# Acute Coronary Artery Occlusion During and After Percutaneous Transluminal Coronary Angioplasty

## Frequency, Prediction, Clinical Course, Management, and Follow-up

Pim J. de Feyter, MD; Marcel van den Brand, MD; GertJan Jaarman, MD; Ron van Domburg, MD; Patrick W. Serruys, MD; and Harry Suryapranata, MD

*Background.* **Acute coronary artery occlusion after percutaneous transluminal coronary angioplasty (PTCA) continues to remain a serious complication despite significant improvement in operator performance and technological advancements. This retrospective study was performed to ascertain the frequency, predictive variables, management, and outcome of acute coronary artery occlusion.**

*Methods and Results.* **The study was based on data from 1,423 consecutive patients who underwent an elective coronary angioplasty between January 1986 and December 1988. Acute coronary artery occlusion occurred in 104 patients (7.3%). Acute occlusion developed during the dilatation procedure in 80 patients (5.6%) and within 24 hours after the procedure in 24 patients (1.7%). Four clinical and 14 angiographic variables predictive for acute coronary artery occlusion were analyzed in these 104 patients with a complicated procedure and were compared with those in 104 representative patients with successful attempts. Multivariate analysis found three independent predictive variables: unstable angina, multivessel disease, and complex lesions. The overall clinical outcome after management of acute coronary artery occlusion including immediate repeat dilatation (95 patients), use of intracoronary streptokinase (34 patients), or autoperfusion catheter (12 patients) was successful (reduction of lumen diameter to <50%, no death, no myocardial infarction [MI], and no emergency surgery) in 42 patients (40%), was a failure without major complication in four patients (4%), and was a failure with major complication (death, MI, and emergency surgery) in 58 patients (56%). The overall mortality rate was 6% (six patients), the overall MI rate was 36% (37 patients), and emergency bypass surgery was required in 30% of patients (31 patients). At 6 months' follow-up of 42 patients with successful management, recurrent angina pectoris due to restenosis occurred in 10 patients (24%), and a late MI occurred in one patient (3%). At 6 months' follow-up of 56 survivors with unsuccessful management (development of MI or need for emergency bypass surgery), recurrent angina occurred in nine patients (16%), and cardiac death in two patients (4%). However, the majority of patients in both groups were either symptom free or had mild angina pectoris.**

*Conclusion.* **Acute coronary artery occlusion during PTCA is often unpredictable, but its frequency is higher in patients with unstable angina, multivessel disease, and complex lesions. Despite immediate redilatation, use of intracoronary streptokinase, and emergency bypass surgery, PTCA is associated with a high mortality and morbidity. (*Circulation* 1991;83:927–936)**

Percutaneous transluminal coronary angioplasty (PTCA) still carries an inherent risk of acute coronary artery occlusion during and after angioplasty despite increased operator experience and technological advancements.[1] The reported incidence of acute coronary artery occlusion varies, depending on the definition, from 2% to 11%.[2–10] It is the major cause of inhospital PTCA morbidity and mortality, and it restricts application of this technique to centers where surgical standby can be provided.[2–10] If in certain patients acute coronary artery occlusion is refractory to repeated balloon dilatation and pharmacological therapy, emergency coronary artery bypass surgery is traditionally recom-

From the Catheterization Laboratory, Thoraxcenter, Erasmus University, Rotterdam, The Netherlands.

Address for correspondence: P.J. de Feyter, MD, Catheterization Laboratory, Thoraxcenter, Erasmus University, PO Box 1738, 3000 DR Rotterdam, The Netherlands.

Received April 24, 1990; revision accepted November 6, 1990.

**TABLE 1. Clinical Characteristics and Angiographic Data of 104 Patients With Acute Coronary Artery Occlusion During Percutaneous Transluminal Coronary Angioplasty and 104 Patients Without Acute Coronary Artery Occlusion Randomly Selected for Comparison**

| | Coronary artery occlusion during PTCA | | | |
|---|---|---|---|---|
| | With | | Without | |
| | *n* | % | *n* | % |
| Male | 79 | 68 | 74 | 71 |
| Mean age (range) (yr) | 56 | (31–78) | 57 | (38–76) |
| Stable angina | 38 | 37 | 69 | 66 |
| Unstable angina | 66 | 63 | 35 | 34 |
| Prior CABG | 6 | 6 | 7 | 7 |
| Previous MI | 55 | 53 | 43 | 41 |
| Multivessel disease | 56 | 54 | 36 | 35 |
| Chronic total occlusion | 10 | 10 | 7 | 7 |
| Supply collaterals | 14 | 14 | 9 | 9 |
| Complex lesion | 57 | 55 | 26 | 25 |
| Intracoronary thrombus | 27 | 26 | 7 | 7 |
| Attempts/vessel | | | | |
| LAD | 60 | 58 | 56 | 54 |
| RCA | 32 | 31 | 27 | 26 |
| Cx | 22 | 21 | 21 | 20 |
| Attempted number of lesions/patient | | | | |
| Single lesion | 74 | 71 | 76 | 73 |
| Multilesion (at least two lesions/vessel) | 21 | 20 | 20 | 19 |
| Multivessel | 9 | 9 | 8 | 8 |
| EF <45% | 7 | 7 | 5 | 5 |

*n*=104 for patients with and without coronary artery occlusion during PTCA. Comparison group (without) was used to analyze clinical and angiographic variables predictive for acute coronary artery occlusion.

