Coronary angioplasty for early postinfarction unstable angina

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ABSTRACT Coronary angioplasty was performed in 53 patients in whom unstable angina had reoccurred after 48 hr and within 30 days after sustained myocardial infarction. Single-vessel disease was present in 64% of the patients and multivessel disease in 36%. The preceding myocardial infarction had been small to moderate in size in the majority of the patients. The left ventricular ejection fraction was more than 50% in 80% of the patients. Forty-five patients were refractory to pharmacologic treatment; eight were initially stabilized but once again became symptomatic with light exertion. Angioplasty was performed in 35 patients 2 to 14 days and in 18 patients 15 to 30 days after infarction (average 12 ± 7 days after infarction). The initial success rate was 89% (47/53). The success rate of the patients treated at 2 to 14 days was lower (29/35, 83%) than that of patients treated at 14 to 30 days (18/18, 100%) but did not reach statistical significance (p < .06). There were no deaths related to the procedure. In four of the six failures, emergency bypass surgery was performed and two patients sustained a myocardial infarction. Furthermore, a myocardial infarction complicated the angioplasty procedure in two other patients; thus the overall procedure-related myocardial infarction rate was 8% (4/53). At 6 months follow-up, 26% (14/53) of all the patients who underwent angioplasty had recurrence of angina, which was successfully treated with repeat angioplasty, bypass surgery, or medical therapy. There were no late deaths. Late myocardial infarction occurred in two patients. Thus the total myocardial infarction rate after angioplasty at 6 months was 11% (6/53 patients). In 42 of the 47 (89%) patients with successful angioplasty, angiography was repeated a mean 3.3 ± 2.5 months after angioplasty. The angiographic restenosis rate was 33%. We conclude that in selected patients, coronary angioplasty for unstable angina occurring 48 hr to 30 days after a myocardial infarction is an effective treatment with an acceptable risk, a high initial success rate, and a sustained beneficial effect.
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THE PRESENCE of ischemic yet viable myocardium in a patient with angina early after an infarction places that patient at increased risk of reinfarction,[1-6] which carries a poor short- and long-term prognosis.[2, 4, 6] Although bypass surgery in patients with postinfarction angina can be performed with good results, the operative mortality and morbidity rates are less favorable than those for stable angina.[7-11] Recently, coronary angioplasty has been shown to be highly effective in the treatment of both stable[12] and unstable angina,[13-15] even in patients who are refractory to optimal pharmacologic treatment.[16] Coronary angioplasty as an alternative to surgery might be an attractive option in the management of patients with early postinfarction unstable angina, since it can avoid the attendant risks of major anesthesia and bypass surgery. Our policy over the last few years has been to offer coronary angioplasty to selected patients with unstable angina who, early after acute myocardial infarction, are either refractory to pharmacologic therapy or who experience angina induced by mild exertion despite initial response to maximal pharmacologic therapy.

In this study we report our experience with coronary angioplasty in the treatment of patients with unstable angina occurring after 48 hr and within 30 days after myocardial infarction.

Patients and methods

From January 1983 through September 1985, 2731 patients were admitted to our coronary care unit at the Thoraxcenter. Of these, 101 patients had postinfarction unstable angina and fulfilled the following criteria: (1) chest pain at rest, lasting for at

TABLE 1

Selection for angioplasty or bypass surgery of patients with postinfarction unstable angina within 30 days of infarction

Postinfarction unstable angina (≤ 30 days)	n	Extent coronary artery disease				Extent left ventricular dysfunction (ejection fraction)					
		One vessel	Two vessels	Three vessels	Left main	>60%	50–59%	40–49%	30–39%	<30%	Unknown
Total	101										
Coronary angioplasty	53	34 (64)	14 (27)	5 (9)	0 (0)	21 (40)	21 (40)	4 (8)	5 (9)	1 (1)	1 (1)
Bypass surgery	48	3 (6)	6 (13)	34 (71)	5 (10)	13 (27)	10 (21)	13 (27)	7 (15)	3 (6)	2 (4)

Data expressed as number of patients, with percent of total in parentheses.

least 15 min, associated with electrocardiographic manifestations of myocardial ischemia and no evidence of further myocardial necrosis; (2) chest pain occurring 2 to 3 days after the acute myocardial infarction; (3) rest pain, or pain on minimal exertion, refractory to optimal medical therapy, including intravenous nitroglycerin, β-adrenergic blockade, or calcium antagonists (if necessary intra-aortic balloon pump support was instituted); (4) suitability for bypass surgery. Patients were selected for coronary angioplasty if the ischemia-related lesion was suitable for dilatation. Patients were selected for surgery if they had multivessel disease with one or more critical stenoses supplying a large area of viable myocardium in addition to the ischemia-related vessel or if they had left main stem disease (table 1). The selection was based only on the coronary anatomy and was not influenced by left ventricular function. Accordingly, 53 patients were selected for angioplasty. Forty-five patients were refractory to pharmacologic treatment consisting of a combination of intravenous nitroglycerin, β-adrenoceptor antagonists, calcium antagonists, and anticoagulants. Eight patients were initially stabilized but had angina after slight exertion despite maximal medical treatment. All underwent coronary angioplasty within 30 days after the onset of the preceding myocardial infarction.

