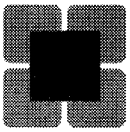

Percutaneous transluminal coronary angioplasty after non-Q-wave infarction

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■ Non-Q-wave infarction is associated with a significant incidence of infarction and death. We followed 52 patients who underwent percutaneous transluminal coronary angioplasty (PTCA) within three months of non-Q-wave myocardial infarction. Thirty-eight patients had single-vessel disease, 12 had double-vessel disease, and two had triple-vessel disease. At follow-up at 28 ± 8 months, all patients were alive. Three suffered a recurrent myocardial infarction during the follow-up period. Nine underwent repeat angioplasty. Four patients had bypass surgery for recurrent symptoms. Only 25% of the patients had angina at follow-up, and all but two were in an improved functional class. PTCA may be used safely in the management of patients after non-Q-wave infarction and results in a favorable long-term prognosis.

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NON-Q-WAVE infarction, formerly thought to be a benign condition,[1–3] has recently been shown to be associated with a mortality rate equal to or greater than that for Q-wave infarction.[4–7] A high incidence of re-infarction and recurrent angina after a patient's discharge from the hospital also has been reported.[8–10]

These previous studies suggest that patients with non-Q-wave infarction require more intensive follow-up and investigation. It seems that there is greater potential to salvage myocardium in these patients than those with Q-wave infarction. Many of these patients have subtotal occlusion of the infarct-related artery[10,11] and may be amenable to percutaneous transluminal coronary angioplasty (PTCA).

In this study, patients who underwent angioplasty for infarct-related arteries soon after non-Q-wave infarction were followed up to document the incidence of cardiac events.

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MATERIALS AND METHODS

Fifty-two consecutive patients with non-Q-wave infarction referred to the Cleveland Clinic between January 1983 and December 1984 and who underwent successful PTCA formed the study group. There were 17 women and 35 men (mean age, 59 years; range, 39–78 years). All patients had a history of chest pain, elevation of serum creatine kinase to twice the upper normal level, and ST-T-wave changes that persisted for more than 24 hours without the appearance of Q waves (≥ 0.03 sec and one third or greater than the amplitude of the R wave). Thrombolytic therapy was given at the discretion of the private physician at the time of the infarction.

Technique

For 24 hours before angioplasty, all patients received dipyridamole and aspirin. Nitrates, beta blockers, and

TABLE 1
ANGINA BY CANADIAN HEART CLASSIFICATION PRIOR TO PTCA AND AT FOLLOW UP

Canadian Heart classification	Pre-PTCA	Follow up
I	9	39
II	9	11
III	12	2
IV	22	0

TABLE 2
CHANGE IN ANGINA BY CANADIAN HEART CLASSIFICATION IN PATIENTS WHO WERE SYMPTOMATIC AT FOLLOW UP

Canadian Heart classification	Pre-PTCA	Follow up
I	1	0
II	1	11
III	3	2
IV	8	0

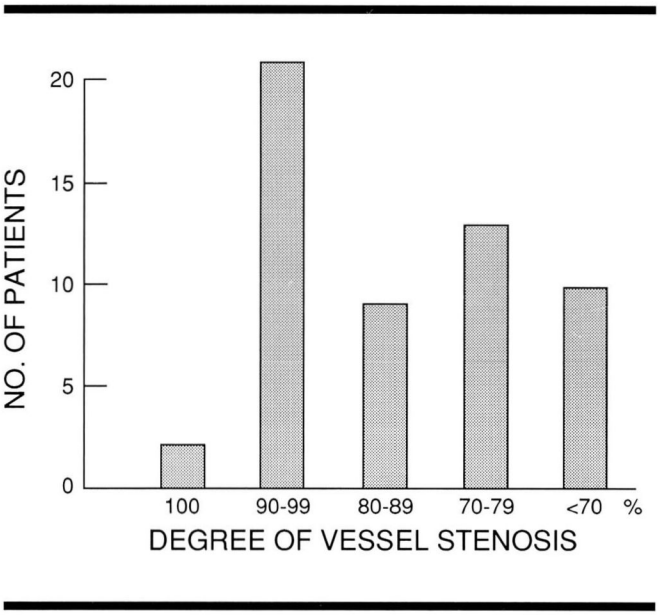

FIGURE 1. Frequency histogram, showing degree of stenosis of infarct-related artery prior to PTCA.

calcium channel blockers were continued as indicated by clinical conditions.

Just before angioplasty, orally administered nifedipine and intravenously administered diazepam were given. After intra-arterial insertion of the catheter, 10,000 units of heparin was administered.

