Efficacy of Percutaneous Transluminal Coronary Recanalization for Preservation of the Post-Infarct Left Ventricular Regional Wall Motion: A Trial of the Evaluation by Weight-ing Coronary Artery Segments

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Abstract

Efficacy of the percutaneous transluminal coronary recannalization (PTCR) therapy was evaluated by weighting infarct-related coronary artery segments in 28 consecutive patients with acute myocardial infarction. The study focused on the influences of the time interval from the onset of chest pain to PTCR (PTCR-Time) and on the post-infarct left ventricular regional wall motion in conjunction with the serum levels of GOT, LDH and CPK and with PTCR-Time. PTCR success rate was 84.0%, and re-occlusion rate was 4.0%. The thrombolysis in myocardial infarction grade 2, however, was observed in 7 (33.3%) of 21 cases with successful PTCR. There was no significant difference in PTCR-Time between the PTCR success and nonsuccess groups. Significant correlations were observed between the PTCR-Time and each peak value of standardized serum levels of LDH and CPK, and between the PTCR-Time and the post-infarct regional wall motion abnormality. There were also significant correlations between the standardized serum level of each of these three enzymes and the post-infarct regional wall motion abnormality. It was clearly demonstrated that the earlier the recannalization of the infarcted artery was achieved, the less extensive the myocardial damage in quantitative and qualitative aspects.

KEYWORDS: myocardial infarction, PTCR, efficacy, regional wall motion, enzymatic levels

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Efficacy of the percutaneous transluminal coronary recannalization (PTCR) therapy was evaluated by weighting infarct-related coronary artery segments in 28 consecutive patients with acute myocardial infarction. The study focused on the influences of the time interval from the onset of chest pain to PTCR (PTCR-Time) and on the post-infarct left ventricular regional wall motion in conjunction with the serum levels of GOT, LDH and CPK and with PTCR-Time. PTCR success rate was 84.0%, and re-occlusion rate was 4.0%. The thrombolysis in myocardial infarction grade 2, however, was observed in 7 (33.3%) of 21 cases with successful PTCR. There was no significant difference in PTCR-Time between the PTCR success and nonsuccess groups. Significant correlations were observed between the PTCR-Time and each peak value of standardized serum levels of LDH and CPK, and between the PTCR-Time and the post-infarct regional wall motion abnormality. There were also significant correlations between the standardized serum level of each of these three enzymes and the post-infarct regional wall motion abnormality. It was clearly demonstrated that the earlier the recannalization of the infarcted artery was achieved, the less extensive the myocardial damage in quantitative and qualitative aspects.

Key words: myocardial infarction, PTCR, efficacy, regional wall motion, enzymatic levels

The early reestablishment of coronary perfusion to the ischemic myocardium is expected to reduce the area and/or the degree of infarction (1-6). In this respect, the reestablishment of coronary circulation by resolving thrombi is a reasonable strategy for treatment of patients with acute myocardial infarction (AMI). The prognostic benefits of intracoronary thrombolytic therapy have now been convincingly demonstrated by the GISSI trial and others (7, 8). Recent developments of the clinical use of tissue plasminogen activator, which may or may not be recombinant, facilitate the thrombolytic therapy. The most remarkable advantage of this procedure will be reduction in time from the onset of pain to the thrombolytic therapy, which is much shorter than the conventional percutaneous transluminal coronary recannalization (PTCR) with urokinase (UK). Consequently, the intravenous thrombolysis procedure is becoming the preferred alternative, and is likely to be commonly used in ordinary medical offices. In these circumstances, it is very important to reevaluate the efficacy of the conventional PTCR therapy in respect to cardiac performance in terms of the left ventricular (LV) regional wall motion of the infarcted area in conjunction with the time interval from the onset of chest pain to PTCR (PTCR-Time).

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Subjects and Methods

Twenty-eight consecutive patients with AMI, who were qualified for our PTCR protocol from March 14, 1985 to March 8, 1991, were admitted to this study; 24 cases were men and 4 cases were women, ranging in age from 43 to 73 (mean 60.8) years. The diagnosis of AMI and protocol entry criteria were as follows: a) continuous chest pain (longer than 30 min) consistent with AMI, b) the time from the onset of chest pain to admission was less than 8 h, c) patients were under 75 years of age, d) electrocardiographic findings suggesting AMI, defined as at least 1 mm ST elevation in two or more adjacent leads (inferior: leads II, III, and aVF; anterior: I, aVL, and V1-V6), e) persistent ST segment elevation after administration of sublingual nitroglycerin (0.4 mg), f) no recent (within 2 weeks) evidence of gastrointestinal and/or cerebral hemorrhage. Laboratory data were also taken into consideration for diagnosis, and serum levels of GOT, LDH, and CPK were routinely measured on admission and every 8 h for a week. The peak values of these enzymes in this period were used as indicators of the infarct size and the degree of myocardial damage.

