Are there differences in late outcome after PTCA for angina pectoris after non-Q wave vs Q wave myocardial infarction?


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Aims Revascularization is thought to improve prognosis better if ischaemia persists after so-called non-Q wave myocardial infarction, than after Q-wave myocardial infarction, because it is assumed that prognosis is better where there is less left ventricular function loss. This study evaluates the differences in clinical outcome between patients with Q wave and those with non-Q wave myocardial infarction who underwent percutaneous transluminal coronary angioplasty because of recurrent ischaemia.

Methods We retrospectively analysed two consecutive groups of patients who underwent percutaneous transluminal coronary angioplasty for ischaemia after either a non-Q wave (n=175) or a Q wave (n=175) myocardial infarction, and who were followed for 4 years.

Results Initial angioplasty success rates were similar in both groups. At follow-up there were no significant differences between the two patient groups in rates of death (9% vs 11%, P=ns), myocardial infarction (3% vs 7%, P=ns) and target vessel revascularization by repeat percutaneous angioplasty (11% vs 15%, P=ns) or coronary bypass surgery (both 7%).

Conclusion We conclude that elective coronary angioplasty in patients with angina pectoris after non-Q wave myocardial infarction does not lead to a better prognosis than after Q wave myocardial infarction. Thus, management strategies after myocardial infarction should not be based on the absence or presence of Q waves on the electrocardiogram.

Key Words: Non-Q wave, myocardial infarction, percutaneous balloon angioplasty.

Introduction

Natural history studies have suggested that patients suffering a non-Q wave myocardial infarction have a more favourable initial prognosis due to less necrosis1-3 and better preserved left ventricular function4-6 than patients who suffer a Q wave myocardial infarction. However, this has been disproved in some studies; where there is a higher incidence of unstable angina and recurrent ischaemia, there is no improvement in late prognosis after non-Q wave myocardial infarction5-7. This being the case, one might expect to prevent recurrent ischaemia or myocardial infarction after non-Q wave myocardial infarction by target vessel revascularization. Currently, however, there are few data on the immediate and long-term results of percutaneous transluminal coronary angioplasty after non-Q wave myocardial infarction.

We retrospectively studied the initial results and late outcome of percutaneous transluminal coronary angioplasty in a consecutive group of patients with recurrent ischaemia after Q wave and non-Q wave myocardial infarction.

Methods

Patients who underwent percutaneous transluminal coronary angioplasty in our centre in 1991 were studied. These comprised the first 175 patients with symptoms and/or signs of ischaemia after Q wave myocardial infarction and the first 175 with symptoms and/or signs of ischaemia after non-Q wave myocardial infarction.
Q wave myocardial infarction was defined as prolonged chest pain characteristic of acute myocardial infarction, the appearance of new Q waves of at least 40 ms in duration and 2 mm in depth in at least two contiguous leads of the electrocardiogram, together with specific cardiac enzyme elevation. Non-Q wave myocardial infarction was defined in this study as prolonged chest pain characteristic of acute myocardial infarction, and specific cardiac enzyme elevation without the appearance of new pathological Q waves, as described above.

Coronary angioplasty procedural success was defined as one in which, on visual inspection, a >20% increase in luminal diameter was achieved, with the final diameter stenosis <50% and without the occurrence of death, acute myocardial infarction, or the need for repeat angioplasty or emergency bypass operation within the first 48 h. Myocardial infarction was defined as an increase in serum creatine kinase levels to at least twice the normal level (and the development of ST or T wave changes that persisted for at least 24 h).

All patients were followed-up at our outpatient clinic, or by the referring cardiologist. If additional information was required, patients were interviewed by telephone. The following events were taken into account during follow-up: death (cardiac or non-cardiac death), myocardial infarction, re-intervention either for restenosis or progression of disease elsewhere, and coronary bypass grafting. The common closure date was 1 April 1995.

Statistical analysis
Continuous data are presented as means ± standard deviations and when appropriate the median. Categorical data are presented as percentages. For the comparison of categorical data the Chi-square test or when appropriate the Fisher exact test was used. Normally distributed data were compared by means of the Student t-test. A P-value of 0.05 was considered statistically significant. For the comparison of right censored end-point data, the Kaplan–Meier method was used to draw the survival curves. Statistical comparison of the Kaplan–Meier curves was performed by means of the log-rank test. The hazard ratios were calculated by means of the Cox proportional hazard model, univariately and multivariately, with corresponding 95% confidence intervals for the indication of precision. This study has a power of more than 80% to reveal a 6% cumulative survival difference at follow-up.

