Effects of Coronary Artery Reperfusion on Relation Between Creatine Kinase-MB Release and Infarct Size Estimated by Myocardial Emission Tomography With Thallium-201 in Man

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The quantitative relations between serum creatine kinase-MB isoenzyme (CK-MB) release and the final infarct size estimated by myocardial emission computed tomography with thallium-201 was assessed in 37 patients with a first acute transmural myocardial infarction who underwent intracoronary thrombolysis using urokinase 4.6 ± 1.9 hours after the onset of symptoms. Serial CK-MB determinations were used to calculate the accumulated release of CK-MB (ΣCK-MB). Myocardial emission tomography with thallium-201 was performed 4 weeks after the onset, and infarct volume was measured from reconstructed tomographic images by computerized planimetry. The results are presented for two groups of patients: 11 patients with unsuccessful thrombolysis (group A) and 26 patients with successful thrombolysis (group B). An excellent linear relation was found for group A (ΣCK-MB = 6.4 x infarct volume + 47.7, r = 0.91), whereas a different linear relation was observed for group B (ΣCK-MB = 10.5 x infarct volume + 89.1, r = 0.80). Moreover, serum CK-MB activity reached a peak at 21.1 ± 2.2 hours after the onset in group A and reached an earlier peak at 12.5 ± 2.9 hours in group B (p < 0.001).

These data suggest that acute coronary recanalization alters the kinetics of CK-MB release, resulting in greater CK-MB release into the serum for equivalent infarct volume estimated by myocardial emission tomography with thallium-201. Thus, serum CK-MB time-activity curves after acute myocardial infarction may be influenced considerably by acute reperfusion, which is an important factor that should be incorporated in the interpretation of enzymatic estimates of infarct size in human patients.

Serial serum creatine kinase (CK) and serum CK-MB isoenzyme activity determinations have been widely used for the noninvasive estimation of myocardial infarct size (1–9). Recently, rapid recanlization of the acutely occluded coronary artery was established with use of intracorony infusion of thrombolytic agents (10–16) and rapid appearance of CK in blood after successful thrombolysis was noted in several clinical studies (11,12,15,16). The early appearance of increased serum enzyme levels after reperfusion has already been reported in experimental animal studies (17). Moreover, experimental studies (18,19) have suggested that the total amount of CK that is released into blood after coronary artery reperfusion may be much greater than expected for the pathologically determined infarct size. In the clinical setting, however, the quantitative relation between enzyme level and the final size of infarcted myocardium after successful recanalization has not yet been clarified.

Because early coronary artery recanalization with thrombolitic agents is finding increasing use in medical practice, a clear understanding of the effects of thrombolysis on the kinetics of enzyme release is needed. Although there is no well established standard for quantifying infarct size in living patients, there is evidence that thallium-201 myocardial
scintigraphy is valid for determining infarct size in human subjects (7,20-25).

In this study, myocardial emission computed tomography with thallium-201 was used to estimate the final size of infarcted myocardium. The purpose of our study was to assess the effect of acute coronary artery recanalization on the relation between serum CK-MB release and infarct size estimated by myocardial emission tomography with thallium-201 in patients with acute myocardial infarction who underwent intracoronary thrombolysis.

Methods

Study patients. The study group comprised 37 patients with a first acute transmural myocardial infarction, in whom intracoronary infusion with urokinase was performed in order to recanalize the obstructed coronary artery during the acute stage of infarction. These patients were selected from a consecutive series of 46 patients who underwent intracoronary thrombolysis from September 1981 to April 1983 on the basis of the following criteria: 1) the patient had a first acute transmural myocardial infarction with neither clinical nor electrocardiographic evidence of a previous infarction; 2) there was no recurrence of myocardial infarction after the completion of enzyme sampling until the time of scintigraphic imaging; and 3) myocardial emission tomography with thallium-201 could be performed 4 weeks after the onset of infarction. Two of the remaining nine patients died during the acute phase of their disease and, therefore, scintigraphic study could not be performed. Five patients had a previous myocardial infarction and two patients had a recurrence of infarction diagnosed by a rise of CK-MB levels until the scintigraphic study.

