Percutaneous Transluminal Coronary Angioplasty for Patients with Unstable Angina Pectoris

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Percutaneous transluminal coronary angioplasty (PTCA) was successful in 91% of 76 patients with unstable angina pectoris refractory to pharmacological treatment. However, the rate of acute occlusion and reocclusion was rather high (95). Restenosis developed in 56.5% of successful cases after initial PTCA, and 29 patients underwent 2nd, and nine 3rd PTCA. Most refractory unstable angina can be controlled by PTCA, which may require repeating in some patients.
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THE clinical syndrome of unstable angina causes great concern to clinicians because of the perceived high risk of progression to myocardial infarction. If medical treatment fails to stabilize unstable angina, its risk increases. Percutaneous transluminal coronary angioplasty (PTCA) has been accepted as an effective treatment for the relief of myocardial ischemia. We have performed PTCA in patients with unstable angina who are refractory to pharmacological therapy. In this paper, we present our data showing that PTCA is effective in controlling refractory angina, although the rates of acute occlusion, acute reocclusion, and restenosis tend to be high.

METHODS

Patients

PTCA was performed in 76 patients (59 men, 17 women, mean age 60.6 years, range 39 to 80) with unstable angina pectoris.

Nineteen patients had had attacks of increasingly frequent or severe pain at rest or on mild daily activities with onset within the previous 2 months. In the other 19 patients, previous stable angina had become unstable with occurrence at rest or on less strenuous activities with a worsening in the frequency or severity of pain within 2 months. The remaining 38 patients had had episodes of prolonged chest pain lasting more than 15 min. The electrocardiograms recorded during chest pain showed reversible ST segment elevations in 11 patients and ST segment depressions in 65 patients. The symptoms could not be stabilized by medical treatment consisting of nitrates, calcium channel blockers, antiplatelet agents (aspirin or ticlopidine) and sometimes nitroglycerin infusions. Patients showing signs of cardiac necrosis as a rise in creatine kinase to twice the normal level or the development of Q waves were not included.

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Fig.1. Clinical course of the patients undergoing PTCA for refractory unstable angina pectoris.

CABG=coronary artery bypass grafting. Rest.=restenosis, Suc.=successful PTCA, Unsuc.=unsuccessful PTCA.

Coronary Arteriography

All patients underwent coronary arteriography through the femoral approach. Heparin (5,000 units) was injected into the femoral artery immediately after introduction of the groin sheath. Isosorbide dinitrate (3 to 5 mg) was administered to the right and left coronary arteries. An intraluminal filling defect of contrast medium was considered to show an intracoronary thrombus.

PTCA

PTCA was performed with standard steerable dilation catheters. It was carried out immediately, within a week, and within 2 weeks after the coronary angiography in 31, 27, and 18 patients, respectively. Before intracoronary manipulations were begun, an intraarterial bolus of 10,000 units heparin was administered. When PTCA was performed immediately after the diagnostic arteriography, an additional 5,000 units of heparin was given. In patients with multivessel disease, only the ischemia-related vessel was dilated. The number of balloon inflations varied depending on the appear-

ance of the lesion as seen during immediate postdilatation angiography. PTCA was considered successful if the severity of obstruction was reduced to less than 50% of the luminal diameter with abolition of acute ischemic symptoms.

Treatment and Follow-up after PTCA

Medications consisting of nitrates, calcium channel blockers, and aspirin or ticlopidine were continued after PTCA. Several patients underwent follow-up angiography approximately 3 months after PTCA or when chest pain recurred. Restenosis was defined as a greater than 50% recurrent stenosis at the previously successful angioplasty site. PTCA was repeated in patients with restenosis. If necessary, coronary artery bypass grafting (CABG) was performed.

Statistics

Values are presented as mean $\pm$ SD. Categorical variables were analyzed using the chi-square test.

RESULTS

One-vessel disease was present in 50 patients (66%), 2-vessel disease in 20 (26%), and 3-vessel disease in 6 (8%). The ischemia-related vessel was the left anterior descending artery in 45 patients (59%), the left circumflex artery in 19 (25%), and the right coronary artery in 12 (16%). The overall clinical course after PTCA is shown in Fig. 1.

1st PTCA

PTCA was initially successful in 69 of the 76 patients (91%). The diameter of the stenosis decreased from $94.3 \pm 5.3\%$ before angioplasty to $29.3 \pm 9.1\%$ after angioplasty. PTCA failure was due to inability to cross the stenosis (2 patients) or to effectively dilate the traversed lesion (2 patients) and to acute occlusion during the procedure (3 patients). The 3 patients with acute occlusion developed acute myocardial infarction and underwent emergent CABG. However, 1 patient died post-operatively.