PTCA, percutaneous transluminal coronary angioplasty; CABG, coronary artery bypass graft surgery; MI, myocardial infarction; LAD, left anterior descending coronary artery; RCA, right coronary artery; Cx, circumflex coronary artery; EF, ejection fraction.

mended for these patients to minimize myocardial damage.[11,12] However, even with emergency coronary artery bypass surgery, acute coronary artery occlusion is associated with a 3–6% mortality and up to a 50% frequency of periprocedural myocardial infarction.[13–18]

This retrospective study describes the frequency, clinical course, management, and long-term outcome of acute coronary artery occlusion during and after PTCA, and retrospectively, risk factors associated with acute coronary artery occlusion are identified.

## Methods

From January 1986 to December 1988, 1,629 consecutive patients underwent coronary angioplasty procedures at the Thoraxcenter, Rotterdam. One hundred forty procedures were performed in the setting of an acute myocardial infarction (typical ischemic chest pain lasting at least 30 minutes, onset of symptoms within 6 hours, and associated electrocardiographic changes), and 66 procedures were performed in saphenous vein bypass grafts. These patients were excluded from this analysis. The remaining 1,423 patients form the basis of this analysis. Of these, 104 (7.3%) patients met the criteria for acute coronary artery occlusion. Acute coronary artery occlusion was defined as clinical or electrocardiographic evidence of myocardial ischemia and a complete or critical reduction in coronary blood flow in the vessel previously dilated, leading to either emergency repeated PTCA, emergency bypass surgery, or myocardial infarction during or after PTCA. Angiographic criteria of critical reduction of antegrade coronary blood flow were defined according to the Thrombolysis in Myocardial Infarction (TIMI) trial[19] in which complete reduction in flow was related to TIMI grade 0 and critical reduction to TIMI grades 1 and 2. Clinical characteristics and angiographic data of patients with an acute coronary artery occlusion are listed in Table 1.

### *PTCA Procedure*

The basic method of angioplasty has been described previously.[18] Patients were kept on their prior medication including platelet inhibitors, β-adrenergic receptor blockers, calcium channel blockers, and nitrates; in addition, all patients received 10,000 units heparin intravenously and 250 mg aspirin intravenously as soon as both the pacing catheter and guiding catheter were in place. An additional 5,000 units heparin was given 1 hour later if angioplasty was not completed by that time.

Intracoronary nitroglycerin was administered before crossing the lesion and was repeated when the operator believed it necessary. Initial balloon infla-

tion pressure was 2.0 atm, with subsequent inflations ranging to 12 atm. Inflation was maintained according to electrocardiographic changes, degree of decrease in blood pressure, or induced pain. The average maximal inflation pressure was 9.1±1.7 atm (range, 6–12 atm). The average inflation duration in patients with initially successful PTCA during the study period was 130±40 seconds. Heparin was never neutralized by the administration of protamine sulfate. It was our policy to observe the patient within the catheterization laboratory for another 15 minutes when the stability of the result immediately after dilatation was in doubt, and such patients were kept on a continuous infusion of heparin in doses sufficient to maintain a partial thromboplastin time at 2–2.5 times control for another 12–24 hours. They were monitored at an intermediate care facility, and the catheters were removed thereafter. In all other cases, the patients were immediately transferred to an intermediate care facility, and the arterial and venous sheaths were left in the puncture site for 6–8 hours after angioplasty. A 12-lead electrocardiogram was obtained immediately after PTCA and daily until discharge. Myocardial enzyme release was monitored for 48 hours. After angioplasty, all patients without contraindications were placed on 60 mg nifedipine p.o. /24 hrs for 36–48 hours and on 500 mg aspirin once daily for 6 months.

### *Management of Acute Coronary Artery Occlusion*

Acute coronary artery occlusion was managed on an individual basis. In general, however, if the acute coronary artery occlusion occurred first, a bolus of intracoronary nitroglycerin was given, and if this was unsuccessful, an attempt was made to redilate the area of occlusion. Initially, a normal-sized balloon was used, and a longer duration of inflation was attempted, usually up to 5 minutes depending on the severity of angina, blood pressure, and electrocardiographic changes. If this was unsuccessful, an "oversized" balloon (0.5 mm larger than the reference area) was used, including a longer duration of inflation. The mean duration of inflation was 410±100 seconds in patients with acute coronary artery occlusion. The mean maximal inflation pressure was 11±1 atm. If this failed, patients were referred for emergency surgery; in patients with ongoing signs of severe ischemia and with suitable coronary anatomy,

**Table of Definitions**

| | |
|---|---|
| Major complications | 1) Death before hospital discharge, 2) nonfatal myocardial infarction, or 3) emergency surgery. |
| Emergency surgery | Need for urgent operation due to refractory acute coronary artery occlusion |
| Myocardial infarction | Presence of two of the three following criteria: 1) prolonged angina, 2) electrocardiographic evidence of infarction, 3) total creatine kinase elevation greater than twice the upper limit of normal values. |
| Unstable angina | Chest pain occurring at rest, accompanied by electrocardiographic ST segment or T wave changes and no evidence of subsequent myocardial necrosis. |
| Clinical success | Reduction of the luminal narrowing to less than 50% without major complication. |
| Multivessel angioplasty | Angioplasty in more than one major coronary artery system (i.e., left anterior descending, left circumflex, or right coronary artery). |
| Intimal dissection | Presehce of angiographically evident intimal damage producing an intraluminal filling defect, extraluminal extravasation of contrast material, linear luminal density, or luminal staining.[1] |
| Stenosis at branch point | Considered present when any part of the lesion was adjacent to a branch vessel of 25% diameter or more of the diameter of the nondiseased native vessel.[5] |
| Stenosis at bend point | Considered present when in any projection the balloon, in position to dilate, appeared to be located in a portion of the vessel that had a 45° or greater angulation at end diastole.[5] |
| Diffuse disease | Presence of luminal irregularities in a coronary artery extending well beyond the segment of the vessel to be dilated. |
| Calcifications | Presence of fixed radiopaque densities having the appearance of calcification noted in the area of the stenosis to be dilated.[5] |
| Collaterals | Presence of collaterals emanating beyond the site to be dilated. |
| Eccentricity | A stenosis asymmetrically positioned in the vessel in any angiographic projection. |
| Complex lesion | Eccentric lesion or a lesion with irregular borders. |
| Thrombus | Presence of a discrete filling defect or an area of contrast staining noted in the area of the stenosis to be dilated. |
| Multivessel disease | Luminal compromise of 50% or more diameter of more than one of the three major coronary arteries. |