PTCA was attempted via the femoral approach, similar to the technique described by Grüntzig et al,[12] an average of 47 days (range, 1–90 days) after the non-Q-wave infarction. Balloon inflations and pressures used in each case varied according to the degree of remaining trans-stenotic gradient and percentage of remaining stenosis after each inflation.

Patients were monitored overnight and, in most cases, discharged one to two days later. Patients in whom angioplasty was successful were discharged on a regimen of nifedipine, dipyridamole, and aspirin, which was continued for at least six months. All patients were advised to undergo repeat coronary angiography six months after angioplasty or earlier if they became symptomatic. After angioplasty, vessel stenosis was measured. The luminal diameter of the vessel at the narrowest portion of the stenosis was expressed as a percentage of the luminal diameter above and below the stenosis. Coronary angioplasty was considered to be a primary success if the stenosis diameter was reduced by at least 40% and the patient did not require emergency bypass surgery within 24 hours of the angioplasty.

Follow-up

After hospital discharge, patients were seen by their own physician and no attempt was made to influence their management. The mean duration of follow-up was 16 months (range, 12 to 41 months) and was 100% complete. Patients were contacted by telephone, and if doubts remained regarding the documentation of cardiac events, the patient's physician was consulted and medical records were obtained. During the follow-up period, a specific attempt was made to determine the incidence of cardiac death, recurrent myocardial infarction, repeat PTCA, coronary artery bypass surgery, recurrent angina and its functional class, and activity status. The presence or absence of recurrence, defined as greater than 50% loss of the initial improvement in the stenosis at angioplasty, was noted on the follow-up angiograms.

RESULTS

The myocardial infarction was anterior in 33 patients (63%), inferior in 17 (33%), and true posterior in two (4%). Thirty-eight patients had single-vessel disease, 12 had double-vessel disease, and two had three-vessel disease. Nine patients had no symptoms at the time of angioplasty, nine had class II angina, 12 class III, and 22 class IV, according to the Canadian Heart Association (*Table 1*). Only 12 patients had angina prior to their non-Q-wave infarction. The left anterior descending artery was the infarct-related vessel in 32 patients; one patient had a circumflex artery dilated as well. Seven-

teen patients had the right coronary artery dilated; one had a circumflex and left anterior descending artery dilated. Two patients had a circumflex artery dilated, and one patient had a left main coronary artery dilated. The mean stenosis before angioplasty was 80% (range, 50%–100%). In two patients, a total occlusion was successfully dilated (*Figure 1*). The mean stenosis immediately after angioplasty was 23% (range, 0%–50%).

Clinical outcome

All patients were alive at follow-up. Four patients underwent coronary artery bypass grafting—two for recurrence shown angiographically and the other two for recurrent angina after their second PTCA. In both of these latter patients, the recurrent ischemia was related to the artery originally dilated.

Nine patients (17%) underwent repeat angioplasty at a mean of 5.3 months (range, 16 days–14 months) after their first angioplasty. Six of these patients had recurrent symptoms, and three underwent repeat angioplasty for recurrence shown angiographically.

Three patients (6%) had recurrent myocardial infarction involving the infarct-related artery at 15 days, two months, and three months, respectively, after the initial angioplasty.

Thirteen patients (25%) had chest pain at follow-up. Only two of these patients were not in an improved functional class (*Table 2*), and these two had proven continued success of the angioplasty at follow-up as determined by angiography.

Follow-up angiography

Follow-up angiography was performed for 36 patients. Most had their repeat study done at six months, but others underwent the study earlier due to recurrent symptoms. Eleven of the 36 patients (31%) showed recurrence angiographically and were managed by repeat angioplasty (nine patients) or medical treatment (two patients). At follow-up, symptom-free status continued for seven; the remaining two eventually underwent coronary artery bypass grafting.

DISCUSSION

In this group of patients who underwent successful PTCA following non-Q-wave infarction, there was no mortality and a low incidence of re-infarction during follow-up. This contrasts markedly with the study by Marmor et al,[13] during which 17% died within nine months after discharge from the hospital. In the series by Hutter et al,[4] a 21% incidence of re-infarction was seen over a two-year follow-up period. This trend toward a high incidence of recurrent cardiac events is repeated in other studies.[6–8] Some other investigators, however, suggest that mortality is lower, especially in younger patients with their first myocardial infarction.[10,11]

In our series, patient selection was highly biased towards the presence of continuing symptoms; 65% had class III or IV angina. Also, our series included many patients over 60 years of age, a group that has been shown to be at even higher risk of recurrent events. One would expect this group to have a higher than average incidence of infarction and death,[9–11] but this was not seen at follow-up.