Reocclusion of the dilated site occurred within 24 h after the angioplasty in 4 of 69 patients with successful PTCA. PTCA was immediately repeated, and was successful in

TABLE 1 RESULTS OF FOLLOW-UP

angina free	66	(86.8%)
angina present	4	(5.3%)
death	4	(5.3%)
cardiac		
after CABG	1	
sudden death	1	
non-cardiac	2	
uncertain	2	(2.6%)

all cases. However, myocardial infarction developed in 2 patients. Thus, the acute occlusion plus reocclusion rate was 9.2% (7/76), and intracoronary thrombus was observed in 3 (43%) of these.

2nd PTCA

Seven patients with successful PTCA did not undergo follow-up arteriography. Six of them were free of angina, but the remaining patient had recurrent chest pain and died suddenly.

Restudy was performed in 62 patients 3.5 ± 1.5 months after the successful 1st PTCA. Restenosis was observed in 35 of 62 patients (56%). The intraluminal thrombus and ST deviations during anginal attacks had no significant effects on the restenosis rate; thrombus positive 52.6% (10/19) vs negative 60.5% (26/43); ST elevation 66.7% (6/9) vs depression 56.6% (30/53).

The second PTCA was carried out in 29 patients, (12 (41%) of whom had developed recurrent unstable angina), and was successful in 24 patients (83%) with a reduction of the diameter stenosis from 89.0 ± 13.6 to $31.5 \pm 10.7\%$. The reasons for unsuccessful PTCA were inadequate dilatation (due to changes in the stenosis morphology) in 4 cases and inability to cross the stenosis in 1 case.

Six patients with restenosis did not undergo PTCA. One of them underwent elective CABG. In the remaining 5 patients medical treatment was continued because collateral channels were well developed (1 patient) or the stenosis was between 50 and 75% (4 patients).

3rd PTCA

Nineteen patients underwent restudy after 2nd PTCA, which revealed restenosis in 9 patients (47%). PTCA was performed in

these 9 patients (3 had recurrent unstable angina) and was successful in all patients. Four of 5 patients who did not undergo follow-up study after successful 2nd PTCA were symptom free.

Late Results

Table I shows the findings at a clinical follow up at an interval of 21.3 ± 11.8 months after the initial PTCA. Acute myocardial infarction had occurred in 5 patients due to acute occlusion (3 patients) during the 1st PTCA, or reocclusion (2 patients) within 24 h after the 1st PTCA. Among them, 3 patients with acute occlusion underwent emergent CABG. Elective PTCA was performed in 2 patients.

DISCUSSION

The present data indicate that PTCA is an effective treatment for unstable angina refractory to pharmacological treatment. However, PTCA in this setting has an increased risk of acute occlusion during the procedure or acute reocclusion early after the successful angioplasty. In our study, 9% of cases developed such complications. A previous report¹ has also demonstrated that the rate of nonfatal myocardial infarction within 24 h after PTCA is significantly higher in patients with unstable angina than in those with stable angina (9.0 vs 4.3%). In 917 PTCA cases performed by Goldbaum et al., unstable angina was present in 20 (80%) of 25 patients with acute reocclusion.² This may be associated with the complex nature of the underlying coronary pathoanatomy including disruption or fissuring of the coronary atherosclerotic plaque,^{3,4} platelet adhesion and aggregation,^{4,5} cyclical generation and lysis of thrombus and coronary arterial spasm.⁵ PTCA may interrupt these events by altering the anatomic substrate with which the hematologic and pathophysiologic factors interact, or by mechanical disruption or dislodgement of intraluminal thrombi. On the other hand, PTCA may potentially aggravate the coronary pathoanatomy and lead to luminal occlusion through extension of intimal dissection and/or thrombus.

Another important aspect to be taken into account is the rate of restenosis. Unstable angina has been shown to be positively

correlated with an increased risk of recurrent restenosis⁶. The rate of restenosis was 56.5% in the present study. This value seems to be higher than that (28%) reported in other studies^{7,8}. The reason for this discrepancy is uncertain. It is likely, however, that the complicated coronary pathoanatomy in refractory angina may easily become the basis for restenosis. Furthermore, 41% of patients who underwent 2nd PTCA due to restenosis developed recurrent unstable angina. This suggests that instability of the coronary arterial lesion may persist in some patients even after the initial PTCA.

Most of the patients with refractory angina and 3 vessel disease were surgically treated in our institution during the period of this study. Therefore, the ratio of 3 vessel disease was rather low (8%) in the present study. This may explain the absence of acute myocardial infarction during the follow-up period.

In conclusion, most refractory angina pectoris can be controlled by PTCA, which may need to be repeated in some patients.

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