The diagnosis of acute transmural myocardial infarction was based on acute onset of chest pain lasting for more than 30 minutes and persistent ST segment elevation lasting for more than 30 minutes and progressing to new Q waves of greater than 0.04 second duration in the standard 12 lead electrocardiogram. Diagnosis was subsequently confirmed by electrocardiographic evolutionary changes and positive CK-MB determinations. Patients who had symptoms for more than 10 hours before entry into the coronary care unit were excluded from the study. All patients gave written informed consent after detailed explanation of the procedure, its possible hazards and possible beneficial effects.

Coronary reperfusion technique. Selective coronary angiography was performed by either the femoral or brachial artery approach. After occlusion of the coronary artery was confirmed, sublingual or intracoronary nitroglycerin (0.2 to 0.4 mg) was administered. Angiography was again performed 1 to 2 minutes later to exclude spastic occlusion. If no change in the angiographic appearance of the vessel was noted, urokinase dissolved in saline solution was infused into the occluded coronary artery through the coronary catheter at a rate of 24,000 units/min. The mean interval from the onset of chest pain to infusion of urokinase was 4.6 ± 1.9 hours (range 2 to 8.5). Coronary angiography was repeated at 5 minute intervals during the infusion of urokinase and the infusion was stopped 5 minutes after recanalization of the occluded vessel. If the coronary vessel was not re-opened after infusion of 840,000 units of urokinase, no further attempt was made. After completion of cardiac catheterization, patients were transferred to the coronary care unit.

Calculation of CK-MB released into serum. Blood samples were obtained immediately after admission, before the urokinase infusion, at 30 minutes and at 1 hour after recanalization of the occluded vessel and every 4 hours for the following 48 hours. Specimens were collected and immediately centrifuged to separate the serum, which was stored at -70°C. CK-MB isoenzyme activity was separated from sera by Diethyl-aminoethyl-Sephalose column chromatography using minicolumns (26). Enzyme activity of the column eluate was determined according to the method of Rosalki (27). A computer was used to generate each serum CK-MB time-activity curve, to fit an exponential to the downslope of the curve using the least squares method and derive an individual disappearance constant (Kd) of CK-MB from serum for each patient. The accumulated release of CK-MB (ΣCK-MB) was calculated using a modification (3,4) of the original formula of Shell et al. (1,2). ΣCK-MB represents the total amount of CK-MB that would appear in the serum in the absence of enzyme disappearance. The peak level of CK-MB was also noted for each patient.

Myocardial emission computed tomography with thallium-201. Myocardial tomograms were obtained 4 weeks after the onset of myocardial infarction, when the patient’s condition had stabilized. Each patient received 2 mCi of thallium-201 at rest and underwent imaging 10 minutes later with a large field of view gamma camera equipped with a high resolution parallel-hole collimator supported by a gantry (General Electric Maxi-400T). Sixty-four different views over 360°, 20 seconds each, provide sampling for every 5.8° of revolution of the detector. Transaxial tomograms were reconstructed into 12 mm thick multiple slices by a filtered back-projection method with Chesler’s filter (28) using a convolution reconstruction algorithm (7,29). Attenuation correction was performed by Sorenson’s method (30). It took the computer (PDP 11, Digital Equipment Corporation) 20 seconds to reconstruct each transaxial slice, which was displayed perpendicular to the long axis of the left ventricle. Thereafter, frontal and sagittal tomograms were organized from a series of transaxial tomograms, so that the frontal and sagittal tomograms closely corresponded to the cross section and longitudinal section in relation to the cardiac axis, respectively (7,29). Each reconstructed slice contained...
150,000 to 200,000 counts. The reconstructed images were displayed in a 64 × 64 matrix.