Details of the PTCR protocol and follow-up catheterization study, including possible complications, were thoroughly explained to all patients, and the written permission was obtained from either of the patient or relatives. An ordinary cardiac catheterization study was done, but the left ventriculography was not performed in the acute phase. Selective coronary angiographic evaluation of the infarct-nonrelated artery was initially performed, and then the infarct-related artery was examined. When a total luminal occlusion of the infarct-related artery was identified, 200 \( \mu g \) of nitroglycerin was intracoronarily administered to exclude the possible coronary artery spasms. If coronary artery occlusion persisted when the second coronary angiography was performed 2 to 3 min later, 24 \( \times 10^4 \) units of UK were intracoronarily infused for 10 min. This procedure was repeated until reperfusion was obtained or the total dose of UK equaled 96 \( \times 10^4 \) units. After completion of PTCR procedure, orthogonal cineangiograms of the infarct-related coronary artery were obtained to permit measurement of the degree of narrowing.

Efficacy of the PTCR procedure was evaluated by the thrombolysis in myocardial infarction (TIMI) trial, which rated the efficacy from grade 0 to 3 (1). The coronary arteries were subdivided into segments according to the schema proposed by the American Heart Association (9), and weighting factors were assigned to each segment by modifying those given by Leaman et al. (10) (Table 1). The peak values of GOT, LDH, and CPK were standardized to reduce the bias of the area supplied by the infarct-related artery; each peak value was divided by each of the coronary segmental weights (CSW), and was expressed as STGOT, ST-LDH, and ST-CPK, respectively.

To reevaluate the infarct-related coronary artery and the cardiac performance after PTCR, especially focusing on the post-infarct LV regional wall motion, the cardiac catheterization

Table 1  The weighting factor assigned to each coronary artery segment (CSW) in the right-dominant and left-dominant coronary artery systems

<table>
<thead>
<tr>
<th>Coronary segments*</th>
<th>LV wall segments</th>
<th>Authors</th>
<th>Leaman</th>
<th>LV wall segments</th>
<th>Authors</th>
<th>Leaman</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>4, 5</td>
<td>2.0</td>
<td>1.0</td>
<td>4, 5</td>
<td>1.0</td>
<td>0.0</td>
</tr>
<tr>
<td>2</td>
<td>4, 5</td>
<td>2.0</td>
<td>1.0</td>
<td>(4, 5)</td>
<td>1.0</td>
<td>0.0</td>
</tr>
<tr>
<td>3</td>
<td>4, 5</td>
<td>2.0</td>
<td>1.0</td>
<td>(4, 5)</td>
<td>1.0</td>
<td>0.0</td>
</tr>
<tr>
<td>4</td>
<td>(4, 5)</td>
<td>1.0</td>
<td>1.0</td>
<td>(4, 5)</td>
<td>1.0</td>
<td>0.0</td>
</tr>
<tr>
<td>5</td>
<td>1, 2, 3, 4</td>
<td>4.0</td>
<td>5.0</td>
<td>1, 2, 3, 4, 5, 6, 7</td>
<td>7.0</td>
<td>6.0</td>
</tr>
<tr>
<td>6</td>
<td>1, 2, 3, (4, 7)</td>
<td>3.5</td>
<td>3.5</td>
<td>3, 4, (7)</td>
<td>2.5</td>
<td>2.5</td>
</tr>
<tr>
<td>7</td>
<td>3, 4</td>
<td>2.0</td>
<td>2.5</td>
<td>(2, 7)</td>
<td>1.0</td>
<td>1.0</td>
</tr>
<tr>
<td>8</td>
<td>3</td>
<td>1.0</td>
<td>1.0</td>
<td>(3)</td>
<td>1.0</td>
<td>1.0</td>
</tr>
<tr>
<td>9</td>
<td>(2, 7)</td>
<td>0.5</td>
<td>1.0</td>
<td>(3)</td>
<td>0.5</td>
<td>0.5</td>
</tr>
<tr>
<td>10</td>
<td>(3)</td>
<td>0.5</td>
<td>0.5</td>
<td>5, 7, (4)</td>
<td>2.5</td>
<td>2.5</td>
</tr>
<tr>
<td>11</td>
<td>7</td>
<td>1.0</td>
<td>1.5</td>
<td>7</td>
<td>1.0</td>
<td>1.0</td>
</tr>
<tr>
<td>12</td>
<td>7</td>
<td>1.0</td>
<td>1.0</td>
<td>5, (4, 7)</td>
<td>1.5</td>
<td>1.5</td>
</tr>
<tr>
<td>13</td>
<td>None</td>
<td>0.0</td>
<td>0.5</td>
<td>4</td>
<td>1.0</td>
<td>1.0</td>
</tr>
<tr>
<td>14</td>
<td>None</td>
<td>0.0</td>
<td>0.5</td>
<td>4</td>
<td>1.0</td>
<td>1.0</td>
</tr>
<tr>
<td>15</td>
<td>None</td>
<td>0.0</td>
<td>0.0</td>
<td>4</td>
<td>1.0</td>
<td>1.0</td>
</tr>
</tbody>
</table>

