不良群（B 群；CI＝0,1）における慢性期左室血行動態，および梗塞部の局所壁運動を定量的に評価した．

A 群（6 例）と B 群（10 例）の間で，心拍数，左室収縮期圧，左室拡張期末圧，心係数はほぼ同じであったが，左室収縮期末容積係数は A 群で有意に小であった（34±9 ml/m^2 vs 60±21 ml/m^2, $p<0.05$)．左室駆出率は A 群で大きく（55±9% vs 39±15%，$p<0.05$)，梗塞部局所壁短縮率も A 群が B 群よりも良好であった（10.8±9.2% vs −0.2±5.4%，$p<0.01$)．

以上より，急性心筋梗塞後に発達した良好な冠動脈側副血行循環は，左室全体機能および梗塞部左室局所壁運動を良好に保つのに貢献すると結論された．

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