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Cardiac Troponin I Level Correlates With Chronic-Phase Left Ventri-cular Function After Successful Direct Percutaneous Transluminal Coronary Angioplasty in Patients With Acute Myocardial Infarction

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Abstract

Objectives. The relationships between cardiac troponin I, various biochemical markers, and chronic-phase left ventricular ejection fraction(LVEF)after successful direct percutaneous transluminal coronary angioplasty(PTCA)were examined in 36 patients with acute myocardial infarction.

Methods. Biochemical markers were measured on admission, immediately after, and from 6 hours to 9 days after PTCA.

Results. The time to peak values were: creatine kinase-MB 9.7 hours, cardiac troponin I 9.8 hours, myoglobin 10.7 hours, creatine kinase 10.6 hours, cardiac troponin T 18.6 hours, and myosin light chain 68.9 hours. Cardiac troponin T, cardiac troponin I and myosin light chain levels were elevated over 9 days after successful direct PTCA. Chronic-phase LVEF inversely correlated with peak values of creatine kinase-MB($r=-0.519$, $p<0.01$), cardiac troponin T($r=-0.500$, $p<0.01$), cardiac troponin I($r=-0.441$, $p<0.05$)and creatine kinase($r=-0.411$, $p<0.05$). The values of cardiac troponin I, cardiac troponin T, creatine kinase and creatine kinase-MB at each sampling point were significantly inversely related to chronic-phase LVEF. The value of cardiac troponin I at each time point for 7 days correlated well with chronic-phase LVEF.

Conclusions. Cardiac troponin I has high specificity for predicting long-term cardiac function after successful direct PTCA when early values are unavailable.

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Key Words

■ **Angioplasty** ■ **Myocardial infarction, pathophysiology** ■ **Troponin** ■ **Ventricular function**

INTRODUCTION

Various biochemical markers for myocardial injury have been used in the early diagnosis and follow-up of patients with acute myocardial infarction[1−3]. Early recanalization to maintain cardiac function in the chronic phase might improve the prognosis for patients with acute myocardial infarction. Recently, direct percutaneous transluminal coronary angioplasty(PTCA)has been used in many institutions worldwide to treat patients with acute myocardial infarction. If chronic-phase car-

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diac function is predicted by the early values of biochemical markers after direct PTCA, these markers might be useful to guide rehabilitation and pharmacological treatment.

Creatine kinase has long been the standard index for myocardial damage. Recently, biochemical markers such as cardiac troponin I, which are more specific for myocardial damage than creatine kinase, have been measured[2,4−6]. Apple *et al*.[7] reported the relationship between cardiac troponin I and cardiac function in the acute phase. Their patients were studied under different conditions, for example with or without PTCA, or systemic thrombolytic therapy. Moreover, patients who underwent coronary angiography were not studied in the chronic phase, as cardiac function was measured only by two-dimensional echocardiography 3 days after myocardial infarction.

We studied the relationship between left ventricular function measured by left ventriculography in the chronic phase (at least 3 to 12 months after acute myocardial infarction) and serum cardiac troponin I as well as other cardiac markers. Further, the time courses of the serum values of various biochemical markers were determined in patients with acute myocardial infarction treated by successful direct angioplasty.

METHODS

Thirty-six consecutive patients (24 men, 12 women, mean age 60.5 ± 10.6 years) with initiation acute myocardial infarction were studied (**Table 1**). All patients were referred to Dokkyo University Hospital between January 1997 and February 1998. All cases of myocardial infarction met the World Health Organization Criteria[8], which requires the presence of symptoms plus either enzymatic elevation or diagnostic electrocardiographic changes. After obtaining informed consent, coronary angiography was performed in all patients. Direct PTCA was performed if patients had at least 90% stenosis of a major coronary artery on coronary angiography after intracoronary injection of nitrate agent. All patients were candidates for direct angioplasty and presented within 6 hours of onset of chest pain, or those with chest pain that persisted after 6 hours of onset when direct angioplasty is necessary for life-saving. These patients did not undergo direct current shocks.