an attempt was first made to place a translesional autoperfusion catheter to bridge the period until emergency surgery could be performed. Special care was taken to prevent clotting by repeated flushing of the perfusion catheter with heparin.

If initial redilation failed and if clot formation was judged to also have played a significant role in the cause of acute occlusion, intracoronary streptokinase was given in an attempt to resolve the clot. Intracoronary clot formation was thought to be present when 1) after an initial successful dilatation, without clear evidence of a dissection, the vessel gradually reoccluded, 2) the presence of an intraluminal central filling defect or lucency surrounded by contrast material was seen in multiple projections, 3) a persistence of contrast material remained in the lumen, or 4) embolization of intraluminal material was noted downstream. Patients were referred for emergency surgery when the combination of redilatation and intracoronary streptokinase failed and when ongoing ischemia persisted. If the patient's condition was hemodynamically unstable, an intra-aortic balloon pump was inserted before surgery. Once acute coronary artery occlusion had been resolved, patients were kept on a treatment regimen with continuous infusions of nitroglycerin and heparin for the following 6 hours.

*Follow-up*

All patients were followed up for at least 6 months. Repeated angiography was only performed in those patients with severe recurrent angina pectoris and a positive exercise test for ischemia (symptoms or ST segment changes).

*Data Collection and Entry*

Clinical information, angiographic, intra-PTCA and post-PTCA complication data, and specific laboratory data including electrocardiographic results and cardiac enzyme levels were recorded during hospitalization and prospectively entered into a computerized data base.

*Statistical Analysis*

One hundred four patients without acute coronary artery occlusion were randomly selected by date of angioplasty (to the nearest week) for comparison with those patients who had an acute coronary artery occlusion. The clinical data and cineangiogram of each patient in both groups were reviewed by two cardiologists unaware of the clinical outcome. The clinical and angiographic data of the comparison group are shown in Table 1.

Univariate and multivariate analyses were performed to determine clinical and angiographic predictors of major complications. Baseline data included age, gender, previous myocardial infarction, clinical history (stable or unstable angina), presence of multivessel disease, ejection fraction, site of lesion, length, eccentricity and severity of lesion, diffuse disease, more than one discrete lesion per vessel, complex lesion (eccentric and irregular borders), calcification, presence of intracoronary thrombus, collaterals to attempted vessel, stenosis at branch point, and stenosis at bend point (>45°). The conditional probability of acute coronary artery occlusion was estimated given a set of variables obtained by the use of a logistic regression function.[20]

## Results

The frequency of acute coronary artery occlusion, occurring at any time during hospitalization, was 7.3% (104 of 1,423 patients). Recurrent chest pain was present in 97% (101 of 104 patients), ST segment elevation was present in 77%, ST segment depression was present in 13%, no significant ST segment changes occurred in 10%, acute hypotension was present in 20%, and ventricular fibrillation occurred in 10% of the patients with acute coronary artery occlusion.

While still in the catheterization laboratory, acute coronary artery occlusion occurred in 80 patients (77%), within 6 hours after dilatation in 13 patients (13%), 6–12 hours after dilatation in five patients (5%), and 12–24 hours after dilatation in six patients (6%). No additional untoward events occurred between the period after 24 hours and hospital discharge.

*Management and Clinical Outcome*

In most patients (95 of 104), an attempt at redilation was performed with either an identical-sized balloon or a larger-sized balloon. Redilation was successful in 35% of the patients (33 of 95 patients) (Figure 1). Four patients were referred directly to emergency surgery without attempting repeated dilatation, two of whom had an uneventful recovery. No attempt to redilate the lesion was undertaken in another five patients, all of whom had acute coronary artery occlusion 2–24 hours after the initial successful dilatation; in two of these patients, progression to myocardial infarction was prevented by intensive medical treatment (nitroglycerin i.v. and streptokinase i.v.) (Figure 1).

A second episode of acute coronary artery occlusion occurred after an initially successful redilatation in only three patients. One of those patients died; one underwent emergency surgery but developed a myocardial infarction; and one recovered uneventfully.

The overall clinical and angiographic outcomes after acute coronary artery occlusion are shown in Table 2. Clinical success after acute coronary artery occlusion was achieved in 40% of the patients. The baseline characteristics of the patients who died or developed a myocardial infarction after acute coronary artery occlusion are shown in Table 3. Procedural death occurred in six patients (6%). Most fatal complications occurred in patients with unstable angina pectoris (five of six patients). Nonfatal myocardial infarction occurred in 37 patients (36%), and emergency surgery was performed in 30% of the patients. Most infarctions were non–Q wave, and based on subsequent enzyme release, most infarctions were small to medium sized.

