Cardioprotective Effects of Ischemic Postconditioning in Patients Treated With Primary Percutaneous Coronary Intervention, Evaluated by Magnetic Resonance

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Background—Postconditioning has been suggested to reduce myocardial damage during primary percutaneous coronary intervention (PPCI) in patients with ST-segment–elevation myocardial infarction. However, because clinical experience is limited, we examined the cardioprotective effects of postconditioning, using cardiac MRI in patients treated with PPCI.

Methods and Results—One hundred eighteen patients with ST-segment–elevation myocardial infarction referred for PPCI were randomly assigned to have either conventional PPCI or PPCI with postconditioning. Postconditioning was performed immediately after obtained reperfusion with 4 balloon occlusions, each lasting 30 seconds, followed by 30 seconds of reperfusion. The primary end point was myocardial salvage after 3 months as judged by delayed enhancement cardiac MRI. We found a 19% relative reduction of infarct size in the postconditioning group (51/11006 16% of total area at risk versus 63/11006 17%, P=0.01), corresponding to a 31% increase in salvage ratio. The number of patients developing heart failure was significantly fewer in the postconditioning group (27% versus 46%, P=0.048). No significant evidence of interaction between the impact of postconditioning and the location of the culprit lesion or size of the myocardium at risk was detected (P=0.21 and P=0.71).

Conclusions—Mechanical postconditioning reduces infarct size in patients with ST-segment–elevation myocardial infarction treated with PPCI. The impact of mechanical postconditioning seems to be independent of the size of myocardium at risk.

Clinical Trial Registration—URL: http://www.clinicaltrials.gov. Unique Identifier: NCT00507156.

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Key Words: myocardial infarction ■ percutaneous coronary intervention ■ postconditioning ■ reperfusion injury ■ cardiac magnetic resonance imaging

ST-segment elevation myocardial infarction (STEMI) is a major cause of death and heart failure in Western society,¹ the recommended therapy for which is primary percutaneous coronary intervention (PPCI). Even though PPCI is the most effective treatment in terms of reduction of long-term mortality and morbidity, acute restoration of myocardial blood flow may jeopardize the cardiomyocytes. This phenomenon is known as reperfusion injury, which contributes to cell death. It has been suggested that reperfusion injury accounts for 50% of the final size of the myocardial infarction,² and because the overall infarct size is an important predictor of long-term outcome in these patients,³ it seems justified to look for means to reduce reperfusion injury.

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Both pre- and postconditioning seems to protect cardiomyocytes during reperfusion therapy.⁴–⁶ Ischemic postconditioning is a sequence of repetitive interruptions of coronary blood flow applied after a period of ischemia. Recently, the effect of postconditioning was proven by Staat et al⁷ and Yang et al,⁸ who reported a 39% and 27% reduction in infarct size in patients with STEMI as measured by means of cardiac biomarkers and single-photon emission computed tomography (SPECT), respectively. The effect on the final infarct size only has been evaluated in 38 patients by Thibault et al⁹ using perfusion defect index measured with SPECT as an...
estimate of the infarct size 6 months after the initial treatment. SPECT is a relatively gross measurement of the infarct size compared with cardiac magnetic resonance (CMR), which has proven superior to SPECT with regard to detection and quantification of myocardial infarction. CMR also has a higher reproducibility than scintigraphy. Furthermore, in the previous studies, patients with symptoms for >6 hours, with preinfarct angina and collateral flow to the myocardium area at risk (AAR) were not enrolled. These patients represent a relatively large proportion of the patients admitted for PPCI, and in the clinical setting they are difficult to distinguish. Therefore, this study used CMR to evaluate for the first time the effect of ischemic postconditioning applied during PPCI on the final infarct size. To mimic daily clinical practice, we did not exclude patients with symptoms for >6 hours, with preinfarct angina, or with collateral flow to the myocardium AAR.

Methods

The study was performed in a prospective, randomized setting according to the Declaration of Helsinki and the European guidelines of Good Clinical Practice. The Danish National Committee on Biomedical Research Ethics approved the protocol. All patients were informed orally before enrollment and in writing after the PCI procedure.