Acute myocardial infarction was diagnosed when patients fulfilled the following criteria: (1) typical history of chest pain, (2) a cardiac enzyme rise to two times control, and (3) evolutionary ST-T changes or development of pathologic Q waves (≥ 0.03 sec). The electrocardiographic ST-T changes associated with chest pain at rest after infarction were categorized as follows: (1) transient ST-T segment elevation (≥ 0.1 mV), (2) transient ST segment depression (≥ 0.1 mV), (3) development of persistent negative T waves (≥ 0.1 mV), or (4) transient minimal ST-T changes (< 0.1 mV) and pseudo normalization of T waves.

Selective coronary angiography was performed in multiple projections, including hemiaxial views. A lesion with a luminal diameter stenosis of more than 50% was considered hemodynamically significant. All films were interpreted visually without knowledge of the clinical status. The estimated percent obstruction was derived from the angiographic view showing the greatest reduction in diameter for the vessel in question. Furthermore, in 29 patients with an initially successful angioplasty the severity of the stenosis before and immediately after angioplasty and at repeat angiography was calculated with the help of a computer-based system for coronary angiography analysis (CAAS).[17] The clinical, electrocardiographic, and angiographic characteristics of the 53 patients with early postinfarction angina are shown in tables 1, 2, and 3.

The majority of the preceding myocardial infarctions were localized to the anterior wall (77%), were nontransmural (65%), and were of small to moderate size (75%) as assessed enzymatically. Half of the patients showed transient ST-T segment elevation during postinfarction angina (table 2). The presence of

single-vessel disease was 64%, and in 74% the left anterior descending artery was the ischemia-related vessel. In 16% (3/19) of the patients with multivessel disease the ischemia-related vessel was found to be "at a distance." Left ventricular function was normal or only slightly abnormal in 80% of the patients (ejection fraction > 50%), whereas only 1% had severely compromised left ventricular function (ejection fraction < 30%).

Coronary angioplasty was performed according to the technique initially described by Grüntzig et al.[12] After January 1983, a steerable dilatation system was used. At the beginning of the procedure a pacing catheter was placed in the right ventricle, 100 mg of heparin and 250 mg of acetylsalicylic acid was administered intravenously, and low molecular weight dextran (500 ml) was infused slowly. To exclude or prevent coronary spasm, nifedipine or nitroglycerin was infused into the coronary artery and this was repeated when necessary.[18] Initial balloon inflation pressure was 2.0 atm, with subsequent inflations ranging to 12 atm. The number of balloon inflations varied depending on the appearance of the lesion as seen during immediate postdilatation angiography and on the residual gradient across the stenosis. Angioplasty was deemed successful when a reduction of the severity of the obstruction to less than 50% luminal diameter narrowing and abolition of acute ischemic symptoms without progression to myocardial infarction or death were achieved. Reduction of the transstenotic pressure gradient index was used as a guide, but not as a sole criterion of success. The

TABLE 2

Clinical characteristics of 53 patients with early postinfarction unstable angina undergoing coronary angioplasty

	No.	%
Age (yr, range)	57.7	(36–74)
Males	44	83
Anterior wall infarction	40	76
Nontransmural	28	53
Transmural	12	23
Inferior wall infarction	13	24
Nontransmural	7	13
Transmural	6	11
Size of previous myocardial infarction[A]		
Small (5×)	31	58
Moderate (5–10×)	9	17
Large (10×)	10	19
Unknown	3	6

[A]Assessed enzymatically: peak creatine kinase level during preceding MI; expressed as compared with upper limit of normal creatine kinase value.

