Original Studies

Primary Stenting in Acute Myocardial Infarction:
A 30-Day Follow Up Study

Upendra Kaul,[1]* MD, DM, **Balbir Singh,**[1] MD, DM, **Dinesh Sudan,**[1] MD, DM, **Rakesh Sapra,**[1] MD, DM,
Ram Dev Yadav,[1] MD, DM, **Tapan Ghose,**[1] MD, DNB, and **N.S. Dixit,**[1] MD, FRCP

Primary coronary stenting is being increasingly used in patients undergoing primary coronary angioplasty for acute myocardial infarction. In this prospective study we evaluated our experience of direct angioplasty in 68 patients with acute myocardial infarction of whom 57 received intracoronary stents using high-pressure deployment ($\geq$12 atmospheres) with adjunct aspirin and ticlopidine therapy without coumadin. All patients underwent pre-discharge follow-up angiography. Stent implantation was successful in all patients. Stent thrombosis was not seen in any patient. However, TIMI grade 3 flow was obtained in only 51 patients (89.6%) with evidence of slow flow present in remaining six patients. Follow-up angiograms showed no stent thrombosis but five out of the six patients (83%) with slow-flow phenomenon persisted to have slow flow. These patients had lower left ventricular ejection fraction as compared to patients with TIMI 3 flow at follow-up angiography (27.5 $\pm$ 10.2% vs. 42.1 $\pm$ 15.2%, $P <$ .001) and a high mortality (two out of six) within 30 days. Primary stenting is safe and feasible in the majority of patients with good short-term outcomes, but persistent slow-flow phenomenon with adverse clinical outcome is seen in a small but significant number of patients. *Cathet. Cardiovasc. Intervent. 46:4–10, 1999.*   © 1999 Wiley-Liss, Inc.

Key words: stenting; slow-flow phenomenon. primary coronary angioplasty

INTRODUCTION

Although intravenous thrombolysis is widely used as a standard therapy for patients presenting with acute myocardial infarction (AMI), primary percutaneous transluminal coronary angioplasty (PTCA) is being used with increasing frequency in this situation [1–5]. Abrupt closure and threatening vessel occlusion complicates PTCA in 2–10% of patients in this setting [5,6]. Primary stenting in AMI has been shown to be feasible with gratifying short-term result in recent studies [7–9]. In this report we present our experience of primary stenting in managing AMI.

MATERIALS AND METHODS

The following patient entry criteria were used. Consecutive patients of any age with chest pain $\leq$12 hours in duration with electrocardiographic change of ST-segment elevation in $\geq$2 contiguous leads were included. Patients with previous bypass surgery and those in cardiogenic shock (systolic blood pressure <80 mm Hg for >30 min not responsive to fluids) were excluded. Patients were also excluded if they had previously received thrombolytic therapy during the same period.

Study Protocol

Patients were treated in the emergency room with 325 mg of chewable aspirin, 500 mg of oral ticlopidine, and 5,000-unit bolus of intravenous heparin. Patients were then transferred immediately to the cardiac catheterization laboratory, and coronary arteriography was performed. Ionic contrast medium (Diatrizoate-Meglumine and Diatrizoate-Sodium Injection) was used in all the instances. PTCA was then performed if appropriate. Medical treatment was instituted if thrombolysis in

[1]Department of Interventional Cardiology, Batra Heart Centre, Batra Hospital & Medical Research Centre, New Delhi, India.