Study Population

Patients were eligible for enrollment if they were aged >18 years and had an STEMI of <12 hours duration from onset of symptoms until arrival at the Department of Cardiology, the Heart Centre, Rigshospitalet (Copenhagen, Denmark). STEMI was defined as ST-segment elevation in 2 contiguous ECG leads of >0.1 mV in V_{1} to V_{3} or limb leads II, III, and aVF or >0.2 mV in lead V_{1} to V_{3}. The patients were excluded if spontaneous reperfusion had occurred before intervention (thrombolysis in myocardial infarction [TIMI] flow >1) or if PCI did not restore normal flow (TIMI flow <2) after vessel wiring or balloon predilatation in the infarct-related coronary artery. In addition, patients were excluded if coronary angiography revealed any other lesion with a diameter stenosis >70%, cardiac shock, left bundle branch block, previous Q-wave myocardial infarction, coronary bypass surgery, severe renal or liver failure, or stent thrombosis.

Experimental Protocol

Patients were pretreated with aspirin (300 mg orally or 500 mg intravenously), clopidogrel (600 mg orally), and heparin (10 000 U intravenously). At hospital admission, coronary angiography was performed to identify the culprit lesion and assess TIMI flow. Reflow was established by introducing a guide wire or inflating a small-sized balloon (1.5 to 2.5 mm). Randomization was performed using a computerized 1:1 randomization sequence. Postconditioning was performed by 4 repetitive low-pressure (4 to 6 atm) balloon inflations, each lasting 30 seconds, followed by a 30 seconds. The deflation episode was done at the site of the lesion in the index coronary artery. This algorithm was chosen because the effect of ischemic postconditioning is diminished by a rapid washout of H^{+} and a subsequent rapid normalization of pH. In contrast, reoxygensation of the tissue stimulates production of reactive oxygen species (ROS), which is important for protection of the mitochondria. Cohen et al suggested that a 30-second algorithm ensures that the pH does not increase abruptly and represents enough time to secure a proper production of ROS. Patients in the control group received no additional intervention during 4 minutes of reperfusion. The choice of stent was left to the discretion of the operator. Balloon angioplasty alone was only allowed if a stent could not be deployed or was considered harmful. Treatment with glycoprotein IIb/IIIa receptor antagonists was administered if no contraindications were present. The study was performed in a prospective, randomized setting.

Determination of Infarct Size

The primary end point was infarct size measured with CMR 3 months after the initial procedure. Three months was chosen because infarct size measured with CMR is thought to remain unchanged from 1 to 6 months after PPCI. If no contraindications were present, a CMR was performed on a 1.5 Tesla scanner. Infarct size was evaluated by delayed enhancement CMR, using 0.2 mg/kg body weight of intravenously injected gadolinium-diethlenetriamine pentaacetic acid as an ECG-triggered inversion-recovery gradient-echo sequence (echo time, 1.4 ms; repetition time, 4.0 ms; slice thickness, 8 mm). The left ventricular (LV) infarct area, defined as the hypoenhanced myocardium, was then identified by outlining the region of elevated signal intensity as shown in Figure 1. The infarct mass was calculated as the infarct mass=\frac{1}{2}(slice thickness\times infarct size\times myocardial density (1.05 g)). The LV mass was calculated as LV mass=\frac{1}{2}(slice thickness\times muscle size\times myocardial density). Infarct size as a percentage of LV mass was then calculated as infarct size (%)=\frac{infarct mass}{LV mass}. An investigator unaware of the treatment allocation performed and analyzed the CMRs.

AAR

Given that the infarct propagates from the subendocardial myocardium and spreads like a wavefront toward the epicardial border, the endocardial surface of the infarct area represents the AAR in patients who have had symptoms of ischemia for >1 hour. The lateral boundaries of the infarct zone are established after 1 hour of symptoms independently of collateral flow. To evaluate the AAR, the endocardial border of the infarct and total endocardial border of the LV were traced in each short axis slice, and the infarct endocardial surface area (ESA) was calculated for each patient as a percentage of the total LV ESA (infarct ESA [%] = ESA of infarct [cm²]/ESA of LV [cm²]) (Figure 2). To contemporarily evaluate different ways of describing AAR, we have compared the size of the myocardial AAR measured with CMR edema imaging (within 1 to 5 days after infarct) with the ESA method (measured 3 months±3 weeks after hospital admission). We have found a very good correlation between the 2 methods (r=0.85, P<0.001; data unpublished). For each patient, the ratio between infarct size and AAR was calculated. An investigator unaware of the treatment allocation analyzed the CMRs.