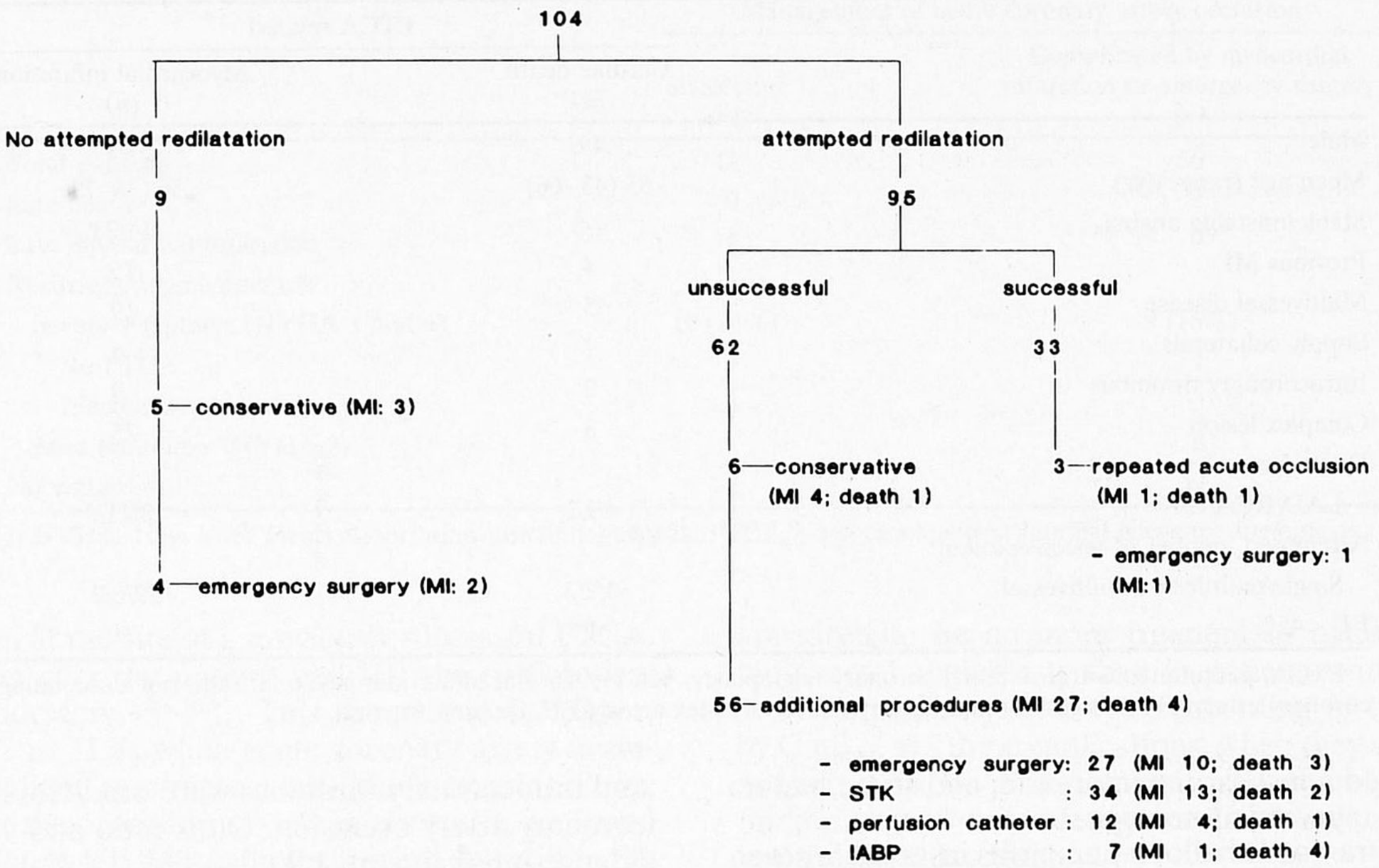


FIGURE 1. *Flow diagram of management and outcome of patients with acute coronary artery occlusion during and after percutaneous transluminal coronary angioplasty. MI, myocardial infarction; STK, intracoronary streptokinase; IABP, intra-aortic balloon pump counterpulsation.*

The outcome of patients in whom acute coronary artery occlusion occurred after leaving the catheterization laboratory was comparable to those with acute occlusion while still in the catheterization laboratory (Table 2).

Intracoronary streptokinase (dose: mean, 750,000 IU; range, 250,000–1.5 million IU) was subsequently used after failure of initial attempts at redilatation in 34 patients. Clinical success was achieved in 18 (53%), and failure with a major complication occurred in 16 (47%) of the patients (Table 4).

In 12 patients, a translesional autoperfusion catheter was placed, and subsequent stabilization of acute ischemia occurred in most of these (Figure 1). Despite emergency bypass surgery that was performed in all these patients, one patient died, four

**TABLE 2. Overall Clinical and Angiographic Outcome After Management of Acute Coronary Artery Occlusion of All Patients and of a Subset of Patients in Whom Acute Occlusion Occurred After Leaving the Catheterization Laboratory**

| | Patients overall | | Patients with acute occlusion after leaving the catheterization laboratory | |
|---|---|---|---|---|
| | *(n)* | (%) | *n* | % |
| Patients with event *(n)* | 104 | | 24 | |
| Success per patient (no death, MI, or CABG) | 42 | 40 | 12 | 50 |
| Angiographic success | 57 | 55 | 16 | |
| With nonfatal MI | 15 | 14 | 4 | |
| Failure without major complication (no death, MI, or CABG) | 4* | 4 | 0 | |
| Failure with major complication | 58 | 56 | 12 | 50 |
| Death (with CABG) | 6 (3) | 6 | 2 (1) | 8 |
| Nonfatal MI (with CABG) | 37 (13) | 36 | 7 (2) | 29 |
| Emergency CABG | 31 | 30 | 6 | 25 |

*All four patients underwent elective bypass surgery.
MI, myocardial infarction; CABG, coronary artery bypass graft surgery.

