Acute coronary occlusion after percutaneous transluminal coronary angioplasty

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■ Acute coronary occlusion following successful percutaneous transluminal coronary angioplasty (PTCA) remains a significant problem and is responsible for a significant percentage of PTCA-related morbidity and mortality. The reported incidence varies from 2.1% to 12.2%. The time of occurrence in most cases is within three hours of angioplasty. Intimal tears and intracoronary thrombus are important angiographic predisposing factors. Redilation appears to be an effective means of managing this complication in most patients.
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PERCUTANEOUS transluminal coronary angioplasty (PTCA) has become an effective alternative to medical treatment or coronary bypass surgery in selected patients with obstructive coronary artery disease. Although technological advances and operator experience have led to improved results, the procedure is associated with short-term complications in approximately 5% of patients.[1] Of these complications, acute closure of the dilated vessel is the main concern. This report reviews the incidence of acute occlusion, predisposing factors, associated morbidity, and treatment.

INCIDENCE

When acute occlusion is defined as angiographic evidence of a complete occlusion at the site of previously successful PTCA, the reported incidence varies from 2.1% to 12.2%, with an average of 3.6% (Table 1).[2–11] The range is this wide because some studies include only acute occlusions occurring after the patient has left the catheterization laboratory, while other groups reporting higher incidences have included coronary occlusions during PTCA. The incidence of this problem may be underestimated because occlusions may develop without signs of myocardial infarction in patients with good collateral supply.

CLINICAL PRESENTATION

Approximately half of the cases of acute occlusion occur while the patient is still in the cardiac laboratory (Table 2), and about 80% within three hours after angioplasty (Table 3).

The clinical presentation in most instances is typical, with a well-defined onset of chest pain associated with ST segment elevation. In the series of Bredlau et al,[6] 82% of patients presented this way, while in our series,[8] 94% of patients did so.

PREDISPOSING FACTORS

A number of clinical and angiographic predisposing factors have been identified. These are: presence of

TABLE 1
INCIDENCE OF ACUTE OCCLUSION FOLLOWING PTCA

Series	No.	%
Hollman et al[2]	20/935	2.1
Marquis et al[3]	18/164	11.0
Cowley et al[4]	151/3079	2.2
Mabin et al[5]	29/238	12.2
Bredlau et al[6]	89/4000	2.2
Shiu et al[7]	20/240	8.3
Goldbaum et al[8]	25/917	2.7
Simpfendorfer et al[9]	32/1500	2.0
Stolz et al [10]	25/240	10.4
Gaul et al[11]	22/714	3.1
TOTAL	431/12017	3.6

TABLE 2
TIME OF OCCURRENCE OF ACUTE OCCLUSION

	No.	During PTCA	After PTCA
Hollman et al[2]	20	4	16
Goldbaum et al[8]	25	20	5
Stolz et al[10]	25	15	10
Gaul et al[11]	22	15	7
TOTAL	92	54 (59%)	38 (41%)

TABLE 3
TIME OF OCCURRENCE OF ACUTE OCCLUSION AFTER PTCA

	Within 3 hours	Latest episode
Hollman et al[2]	14/20 (70%)	56 hr
Simpfendorfer et al[9]	25/32 (78%)	72 hr
Gaul et al[11]	17/22 (77%)	24 hr

intimal tear,[2,7,9,10] intracoronary thrombus,[5,8] eccentric lesions,[4] lesions on bends,[12] multivessel angioplasty,[12] coronary spasm,[2,7] episodes of hypotension following PTCA,[2,11] female sex,[4,12] dilatations of the right coronary artery,[12] and history of unstable angina.[8]

Proposed mechanisms of acute closure include occlusive intimal tears, coronary thrombosis, and coronary spasm. Many investigators[2,3,7,9,10] have found an intimal tear to be the most significant angiographic finding associated with acute occlusion after PTCA. This finding and the lack of response to intracoronary administration of nitroglycerin suggests that the mechanism of acute occlusion is probably a progressively occlusive intimal dissection rather than coronary artery spasm. Others[5,7,8] have found a correlation between intracoronary filling defects and acute coronary occlusion. Coronary thrombosis is an important element in the cascade leading to coronary occlusion. It may represent a secondary event that contributes to the coronary occlusion when superimposed on occlusive intimal disruption.