Other End Points

To assess global LV function on the CMR multiple slice of an ECG-triggered steady-state imaging pulse sequence (echo time, 1.6 ms; slice thickness, 8 mm; 20 cardiac phase) was performed, covering the entire left ventricle. The diastolic and systolic frames were identified.
according to the ventricular blood pool area. The LV ejection fraction (LVEF) was calculated by manually tracing the endocardial borders at end systole and end diastole for each short axis slice. Troponin T was used as biochemical marker and was obtained based on clinical routines, with blood samples 3 times within the first 24 hours after PPCI.

The incidence of clinical events, including evaluation of functional class (classified 1 to 4 on the New York Heart Association scale [NYHA]), angina pectoris (classified 0 to 4 according to Canadian Cardiovascular Society), myocardial infarction, need for reperfusion therapy in the target vessel or other vessel, and death were obtained during 3 months follow-up. An investigator unaware of the treatment assignment evaluated the clinical symptoms.

Statistical Analysis

The statistical analysis was done with SPSS version 15. Categorical variables were compared by Student t test or Fisher exact test. Continuous variables were compared by Student t test. To compare the relationship between the AAR and the infarct size, a regression analysis was performed with treatment modality as a fixed factor. To identify interaction between subgroups and the treatment modality, a 2-way ANOVA was used. Nonsignificant variables were eliminated from the model by backward stepwise elimination. A P<0.05 was considered statistically significant. With a risk of type 1 error of 5% and type 2 error of 20%, we wanted to find a difference of 20% in infarct size. Assuming an average infarct size of 20% of the total LV mass and an SD of 8, we needed 63 patients.

Results

Study Population and Treatment

From July 2007 to July 2008, 546 patients with STEMI were admitted to the hospital and assessed for treatment with PPCI. Of these, 428 were not eligible for the study protocol due to the following reasons: TIMI flow >1 (n=123), multiple vessel disease (n=44), did not understand information content (n=39), other heart disease (n=33), no myocardial infarction (n=31), duration of symptoms >12 hours (n=28), cardiogenic shock (n=28), unconsciousness (n=23), study refusal (n=17), stent thrombosis (n=12), did not meet inclusion criteria (n=38), no reason reported (n=24). Thus, 118 patients (21.6%) were eligible for enrollment. The groups were well matched with regard to demographic, angiographic, and procedural data, and there was no difference between treatment groups with regard to number of patients with collaterals to the infarct-related artery and with preinfarct angina (Tables 1 and 2). In this study, only 2 patients had symptoms for <1 hour, hence measuring the infarct ESA was a sufficient method to evaluate AAR. The patients were divided into subgroups according to whether their lesion was located in the left anterior descending artery, the right coronary artery, or a minor vessel. A minor vessel was defined as the left circumflex artery, posterior descending artery, posterior lateral artery, diagonal branches, or marginal branches. In addition, the patients were divided into 3 subgroups according to the size of AAR (<20%, 20% to 35%, and >35% of the total myocardium).

Two patients died within the first 3 months of follow-up. Of the remaining 116 patients, a CMR was not performed in 30 because of patient refusal (n=15), inability to cooperate during the CMR examination (n=4), contraindications (n=8), and technical difficulties (n=3). Thus, a CMR was performed in 86 patients (43 each in the postconditioning and control groups) within similar time frames from the PPCI treatment.

Infarct Size

The effects of postconditioning 3 months after PPCI (evaluated with CMR) are summarized in Table 3. An 18% relative reduction of infarct size was calculated as a percentage of the LV mass (P=0.04), and a 19% relative reduction of infarct size was calculated as a percentage of the total myocardium at risk in the patient group who had postconditioning per-
Effect of ischemic postconditioning on infarct size by CMR

This randomized study is the first to indicate a cardioprotective effect of ischemic postconditioning on infarct size by CMR. We found a significantly smaller peak of blood concentrations of creatine kinase and perfusion defect index measured with SPECT after 6 months. Using SPECT, Yang et al\textsuperscript{8} reported a 27% reduction in infarct size 7 days after postconditioning. In a study of 94 patients, Ma et al\textsuperscript{20} found a significantly smaller peak of blood concentrations of creatine kinase MB in patients treated with postconditioning.

Previously, Staat et al\textsuperscript{7} and Thibault et al\textsuperscript{9} enrolled 38 patients and found a statistically significant reduction in infarct size of 36 to 39% using the area under the curve of blood concentrations of creatine kinase and perfusion defect index measured with SPECT after 6 months. Using SPECT, Yang et al\textsuperscript{8} reported a 27% reduction in infarct size 7 days after postconditioning. In a study of 94 patients, Ma et al\textsuperscript{20} found a significantly smaller peak of blood concentrations of creatine kinase MB in patients treated with postconditioning.

