Intracoronary stents were implanted in 15 patients (pts) after unsuccessful percutaneous transluminal coronary angioplasty (PTCA) in the setting of acute myocardial infarction (AMI). The stented vessel was the left anterior descending (LAD) in II pts, the right coronary artery (RCA) in 3 pts, and a venous bypass graft to the LAD in a single pt. A total of 16 stents were implanted (15 Palmaz-Schatz, Johnson and Johnson; and 1 Wiktor, Medtronic). During follow-up, 1 pt died 10 days after stent implantation as a result of renal failure and cardiogenic shock. Subacute thrombosis occurred in 2 pts, 5 and 15 days after stent implantation; both underwent successful emergency coronary artery bypass grafting (CABG). The remaining 12 pts were free from major ischemic events (death, AMI, and further revascularization) after a mean follow-up of 18.7 ± 4.1 mo. We conclude that long-term results of intracoronary stenting in AMI after failed PTCA are favorable.

ABRUPT OR THREATENED vessel closure is a serious complication of percutaneous transluminal coronary angioplasty (PTCA). It is usually caused by dissection or elastic recoil of the vessel. Intracoronary stenting, because of its scaffolding effect, has become an accepted technique to seal dissections and prevent as well as reverse most cases of abrupt vessel closure. Because stent thrombosis still remains a potential complication of elective coronary setting, it seems reasonable to avoid stent implantation in the setting of acute myocardial infarction (AMI).

However, when infarct vessel angioplasty is complicated by extensive arterial dissection with threatened or abrupt vessel closure, stenting may be the only means of restoring vessel patency in spite of the thrombogenic environment. We studied retrospectively the early and long-term results of coronary stenting in the setting of AMI after failed PTCA.

Methods

The study population consisted of all patients who received intracoronary stents after unsuccessful PTCA in AMI between January 1991 and January 1995. The clinical and procedural data were obtained by review of the hospital and outpatient medical records. All patients received 80 mg of aspirin on admission except for a single patient who received aspirin after the procedure. Heparin 10,000 U intravenously was given as a bolus at the beginning and 5,000 U at the end of the procedure. Low-molecular weight dextran 40 (500 mL) intravenous infusion was started during the procedure after a decision was made to implant a stent. After the procedure, femoral sheaths were removed when the activated partial thromboplastin time (APTf) was < 80 s. Heparin intravenously was restarted 2h after sheath removal in a dose of 1,000 units/h in order to maintain APTf between 80 to 120 s. Coumarin and dipyridamol 2 x 150 mg were started on the day of the procedure. Heparin was continued until a therapeutic level of coumarin was achieved (international normalized ratio [INR] > 3). Coumarin and dipyridamol were given for 3 mo. Aspirin 300 mg daily was given for 3 mo and 80 mg daily thereafter indefinitely.
Angiographic Review

All angiograms were analyzed for baseline-lesion characteristics of the culprit vessel including the presence or absence of intraluminal thrombus, dissection, and TIMI (thrombolysis in myocardial infarction) perfusion grade. The same characteristics were also evaluated after PTCA and immediately after stent implantation. Computerized quantitative coronary angiographic measurements (using the cardiovascular measurement system, Medis, Nuenen, The Netherlands) were used to determine the reference vessel diameter, minimal luminal diameter (MLD), and the percentage diameter stenosis before and after PTCA and immediately after stent implantation.

Baseline Characteristics

Fifteen patients (mean age, 57.5 ± 2.6 years), consisting of 14 males and 1 female, received 16 coronary stents in the setting of AMI. The indication for PTCA was primary in 5 patients, whereas in 10 patients angioplasty was performed because of failed reperfusion with ongoing or recurrent myocardial ischemia after intravenous thrombolysis (rescue PTCA). Indication for stent implantation was abrupt vessel closure in 4 patients and threatened closure in 11 patients (ten with extensive dissection and one with elastic recoil).