**TABLE 3. Baseline Characteristics of Patients With Percutaneous Transluminal Coronary Angiography Who Developed Periprocedural Acute Occlusion That Resulted in Myocardial Infarction or in Cardiac Death**

| | PTCA related | |
|---|---|---|
| | Cardiac death *(n)* | Myocardial infarction *(n)* |
| Male | 4/6 | 31/37 |
| Mean age (range)(yr) | 55 (43–66) | 56 (38–78) |
| Stable/unstable angina | 1/5 | 14/23 |
| Previous MI | 4 | 15 |
| Multivessel disease | 5 | 19 |
| Supply collaterals | 2 | 6 |
| Intracoronary thrombus | 2 | 9 |
| Complex lesion | 3 | 25 |
| Dilated vessel | | |
| LAD/RCA/Cx | 4/1/1 | 23/11/3 |
| Attempted number of lesions/patient: | | |
| Single/multilesion/multivessel | 2/2/2 | 29/6/2 |
| EF <45% | 1 | 4 |

PTCA, percutaneous transluminal coronary angioplasty; MI, myocardial infarction; LAD, left anterior descending coronary artery; RCA, right coronary artery; Cx, circumflex artery; EF, ejection fraction.

developed a myocardial infarction, and seven had no signs of myocardial necrosis.

An intra-aortic balloon pump was inserted in seven patients in an attempt to stabilize hemodynamics. Two patients died before attempted surgery; two patients died after emergency surgery; one patient developed a myocardial infarction; and two patients had an uneventful recovery (Figure 1).

Emergency bypass surgery was performed in 31 patients (Table 5). Nevertheless, 13 of 31 patients (42%) had a Q wave myocardial infarction, and three patients (10%) died perioperatively.

The 6-month follow-up data of all hospital survivors are shown in Table 6. Recurrent angina pectoris due to restenosis occurred in 24% of the patients with an initially successful redilatation, and recurrent or persistent angina pectoris occurred in 16% of the patients who had a procedural complication.

### *Predictors of Acute Coronary Artery Occlusion*

Univariate analysis showed that the presence of multivessel disease, unstable angina, complex lesion, and intracoronary thrombus were predictors of acute coronary artery occlusion. Odds ratio and 95% confidence interval were 1.9 (1.1–3.3), 3.7 (2.1–6.6), 4.0 (2.1–7.5), and 4.8 (2.0–11.7), respectively. Logistic regression analysis showed that multivessel disease, unstable angina, and complex lesion were independent predictors with a relative risk of 1.8, 2.9, and 2.9, respectively. The probability of acute coronary artery occlusion, given a prevalence of 7% of acute coronary artery occlusion in a population, varied from 2% (no variable present) to 25% (all three variables present).

## Discussion

### *Frequency of Acute Coronary Artery Occlusion*

Despite the improvements in equipment and technique that have made it possible to cross and dilate more than 90% of the lesions attempted, acute coronary artery occlusion remains one of the most feared complications of coronary angioplasty. A frequency from 1% to 2% has been reported when acute coronary artery occlusion is defined as complete

**TABLE 4. Clinical and Angiographic Outcome of Additional Use of Intracoronary Streptokinase After Initial Failure to Redilate an Acute Coronary Artery Occlusion**

| | *n* | % |
|---|---|---|
| Total patients | 34 | 100 |
| Clinical success | | |
| (no death, MI, or CABG) | 18 | 53 |
| Angiographic success | 22 | 65 |
| With MI | 4 | 12 |
| Failure with major complication | 16 | 47 |
| Death (with CABG) | 2 (1) | 6 |
| Nonfatal MI (with CABG) | 13 (3) | 38 |
| Emergency CABG | 5 | 15 |

MI, myocardial infarction; CABG, coronary artery bypass graft surgery.

**TABLE 5. Clinical Outcome of Emergency Bypass Surgery After Refractory Acute Coronary Artery Occlusion**

| | All patients | Patients without streptokinase *(n)* |
|---|---|---|
| Total patients | 31 | 26 |
| Time interval failure to total perfusion (mean±SD) (min) | 56±28 | 52±30 |
| LIMA | 5 | 5 |
| Death | 0 | 0 |
| Nonfatal MI (Q wave) | 1 | 1 |
| Aortosaphenous graft | 26 | 21 |
| Death | 3 | 2 |
| Nonfatal MI (Q wave) | 12 | 9 |

LIMA, left internal mammary artery implantation; MI, myocardial infarction.

**TABLE 6. Six Months of Follow-up After Percutaneous Transluminal Coronary Angioplasty Complicated by Acute Coronary Artery Occlusion**

| | Management of acute coronary artery occlusion | |
|---|---|---|
| | Successful *(n)* | Complicated by myocardial infarction or emergency surgery *(n)* |
| Total patients | 42 | 56 |
| Late death | 0 | 2 |
| Late myocardial infarction | 1 | 0 |
| Recurrent angina pectoris | | |
| Severe symptoms (NYHA 3 and 4) | 10 (24%) | 9 (16%) |
| Re-PTCA | 7 | 4 |
| Elective surgery | 3 | 5 |
| Mild symptoms (NYHA 2) | 4 | 6 |
| No symptoms | 28 | 39 |

NYHA, New York Heart Association functional classes; PTCA, percutaneous transluminal coronary angioplasty.