The reduction of infarct size was smaller in this study than reported in the previous studies. This discrepancy could be explained by some important differences among the studies. First, contrary to existing studies, we did not exclude the patients if the angiography revealed a collateral flow to the myocardium at risk. However, in our study population, 74% of the patients had no collaterals with equal distribution in the 2 groups. In addition, we excluded patients with multiple-vessel disease to ensure exclusion of patients with expected extensive collateral development. Second, patients with pre-infarction angina were included in this study; therefore, these patients could have been inadvertently preconditioned before the experimental protocol. Third, patients with symptoms lasting \(\leq 12\) hours were included in this study, whereas in other studies, the cutoff point was 6 hours. The 12-hour cutoff point was chosen based on the assumption that viable cardiomyocytes are present up to 12 hours of infarct duration and on the fact that postconditioning has been shown to protect the cardiomyocytes against lethal reperfusion injury.\textsuperscript{6}

Therefore, we hypothesized that as long as viable cardiomyocytes exist in the ischemic myocardium at the time of reperfusion, they will benefit from postconditioning. However, a long duration of ischemia leads to damage to a larger number of cardiomyocytes and, thus, to a larger infarct size,\textsuperscript{3} so the effect of postconditioning might be less pronounced in these patients. Fourth, in this study, the patients were not directly stented as in the previous studies; however, predilatation only was allowed with a small-sized balloon and only formed \((P<0.01)\). Furthermore, the hearts in the postconditioning group were significantly heavier due to a significantly higher loss of myocardium in the control group. Importantly, in the regression analysis, the line for the postconditioning group lies significantly below the line for the control group (Figure 3). There was no significant evidence of interaction between the impact of postconditioning and the location of the culprit lesion or size of the myocardium at risk (Figures 4 and 5).

Other End Points

No significant difference was observed between the groups with regard to the peak troponin T value (Table 4). The clinical outcomes after 3 months are summarized in Table 4. The number of patients with NYHA class 2 to 4 was significantly lower in the postconditioning group, despite a similar LVEF, LV end-systolic volume, and LV end-diastolic volume in the 2 groups (Table 3). During the 3 months of follow-up, 1 patient died the day after PPCI due to pump failure, and 1 patient died 7 days after PPCI due to probable stent thrombosis according to the Academic Research Consortium definition (both from the postconditioning group).

Table 2. Angiographic and Procedural Results

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control (n=59)</th>
<th>Postconditioning (n=59)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Use of glycoprotein Iib/IIa inhibitor</td>
<td>49 (83)</td>
<td>49 (83)</td>
<td>1</td>
</tr>
<tr>
<td>Use of bivalirudin</td>
<td>7 (12)</td>
<td>4 (7)</td>
<td>0.34</td>
</tr>
<tr>
<td>Infarct-related artery</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RCA</td>
<td>25 (42)</td>
<td>28 (47)</td>
<td>0.27</td>
</tr>
<tr>
<td>LAD</td>
<td>23 (39)</td>
<td>26 (44)</td>
<td></td>
</tr>
<tr>
<td>LCX/PDA/PLA</td>
<td>11 (19)</td>
<td>5 (8)</td>
<td></td>
</tr>
<tr>
<td>No. of diseased vessels</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>49 (83)</td>
<td>46 (78)</td>
<td>0.66</td>
</tr>
<tr>
<td>2</td>
<td>8 (14)</td>
<td>9 (15)</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>2 (3)</td>
<td>4 (7)</td>
<td></td>
</tr>
<tr>
<td>Collaterals*</td>
<td>15 (26)</td>
<td>15 (26)</td>
<td>1</td>
</tr>
<tr>
<td>Type of stent</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>None</td>
<td>10 (17)</td>
<td>9 (15)</td>
<td>0.58</td>
</tr>
<tr>
<td>BMS</td>
<td>7 (12)</td>
<td>3 (6)</td>
<td></td>
</tr>
<tr>
<td>DES</td>
<td>42 (71)</td>
<td>47 (80)</td>
<td></td>
</tr>
<tr>
<td>Diameter of final balloon, mm</td>
<td>3.3</td>
<td>3.3</td>
<td>0.62</td>
</tr>
<tr>
<td>Length of stent, mm</td>
<td>18.1</td>
<td>19.6</td>
<td>0.43</td>
</tr>
<tr>
<td>Thrombectomy</td>
<td>10 (17)</td>
<td>14 (24)</td>
<td>0.44</td>
</tr>
<tr>
<td>Maximum deployment</td>
<td>16.2</td>
<td>17.6</td>
<td>0.32</td>
</tr>
<tr>
<td>TIMI grade 3 after procedure</td>
<td>52 (88)</td>
<td>56 (95)</td>
<td>0.32</td>
</tr>
</tbody>
</table>

Data are presented as n (%) unless otherwise indicated. BMS indicates bare metal stent; DES, drug-eluting stent; LAD, left anterior descending artery; LCX, left circumflex artery; PDA, posterior descending artery; PLA, posterior lateral artery; RCA, right coronary artery.