Onset time of myocardial infarction was defined as the time when chest pain did not resolve spontaneously. Successful angioplasty was defined as the presence of residual stenosis of less than 25% in the target lesion. The condition of the target lesion was re-evaluated by coronary angiography and left ventricular function calculated using the Siemens densitometric autotrace system (Siemens Co.) from a 30-degree right anterior oblique ventriculogram from 3 to 12 months (defined as the chronic phase) after direct angioplasty. Patients were excluded if angioplasty was unsuccessful or the target lesions for coronary angioplasty were totally occluded 3 to 12 months after direct angioplasty.

Creatine kinase, creatine kinase-MB, cardiac troponin I, cardiac troponin T, myosin light chain and myoglobin levels were measured in the serum on admission, immediately after, and at 6, 12, 18, 24, 48, and 72 hours and 5, 7, and 9 days after angioplasty. The Hitachi 7170 automatic analyzer (Hitachi Co.) was used to measure creatine kinase and creatine kinase-MB activity using Merck auto creatine kinase and creatine kinase-MB (Merck Co.). The ES 6000 automatic analyzer (Beringer-Manheim Co.) was used to measure cardiac troponin T using the enzymatic immunoassay method. The ARC-950 γ-scintillation counter (Aloka Co.) was used to measure cardiac troponin I with iodine-125-anti-cardiac troponin I monoclonal antibody using the immunoradiometric assay. Myosin light chain and myoglobin levels were measured using the myosin LI Kit (Yamasa Co.) and the myoglobin Kit III (Daiichi Radioisotope Lab.), respectively. The normal cut-off values of creatine kinase, creatine kinase-MB, cardiac troponin I, cardiac troponin T, myosin light chain and myoglobin were 119, 25 IU/*l*, 0.33, 0.25, 2.5 and 60 ng/m*l*, respectively. The cut-off ratio was calculated in each sample.

$$\text{Cut-off ratio} = \text{blood concentration/cut-off value}$$

All data are expressed as mean ± standard deviation. The correlation coefficient was calculated for paired data. Statistical analysis was conducted with a commercially available statistical software (STATVIEW, Abacus Concepatients). Significant differences were determined by one way analysis of variance and non-parametric statistical analysis. A value was considered to be statistically significant when $p < 0.05$.

RESULTS

The mean time from the appearance of clinical

Table 1 Patient characteristics

Patient No.	Age (yr)	Sex	Culprit lesion	Time to hospital (hr)	Coronary risk
1	52	F	LCX	9	Smoking, HT
2	70	M	LCX	5	Smoking
3	46	M	LCX	3	DM, HT
4	61	M	LCX	23	DM
5	56	F	LAD	2	Smoking, HT
6	55	F	LAD	12	Smoking, DM, HT
7	50	F	LAD	2	Smoking
8	73	F	LAD	4	HT
9	52	F	LAD	9	
10	47	M	LAD	5	HL
11	58	M	LAD	6	Smoking, DM, HL
12	60	M	LAD	3	Smoking, DM
13	74	M	LAD	6	
14	65	M	LAD	4	Smoking, DM, HL
15	49	M	LAD	6	Smoking
16	64	M	LAD	8	Smoking
17	65	M	LAD	19	HT
18	56	M	LAD	2	HT
19	68	M	LAD	12	DM
20	49	M	LAD	6	Smoking, HT
21	69	M	LAD	2	Smoking, HT
22	78	M	LAD	22	
23	49	M	LAD	9	
24	61	F	RCA	15	DM, HT, HL, FH
25	72	F	RCA	2	
26	46	F	RCA	2	DM, HT, HL
27	71	F	RCA	5	HT
28	75	F	RCA	9	
29	67	F	RCA	5	HT
30	79	M	RCA	11	Smoking, HT, FH
31	53	M	RCA	13	Smoking
32	39	M	RCA	3	Smoking, HT
33	71	M	RCA	17	
34	48	M	RCA	7	Smoking, HT, HL
35	66	M	RCA	4	DM, HT
36	65	M	RCA	10	HT
Mean±SD	60.5±10.6			7.8±5.7	

F=female; M=male; AMI=acute myocardial infarction; LCX=left circumflex coronary artery; LAD=left anterior descending coronary artery; RCA=right coronary artery; DM=diabetes mellitus; HT=hypertension; HL=hyperlipidemia; FH=family history.