To estimate the size of the perfusion defect, we analyzed the tomograms in the most appropriate section that showed the defect transversely, selecting transaxial sections in the anterior myocardial infarct and frontal sections in the inferior infarct. Our method to measure the size of the perfusion defect from tomographic images has been described in detail previously (7). Briefly, after each image was subtracted by 45% of the maximal radioactivity, the myocardial contour was outlined with a light pen and the area showing the defect was measured by computerized planimetry. A defect region was defined as one with thallium-201 uptake less than 45% of the maximal uptake in any image element (Fig. 1). The number of image elements occupied by perfusion defects in all reconstructed slices was multiplied by the size factor (0.432 ml per image element) to determine the volume of infarcted myocardium. Infarct volume was expressed in milliliters. Thallium-201 myocardial tomograms were assessed independently by two of us, neither of whom had prior knowledge of the results of the enzymatic analysis.

The reproducibility of the quantitative tomographic analysis by these methods described was also tested in 37 patients. When the same observer recalculated the same data in 37 patients 1 month later, the intraobserver variation in measurement of infarct volume was 1.8 ± 1.3 ml (range 0.3 to 5.0). When a second observer performed these 37 studies without knowledge of the results of the first observer, the interobserver variation was 2.1 ± 2.0 ml (range 0.4 to 8.1) for calculations of infarct volume.

Statistical analysis. We found linear correlations by the method of least squares analysis and used the unpaired t test to assess differences between groups of unpaired data. A probability (p) value of less than 0.05 was considered significant. Data are expressed as mean ± 1 standard deviation.

Results

Patient groups (Table 1). Thirty-seven patients met the previously described criteria and were included in the study. Thirty-three were men and four were women. Their mean age was 59 years (range 39 to 77). The patients were classified into two groups. Group A consisted of 11 patients in whom the attempt to recanalize the occluded coronary artery failed (unsuccessful thrombolysis) and group B consisted of 26 patients with reopening of the occluded coronary artery after intracoronary infusion of urokinase (successful thrombolysis). All patients in group A had total occlusion of the infarct-related coronary artery and the total occlusion remained unchanged even after intracoronary urokinase infusion. The occluded vessel was the right coronary artery in four patients and the left anterior descending coronary artery in seven patients. In group B before thrombolysis, 22 patients had total occlusion of the infarct-related vessel and reopening of occlusion after urokinase infusion. The remaining four patients had subtotal occlusion of the infarct vessel with sluggish flow and improved runoff after intracoronary urokinase administration. The occluded vessel was the right coronary artery in 6 patients, the left circumflex coronary artery in 2 patients and the left anterior descending coronary artery in 18 patients. Recanalization was achieved 5.3 ± 2.0 hours after the onset of symptoms. Adequate collateral supply to the occluded vessel was observed in one patient in group A and in two patients in group B. The time interval between the onset of symptoms and initiation of intracoronary urokinase infusion was 4.3 ± 1.7 hours in group A and 4.7 ± 1.9 hours in group B (difference not significant).

All patients were treated medically after intracoronary thrombolysis. One patient in group A had congestive heart failure, which required afterload-reducing agents in addition to diuretic drugs. Two patients in group B had postinfarction angina but responded well to medical therapy. The remaining 34 patients did not have chest pain or exhibit signs of heart failure during the hospitalization after the acute intervention. No patient in group A or B had enzymatic or electrocardiographic evidence of recurrence of acute myocardial infarction between the acute intervention and the late scintigraphic study. All patients were in stable condition when scintigraphic imaging was performed.

Serum CK-MB kinetics (Fig. 2 and 3). Serum CK-MB time-activity curves in patients in group B (successful thrombolysis) differed from those in patients in group A (unsuccessful thrombolysis). In group A, serum CK-MB activity increased gradually to a peak at 21.1 ± 2.2 hours after the onset of the infarct (Fig. 2). In contrast, in group B serum CK-MB activity increased sharply immediately after intracoronary urokinase infusion.
after recanalization and reached a peak at 12.5 ± 2.9 hours after the onset (Fig. 3), occurring significantly earlier than in group A (p < 0.001). There were no significant group differences in $\Sigma$CK-MB (group A, 233.0 ± 120.4 IU/liter, group B, 304.5 ± 186.9 IU/liter) and Kd (group A, $-85.2 \pm 18.4 \times 10^{-3}$ hour$^{-1}$; group B, $-86.8 \pm 19.1 \times 10^{-3}$ hour$^{-1}$) (Table 1).