Fourteen patients received 15 Palmaz-Schatz stents (Johnson and Johnson Interventional Systems, Warren, New Jersey) and 1 patient received a Wiktor stent (Medtronic Inc., Minneapolis, Minnesota). Thirteen patients received the stent on the same day of infarction (< 24 h) and 2 patients on the second day (< 48 h). The location of the infarction was anterior in 12 patients, inferior in 2, and inferoposterior with right ventricular infarction in 1 patient. Two patients were in cardiogenic shock. Nine patients had single-vessel disease, 4 patients had two-vessel disease, and 2 patients had three-vessel disease. The culprit vessel was the left anterior descending artery (LAD) in II, the right coronary artery in 3, and a venous bypass graft to the LAD in 1 patient. Ten patients received intravenous thrombolytic therapy prior to PTCA - streptokinase in 8 patients, streptokinase and tissue plasminogen activator in 1 patient, and anisoylated streptokinase plasminogen activator complex (APSAC) in 1 patient. Three patients received intracoronary streptokinase before PTCA (two received an extra bolus of intracoronary streptokinase after PTCA because of acute closure). Two patients did not receive thrombolytic therapy at all.

The angiographic diameter stenosis of the initial lesion ranged between 70% to 100% (mean, 93.7 ± 2.87%) and MLD was 0.21 ± 0.09 mm. Intraluminal filling defect suggestive of thrombus was present in 13 patients.

Immediate Angiographic Results

Successful stent deployment was achieved in all patients; 2 stents were required in 1 patient to cover a long spiral dissection after PTCA. The mean balloon size used to expand the stents was 3.5 ± 0.084 mm and the mean inflation pressure was 10.13 ± 0.49 atmospheres. The MLD increased from 1.71 ± 0.14 mm after PTCA to 3.17 ± 0.12 mm after stenting, and the percentage diameter stenosis decreased from 51.2 ± 3.48% to 15.2 ± 1.76%. All vessels had TIMI 3 flow (Table 1).

After stent deployment haziness in the stent was seen in 1 patient. and residual uncovered dissection was present in 3 patients (proximal to the stent in two and distal in one).

Inhospital Events

One patient died 10 days after stent implantation. He suffered from an old inferior myocardial infarction and severely impaired left ventricular function before presenting with a recent anterior myocardial infarction. He developed signs of left

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<th>Table I. Angiographic results.</th>
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<td>Before</td>
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<td>RD (mm)</td>
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<td>MLD (mm)</td>
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<td>DS (%)</td>
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<td>TIMI flow 0</td>
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<td>TIMI flow 1-2</td>
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<td>TIMI flow 3</td>
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<td>Visible thrombus</td>
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<td>Dissection</td>
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*Values are expressed as mean ± standard deviation.

PTCA = percutaneous transluminal coronary angioplasty; RD = reference vessel diameter; MLD = minimal lumen diameter; DS (%) = percentage diameter stenosis; TIMI = thrombolysis in myocardial infarction.
ventricular failure 5 h after stent implantation followed by severe hemoptysis, and hypotension occurred 3 days later that necessitated the stoppage of the anticoagulation regimen. The course was further complicated by renal failure, followed by cardiogenic shock and death. Two patients suffered subacute thrombosis 5 and 15 days after stent implantation. One belonged to the threatened closure group with two tandem stents, and residual uncovered proximal dissection developed subacute thrombosis despite adequate anticoagulation. The other patient belonged to the acute closure group. He received a stent in a venous graft with angiographic evidence of intraluminal thrombus before and after stent implantation. Anticoagulation level was subtherapeutic on two occasions after stent implantation. These 2 patients underwent successful emergency coronary artery bypass surgery (CABO) with signs of reinfarction. Femoral hematoma occurred in 2 patients, one required blood transfusion due to hypovolemic shock.

**Follow-up**

At a mean follow-up of 18.7 ± 4.1 mo, all 12 patients who were successfully discharged from the hospital without complications after intracoronary stenting were free from major ischemic events (death, AMI, and further revascularization).

Although II patients had no anginal complaints, 1 patient suffered from mild effort angina (Canadian Cardiovascular Society Class I). Nine patients underwent stress testing on a bicycle ergometer during follow-up without evidence of myocardial ischemia. Two patients underwent repeat coronary angiography 4 and 18 mo after stenting without angiographic evidence of restenosis (residual stenosis was less than 35%).