Our new, modified coronary segmental weights (CSWs) are presented together with the original values proposed by Leaman et al. (10), and LV wall segments, which are considered to possibly be influenced in terms of regional wall motion when the corresponding coronary segment is occluded, are also presented; wall segments, which may partially be affected, are indicated in parentheses. *: Segments according to the American Heart Association Report (9).

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study was repeated 44.1±11.6 days after PTCR in surviving patients (n=24); two patients refused the repeated catheterization, and the other two patients had succumbed at the acute phase. In addition to the emergency catheterization, biplane left ventriculography was done in the 30 degree right anterior oblique and orthogonally left anterior oblique projections. The regional wall motion of the territory supplied by the infarct-related artery was segmentally analyzed using the seven wall segments proposed by the American Heart Association (9). Regional wall motion was defined as normal, slightly hypokinetic, hypokinetic, akinetic, dyskinetic, and aneurysmal at each segment, and each motion was assigned a point value of 0.0, 0.5, 1.0, 3.0, 5.0 and 5.0, respectively, representing the segmental score of the regional wall motion abnormality. A sum of these scores for all segments supplied by the infarct-related artery was divided by the CSW. The resulting value represented the regional wall motion abnormality score (MA-score). With these parameters, the influences of PTCR-Time on the post-infarct LV regional wall motion were evaluated in conjunction with the peak serum enzyme levels.

Statistical analysis was performed using paired or unpaired Student’s t-tests to compare the parameters. The Chi-square test was employed to test the incidence difference, and a simple linear regression analysis was performed to evaluate the correlation between the two parameters. All values are expressed as means ± SD. The difference was considered statistically significant, when p values were less than 0.05.

Table 2  Clinical data

<table>
<thead>
<tr>
<th>Infarct-related artery</th>
<th>Number of cases</th>
<th>PTCR-Time (min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute myocardial infarction</td>
<td>28</td>
<td>175.4±104.7</td>
</tr>
<tr>
<td>Anterior infarction</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Segment 6</td>
<td>11 (39.3)</td>
<td></td>
</tr>
<tr>
<td>Segment 7</td>
<td>3 (10.7)</td>
<td></td>
</tr>
<tr>
<td>Inferior infarction</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Segment 1</td>
<td>17 (50.7)</td>
<td></td>
</tr>
<tr>
<td>Segment 2</td>
<td>4 (14.3)</td>
<td></td>
</tr>
<tr>
<td>Segment 3</td>
<td>6 (21.4)</td>
<td></td>
</tr>
<tr>
<td>Segment 4</td>
<td>4 (14.3)</td>
<td></td>
</tr>
</tbody>
</table>

a: Coronary artery segment postulated by the American Heart Association (9); b: Time interval from chest pain onset to percutaneous transluminal coronary recannalization (PTCR) therapy ranging from 45 to 480 min. Numbers in parentheses indicate percentages.

<table>
<thead>
<tr>
<th>TIMI Before</th>
<th>PTCR</th>
<th>PTCR</th>
<th>Follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>grades</td>
<td>(n=28)</td>
<td>(n=28)</td>
<td>(n=24*)</td>
</tr>
<tr>
<td>III</td>
<td>4 (14.3)</td>
<td>18 (54.3)</td>
<td>18 (75.0)</td>
</tr>
<tr>
<td>II</td>
<td>2 (7.1)</td>
<td>6 (21.4)</td>
<td>4 (16.7)</td>
</tr>
<tr>
<td>0 ~ I</td>
<td>22 (78.6)</td>
<td>4 (14.3)</td>
<td>2 (8.3)</td>
</tr>
</tbody>
</table>

Fig. 1  Results of PTCR and follow-up catheterization study. PTCR: percutaneous transluminal coronary recannalization, TIMI grades: the grades proposed by the thrombolysis in myocardial infarction trial (1). Cath.: catheterization. *: Two patients succumbed at an acute phase and other two patients refused the follow-up study. Numbers in squares indicate the cases, and those in parentheses indicate percentages.