*Patients in whom the coronary angiography revealed collaterals to the infarct-related artery.

Discussion

This randomized study is the first to indicate a cardioprotective effect of ischemic postconditioning on infarct size by CMR scanning and on clinical end points. In this study, we treated patients with STEMI with ischemic postconditioning during PPCI and found a significant reduction in myocardial infarct size of 18% and a 19% reduction of myocardial infarct size expressed as a percentage of the AAR. We evaluated the infarct size using CMR, which has been proven to accurately determine the infarct size.\textsuperscript{18,19} In addition, we found an improvement in functional class in the patients treated with postconditioning.

Table 3. Outcomes Evaluated With CMR

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Control (n=43)</th>
<th>Postconditioning (n=43)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infarct size, g</td>
<td>25±13</td>
<td>23±13</td>
<td>0.30</td>
</tr>
<tr>
<td>LV mass, g</td>
<td>150±33</td>
<td>165±41</td>
<td>0.05</td>
</tr>
<tr>
<td>Infarct size, %*</td>
<td>17±8</td>
<td>14±7</td>
<td>0.04</td>
</tr>
<tr>
<td>Area at risk, %†</td>
<td>29±16</td>
<td>29±16</td>
<td>0.92</td>
</tr>
<tr>
<td>Infarct size/AAR, %</td>
<td>63±16</td>
<td>51±16</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>LVEF, %</td>
<td>53±10</td>
<td>53±10</td>
<td>1</td>
</tr>
</tbody>
</table>

All data are presented as the mean±SD. Total number of patients was 86.

*The mass of the infarct as a percentage of the LV mass.
†The ESA of the infarct as a percentage of the total LV ESA.
until reperfusion was obtained. Using this setup, we find it unlikely that patients could have been inadvertently postconditioned, even without the use of a direct stenting technique. Finally, and perhaps, most importantly, there are differences in the measurements of the infarct size among the different studies. In the present trial, CMR was used because this technique is able to identify an irreversibly injured myocardium with great precision. Compared with scintigraphy, CMR has a considerably higher resolution, has proven superior to SPECT with regard to detection and quantification of smaller areas of myocardial infarction, and appears to have a higher reproducibility than scintigraphy. Taken together, including patients with collaterals, preinfarct angina, and symptoms of ≤12 hours might diminish the effect of postconditioning. However, it is important to emphasize that a significant reduction in infarct size in this study population was observed, which supports that postconditioning is beneficial in these patients.

No difference in peak troponin T was observed in the present study. However, the troponin T values were collected in a very inconsistent manner, which makes the use of peak troponin T invalid as a precise surrogate measurement of the myocardial damage. This drawback might be more pronounced in ischemic postconditioning settings because this procedure has been shown to change the coronary blood flow, which in turn might change the washout of troponin T.

In addition, we found a significant improvement in NYHA class in the patients treated with postconditioning, which is a

Figure 3. Infarct size versus AAR. Infarct size as a percentage of the total LV mass was plotted against the myocardium AAR measured by the ESA method. The line for the postconditioning group lies significantly below the line for the control group (P<0.01). In both groups, the infarct size correlates with the AAR (r²=0.699 and 0.684, respectively).