symptoms of myocardial infarction to admission to our hospital was 7.8±5.7 hours. Thirteen culprit lesions were observed in the right coronary artery, 19 in the left anterior descending coronary artery, and 4 in the left circumflex coronary artery. The time courses of each serum biochemical marker are shown in **Fig. 1**. The mean cut-off ratio of cardiac troponin I was observed to be about 10-fold greater

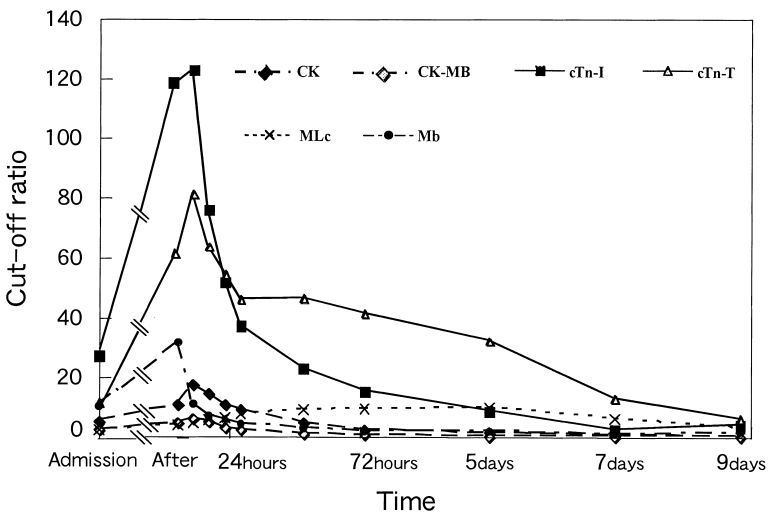

Fig. 1 Time course of each cardiac marker
After: After percutaneous transluminal coronary angioplasty. Cut-off ratio: Blood concentration/cut-off value.
CK = creatine kinase; CK-MB = creatine kinase-MB; cTn-I = cardiac troponin I; cTn-T = cardiac troponin T; MLc = myosin light chain; Mb = myoglobin.

Table 2 Time after the onset of myocardial infarction to the peak value and the recovery time of each biochemical marker

	CK	CK-MB	cTn-I	cTn-T	MLc	Mb
Peak time (hr)	10.6±6.8	9.7±6.5	9.8±6.6	18.6±21.0	68.9±49.1	10.7±13.7
Recovery time (hr)	116.8±8.9	86.6±6.8	216 over	216 over	216 over	119.2±15.9

Continuous values are mean±SD.
Recovery time: Time of return to normal range.
Abbreviations as in Fig. 1.

than the values of the creatine kinase-MB. Creatine kinase peaked at 10.6 ± 6.8 hours after onset, creatine kinase-MB at 9.7 ± 6.5 hours, cardiac troponin I at 9.8 ± 6.6 hours and myosin light chain at 68.9 ± 49.1 hours. The time course of the cardiac troponin T was biphasic, peaking first at 18.6 ± 21.0 hours and again at 48 hours. Myoglobin peaked at 10.7 ± 13.7 hours, and the abnormal value remained elevated until 119.2 ± 15.9 hours. The abnormal creatine kinase and creatine kinase-MB values remained elevated until 116.8 ± 8.9 and 86.6 ± 6.8 hours, respectively. Cardiac troponin T, cardiac troponin I and myosin light chain values declined rapidly although they remained elevated over 9 days. The times after the onset of myocardial infarction to the peak value and the recovery time (the time that the value returned to the normal range) of each biochemical marker are shown in **Table 2**. The order of the recovery time was creatine kinase-MB < creatine kinase < myoglobin. The cardiac troponin I, cardiac troponin T and myosin light chain values did not recover by 9 days.