occlusion at the site of a previously successful PTCA, occurring *after* the patient had left the catheterization laboratory.[2,4,6,10,21] This frequency is higher, from 4% to 11%, when acute coronary artery occlusion is defined as complete occlusion occurring *during* and *after* the procedure.[3,5,6–10] In our study, we found an incidence of 5.6% while the patient was still in the catheterization laboratory and of 1.7% after the patient had left the catheterization laboratory. These findings are similar to the acute occlusion rate reported by the 1985–1986 National Heart, Lung, and Blood Institute (NHLBI) PTCA Registry: 4.9% periprocedural occlusion in the catheterization laboratory and 1.9% outside the laboratory.[10]

*Risk Factors of Acute Coronary Artery Occlusion*

In a large study of 4,772 procedures, multivariate analysis of 16 clinical variables disclosed female gender as an independent factor, and multivariate analysis of 35 angiographic variables disclosed the following as independent factors: stenosis length of at least twice the luminal diameter, stenosis at a bend point of 45° or more, stenosis at a branch point, stenosis-associated thrombus, other stenosis in the same vessel, and multivessel disease.[5] The 1985–1986 NHLBI PTCA Registry study[10] analyzed 1,801 patients and disclosed that baseline patient factors independently associated with increased occlusion rates included triple-vessel disease, high-risk status for surgery, and acute coronary insufficiency and disclosed that lesion characteristics included diffuse or multiple discrete morphology, thrombus, and collateral flow from the lesion. In our study, unstable angina, multivessel disease, and complex lesions were independently associated with acute coronary occlusion.

From these three large studies, it appears that gender (female), symptomatic status (unstable angina) and severity, and extent and complexity of coronary artery lesions are important factors associated with risk of acute coronary artery occlusion. Unexpectedly, in our study and in that by Gaul et al,[7] the occurrence of acute coronary artery occlusion appeared to be no more frequent in patients with multivessel angioplasty than in patients with single-vessel angioplasty. However, according to the study by Gaul et al,[7] the complications, when they do occur, appeared to be more severe, and therefore, this finding should not infer generalized indication for multivessel dilatation.

Like Sugrue et al,[22] we confirmed that the presence of an angiographically detectable thrombus at the dilatation site increased the risk of acute occlusion. However, the presence of intracoronary thrombus is known to be associated with unstable angina,[23–25] and it was shown that, with a logistic regression model, the presence of intracoronary thrombus was not an independent variable to predict acute coronary artery occlusion.

In our study, we found that multivessel disease, unstable angina, and complex lesions were strong independent risk factors of acute coronary artery occlusion. The risk associated with the absence or the presence of one or more of these factors emphasizes 1) the low probability (2%) of acute coronary artery occlusion in patients without risk factors, 2) the additive effect of these risk factors, and 3) the probability of acute coronary artery occlusion in approximately 25% of patients when all three risk factors were present.

We did not attempt to analyze procedural variables for acute coronary artery occlusion because angiography is a poor method to definitely distinguish between the most important potential variables such as dissection, thrombus, and spasm. For obvious reasons, it would be important if one could predict the occurrence of acute coronary artery occlusion after initially successful PTCA after the patients leave the catheterization laboratory. The frequency of this untoward event is low (approximately 2%), and the small number of patients with this event in our study precluded meaningful analysis of predictive variables. However, in accordance with the 1985–1986 NHLBI PTCA Registry study,[10] an intimal tear and a "hazy" appearance of the segment were the

most frequent angiographic findings after PTCA associated with acute coronary artery occlusion.

*Management and Clinical Course of Acute Coronary Artery Occlusion*

Acute coronary artery occlusion results largely from plaque disruption and dissection, with coronary spasm, thrombosis, and expanding subintimal hematoma being secondary events. Although it is frequently not possible to definitely distinguish among coronary spasm, coronary thrombosis, and subintimal hemorrhage by angiographic criteria, arterial spasm is probably important because this is the common response of an artery to injury.[26] Intracoronary administration of nitroglycerin is the first step in the management of acute coronary artery occlusion.[27] The occurrence of acute coronary artery occlusion at a time of diminishing anticoagulation,[21] the fact that in a substantial number of patients additional treatment with streptokinase has been successful to manage acute coronary artery occlusion,[11] and the known tendency of clot formation in a vessel with slow flow, as occurs after extensive dissection, suggest that thrombus formation as a primary or secondary event may be an important mechanism. Addition of thrombolytic treatment to manage acute coronary artery occlusion is, therefore, an established option in patients with suspected thrombus formation.[11]

Inadequate response after nitroglycerin is followed by immediate repeated PTCA with the same balloon or an "oversized" balloon. Repeated dilatation will result in a success rate between 36% and 56%,[2–4,6,7,10,21] which is in keeping with our success rate of 36%. Patients refractory to repeated dilatation should be referred for emergency surgery. However, in individual patients, depending on the clinical condition, the angiographic findings, and the availability of immediate surgical standby, additional interventions or procedures such as insertion of an intra-aortic balloon pump,[28] administration of thrombolytic treatment,[11] or placement of an autoperfusion catheter[29,30] may be performed first.

Additional use of intracoronary streptokinase was successful in 50% of selected patients, whereas all failures were associated with either death (6%) or development of a myocardial infarction (38%). However, the observed high frequency of perioperative mortality (20%) and myocardial infarction (60%) may be due to the inevitable delay if this approach fails. Clearly, additional thrombolytic treatment is associated with substantial morbidity and mortality and cannot be considered an effective treatment. However, the alternative, immediate referral of patients to emergency surgery, is also associated with a high perioperative mortality rate (18%) and myocardial infarction rate (39%).

There appeared to be no difference in outcome of acute coronary artery occlusion whether it occurred while the patient was outside the catheterization laboratory or while the patient was in the catheterization laboratory. This is somewhat surprising because a poorer outcome is expected in patients outside the catheterization laboratory, primarily because of the time needed to mobilize the catheterization laboratory and because of difficulties in obtaining adequate redilatation.