Figure 4. Size of myocardial infarction according to treatment and culprit vessel. This graph shows the size of myocardial infarction as a percentage of the total LV mass. The results are divided into groups according to location of the lesion and treatment. The insignificant P value shows that there is a lack of interaction between the impact of postconditioning and location of the lesion. LAD indicates left anterior descending artery; Postcon, postconditioning; and RCA, right coronary artery. *Minor vessel is defined as the left circumflex artery, diagonal branches, or marginal branches.
clinical symptom of left-side heart failure. Clinical heart failure is an important prognostic factor with respect to mortality independent of the LVEF. On the other hand, NYHA classification is a relatively inaccurate and a subjective measurement, and the number of patients in this study is relatively small. Furthermore, the difference in NYHA classification between treatment groups was observed despite similar levels of LVEF and LV dimensions in the 2 groups. However, it is now recognized that many patients with symptoms and signs of heart failure have preserved LVEF and LV dimensions. Taken together, the shift in NYHA class, as observed in this study, should be taken with precaution, and whether it is due to the effect of postconditioning is still to be settled. The similarity in LVEF and LV dimensions was observed despite of the significant difference in infarct size. However, LV dimensions and LVEF are gross measures of the postinfarction LV damage partly because compensatory hyperkinesias of the noninfarcted myocardium contribute to a preserved LVEF. Furthermore, the infarct size is suggested to be superior to LV dimensions and LVEF as single predictors for long-term mortality. Thus, the discrepancy between no difference in LVEF and LV dimensions on one side and a reduction in infarct size and a shift in NYHA class on the other side might be explained by a shift in the hemodynamic pattern of LV ejection following infarction. However, it is beyond the scope of this article to explain the pathophysiological pattern of heart failure with preserved LVEF. Taken together, whether the positive impact of ischemic postconditioning will translate into an improved long-term prognosis still needs to be settled.

Considering the relatively high frequency of balloon angioplasty without stenting (16%), a higher incidence of reocclusion during 3 months of follow-up might have been expected and overlooked because we did not perform control angiography. However, reocclusion is an acute cardiac event and would lead to symptoms and immediate hospital readmission. Therefore, even without a control angiography, it is highly unlikely that any patients with reocclusion were missed during the 3-month follow-up.

Thrombectomy was performed in relatively few patients (20%) but equally in the 2 treatment groups. This relatively low frequency was partly due to the fact that the importance of thrombus aspiration during percutaneous coronary intervention in acute myocardial infarction was revealed after the performance of this study. However, it is possible that a more consequent use of thrombectomy might add to the effect of postconditioning.

**Study Limitations**

First, of the 118 patients enrolled in this study, 32 did not undergo a CMR mostly because of patient refusal, which is a
calculated risk when using CMR to determine a primary end point. Second, the operators were not blinded to the treatment sequence during PPCI; on the other hand, the investigators evaluating the primary end point and CMR data and those performing the clinical evaluations were unaware of the treatment sequence. Third, the study was not powered to show a possible interaction between treatment modalities and location of the culprit lesion. Fourth, the ESA method to evaluate AAR has been validated previously against other angiographic methods, such as the Myocardial Jeopardy Index and Alberta Provincial Project for Outcome Assessment in Coronary Heart Disease, but not against scintigraphy, which is the gold standard for measuring AAR. Furthermore, this method might underestimate the AAR. Finally, a more frequent collection of blood samples would have provided more accurate information with regard to rise and fall in biochemical markers of ischemia.

Conclusion

Our results show that ischemic postconditioning reduces the myocardial infarct size in 18% of patients with STEMI with TIMI flow of 0 to 1 who were treated with PPCI. In addition, it might improve the functional class in these patients. Future research is warranted to settle the impact of ischemic postconditioning on long-term clinical outcome, which protocol should be preferred, and which patients benefit from this treatment.

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Disclosures

None.

References


**CLINICAL PERSPECTIVE**

Acute restoration of myocardial blood flow with primary percutaneous coronary intervention in itself jeopardizes the cardiomyocytes. In some cases, this phenomenon accounts for 50% of the final size of the myocardial infarction. Therefore, it is important to look for means to protect the myocardium during reperfusion. Ischemic postconditioning has been suggested as such a method. Few small studies have demonstrated a beneficial effect of ischemic postconditioning, but the effect on the final infarct size only has been assessed in 38 patients with perfusion defect index measured by scintigraphy as a surrogate measurement for the infarct size. Ischemic postconditioning is simple, cheap, not time consuming, and a safe adjuvant to primary percutaneous coronary intervention, and the method can be introduced in the catheterization laboratories almost overnight. However, the possible introduction of this modality in our view should be demonstrated in a substantial number of patients before taken into consideration. With the use of cardiac magnetic resonance to measure final infarct size in 86 patients, this article demonstrates a decrease in infarct size of 18% with ischemic postconditioning. Being the first to evaluate effect of ischemic postconditioning by cardiac magnetic resonance, we believe that this study makes an important contribution. Furthermore, it is the first, to our knowledge, to suggest an effect on functional status evaluated by New York Heart Association classification.
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