TABLE 3

The electrocardiographic manifestations of ischemia associated with early postinfarction unstable angina in 53 patients

	No.	%
ST segment elevation ($\geq$0.1 mV)	26	49
ST segment depression ($\geq$0.1 mV)	5	9
Negative T wave ($\geq$0.1 mV)	12	23
Minimal ST-T changes ($<$0.1 mV) or apparent "normalization" of T wave	10	19

transstenotic gradient index was calculated as the mean proximal pressure minus the mean distal pressure divided by the mean aortic pressure.[19] In patients with multivessel disease, only the ischemia-related vessel was dilated.[20] After the procedure, patients were monitored for 24 hr in the coronary care unit, where serial electrocardiograms and enzyme levels were obtained, and were discharged after 2 to 3 days. They were kept on treatment with nifedipine 40 to 60 mg daily and acetylsalicylic acid 500 mg daily over a period of 6 months. During the procedure a cardiac surgical team was available for standby. If emergency bypass surgery was required, perioperative myocardial infarction was diagnosed when new pathologic Q waves developed.

Clinical follow-up information was obtained by personal interview, from the referring physician, or by questionnaire. Patients were evaluated for the occurrence of myocardial infarction and recurrence of angina pectoris. The majority underwent electrocardiographic exercise testing with thallium-201 scintigraphy and repeat angiography. Patients performed symptom-limited exercise on the bicycle ergometer with stepwise increments of 20 W every minute. The three orthogonal leads XYZ of the Frank lead system were recorded. An ischemic response was defined as at least a 0.1 mV ST segment depression, occurring 0.08 sec after the J point. The maximum workload achieved was expressed as a percentage of the normal workload predicted for age, sex, and height. Thallium scintigraphic imaging was performed in the 45 and 65 degree left anterior oblique views, immediately after injection of 1.5 mCi of thallium-201 at peak stress. The postexercise images were obtained 4 hr later. Images were obtained with a Searle Phogamma V camera and processed with computer interface as previously described.[21] Defects with redistribution were considered to represent exercise-induced ischemia. Persistent defects without redistribution were considered to represent infarcted myocardium. Repeat angiograms were obtained in multiple views (including hemiaxial views for the left coronary artery) and were interpreted visually without knowledge of the clinical status. Restenosis was defined as an increase of the luminal diameter stenosis of the dilated lesion above 50%. Results are presented as mean ± SD.

Statistics. The significance of the difference between groups was measured by the chi-square test or Fisher's exact test where appropriate. All p values were calculated by a two-tailed test. A p value of less than .05 was considered significant.

Results

Coronary angioplasty was performed a mean 11.9 ± 7.2 days (range 2 to 30) after the preceding myocardial infarction. Eighteen patients (34%) underwent angioplasty 2 to 7 days after infarction, 17 (32%) 8 to 14 days, 13 (25%) 15 to 21 days, and five (9%) 22 to 30

days after infarction. Coronary angioplasty was initially successful in 89% (47/53). The initial success rate for dilatation was 78% (14/18) 2 to 7 days after infarction, 88% (15/17) after 8 to 14 days, and 100% (18/18) after 15 to 30 days. The initial success rate of dilatation 2 to 14 days after infarction of 83% (29/35) was lower than the 100% success rate 15 to 30 days after infarction but did not reach a statistically significant level (p < .06). The initial success rate was 87% (34/39) for the left anterior descending artery, 100% (9/9) for the right coronary artery, and 80% (4/5) for the left circumflex artery. The inflation pressure per procedure was a mean of 10 ± 2.2 atm (range 6 to 12). The number of inflations per procedure was a mean of 4.2 ± 1.5; the average inflation duration was a mean of 45 ± 16 sec.

The transstenotic pressure gradient was reliable in 39 of the 47 patients who had a successful angioplasty, dropping from 0.58 ± 0.15 before angioplasty to 0.14 ± 0.10 after angioplasty. A computer-derived measurement of the dilated stenosis before and after angioplasty and at repeat angiography was available in 29 patients with successful angioplasty. The computer-calculated diameter stenosis was 63 ± 12% before angioplasty, 29 ± 10% immediately after angioplasty, and 28 ± 13% at repeat angiography performed 2.9 ± 2 months later.

In six patients the procedure was not successful. In four the vessel totally occluded after manipulation of the guidewire. All underwent emergency bypass surgery, but two nevertheless sustained a myocardial infarction. In two patients the procedure was complicated by a prolonged period of angina, and despite eventual angiographic success both patients showed enzymatic and electrocardiographic evidence of new myocardial infarction. There were no deaths related to the procedure. It was not possible in this rather small study population to identify a variable such as age, gender, size or location of previous infarction, severity and location of ischemia related lesion, or interval between coronary angioplasty and myocardial infarction that was predictive for unsuccessful angioplasty.