*Correspondence to: Prof. Upendra Kaul, Director Interventional Cardiology & Cardiac Electrophysiology, Batra Hospital & Medical Research Centre, 1, Tughlakabad Institutional Area, Mehrauli Badarpur Road, New Delhi-110 062, India.
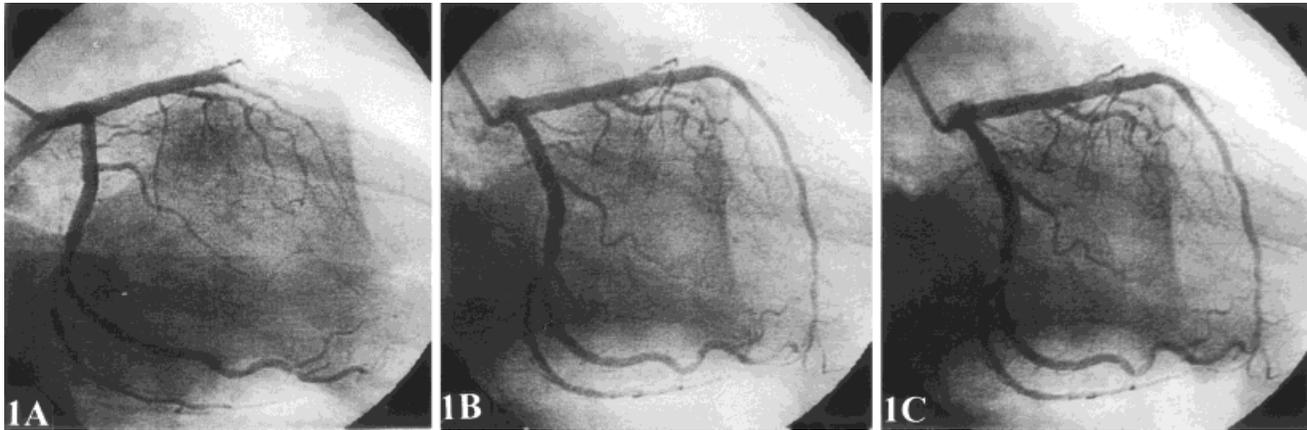
Fig. 1.   Coronary angiogram in RAO view (A) before PTCA showing mid-LAD total occlusion, (B) immediately after PTCA and stenting showing restoration of TIMI-3 flow, and (C) a predischarge angiogram showing persistence of TIMI-3 flow with no residual stenosis.

myocardial infarction (TIMI) grade 3 flow was present with an infarct-related artery (IRA) stenosis <70% or if the IRA was very small. PTCA was deferred for urgent coronary artery bypass surgery if TIMI grade 3 flow was present in the IRA with severe three-vessel disease or other lesion characteristics unfavourable for PTCA.

Before PTCA, additional heparin was given to achieve an activated clotting time (ACT) of >300 seconds. PTCA was then performed using the standard technique with floppy guidewires and rapid exchange or over the wire balloons. After restoration of flow the lesion was assessed for stent deployment. Stent was deployed in all the instances unless the vessel was <2.75 mm in diameter. No lesion was excluded because of the presence of thrombus before or after PTCA. There was no predetermined choice of the stent, and a variety of stents were used depending upon the vessel characteristics, availability of the stents, and choice of the operator. Abciximab was not used in this study.

The stents were deployed at high pressure (>12 atmospheres). Any persistent dissection was treated with additional stents. An angiographic filling defect consistent with thrombus after stenting was treated with additional PTCA. Intracoronary thrombolysis was not used in any case. Slow-flow or no-flow phenomena, if encountered, were treated with intracoronary nitroglycerin injection (200 µg) followed by intracoronary Verapamil (150–250 µg) if the slow flow persisted. All lesions with a stenosis ≥70% either proximal or distal to the infarct-related lesion that might represent a significant inflow or outflow obstruction were subjected to balloon angioplasty and stented if the result was suboptimal.

After the procedure, sheaths were removed when the ACT fell below 160 seconds. Six hours later patients were started on low-molecular-weight heparin (Enoxaparin sodium) at a dose of 1 mg/kg subcutaneously 12 hourly for 72 hours. All patients were treated with oral aspirin 100 mg per day indefinitely and ticlopidine 250 mg twice daily for 4 weeks. β-Adrenergic blocking agents and angiotensin converting enzyme inhibitors were used in all cases unless contraindicated. Oral anticoagulants were not used in any patient in this study.