Relation between CK-MB release and infarct size estimated by myocardial emission tomography with thallium-201 (Fig. 4). An excellent linear relation between $\Sigma$CK-MB and infarct volume estimated by myocardial emission tomography with thallium-201 was observed in group A (unsuccessful thrombolysis) ($\Sigma$CK-MB = 6.4 × infarct volume + 47.7, correlation coefficient $r = 0.91$, standard error of the estimate [SEE] = 55.7, p < 0.001). On the other hand, the relation remained linear but it was shifted upward in group B (successful thrombolysis) ($\Sigma$CK-MB = 10.5 × infarct volume + 89.1, $r = 0.80$, SEE = 116.3, p < 0.001). The slope was steeper than observed for patients in group A, suggesting that more CK-MB appeared in serum per unit of infarcted myocardium after successful thrombolysis. This is supported by the observation that the average ratio of $\Sigma$CK-MB to infarct volume estimated by myocardial emission tomography with thallium-201 (16.1 ± 6.8) was significantly greater than in patients in group A (8.6 ± 2.2) (p < 0.01), although alternations of the ratio of $\Sigma$CK-MB to infarct volume had a wide variation.

Representative thallium-201 tomographic images from one patient in group A (Patient 2) and one in group B (Patient 25) are shown in Figures 2 and 3, respectively. Both had inferior myocardial infarction. Despite greater $\Sigma$CK-MB, the size of the inferior wall perfusion defects seen on tomograms from Patient 25 (successful thrombolysis) was smaller than that in Patient 2 (unsuccessful thrombolysis).
Table 1. Clinical, Enzymatic and Scintigraphic Data in 37 Patients

<table>
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<tr>
<th>Case</th>
<th>Age (yr) &amp; Sex</th>
<th>Infarct Vessel/Residual Stenosis (%)</th>
<th>Time to Peak CK-MB (h)</th>
<th>Kd ((\times 10^{-3} \text{ h}^{-1}))</th>
<th>(\Sigma\text{CK-MB} \text{ (IU/liter)})</th>
<th>Infarct Volume Based on Tl-201 ECT (ml)</th>
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<tr>
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<td>2.2</td>
<td>18.4</td>
<td>120.4</td>
<td>17.1</td>
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Group A. Patients With Unsuccessful Thrombolysis

Group B. Patients With Successful Thrombolysis

*Significantly different from group A \((p < 0.001)\). CK-MB = creatine kinase-MB isoenzyme; F = female; Kd = disappearance constant; LAD = left anterior descending coronary artery; LCx = left circumflex coronary artery; M = male; RCA = right coronary artery; SD = standard deviation; \(\Sigma\text{CK-MB} = \) the accumulated release of CK-MB; Tl-201 ECT = myocardial emission tomography with thallium-201.

Although infarct volume estimated by myocardial emission tomography with thallium-201 showed marked variability in both groups, there was no significant group difference in infarct volume \((\text{group A, 28.9} \pm 17.1 \text{ ml, range 7 to 74; group B, 20.5} \pm 14.3 \text{ ml, range 5 to 74)} \) (Table 1).