The low incidence of recurrent angina (25%) after angioplasty in our study is gratifying and contrasts with the study by Nicholson et al,[10] in which 62% of patients had angina at follow-up. The most significant angiographic predictor of recurrent angina in their group seemed to be an obstruction of a proximal left anterior descending artery. Patients with this type of obstruction were the majority in our group. Despite this fact, severe angina at follow-up was unusual.

Three patients in our study had recurrent infarction involving the artery that had undergone angioplasty. In one patient, infarction recurred at only 15 days. This is of some concern and may be related to rapid re-stenosis or spasm at the site of angioplasty despite the use of calcium blocking drugs.[14] Thrombotic occlusion despite the use of antiplatelet agents is another possible explanation.

Our recurrence rate (31%), as shown angiographically after six months, is similar to the recurrence rate other investigators have noted after elective single-vessel angioplasty.[15–17] In other series of patients with non-Q-wave infarction,[10,11] most had subtotal occlusion of the infarct-related artery as evidenced by early angiographic study. In our series, only two patients underwent angioplasty of a totally occluded artery and only 10 patients had stenosis of less than 70% at the time of angioplasty. There is still doubt whether, given time, all arteries in patients with non-Q-wave infarction would show a subtotal rather than total occlusion. It would appear that the earlier the artery recanalizes after a non-Q-wave infarction, the greater the potential for further problems, such as infarction, angina, or death after the first event.

The widespread use of thrombolytic drugs, such as streptokinase or tissue plasminogen activator, might increase the number of nontransmural myocardial infarctions. Re-infarction or death followed successful thrombolytic therapy despite anticoagulation in 19% of

patients in one series.[18] Early reocclusion following successful thrombolysis is between 15% and 35%.[18,19]

The use of PTCA after successful thrombolytic therapy has reduced the re-infarction rate during initial hospitalization.[20] Furthermore, the presence of a severe stenosis after successful thrombolytic therapy has been associated with a higher incidence of early reocclusion.[21,22]

Data from our series suggest that angioplasty has a favorable effect on the prognosis in the first year after nontransmural myocardial infarction. When thrombolytic therapy is successful in salvaging myocardium and creating a nontransmural myocardial infarction, angioplasty for the underlying atherosclerotic obstruction may be needed in many cases to prevent repeat infarction and death.

The results of angioplasty compare favorably with coronary artery bypass surgery for the treatment of this condition. In an uncontrolled study, Madigan et al[23] reported no mortality or re-infarction at 16 months in a group of patients undergoing operation after non-Q-wave infarction. Only 22% of these patients had angina at the time of follow-up. The CASS study[24] was a controlled trial of surgery *v* medical treatment, but did not examine the subset of patients with non-Q-wave infarction. Most of the post-infarction patients in the CASS study were asymptomatic. Also, Q wave and non-Q wave were considered in the same subgroup for the purposes of analysis.

Limitations of the study

Ours is not a prospective study, and the patients were a selected group biased towards those with recurrent angina and subtotal rather than total occlusions of the infarct-related artery. We believe, however, that because of these biases, these patients had a greater chance of recurrent infarction and further symptoms. We contend that PTCA reduces mortality and re-infarction, although this can be truly tested only by a randomized trial. Yet such a trial would be difficult to perform because of the symptomatic benefit to the patient after angioplasty in this setting. It is important to continue to follow these patients to see if the frequency of cardiac events increases after the first year. In our series, PTCA was initially successful for all patients. Cases were not excluded, but due to chance, all cases happened to be successful. At the Cleveland Clinic, we would expect 95% to have a successful procedure and 2%–3% to undergo emergency coronary bypass surgery.

Only two of our patients (4%) had total occlusion of their infarct-related vessel despite the relatively late restudy time (mean, 47 days). This is in contrast to data from DeWood et al[25] who demonstrated an increasing incidence of total occlusion with time from infarction such that 40% of patients studied angiographically had occlusion of their infarct-related vessel by 72 hours to one week following myocardial infarction. Their study was clinical, with different selection biases, but probably represents a more accurate incidence of vessel occlusion. Our study, in which most patients were symptomatic and had lesions suitable for PTCA, is biased in favor of patients with patent vessels and high-grade obstruction. Subsequently, this should lead to a higher complication rate.

CONCLUSION

This study suggests that there is a long-term benefit to patients who undergo angioplasty after having suffered a non-Q-wave infarction. The implications are immense, particularly when applied to the increased numbers of patients with nontransmural myocardial infarction following successful thrombolytic therapy, and could well influence routine in-hospital therapy of acute myocardial infarction.

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