Clinical data
Baseline characteristics were compared in both groups.

Angiographic data
Prior to balloon angioplasty, all patients underwent coronary angiography and left ventricle angiography.

The ventriculogram was evaluated using the Coronary Artery Surgery Study system[9]. This left ventricular function score provides a quantitative assessment of the segmental abnormalities of left ventricular function.

Results

Baseline characteristics
The baseline characteristics were comparable in both groups (see Table 1). For age, sex, time between myocardial infarction and percutaneous transluminal coronary angioplasty, thrombolysis, previous coronary artery bypass surgery, and the duration of follow-up there were no statistically and clinically significant differences between the two subgroups (Table 1).

Angiographic data
The number of patients with or without collaterals were the same in both groups, with a P value of 0.267. Stenosis severity prior to percutaneous transluminal coronary angioplasty was not statistically significantly different between the two groups on a categorial basis \( P=0.057 \) when categorized as percentages documented between 50–70%, 70–90%, 90–99%, and 100%.

In Table 2, the Coronary Artery Surgery Study classification of left ventricular function is shown. Only three non-Q wave myocardial infarction patients had a poor left ventricular function (Coronary Artery Surgery Study score 18 or 19), and one Q wave myocardial infarction patient had a poor left ventricular function (Coronary Artery Surgery Study score 18). Because of the small number of patients in these categories, these patients were classified in the group with moderate left ventricular dysfunction.

The Coronary Artery Surgery Study score for ventricular function was assessed and found to be statistically significantly different for the Q wave myocardial infarction patients as compared to the non-Q wave myocardial infarction patients. Fifteen of the 175 non-Q wave myocardial infarction patients had moderate left ventricular function as opposed to 30 in the Q wave myocardial infarction patients, \( P=0.016 \). The Cox proportional hazards model showed virtually identical hazards ratios for the Q wave and non-Q wave myocardial infarction patients when age, gender and left ventricular function score were incorporated in the model as compared to the univariate analyses. Only univariate hazard ratios are depicted in Fig. 1.

There were significantly more total occlusions with a \( P \) value of 0.02 in the Q wave patients (51%) as compared to the non-Q wave patients (29%). Other angiographic data including calcifications, number of diseased vessels and the localization of the myocardial infarction are given in Table 2.

In the 175 patients who underwent percutaneous transluminal coronary angioplasty for angina pectoris...
after Q wave myocardial infarction, 265 lesions were dilated (1-5 lesions/patient). In the group with a non-Q wave myocardial infarction, 251 lesions were attempted (1-4 lesions/patient). Success rates per patient in the Q wave group was 94% (164/175), and in the non-Q wave myocardial infarction group 97% (169/175) ($P=0.21$).

Myocardial infarction as a complication of the angioplasty procedure occurred in 10 (5-7%) of the Q
The Kaplan-Meier event-free survival curves for non-Q vs Q wave infarction after successful percutaneous transluminal coronary angioplasty for post-myocardial infarction ischaemia. Freedom from all-cause mortality. The log rank P value is 0.3712. Non-Q wave myocardial infarctions are indicated by broken lines, Q wave myocardial infarctions by solid lines.

Table 3 Initial results

<table>
<thead>
<tr>
<th></th>
<th>Q wave (%)</th>
<th>Non-Q wave (%)</th>
<th>P value</th>
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<tbody>
<tr>
<td>Dilated lesions</td>
<td>265</td>
<td>251</td>
<td>0.76</td>
</tr>
<tr>
<td>Lessons per patient</td>
<td>1.51</td>
<td>1.43</td>
<td></td>
</tr>
<tr>
<td>Success rates</td>
<td>164 (94)</td>
<td>169 (97)</td>
<td>0.21</td>
</tr>
<tr>
<td>Stenosis pre-PTCA SD</td>
<td>85 (12-6)</td>
<td>82 (11-8)</td>
<td></td>
</tr>
<tr>
<td>Stenosis post-PTCA SD</td>
<td>14 (21-7)</td>
<td>11 (17-8)</td>
<td></td>
</tr>
<tr>
<td>Gain</td>
<td>-71</td>
<td>-71</td>
<td>0.19</td>
</tr>
<tr>
<td>Complication &lt;48 h</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Dissection</td>
<td>34</td>
<td>34</td>
<td></td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>10</td>
<td>13</td>
<td>0.7</td>
</tr>
<tr>
<td>Redilatation</td>
<td>6</td>
<td>3</td>
<td>0.5</td>
</tr>
<tr>
<td>Bypass surgery</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Death</td>
<td>0</td>
<td>0</td>
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</table>

PTCA = percutaneous transluminal coronary angioplasty; SD = standard deviation.