All patients were followed for at least 6 months after angioplasty, the majority of them for 12 months (table 4). There were no late cardiac deaths; however, one patient died of lung carcinoma. Two patients developed late myocardial infarction in the territory supplied by the dilated vessel. Thus the total incidence of a reinfarction after postinfarction angioplasty for unstable angina (either procedure related inclusive of emergency surgery or late reinfarction) at 6 months follow-up was 11% (6/53). Recurrent angina was initially

TABLE 4

Clinical follow-up (6 and 12 mo) after initially successful coronary angioplasty in 53 patients with early postinfarction unstable angina

	Successful angioplasty (n = 47)		Unsuccessful angioplasty (n = 6)	
	6 mo	12 mo	6 mo	12 mo
No. of patients followed	47 (17)	42 (15)	6 (2)	6
Late cardiac death	0	0	0	0
Late reinfarction	1	0	1	0
Recurrent angina	13 (8)	0	1	0
Repeat angioplasty	3 (7)	0	0	0
Bypass surgery	6 (4)	0	0	0
Pharmacologic treatment	4 (3)	0	1	0

Numbers in parentheses denote patients with multivessel disease and dilatation of only the ischemia-related vessel.

treated pharmacologically in all patients. If there was an inadequate response to this initial treatment, patients with single-vessel disease were offered repeat angioplasty and those with multivessel disease were offered bypass surgery. Fourteen patients had recurrent angina within 6 months of the procedure; they were successfully treated by repeat coronary angioplasty (three patients), bypass grafting (six patients), or pharmacologic treatment (five patients). Recurrent angina occurred more frequently in patients with multivessel disease who underwent dilatation of the ischemia-related vessel only (8/17, 47%) than in patients with single-vessel disease (6/30, 20%). This difference was not statistically significant (p < .07). Thus at 6 months follow-up, 79% (37/47) had sustained improvement after an initial successful angioplasty, including a repeat angioplasty, and at 6 months after the initial angioplasty attempt 91% (48/53) of patients were in NYHA class I and 9% (5/53 patients) were in class II.

Results of electrocardiographic exercise testing and thallium-201 scintigraphy after successful coronary angioplasty were available in 92% (43/47) of the patients and were performed a mean 1.8 ± 0.9 months after the procedure. A maximum workload of more than 80% predicted for age, sex, and height was achieved in 86% of the patients. Eighty-seven percent of the patients were symptom free during the test; an ischemic ST-T segment depression was induced in 11%, and a reversible thallium-201 perfusion defect was present in 28%. Repeat angiograms were available in 89% (42/47) of patients having an initially successful angioplasty. In five patients angiography was not repeated; one had recurrent angina and four were

asymptomatic. The angiography was performed a mean 3.3 ± 2.5 months after the procedure. Restenosis occurred in 14 patients (33%), of whom four were asymptomatic.

Discussion

The incidence of angina recurring after acute myocardial infarction during hospitalization varies from 18% to 57%.[2, 6, 22–25] In many such patients, a subtotal or total occlusion of the infarct-related vessel is found at cardiac catheterization.[26] Recently a number of reports have suggested that appropriately applied pharmacologic therapy may reduce the size of myocardial infarction, which implies that jeopardized myocardium may remain in or near the infarcted area and this salvageable tissue may be subject to recurrent or persistent ischemia.[25] As our ability to interrupt the process of myocardial necrosis continues to improve, the number of patients with symptoms of angina early after infarction may increase.

Persistent or recurrent pain after infarction implies that the cardiac event is not yet complete and suggests that more aggressive invasive management might improve clinical status and might salvage myocardium. Postinfarction ischemia may be localized to either the border zone of the infarct ("ischemia in the infarct zone") or to a distant vascular bed ("ischemia at a distance").[25] In our study, the ischemia-related vessel in patients with multivessel disease was found to be at a distance in 16% of these patients.

The mechanisms of early postinfarction unstable angina are largely unknown. Apart from coronary artery spasm or increased vasomotor tone, recanalization of thrombus followed by the formation of a new thrombus may be responsible.[27, 28] Such early postinfarction angina carries a poor short- and long-term prognosis.[2, 4, 6, 25] Despite marked improvement in pharmacologic therapy, the reinfarction rate remains high at 10% to 34%[2, 6] and is associated with a poor prognosis.[4, 5]

Management of early postinfarction angina poses a problem to the clinician. The occurrence of intractable postinfarction angina has been a primary indication for urgent revascularization. First reports of patients undergoing surgical revascularization early after infarction noted an increased operative mortality compared with that in other patients undergoing revascularization months or years after infarction.[29–33] Despite the substantial improvement in surgical technique and postoperative care and the consequent lower mortality and better long-term results,[7–11] there is still no consensus as to the safety of surgery in the early postinfarction phase and many centers continue to manage these

patients medically.[34] It has been our policy to endeavor to postpone surgery for postinfarction angina to at least 6 weeks after the acute initial event by means of intensive pharmacologic therapy and, if needed, by intra-aortic balloon counterpulsation. This policy still holds for patients sustaining a large myocardial infarction with extensive left ventricular dysfunction.