The 6-month follow-up of patients with successful redilatation was satisfactory and is comparable to the reported 6-month follow-up results of patients after successful coronary angioplasty.[31–34] There also was no indication of excess mortality or morbidity during the 6-month follow-up of survivors with an unsuccessful attempt at redilatation after acute coronary artery occlusion.

*Limitations of This Study*

This study is a retrospective analysis of the incidence and management of acute coronary artery occlusion during PTCA from a consecutive series of patients undergoing PTCA from 1986 through 1988. There was no rigid, prospectively designed protocol to manage this complication, although in most patients, the initial step was the use of intracoronary vasodilators followed by redilatation of the occluded segment. If redilatation failed, the next steps were based on the judgement of the responsible operator regarding the symptomatic and hemodynamic status of the patient and on the characteristics of the coronary anatomy and the occluded segment and on the use of intra-aortic balloon pumping, perfusion catheter, thrombolytic treatment, or immediate referral for surgery.

Because the field of coronary angioplasty is rapidly changing, the findings of this study must be placed in the context of current clinical experience. Future application of intracoronary stents, laser balloon welding, and atherectomy may further increase the ability to salvage initially failed angioplasty and may diminish the need for emergency bypass surgery. It is not possible to make inferences from the present results on the current best course of treatment after periprocedural coronary occlusion.

*Conclusion*

Advances in catheter technology and increased operator experience have allowed a rapid growth in apparent indications and in the application of PTCA. Acute coronary artery occlusion after PTCA is a feared complication, which carries a high mortality and morbidity rate and which requires the need for the availability of emergency bypass surgery. As more patients with multivessel disease and diffuse disease undergo angioplasty, the problem of acute coronary artery occlusion increases. In considering a patient for angioplasty, one would like to be able to estimate the probability of acute coronary artery occlusion. The risk factors predictive of acute coronary artery occlusion after elective percutaneous transluminal coronary angioplasty include female gender, unstable angina pectoris, multivessel disease, and complex lesions. In the absence of any of these risk factors, the risk of acute coronary artery occlusion is small,

whereas if all these factors are present, the risk is substantial and may be such that alternative forms of revascularization should be considered.

Acute coronary artery occlusion should be managed initially by emergency repeated dilatation in most patients. The 6-month follow-up is satisfactory after successful redilatation. If this approach fails, even with immediate referral for emergency surgery and with support by intra-aortic balloon pump, autoperfusion catheter, or addition of thrombolytic treatment, the outcome is poor and is associated with a high mortality and morbidity rate.

## Acknowledgment

We thank Claudia Sprenger de Rover for manuscript preparation.