Twenty-eight of 36 patients underwent follow-up cardiac catheterization during the chronic phase. The mean follow-up interval was 8.7 months. Eight patients dropped out of the chronic-phase study (one patient had renal dysfunction, 3 were too old to qualify for restudy, 2 died of ventricular tachycardia, and 2 were lost to follow-up). The 2 patients who died after ventricular tachycardia did not have higher biochemical markers than the other patients. The relationships between the peak values of each biochemical marker and the chronic-phase left ventricular ejection fractions in the 28 patients are shown in **Fig. 2**. The left ventricular ejection fraction was inversely correlated with peak creatine kinase-MB ($r = -0.519$, $p < 0.01$), cardiac troponin T ($r = -0.500$, $p < 0.01$), cardiac troponin I ($r = -0.441$, $p < 0.05$), creatine kinase ($r = -0.411$, $p < 0.05$). There was no correlation between the left ventricular ejection fraction and myoglobin or myosin light chain levels.

The correlation coefficients between each biochemical marker at each time point and the chronic-phase left ventricular ejection fraction are given

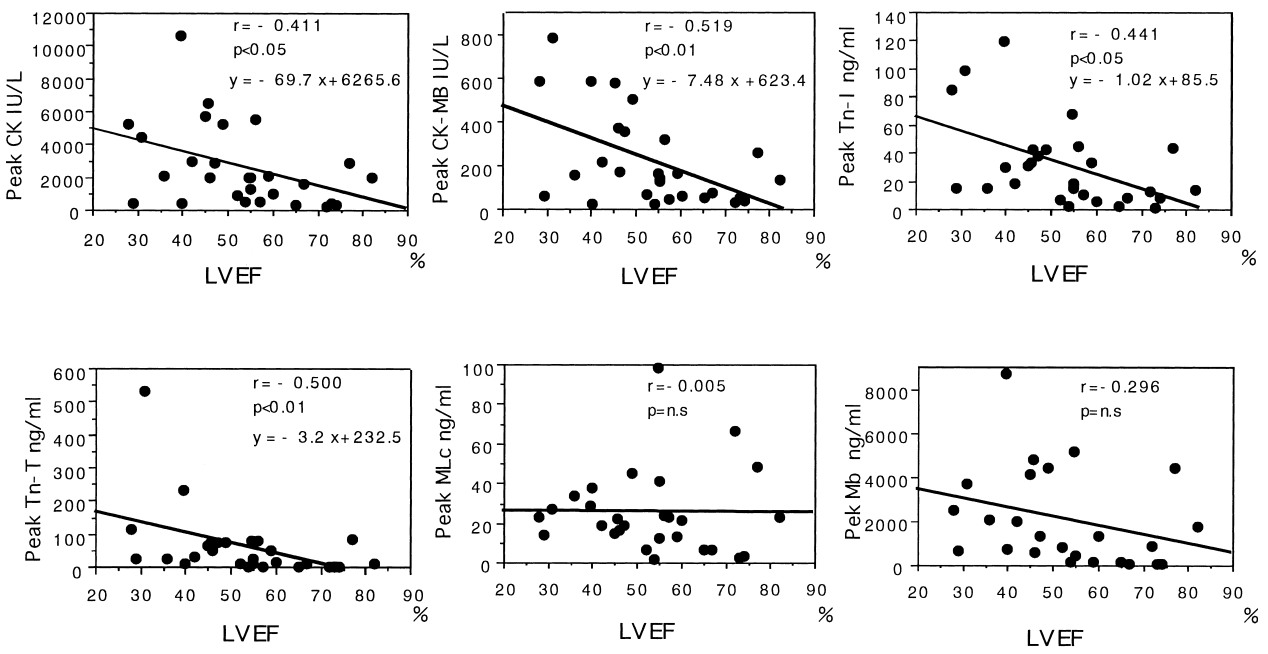

Fig. 2 Relationships between the peak value of each cardiac marker and the left ventricular ejection fraction in the chronic phase after percutaneous transluminal coronary angioplasty
LVEF = left ventricular ejection fraction. Other abbreviations as in Fig. 1.