More recently we have referred patients with early postinfarction angina and preserved left ventricular function for surgery and have indeed shown surgery to be relatively safe and more effective than medical treatment for relief of angina during the first year after infarction,[35] results that have been confirmed by others.[7-11] Yet, it is also clear that these results fall somewhat short of the excellent results of elective surgery. Since the introduction of coronary angioplasty for stable angina,[12] the strict initial indications have been expanded and now include angioplasty for unstable angina refractory to optimal pharmacologic therapy. In an earlier study we reported the results of angioplasty in 60 patients with refractory unstable angina.[16] Of these 60, 11 patients underwent angioplasty within 30 days of a myocardial infarction and the majority 3 to 4 weeks after infarction. This number of patients was too small to be considered for a meaningful separate analysis. Since then we have begun to accept patients for angioplasty with angina occurring very early after infarction, and these patients, together with those previously described, constitute the present study population. It is now our belief that early dilatation in patients with postinfarction angina is effective and might be accomplished with an acceptable risk.

Angioplasty offers several advantages over bypass surgery: (1) angioplasty is easier to implement, (2) it avoids the intrinsic risks of major anesthesia and major surgery, (3) it avoids the risks of intra-aortic balloon pumping which might be instituted in refractory patients in the preoperative period,[36] (4) it considerably reduces the hospital stay, and (5) it reduces costs.[37]

The results of angioplasty in this group of patients compared favorably to those of dilatation in patients with stable angina. The initial success rate of dilatation within 30 days after an infarction was 89%, which is slightly lower than the recently reported rate of well above 90% for stable angina.[38] There was a trend for the success rate to be lower if the dilatation was performed within 14 days of infarction when compared with dilatation performed within 15 to 30 days (83% vs 100%; $p < .06$). In the majority of our patients the postinfarction ischemia was localized to the border zone of the infarct ("ischemia at the infarct zone").[25] A possible explanation that the initial success rate was

lower earlier after infarction may be that the coronary lesion leading to the infarction is less well healed earlier after infarction, which renders these lesions more vulnerable to intracoronary manipulation with guidewires, leading to a total occlusion, as has occurred in some of our patients. Emergency bypass surgery was deemed necessary for four patients (8%), yet two of these developed myocardial infarction. There were no deaths related to the procedure itself, and thus the overall procedure-related myocardial infarction rate was 8% (4/53). This rate is substantially higher than the 2.6% reported for elective procedures.[38]

The preceding myocardial infarction was of small or moderate size in the majority of the 53 patients. Presumably this is a reflection of the selection process of patients with postinfarction angina for angioplasty. Patients with single-vessel disease and well-preserved left ventricular function tend to be selected for angioplasty, whereas those with multivessel disease and severely compromised left ventricular function undergo surgery, even though severely compromised left ventricular function was not considered an exclusion criterion for angioplasty, since patients were selected only according to coronary anatomy. Thus it is yet unknown whether angioplasty in patients with unstable angina complicating larger infarcts could be performed with similar results.

During at least 6 months follow-up of all patients who underwent angioplasty (either successful or failed), recurrent angina occurred in 26% (14/53). All were satisfactorily treated by repeat angioplasty, coronary bypass surgery, or pharmacologic therapy. There were no late cardiac deaths, and late myocardial infarction developed in two patients. Thus the overall reinfarction rate at 6 months follow-up (procedure related and late reinfarction) was 11%. The angiographic restenosis rate after successful angioplasty was 33%, a figure similar to that reported for stable angina.[39]

At 6 months follow-up after attempted angioplasty, 91% (48/53) of the patients were in NYHA class I and 9% (5/53) were in NYHA class II. During the relatively short follow-up period of the present series, early coronary angioplasty appears to relieve angina satisfactorily. However, the long-term benefits (beyond the first year) in terms of preventing recurrent infarction or cardiac death remain in question. Furthermore, a prospective randomized study comparing aggressive medical therapy, early angioplasty, and early surgery should provide clear insight into the relative merits of each treatment. Nevertheless, these data show that coronary angioplasty can be performed in selected patients with early postinfarction angina who have a rea-

sonably well-preserved left ventricular function and who have predominantly single-vessel disease with an acceptable risk and with a high initial success rate.

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