## References

1. Dorros G, Cowley MJ, Simpson J, Bentivoglio LG, Block PC, Bourassa M, Detre K, Gosselin AJ, Gruntzig AR, Kelsey SF, Kent KM, Mock MB, Mullin SM, Myler RK, Passamani ER: Percutaneous transluminal coronary angioplasty: Report of complications from the National Heart, Lung, and Blood Institute PTCA Registry. *Circulation* 1983;67:723–730
2. Hollman J, Gruentzig AR, Douglas JS Jr, King SB III, Ischinger T, Meier B: Acute occlusion after percutaneous transluminal coronary angioplasty—A new approach. *Circulation* 1983;68:725–732
3. Marquis JF, Schwartz L, Aldridge H, Majid P, Henderson M, Matushinsky E: Acute coronary artery occlusion during percutaneous transluminal coronary angioplasty treated by redilation of the occluded segment. *J Am Coll Cardiol* 1984;4: 1268–1271
4. Simpfendorfer C, Belardi J, Bellamy G, Galan K, Franco I, Hollman J: Frequency, management, and follow-up of patients with acute coronary occlusions after percutaneous transluminal coronary angioplasty. *Am J Cardiol* 1987;59:267–269
5. Ellis SG, Roubin GS, King SB III, Douglas JS, Weintraub WS, Thomas RG, Cox WR: Angiographic and clinical predictors of acute closure after native vessel coronary angioplasty. *Circulation* 1988;77:372–379
6. Sinclair JN, McGabe CH, Sipperly ME, Baim DS: Predictors, therapeutic options and long-term outcome of abrupt reclosure. *Am J Cardiol* 1988;61:61G–66G
7. Gaul G, Hollman J, Simpfendorfer C, Franco I: Acute occlusion in multiple lesion coronary angioplasty: Frequency and management. *J Am Coll Cardiol* 1989;13:283–288
8. Shiu MF, Silverton NP, Oakley D, Cumberland D: Acute coronary occlusion during percutaneous transluminal coronary angioplasty. *Br Heart J* 1985;54:129–133
9. Meyerovitz MF, Friedman PL, Ganz P, Selwyn AP, Levin DC: Acute occlusion developing during or immediately after percutaneous transluminal coronary angioplasty: Nonsurgical treatment. *Radiology* 1988;169:491–494
10. Detre KM, Holmes DR, Holubkov R, Cowley MJ, Bourassa MG, Faxon DP, Dorros GP, Bentivoglio LB, Kent KM, Myler RK, coinvestigators of the NHLBI PTCA Registry: Incidence and consequences of periprocedural occlusion: The 1985–1986 NHLBI PTCA Registry. *Circulation* 1990;82:739–750
11. Suryapranata H, de Feyter PJ, Serruys PW: Coronary angioplasty in patients with unstable angina pectoris: Is there a role for thrombolysis. *J Am Coll Cardiol* 1988;12:69A–77A
12. Bredlau CE, Roubin GS, Leimgruber PP, Douglas JS, King SB, Gruntzig AR: Inhospital morbidity and mortality in patients undergoing elective coronary angioplasty. *Circulation* 1985;72:1044–1052
13. Reul GJ, Cooley DA, Hallman GL, Duncan JM, Livesay JJ, Frazier OH, Ott DA, Angelini P, Massumi A, Mathur VS: Coronary artery bypass surgery for unsuccessful percutaneous transluminal coronary angioplasty. *J Thorac Cardiovasc Surg* 1984;88:685–694
14. Cowley MJ, Dorros G, Kelsey SH, Raden MV, Detre KM: Emergency coronary bypass surgery after coronary angioplasty: The NHLBI Institute's Percutaneous Transluminal Coronary Angioplasty Registry experience. *Am J Cardiol* 1984; 53:22C–26C
15. Killen DA, Hamaker WR, Reed WA: Coronary artery bypass following percutaneous transluminal coronary angioplasty. *Ann Thorac Surg* 1985;40:133–138
16. Golding LAR, Loop FD, Hollman JL, Franco I, Borsh J, Stewart RW, Lytle BW: Early results of emergency surgery after coronary angioplasty. *Circulation* 1986;74(suppl III): III-26–III-29
17. Talley JD, Jones EL, Weintraub WS, King S: Coronary artery bypass surgery after failed percutaneous transluminal coronary angioplasty. *Circulation* 1989;79(suppl I):I-126–I-131
18. de Feyter PJ, Serruys PW, van den Brand M: Emergency coronary angioplasty in refractory unstable angina. *N Engl J Med* 1985;313:342–347
19. TIMI Study Group: The Thrombolysis in Myocardial Infarction Trial. *N Engl J Med* 1985;312:932–936
20. Maisels MJ, Gifford K, Antle CE, Leib GR: Jaundice in the healthy new born infant: A new approach to an old problem. *Pediatrics* 1988;81:505–511
21. Gabliani G, Deligonul U, Kern MJ, Vandormael M: Acute coronary occlusion occurring after successful percutaneous transluminal coronary angioplasty: Temporal relationship to discontinuation of anticoagulation. *Am Heart J* 1988;116:696–700
22. Sugrue DD, Holmes DR, Smith HC, Reeder GS, Lane GE, Vlietstra RE, Bresnahan JF, Hammes LN, Pichler JM: Coronary artery thrombus as a risk factor for acute vessel occlusion during percutaneous transluminal coronary angioplasty: Improving results. *Br Heart J* 1986;56:62–67
23. Davies MJ, Thomas AC: Plaque fissuring: The cause of acute myocardial infarction, sudden ischaemic death, and crescendo angina. *Br Heart J* 1985;53:363–373
24. Falk E: Unstable angina with fatal outcome, dynamic coronary thrombosis leading to infarction and/or sudden death: Autopsy evidence of recurrent mural thrombosis with peripheral embolization culminating in total vascular occlusion. *Circulation* 1985;71:699–708
25. Fuster V, Badimon L, Cohen M, Ambrose JA, Badimon JJ, Chesebro JH: Insights into the pathogenesis of acute ischemic syndromes. *Circulation* 1988;77:1213–1220
26. Fischell TA, Derby G, Tse TM, Stadius ML: Coronary artery vasoconstriction routinely occurs after percutaneous transluminal coronary angioplasty: A quantitative arteriographic analysis. *Circulation* 1988;78:1323–1334
27. Topol EJ: Emergency strategies for failed percutaneous transluminal coronary angioplasties. *Am J Cardiol* 1989;63:249–250
28. Murphy DA, Craver JM, Jones EL, Curling PE, Guyton RA, King SB, Gruntzig AR, Hatcher CR: Surgical management of acute myocardial ischemia following percutaneous transluminal coronary angioplasty: Role of the intra-aortic balloon pump. *J Thorac Cardiovasc Surg* 1984;87:332–339
29. Ferguson TB, Hinohora T, Simpson J, Stack R, Wechsler AS: Catheter reperfusion to allow optimal coronary bypass grafting following failed transluminal angioplasty. *Ann Thorac Surg* 1986;42:399–405
30. Sundram P, Harvey JR, Johnson RG, Schwartz MJ, Baim DS: Benefit of the perfusion catheter for emergency coronary artery grafting after failed percutaneous transluminal coronary angioplasty. *Am J Cardiol* 1989;63:282–285
31. Gruentzig AR, King SB, Schlumpf M, Siegenthaler W: Long-term follow-up after percutaneous transluminal coronary angioplasty: The early Zurich experience. *N Engl J Med* 1987; 316:1127–1132
32. Rosing DR, Cannon RO, Watson RM, Bonow RO, Mincemoyer R, Ewels C, Leon MB, Lakatos E, Epstein SE, Kent KM: Three year anatomic, functional, and clinical follow up after successful percutaneous transluminal coronary angioplasty. *J Am Coll Cardiol* 1987;9:1–7

33. Kamp O, Beatt KJ, de Feyter PJ, van den Brand M, Suryapranata H, Luijten HE: Short-, medium-, and long-term follow-up after percutaneous transluminal coronary angioplasty for stable and unstable angina pectoris. *Am Heart J* 1989;117:991–996
34. Serruys PW, Luijten HE, Beatt KJ, Geuskens R, de Feyter PJ, van den Brand M, Reiber JHC, ten Katen HJ, van Es GA, Hugenholtz PG: Incidence of restenosis after successful coronary angioplasty: A time-related phenomenon: A quantitative angiographic study in 342 consecutive patients at 1, 2, 3, and 4 months. *Circulation* 1988;77:361–371

KEY WORDS • percutaneous transluminal coronary angioplasty • occlusion, acute coronary artery • abrupt occlusion syndrome