Table 3 Correlation coefficient between each biomedical marker at each time point and the chronic-phase left ventricular ejection fraction

	CK	CK-MB	cTn-I	cTn-T	MLc	Mb
On admission	−0.13	−0.13	−0.30	−0.19	−0.16	−0.10
After PTCA	−0.48**	−0.52**	−0.43*	−0.55**	−0.21	−0.30
6 hours	−0.39*	−0.30	−0.43*	−0.4*	−0.12	−0.30
12 hours	−0.34	−0.38	−0.36	−0.30	0.01	0.01
18 hours	−0.41*	−0.47*	−0.40*	−0.22	0.06	−0.40
24 hours	−0.40	−0.42	−0.44*	−0.24	0.06	0.00
48 hours	−0.35	−0.36	−0.54**	−0.20	−0.01	0.07
72 hours	−0.32	−0.22	−0.48*	−0.13	0.09	0.24
5 days	−0.11	−0.11	−0.42	−0.06	0.09	0.11
7 days	−0.15	−0.03	−0.45*	−0.17	0.08	0.19
9 days	−0.20	−0.25	−0.39	−0.16	0.06	0.22

**$p<0.01$, *$p<0.05$.
PTCA = percutaneous transluminal coronary angioplasty. Other abbreviations as in Fig. 1.

in **Table 3**. The following values were significant: creatine kinase and creatine kinase-MB values from the time of PTCA to 18 hours after the procedure, the cardiac troponin I values at all times except on admission and 12 hours and 5 and 9 days after the procedure, and the cardiac troponin T values from the time of angioplasty to 6 hours after the procedure. The myosin light chain and myoglobin values were not significant at any time point. Therefore, creatine kinase-MB, creatine kinase, cardiac troponin T and myoglobin values in the early phase of acute myocardial infarction correlated with the left ventricular ejection fraction but were not constant over the late phase. Cardiac troponin I correlated with the left ventricular ejection fraction in the early phase and the late phase.

DISCUSSION

Creatine kinase, creatine kinase-MB, cardiac troponin T, cardiac troponin I and myoglobin values peaked within 12 hours after direct PTCA and the values of cardiac troponin I, cardiac troponin T and myosin light chain remained elevated 9 days after angioplasty. Therefore, creatine kinase, creatine kinase-MB and myoglobin may dissipate in the blood shortly after direct angioplasty. In contrast, myosin light chain, cardiac troponin T and cardiac troponin I were detected in the blood serum for a long period after angioplasty. Moreover, the peak values of myoglobin and myosin light chain were observed during the late phase. Creatine kinase, creatine kinase-MB and myoglobin have been considered as early diagnostic markers, and lactate dehydrogenase, cardiac troponin I, cardiac troponin T and myosin light chain as late diagnostic markers[1,2,9−11]. However, in the present study, most markers peaked within 12 hours after direct angioplasty. The washout phenomenon, induced by early reperfusion in the infarcted myocardium, may be considered as the mechanism[12,13]. Based on analysis of the correlation between the chronic-phase left ventricular ejection fraction and each biochemical marker at every time point, only creatine kinase, creatine kinase-MB and cardiac troponin T in the early phase were related to left ventricular ejection fraction. The correlation coefficient between the peak value of cardiac troponin I and acute-phase left ventricular ejection fraction was −0.46[7]. Our results demonstrated that the correlation coefficient between peak cardiac troponin I and chronic-phase left ventricular ejection fraction in patients with successful direct angioplasty is −0.44(**Fig. 2**).

Thus, the peak value of cardiac troponin I may be a useful index that predicts chronic-phase ejection fraction as well as acute-phase ejection fraction. Furthermore, it was very interesting that every sampling value of cardiac troponin I after PTCA correlated with chronic-phase left ventricular ejection fraction(**Table 3**). Creatine kinase and creatine kinase-MB have been used clinically to diagnose acute myocardial infarction. However, the myocardial specificity of these substances to myocardial injury is questionable. Creatine kinase is present not only in the myocardium but also in the skeletal muscle and the brain[14], and increased values are observed in the presence of muscle, cerebral, and thyroid disease[15]. Although creatine kinase-MB is present mostly in the myocardium, creatine kinase-MB values are also elevated in these diseases[14−16]. Furthermore, the peak creatine kinase value is observed shortly after onset of acute myocardial infarction, and the value becomes extremely high in patients who have undergone successful myocardial reperfusion because creatine kinase flows into the blood without loss in the myocardium with successful early reperfusion[4]. Myoglobin, also a useful early diagnostic biochemical marker, is a carrier protein present in the cytoplasm of muscle cells, but is nonspecific as a biochemical marker for myocardial infarction because its value also increases during skeletal muscle disease, muscle damage, countershock, and renal failure[10].