Results

Among the 28 patients, anterior and inferior AMIs were observed in 17 (60.7 %) and 11 (39.3 %) cases, respectively (Table 2). Infarct-related coronary artery segments are also shown. The PTCR-Time was determined as 175.4±104.7 min (n=26, range from 45 to 480 min); in two cases with inferior infarction the time interval was obscure.

Complete occlusion of the involved coronary artery was observed at the first coronary angiography during the acute phase in 22 (78.6 %) cases. Spontaneous recannalization was observed in 6 (21.4 %) cases; 2 cases with TIMI grade 2, and 4 cases with TIMI grade 3 (Fig. 1). PTCR protocol was performed in 27 cases; one case presented severe dysrhythmia with profound circulatory deterioration, and succumbed before completion of PTCR procedure. The results of PTCR therapy are summarized in Fig. 1. Four cases of spontaneous recannalization with TIMI grade 3 were excluded from the calculation of the PTCR success rate. Two cases with spontaneous recannalization with TIMI grade 2, which improved to grade 3 after PTCR procedure, and one case in which PTCR was performed a second time to correct a re-occlusion of the infarct-related artery were added to calculate the PTCR success rate. Thus, PTCR success rate was determined to be 84.0 % [21/25 (28-4+1)], and the re-occlusion rate was 4.0 % (1/25). However, TIMI grade 2 was observed in 7 (33.3 %) of 21 cases with PTCR success. This last group included the case which required a second PTCR.

The PTCR-Time in the cases with PTCR success
was 185.6 ± 113.6 min, and in those without success was 150.0 ± 74.5 min; the difference was not statistically significant.

The peak serum levels of GOT, LDH, and CPK were 365.1 ± 218.8, 1812.8 ± 894.7, and 2615.6 ± 1691.2 IU/L, respectively. Each value significantly exceeded the respective normal range in all cases, proving the presence of myocardial infarction.

The correlations between the PTCR-Time, ST-values of the studied enzymes and MA-scores were investigated in 15 cases, in which PTCR succeeded and the PTCR-Time was determined. The results are summarized in Fig. 2. The PTCR-Time significantly correlated with ST-LDH (p < 0.01) and ST-CPK (p < 0.01). The PTCR-Time also correlated significantly with MA-score (p < 0.01). MA-score was also observed to correlate well with ST-GOT (p < 0.02), ST-LDH (p < 0.01), and ST-CPK (p < 0.01) (Fig. 2). Normal LV regional wall motion (MA-score = 0) was observed in 4 (57.1%) of 7 cases that underwent PTCR within 120 min after the onset of chest pain, but not observed in the cases subjected to the PTCR protocol after 120 min. This difference was not statistically significant (p < 0.1).

Discussion

Although the coronary artery scoring method de-
scribed by Leaman et al. (10) may not generally be accepted, there have not been any studies which attempt to weight the individual coronary arteries based on blood flow volumes to evaluate the severity of coronary artery stenoses or post-infarct LV regional wall motion. The present study is such an attempt. It should be noted that weights were not assigned to the right coronary arterial segments when the left coronary artery was dominant (Table 1). It was often observed that the motion of the LV wall segment 4 and/or 5 was considerably reduced in cases with an occluded right coronary artery even when the left coronary artery system was dominant. When the right artery was dominant, wall segments 4 and 5 were influenced if any of the coronary segments 1, 2, or 3 was occluded. When the left artery was super-dominant, the weights on the right coronary segments might be unnecessary. Such cases were not included in this series. Another major modification was the weight assigned to the coronary segment 6 which was 5.0 in the present study and was 3.5 in the report of Leaman et al. when the left coronary system was dominant. If coronary segment 6 was occluded in such the cases, the number of the wall segments which could be influenced was considered to be five; segments 1–3, 6, and one half of segments 4 and 7. Therefore, we assigned the weight of 5.0 to the coronary segment 6. The CSWs in our report were derived from the scoring modifications associated with the number of the LV wall segment(s) which was/were thought to have been supplied from the infarct-related coronary segment (Table 1).