ASSOCIATED MORBIDITY AND MORTALITY

The importance of acute occlusion as a complication of PTCA cannot be overemphasized. In the initial NHLBI Registry,[4] acute occlusion accounted for 53% of all emergency bypass surgery, 36% of all acute myocardial infarctions, and 28% of all PTCA-related deaths. The incidence of emergency bypass surgery, acute myocardial infarction, and mortality associated with acute occlusion reported in seven series is depicted in *Table 4*. On the average, emergency coronary bypass surgery was required in 56% of patients with acute occlusion, Q-wave evidence of a new myocardial infarction developed in 35% of them, and the mortality rate was 4.5%.

MANAGEMENT

In patients considered to be at high risk for acute occlusion, i.e., those having intracoronary thrombus or large intimal tears following angioplasty, it is advisable to leave the arterial sheath in place for a minimum of six hours for rapid access, since most coronary occlusions occur during this period. Although there are no data indicating that heparin will prevent or reduce the incidence of acute occlusion, empirically most centers treat high-risk patients with continuous heparin infusion for 24 hours. In the only randomized trial using a continuous heparin infusion for 18 to 24 hours, Ellis et al[13] found a 2.2% incidence of acute closure in the heparin-treated group compared with 2.8% in the control group. Patients with large dissections were excluded from this study.

Once coronary occlusion occurs, a decision must be made regarding surgical or nonsurgical treatment. In all cases arrangements should be made for surgical support. Patients considered to be at high risk for repeat PTCA or those patients in whom PTCA seems unlikely to be successful (technically difficult initial PTCA and those with poor left ventricular function) should be sent directly to surgery.

All other patients are best managed by repeat PTCA. Since the report by Hollman et al[2] describing treatment of this complication by repeat PTCA, this approach has been used with increasing frequency. *Table 5* shows in chronological order seven series reporting patients with acute occlusion. In these series, an average of 62% were

TABLE 4
IMPACT OF ACUTE OCCLUSION AFTER PTCA ON COMPLICATION RATES

Series	No.	Emergency CABG	Acute MI	Deaths
Hollman et al[2]	20	5 (25%)	5 (25%)	0 (0%)
Marquis et al[3]	18	11 (61%)	0 (0%)	0 (0%)
Cowley et al[4]	151	108 (72%)	62 (41%)	8 (5.3%)
Shiu et al[7]	20	14 (70%)	5 (25%)	2 (10%)
Simpfendorfer et al[9]	32	13 (41%)	14 (44%)	0 (0%)
Stolz et al[10]	25	5 (20%)	9 (36%)	3 (12%)
Gaul et al[11]	22	6 (27%)	6 (27%)	0 (0%)
TOTAL		56%	35%	4.5%

CABG = coronary artery bypass graft; MI = myocardial infarction

TABLE 5
TREATMENT OF ACUTE OCCLUSION WITH REPEAT PTCA

Series	Acute occlusion	Repeat PTCA	Primary success	Discharged without CABG
Hollman et al[2]	20	11 (55%)	8 (73%)	6 (55%)
Marquis et al[3]	18	7 (39%)	7 (100%)	7 (100%)
Bredlau et al[6]	89	42 (47%)	38 (90%)	31 (74%)
Shiu et al[7]	20	10 (50%)	6 (60%)	5 (50%)
Goldbaum et al[8]	25	17 (68%)	15 (88%)	—
Simpfendorfer et al[9]	32	31 (97%)	27 (87%)	18 (58%)
Gaul et al[11]	22	22 (100%)	21 (95%)	16 (73%)
TOTAL		62%	87%	67%

CABG = coronary artery bypass graft

treated initially by repeat angioplasty. The primary success of this approach has varied from 60% to 100% with a mean of 87%. Approximately one third of patients in whom redilation is successful will eventually require emergency bypass surgery because of a tendency to reocclusion.[9] If we take this into account, we can see that of patients for whom repeat PTCA is chosen to treat complete occlusion, 67% will be discharged from the hospital with no other intervention.

Underscoring the importance of thrombosis in the cascade leading to coronary occlusion, there are reports showing resolution of acute occlusion by intracoronary administration of thrombolytic agents[14] or bolus of intravenous heparin.[15] The application of intracoronary stents to treat acute occlusion is presently under investigation.[16]

CONCLUSION

Acute closure of the dilated vessel during or after PTCA is a serious but, fortunately, infrequent complication. The presence of an intimal tear after PTCA appears to be the most frequent predisposing factor. Although optimal treatment of patients with acute occlusion remains to be determined, based on accumulated experience, redilation appears to be a fast and highly successful means of managing this complication.

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