Cardiac troponin I, cardiac troponin T, and myosin light chain are the most specific biochemical markers for myocardium. Myosin light chain is the structural protein present in myocardial cells, and the peak myosin light chain value can be used to estimate infarction size without influencing reperfusion in acute myocardial infarction[12,13,17,18]. However, in the present study, myosin light chain was not an indicator of the chronic-phase left ventricular ejection fraction after successful direct PTCA. Measurement of the myosin light chain is also not useful in patients with muscular dystrophy and renal disease. In contrast, cardiac troponin I and cardiac troponin T are also myocardial structural proteins and highly specific biochemical markers for myocardium[1,2,4−7,10,12,19]. Cardiac troponin is present in the blood after myocardial injury from the early to the late phase. Cardiac troponin is detectable in blood over the long term because it flows into the blood from the cytoplasm in high concentrations during the early ischemic phase and after collapse of the myocardial muscle fiber in the late ischemic phase[1,10]. The pattern of increasing cardiac troponin T was biphasic in our study, as previously reported[3−5,11−15,18,20−23], but the value did not correlate at any time point with the chronic-phase left ventricular ejection fraction in the present study. Several studies reported that first-generation cardiac troponin T increases during renal disease[11,23]. However, in the present study, cardiac troponin I peaked once, and was significantly inversely related to the chronic-phase left ventricular ejection fraction at each time point up to 7 days after myocardial infarction. Acute-phase cardiac troponin I values might predict chronic-phase left ven-

tricular function even if early serum values are unavailable[24].

This study has certain limitations. Patients presented at a mean of 7.2 hours after the onset of myocardial infarction and the duration from the onset of myocardial infarction to direct PTCA varied; we did not exclude patients with previous angina who had myocardial preconditioning; and the influence on left ventricular function by location of myocardial infarction was not the same.

In conclusion, the value of cardiac troponin I might predict chronic-phase cardiac ejection fraction in early- and late-phase blood samples after successful direct angioplasty in patients with myocardial infarction. This study suggests that cardiac troponin I is useful for estimating myocardial injury and might replace creatine kinase and creatine kinase-MB when early-phase measurements of these indicators are unavailable.

要　　約

急性心筋梗塞患者の経皮的冠動脈形成術成功例における慢性期心機能と心筋トロポニンIとの関係

原　すおみ　　堀中　繁夫　　土谷　範昭

矢部　彰久　　浅川　洋　　八木　博

目　的: 我々は，経皮的冠動脈形成術（PTCA）成功例の急性心筋梗塞患者36例における血清心筋トロポニンI，およびその他の血液生化学的マーカーと，慢性期の左室駆出率との関係を調べた．

方　法: 各生化学的マーカーを，入院時，PTCA直後，6時間後から9日後まで測定した．

結　果: 最大値への到達時間は以下の通りであった．クレアチンキナーゼMBは9.7時間，心筋トロポニンIは9.8時間，ミオグロビンは10.7時間，クレアチンキナーゼは10.6時間，心筋トロポニンTは18.6時間，ミオシン軽鎖は68.9時間であった．心筋トロポニンT，心筋トロポニンIとミオシン軽鎖はPTCA後第9病日までその高値が継続した．マーカーの最高値と慢性期左室駆出率の関係では，クレアチンキナーゼMB（$r=-0.519$，$p<0.01$），心筋トロポニンT（$r=-0.500$，$p<0.01$），心筋トロポニンI（$r=-0.441$，$p<0.05$），クレアチンキナーゼ（$r=-0.411$，$p<0.05$）が有意に負の相関を示した．各採血時点でのマーカー値と慢性期左室駆出率との関係では，心筋トロポニンI，心筋トロポニンT，クレアチンキナーゼMB，クレアチンキナーゼが慢性期の左室駆出率と相関した．これらの指標のうち，測定期間7日間の長期にわたり慢性期の左室駆出率と相関を示したものは，心筋トロポニンIのみであった．

結　論: 心筋トロポニンIは急性心筋梗塞例において，その初期値が不明な場合でもPTCA後の慢性期の心機能を予測する指標となることが示唆された．

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