Leaman et al. focused on the correlation between the amount of coronary blood flow and frequency of angina pectoris and/or compromised LV function. Therefore, they assigned weighting values on the basis of blood flow, but not on the basis of the area of infarction. We considered it necessary to modify their weighting values of the number of the LV wall segments that are fed from the infarcted coronary segment so that we could rationally measure the efficacy of PTCR in respect to salvage of myocardium. As in the study by Leaman et al., the problem of collateral flow to the infarct-related artery was not taken into account in assigning the weights because the true significance of this problem is not yet clear (10, 11).

We assumed that the serum levels of the studied enzymes were dependent on the infarct size defined by the number of the wall segments that were involved during the ischemic period. The serum levels also depend on the degree of myocardial damage when the number of the wall segments subjected to ischemia is equal. Therefore, we thought that the serum level of the enzymes could be standardized by dividing the value by the CSW, eliminating the bias of the difference of the coronary artery segment involved, and preserving the influence of the degree of myocardial damage.

The efficacy of PTCR has been studied in respect to the prognostic benefits and post-infarction cardiac performance, but the results are controversial (6–8, 11–14). Furthermore, the influences of the PTCR-Time on the global and regional cardiac performance have been investigated, but the results are not definitive (15–20).

We wanted to study how the sustained coronary reperfusion could salvage myocardium by evaluating the influence of the PTCR-Time on the LV regional wall motion of the infarcted zone. The centerline method is usually used today to evaluate the LV regional wall motion. An although it is a useful mean to assess the LV regional wall contractility, it is rather difficult to evaluate the abnormal motion of the segments with dyskinesis and/or aneurysm. This is particularly so when a comparative evaluation is not possible, as in the present study when the left ventriculography was not performed at the acute phase because it did not benefit the patients and might have even hazardous at that time. The MA-score was devised to evaluate the regional wall motion abnormality qualitatively as well as quantitatively.

The same weight was assigned to dyskinetic and aneurysmal motions because we felt they might be similar with respect to the degree of myocardial damage. Their different clinical manifestations might be the result of infarct size and/or post infarct care. In our study, the degree of qualitative expansion of infarction was evaluated by the segmental score of the wall motion abnormality, and the degree of quantitative expansion of infarction was evaluated by the MA-score, which was standardized by the CSW. There is a controversy regarding the length after the onset of pain during which coronary reperfusion is beneficial. Lavallee et al. (4), in an animal experiment, described that reperfusion obtained within 2h after the total occlusion of a coronary artery was beneficial for myocardial salvage. Mathey et al. (15) reported that almost all patients reperfused within 2h after the onset of pain had improved regional LV function. Rentrop et al. (16), and Nobuyoshi et al. (17) described similar results in patients without collateral circulation to the infarcted artery as such that coronary reperfusion improved global
and regional LV functions if sustained recannalization was achieved within 3h or less after the onset of pain. Kamatsuue et al. (18), and Rogars et al. (19) stressed the importance of collaterals, that had developed before PTCR, to the infarcted coronary artery for improvement of cardiac function. Stack et al. (5) also addressed the importance of early sustained reperfusion for the improvement of the regional LV function and reduction of the infarct size. Ritchie et al. (13), and Smalling et al. (20), in contrast, reported that PTCR-Time did not correlate with improvements in global and regional LV functions.

In many of the above studies, however, the efficacy of PTCR was compared between groups with and without coronary reperfusion. None of the studies used a protocol in which the individual coronary artery was weighted. The studies did not correlate the PTCR-Time and the qualitative and quantitative evaluations of infarction cases with sustained coronary reperfusion. Our major concern is the significance of collateral circulation to infarct-related coronary artery as addressed by some authors (18, 19). But coronary angiograms taken at the acute and chronic phases did not document such significant collateral blood flow in all the cases subjected to the present evaluation of the time influences.

In the present study, we can clearly demonstrate that the PTCR-Time significantly correlated with the enzymatic serum levels, which indicated the degree of myocardial damage, significantly influenced post-infarct LV regional wall motion. The earlier the recannalization of the infarcted artery was achieved, the less the extent of the myocardial infarction. Normal wall motion tended to be retained when sustained recannalization was achieved within 2h after the onset of chest pain. Accordingly, the recent intravenous thrombolysis therapy may have advantages over the conventional PTCR procedure particularly in respect to the time-benefit. This study series, however, was too small to draw a definitive conclusion. Similar studies in a large series are required to corroborate our findings.

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