Wave myocardial infarction patients and in 13 (7.4%) of the non-Q wave myocardial infarct patients, P = 0.52. Six patients from the Q wave group needed acute redilatation, vs three in the non-Q wave group (P = 0.5). No patient died or needed emergency bypass surgery in the first 48 h (Table 3).

Follow-up

Mean follow-up was 44 months in both groups. At the common closing date, 114 out of 175 patients in the Q wave group had remained free from any event (65.1%), compared to 101 in the non-Q wave group (57.8%). Angina pectoris recurred in 15 patients in the Q wave group and in 14 patients in the non-Q wave group (P = 0.85).

Twenty patients died in the Q wave group, 14 for cardiac reasons; 15 died in the non-Q wave group, seven for cardiac reasons (P = 0.53). Myocardial infarction relating to the vessel in which percutaneous transluminal coronary angioplasty was performed occurred in six patients in the non-Q wave group and in five patients in the Q wave group (P = 0.76). Myocardial infarction not related to the vessel in which percutaneous transluminal coronary angioplasty was performed was only seen in the non-Q wave myocardial infarction group (n = 7) (Table 4).

Surgical myocardial revascularization was performed in 13 patients (7%) and repeat coronary angioplasty was performed in 13 patients (7%) in the Q wave group. In six patients (3%) in the Q wave group coronary angioplasty was performed in a vessel not related to the previous myocardial infarction area. In the non-Q wave group, 12 patients (7%) had surgical myocardial revascularization; repeat coronary angioplasty was performed in 19 patients (11%) in the related vessel, and in
Table 4  Follow-up

<table>
<thead>
<tr>
<th></th>
<th>Q wave (%)</th>
<th>Non-Q wave (%)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean follow-up (months)</td>
<td>43-32</td>
<td>45-02</td>
<td>0-83</td>
</tr>
<tr>
<td>No event</td>
<td>114</td>
<td>101</td>
<td>0-54</td>
</tr>
<tr>
<td>Event</td>
<td>61</td>
<td>74</td>
<td>0-40</td>
</tr>
<tr>
<td>Cardiac death</td>
<td>14 (8)</td>
<td>7 (4)</td>
<td>0-12</td>
</tr>
<tr>
<td>Non-cardiac death</td>
<td>6 (3)</td>
<td>8 (5)</td>
<td>0-59</td>
</tr>
<tr>
<td>re-MI in PTCA vessel</td>
<td>5 (3)</td>
<td>6 (3)</td>
<td></td>
</tr>
<tr>
<td>re-MI in non PTCA vessel</td>
<td>0 (0)</td>
<td>7 (4)</td>
<td>0-76</td>
</tr>
<tr>
<td>Repeat PTCA of the same vessel</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Related vessel</td>
<td>13 (7)</td>
<td>19 (11)</td>
<td>0-27</td>
</tr>
<tr>
<td>Not related vessel</td>
<td>6 (3)</td>
<td>9 (5)</td>
<td>0-43</td>
</tr>
<tr>
<td>Bypass surgery</td>
<td>13 (7)</td>
<td>12 (7)</td>
<td>0-84</td>
</tr>
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QI=myocardial infarction; PTCA=percutaneous transluminal coronary angioplasty.

Figure 2 The Kaplan–Meier event-free survival curves for non-Q vs Q wave infarction after successful percutaneous transluminal coronary angioplasty for post-myocardial infarction ischaemia. Non-Q wave myocardial infarctions are indicated by broken lines, Q wave myocardial infarctions by solid lines. Freedom from cardiac death. The log rank P value is 0-115. Symbols as in Fig. 1.

nine patients (5%) coronary angioplasty was performed in a vessel not related to the previous myocardial infarction area (Table 4).

Discussion

Our results indicate that the initial and long-term outcome after percutaneous transluminal coronary angioplasty for angina pectoris following acute myocardial infarction is similar in patients who suffer a non-Q wave myocardial infarction or a Q wave myocardial infarction. The literature on outcome after Q wave and non-Q wave myocardial infarction is, in many respects, conflicting.