**Discussion**

Previous studies relating creatine kinase (CK) and CK-MB release to infarct size. Of the methods available for living patients, quantification of the release of CK by serial serum sampling is a widely used method of assessing the extent of myocardial damage (2–9). Experimental stud-
ies (1,4) have shown that infarct size determined from CK released into the serum correlates closely with that determined by myocardial CK depletion and by morphologic features in dogs after 24 hours of coronary occlusion. Studies of acute myocardial infarction in human subjects have demonstrated reasonable agreement between infarct size estimated by serum CK analysis and prognosis (2,3), left ventricular hemodynamics (5) and postmortem measurement of infarct size (5). Estimation has been improved by quantifying the CK-MB isoenzyme (4). Recent studies comparing infarct size estimated from serum CK-MB release with other independent descriptors of infarct size, such as left ventricularulography (6), myocardial emission computed tomography with thallium-201 (7) or positron emission tomography with C-11-labeled palmitate (8), have added support to these observations. In a study performed by Grande et al. (9), infarct size was estimated from CK-MB release and correlated closely with morphologic estimates obtained in 22 patients who died a mean of 6.4 days after myocardial infarction. Thus, the majority of data indicate that the CK-MB system has at least semiquantitative validity in human subjects, although the experimental and mathematical basis for the enzymatic models as a measure of infarct size has been a matter of controversy (31–34).

Effects of coronary artery reperfusion on infarct size calculated from serum CK-MB release. In our patients with successful thrombolysis serum CK-MB activity increased sharply immediately after recanalization and reached a peak after the onset, significantly earlier than occurred in patients with unsuccessful thrombolysis (Fig. 2 and 3). This finding is in agreement with the results of prior animal studies (17–19) and recent clinical studies (11,12,15,16) suggesting that acute reperfusion washes CK or CK-MB into the blood. Serial changes in total serum CK activity may be distorted because of potential release of CK from noncardiac sources after cardiac catheterization (35). Therefore, we analyzed serum CK-MB time-activity curves to assess the influence of thrombolysis on enzyme release from the myocardium. The shape of the serum CK-MB time-activity curve depends not only on the rate of release but also on the CK-MB disappearance rate (36). In this study, however, the mean CK-MB disappearance rate in patients with successful recanalization was similar to that in patients with unsuccessful recanalization (Table 1). In patients with unsuccessful recanalization, an excellent linear relation was observed between $\Sigma$CK-MB and infarct volume estimated by myocardial emission tomography with thallium-201 ($r = 0.91$). Although the relation remained linear even in patients with successful recanalization ($r = 0.80$), it was shifted because of relatively greater CK-MB release into the blood at a given infarct volume estimated by myocardial emission tomography with thallium-201 (Fig. 4).

In experimental dogs with coronary artery reperfusion, the relation between histologic extent of infarction and enzymatic estimate was studied by several investigators. Jarmakani et al. (18) noted early appearance of CK after reperfusion and showed the possible effects on CK release of reperfusion, distorting infarct size calculations. Vatner et al. (19) showed that the enzymatic estimate of infarct size correlated closely ($r = 0.94$) when the coronary vessel was permanently occluded. They also reported that these estimates correlated significantly ($r = 0.90$) even after reperfusion, although the enzymatic values were greater than expected for the histologic infarct size, because early reperfusion may facilitate enzyme washout from infarcted myocardium. It has been shown that cardiac lymphatics are the major escape route of myocardial enzymes to the distribution space and CK is inactivated in lymph (37,38). Occlusion of cardiac lymphatics induced during myocardial infarction results in a 50% decrease of the amount of CK appearing in blood (38). Therefore, it would be reasonable to postulate that more rapid transport of CK into the systemic
circulation due to reperfusion results in relatively less local degradation of CK and an increase in the amount of CK released into the blood.

In contrast to experimental data (19), alternations of the ratio of \( \Sigma CK-MB \) to infarct size estimated by myocardial emission tomography with thallium-201 in the present study have a wide variation, indicating that the degree of reperfusion achieved by thrombolysis is quite inconstant. Factors determining the degree of washout effect are the timing of reperfusion and the amount of blood delivered to the infarcted area. Experimental reperfusion could be achieved by properly timed and complete release of a mechanical obstruction. In patients who underwent intracoronary thrombolysis, these are likely to be more variable than in animal models because of differences in the degree of residual coronary stenosis and the duration of ischemia. Therefore, varying degrees of reperfusion achieved by thrombolysis could result in variation of recovery of the enzyme in blood. Moreover, experimental reperfusion leads to reactive hyperemia (17,39), whereas our technique results in gradual restoration of flow without reactive hyperemia. These factors may account for the differences between our results and data from animal studies.