**Discussion**

Primary angioplasty has been shown to be an effective reperfusion strategy in patients with AMI. It is superior to thrombolysis as regards to clinical outcome and C0St.6,7 It is also the preferred approach in patients with a contraindication to thrombolysis, at high risk of mortality, or in cardiogenic shock.6,8,9

Despite the improved clinical outcome with thrombolytic therapy, multiple inherent limitations remain. Following thrombolytic therapy, angiographic studies have demonstrated patency of the infarct-related artery in 60% to 80% of patients.10,11 When thrombolysis is achieved a residual high-grade stenosis remains in 70% to 80% of patients, placing them at high risk for recurrent ischemia and reinfarction and resulting in blunted myocardial recovery.12,13 When thrombolytic therapy is unsuccessful in recanalizing the infarct-related artery, myocardial damage and inhospital mortality are both increased.14 In an attempt to improve the outcome of patients after failed thrombolysis, rescue PTCA has been performed with a procedural success rate exceeding 90% with a significant reduction of mortality.6

However, despite the high success rate of angioplasty in restoring vessel patency, inhospital reocclusion rate occurs in up to 18% and mortality rate rises up to 39% if PTCA failed.15 This means that persistent patency of the infarct-related artery is very important in limiting infarct size, reducing left ventricular remodeling, preserving left ventricular function, and subsequently improving patient survival.16

Coronary stenting has become an accepted modality for the management of threatened and abrupt vessel closure after PTCA.15 Stenting is the only intervention that can prevent vessel collapse and tacks back the intimal and medial tears, sealing the subintimal thrombogenic space. Stent thrombosis, however, is still a major problem after stent implantation even in the elective situation in vessels with no evidence of intraluminal thrombus4,17 (incidence, 5% to 8.7%).

Angiographic evidence of thrombus has been indentified as one of the predictors of stent thrombosis, along with small vessel size and residual uncovered dissection after stent implantation. The reports of stent implantation to seal extensive coronary dissection in the setting of AMI are few. Wong et al18 described 3 cases of stent implantation after acute occlusive dissection occurred during direct infarct coronary angioplasty with good immediate and long-term results. Iyer et al19 studied 46 patients who received flexible coil stents to stabilize abrupt or threatened vessel closure complicating PTCA performed 0 to 15 days following AMI. Five patients suffered inhospital stent thrombosis, six suffered AMI, and four died. In our group, 2 patients suffered subacute stent thrombosis which might have been prevented by properly covering the dissection in the first case and
adequate anticoagulation regimen in the second case. The only patient who died (aged 74 years) had poor left ventricular function which indicates a predictor of procedural-related mortality in angioplasty.20 Death, however, was related to stent thrombosis.

The other limiting factor of PTCA is the high incidence of restenosis rate between 30% to 40% in elective situations7 which increase to 45% after primary angioplasty in AMI. Since the process of restenosis includes elastic recoil, neointimal hyperplasia, and vessel remodeling, coronary stenting might influence all these factors, especially elastic recoil.4,5 It is clear that intracoronary stenting reduces the angiographic restenosis rate by creating a larger vessel lumen which may accommodate subsequent neointimal hyperplasia. A number of large trials using quantitative angiography have shown that stent implantation indeed results in a significant further improvement in stenosis geometry and consequently reduces restenosis rate as compared to PTCA.5,10 In our series, there was a significant increase in the MLD after stent implantation. This is reflected in the favorable clinical follow-up. At a mean follow-up period of 18.7 ± 4.1 mo, all patients who underwent successful stent implantation had event-free survival without evidence of clinical restenosis.

Conclusion

Coronary stenting in the setting of failed angioplasty for AMI restored vessel patency and myocardial perfusion. Despite the highly thrombogenic nature of the lesions, the long-term results were favorable. Recent development of heparin-bonded stents, local drug delivery systems, as well as newer anticoagulation regimens should further diminish the risks of stent thrombosis in patients with AMI.

References