In a non-selected group of patients with acute myocardial infarction, the occurrence of a non-Q wave myocardial infarction is probably much higher than previously reported9,10. This may be because the more sensitive creatine-kinase-MB assay improves recognition of myocardial infarction, or that medical treatment, specifically thrombolytic therapy, leads to early reperfusion, itself a mechanism of non-Q wave myocardial infarction generation4,9,10,12.

Non-Q wave myocardial infarction is more likely to affect an older population, with a higher proportion of women, and with more previous coronary events9. In our patient groups, age and percentages of women were equal. Aguirre et al. showed that in non-Q wave, as opposed to Q wave patients there were more women and fewer anterior wall infarctions, and that the left ventricular function was better11.

Non-Q wave myocardial infarction is usually characterized by partial perfusion of the infarct-related artery by either collateral or antegrade flow, and by a lower incidence of intracoronary thrombus than in Q wave acute myocardial infarction4,14,15,18,19. At angiography following non-Q wave myocardial infarction,
Figure 3 The Kaplan–Meier event-free survival for non-Q vs Q wave infarction after successful percutaneous transluminal coronary angioplasty for post-myocardial infarction ischaemia. Freedom from myocardial infarction. The log rank \( P \) value is 0.086. Symbols as in Fig. 1.

Figure 4 The Kaplan–Meier event-free survival curves for non-Q vs Q wave infarction after successful percutaneous transluminal coronary angioplasty (PTCA) for post-myocardial infarction ischaemia. Freedom from related PTCA. The log rank \( P \) value is 0.257. Symbols as in Fig. 1.

arterial occlusion is usually subtotal, probably because (spontaneous) reperfusion has occurred. The size of non-Q wave myocardial infarction is therefore generally less extensive than Q wave myocardial infarction, as was shown by enzymatic, scintigraphic and angiographic data. The same phenomenon of early reperfusion explains why the results of exercise stress testing and thallium myocardial scintigraphy suggest that residual myocardial ischaemia is more frequent and extensive after non-Q wave myocardial infarction. Long-term prognosis of non-Q wave myocardial infarction is dependent on the amount of viable myocardial tissue at risk and the extent of collateral coronary circulation. So, although patients with non-Q wave myocardial infarction have a favourable short term prognosis, late prognosis is poor due to a high incidence of recurrent unstable angina pectoris or myocardial infarction. Therefore, it is not surprising that mortality 1 year after the myocardial infarction is similar for both patients with Q wave and non-Q wave myocardial infarction. When infarction recovers after a non-Q wave myocardial infarction, this has a deleterious effect on survival.

Thus, is it possible to select and treat those patients who have an unfavourable long-term outcome? Post-infarction angina could be identified as an important risk variable for adverse long-term outcome in patients with non-Q wave myocardial infarction. One study showed that only 25% of the patients have angina pectoris after a non-Q wave myocardial infarction.
Late outcome after PTCA

Figure 5 The Kaplan-Meier event-free survival curves for non-Q vs Q wave infarction after successful percutaneous transluminal coronary angioplasty for post myocardial infarction ischaemia. Freedom from coronary artery bypass grafting (CABG). The log rank \( P \) value is 0.808. Symbols as in Fig. 1.

Figure 6 The Kaplan-Meier event-free survival curves for non-Q vs Q wave infarction after successful percutaneous transluminal coronary angioplasty (PTCA) for post-myocardial infarction ischaemia. Freedom from CABG or related PTCA. Symbols as in Fig. 1.

Infarction\(^{27}\) Mickley et al.\(^{28}\) showed that in non-Q wave myocardial infarction the presence of ST segment depression on ambulatory electrocardiography recordings and exercise testing had a statistically significant predictive value for the development of future angina pectoris, whereas patients at increased risk for subsequent non-fatal reinfarction or cardiac death were not identified. In survivors of a first non-Q wave acute myocardial infarction, routine coronary angiography is not indicated if they are asymptomatic below the age of 60 years and after a first non-Q wave myocardial infarction\(^{27}\). Batalha et al.\(^{29}\) concluded from their study that it is not necessary to use invasive studies in every patient who has suffered a non-Q wave myocardial infarction without complications, since stress testing shows a high sensitivity (94-4%) and specificity (75%), and a high predictive positive value (100%) for predicting the risk of recurrent ischaemia. Any type of cardiac event occurred in 12% of patients with normal technetium-99m sestamibi stress testing compared with 39% of those with an abnormal test after medically treated unstable angina pectoris\(^{30}\). Patients should be considered for angiography and revascularization when they continue to have myocardial ischaemia, ST-depression, or thallium 201 perfusion defects on exercise\(^{31}\), especially if they already have reduced ventricular function\(^{32}\) and this policy has been adopted by our institution.