Limitations of the study. The limitations of myocardial emission tomography with thallium-201 used as a method to estimate infarct size in this study must be considered. Experimental studies have shown that myocardial uptake of thallium-201 correlates inversely with myocardial creatine kinase depletion (22) and relative decrease of myocardial blood flow (20). Necrotic tissue fails to accumulate thallium-201 (21). Defects identified by imaging performed at rest in stable patients usually represent myocardial scar rather than ischemia (25). However, thallium-201 perfusion defects undoubtedly change with time (23,24). It is possible that thallium-201 defects in the early stage of infarction represent ischemic as well as necrotic tissue. The greatest changes were noted 4 to 11 days after the onset of infarction (23). To minimize this problem and estimate the final size of necrotic myocardium, myocardial tomograms were obtained at rest 4 weeks after the onset in stable patients with a first myocardial infarction. The close correlation between CK-MB release and tomographic estimate in this study suggests that myocardial ischemia does not play a large role.

Clinically, it would be useful to express scintigraphic infarct size as a percent of left ventricle infarcted (24,25). However, because the enzymatic estimate in this study was presented as an absolute value (the quantity of CK-MB released) rather than as an index of the proportion of the left ventricle infarcted (3,6–9), the estimated size by emission tomography was expressed as infarct volume, which could be compared with \( \Sigma CK-MB \).

The major advantage of emission tomography is in eliminating radioactivity from superimposed structures and making improved quantification possible. Accurate attenuation correction is not available at this time; however, recent animal studies have shown that emission tomographic imaging of thallium-201 is accurate in measuring viable or infarcted myocardial mass (40) and that the emission tomographic system accurately reflects regional distribution of myocardial blood flow (41). Although the definition of boundaries of infarcted myocardium may have limitations, our previous reports showed that this approach can provide improved quantification of the extent of perfusion abnormalities (7) as well as improved detection of perfusion defects (29) in comparison with planar imaging in phantom and clinical studies.

Recently, thallium-201 myocardial scintigraphy has been used to assess the effect of intracoronary thrombolysis on myocardial salvage (11,14–16). It is beyond the scope of the present study to answer the crucial question whether early thrombolysis can preserve the myocardial structure. There was no statistically significant group difference in infarct volume estimated by emission tomography with thallium-201. Myocardial emission tomography can provide geometrically reliable three-dimensional distribution of thallium-201. We feel encouraged to apply this procedure to assessment of the effectiveness of further randomized trials of thrombolysis in evolving acute myocardial infarction.

Clinical implications. The finding that successful recanalization alters the relation between accumulated release of CK-MB and infarct size indicates the need for caution in interpreting enzymatic analysis. In patients with acute myocardial infarction, early spontaneous reperfusion may occur from reopening of the occluded vessel, native collateral flow, reversal of spasm and various interventions during the period of CK release. DeWood et al. (42) reported that in a substantial number of patients, recanalization occurs within the first 24 hours. In the present study, spontaneous recanalization of the infarct-related vessel was observed in four patients and they had early peak of the CK-MB curves. Under these conditions, the enzymatic values may overestimate the extent of myocardial injury.

In conclusion, successful thrombolysis alters the kinetics of CK-MB release, resulting in relatively greater CK-MB release into the serum at a given infarct size estimated by myocardial emission tomography with thallium-201. Thus, serum CK-MB time-activity curves after acute myocardial infarction may be influenced considerably by acute reperfusion, one important factor that should be incorporated when interpreting enzymatic estimates of infarct size. This fact should also be noted in cases of spontaneous reperfusion after acute myocardial infarction.

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References