Cardiac revascularization by means of coronary artery bypass grafting or percutaneous transluminal
coronary angioplasty is often recommended for ischaemia after myocardial infarction, even more in the case of non-Q wave myocardial infarction because there is greater potential to salvage myocardium than in those with Q wave myocardial infarction[33,43]. Several investigators suggest that coronary bypass surgery or percutaneous transluminal coronary angioplasty is feasible and safe for patients with angina pectoris after non-Q wave acute myocardial infarction[33,36].

Systolic left ventricular function has an important influence on subsequent survival, particularly in patients with more extensive coronary disease[8,37-39]. Using percutaneous transluminal coronary angioplasty to achieve improvement in ventricular function therefore seems to be justified, but it remains unclear whether complete revascularization is indicated, although in our group we tried as often as possible to achieve complete revascularization, as is indicated by the number of 1-4-1-5 lesions dilated per patient. In a recent follow-up study, Weintraub et al. showed that associated disease in non-dilated segments was the strongest predictor of late events including death, myocardial infarction and need for a repeat revascularization[40]. However others have indicated that patients are at an increased risk for recurrent ischaemia usually in the non-Q wave myocardial infarction area[41,43].

Thus, although there is greater potential to salvage myocardium in patients with non-Q wave myocardial infarction, in our study group the clinical results after 4 years were not better than in the Q-wave myocardial infarction group. These somewhat surprising findings may, in part, be explained by careful patient selection, because the procedure was limited to lesions technically suitable for percutaneous transluminal coronary angioplasty. In addition, our results are based on a relatively small number of patients, thereby raising the possibility of a selection bias. Moreover the different mechanisms which may lead to a non-Q wave myocardial infarction may have influenced patient selection. Most of the patients can be thought of as patients with an aborted Q wave myocardial infarction. If a Q wave myocardial infarction has been aborted this may be due to autolysis, thrombolytic therapy or collaterals, but in these respects the patients in our non-Q wave myocardial infarction group should have been doing better, because of the lower number of total occlusions, the equal number having received thrombolitics and because more collaterals were present. Also, a non-Q wave acute myocardial infarction may be considered a small but complete acute myocardial infarction. In our patient group, however, all patients had angioplasty in the major coronary arteries, and not in the small sidebranches, and all (but 3%) had recurrent ischaemia, necessitating angioplasty. The results of this study cannot be extrapolated to all patients with previous non-Q wave and Q wave myocardial infarction. The study was not randomized and alternative treatment could have been as good as percutaneous transluminal coronary angioplasty. However, we included a group of consecutive patients with a long-term follow-up of 4 years, thus allowing validation of the clinical approach used in our institution.

Our data confirm that percutaneous transluminal coronary angioplasty is an effective means for treating patients with ischaemia after non-Q wave myocardial infarction, as well as after Q wave myocardial infarction. Percutaneous transluminal coronary angioplasty in the clinical setting provides not only a high primary success rate, but also a favourable outcome at 4 years, but with no clinical advantage to the non-Q wave myocardial infarction group.

We conclude that percutaneous transluminal coronary angioplasty for angina pectoris after non-Q wave myocardial infarction is often recommended for ischaemia after myocardial infarction, even more in the case of non-Q wave myocardial infarction because there is greater potential to salvage myocardium than in those with Q wave myocardial infarction. Several investigators suggest that coronary bypass surgery or percutaneous transluminal coronary angioplasty is feasible and safe for patients with angina pectoris after non-Q wave acute myocardial infarction.

**Figure 7** The Kaplan-Meier event-free survival curves for non-Q vs Q wave infarction after successful percutaneous transluminal coronary angioplasty for post myocardial infarction ischaemia. Freedom from death, myocardial infarction (MI), coronary artery bypass grafting (CABG), or related percutaneous transluminal coronary angioplasty (PTCA). The log rank $P$ value is 0.399. Symbols as in Fig. 1.
wave myocardial infarction has a similar initial and long-term outcome in patients with non-Q wave myocardial infarction. This study suggests that percutaneous transluminal coronary angioplasty appears not to improve prognosis beyond the prognosis after Q wave myocardial infarction. This indicates that management strategies after myocardial infarction should not be based on the absence or presence of Q waves at the ECG.

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