Patients were ambulated and discharged when stable. In general, stable low-risk patients were discharged on hospital day 3 or 4 and stable high-risk patients were discharged on day 5–7. A repeat coronary angiogram (Figs. 1–3) and left ventriculogram were performed before discharge in all the patients. After discharge, there was a follow-up visit at 1 month.

Anterograde flow was assessed by TIMI flow grade [10]. TIMI grade 3 flow was defined as complete opacification of the distal coronary bed by the third cardiac cycle.

Clinical End Points

Intraprocedural complications including abrupt closure, repeat angioplasty of the original lesion before discharge, stent thrombosis, persistence of slow flow and need for bypass surgery owing to renarrowing of the original lesion, and death were considered as major clinical events.

Ethical Considerations

Before the start of this study, the study protocol was examined and approved by the ethical committee of our hospital. The possible risks and benefits of coronary angioplasty and stent implantation were fully explained to each patient and/or his family, and written consent was

Fig. 2. Coronary angiogram in LAO caudal view (A) before PTCA showing proximal LAD total occlusion, (B,C) immediately after PTCA and stenting showing slow flow phenomenon in LAD. B: Slow flow in the distal LAD at the end of third cardiac cycle. C: The distal LAD fills only at the end of fifth cardiac cycle.
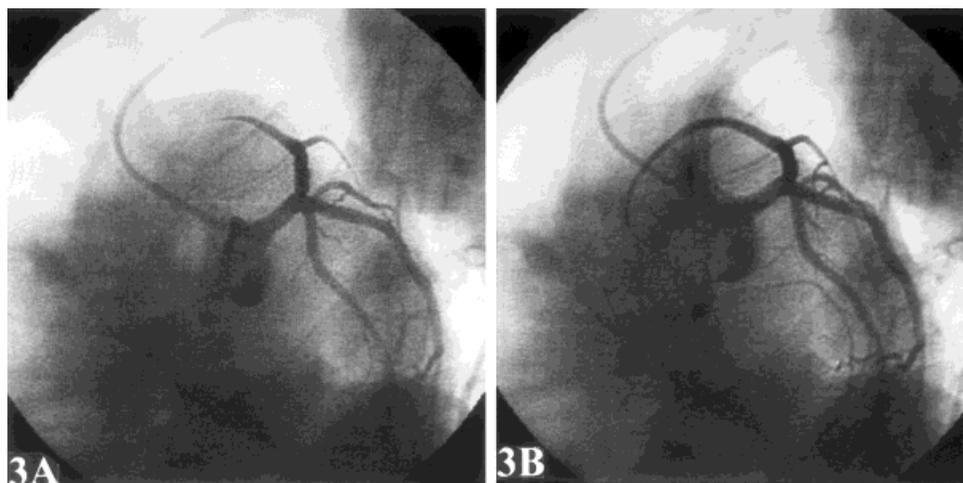
Fig. 3. Predischarge coronary angiogram of the same patient as in Figure 2 showing persistence of slow-flow phenomenon in LAD. (A): Slow flow in distal LAD at the end of third cardiac cycle. B: The distal LAD fills only during fourth cardiac cycle.

obtained before the patients were sent to the cardiac catheterization laboratory for emergent catheterization.

Measurements and Statistics

Vessel diameter was measured using the automated edge-detection method which is a part of the Philips digital angiography system (Integris-3000). Calibration was done using a 7F or 8F guiding catheter before dye filling. The measurements of the lesion diameter were made during diastole using the image in a single ''worst view.'' Measurement of the reference vessel diameter was made at the proximal part of the vessel nearest to the initial occlusion. Data were entered into a computerized data base, and statistical analysis was performed with a

commercially available package (Starview 4.5, Abacus concepts, Sunnyvale, CA).

Data were expressed as the mean value ± SD. Comparisons of continuous and categorical variables were calculated by the Student t-test and Fischer exact test, respectively.

RESULTS

Between January 1997 and April 1998, primary PTCA was performed in 68 consecutive patients with an AMI meeting the enrollment criteria. After initial PTCA, stenting was attempted in 57 patients (84%). The reasons for not stenting the remaining 11 patients were vessel size
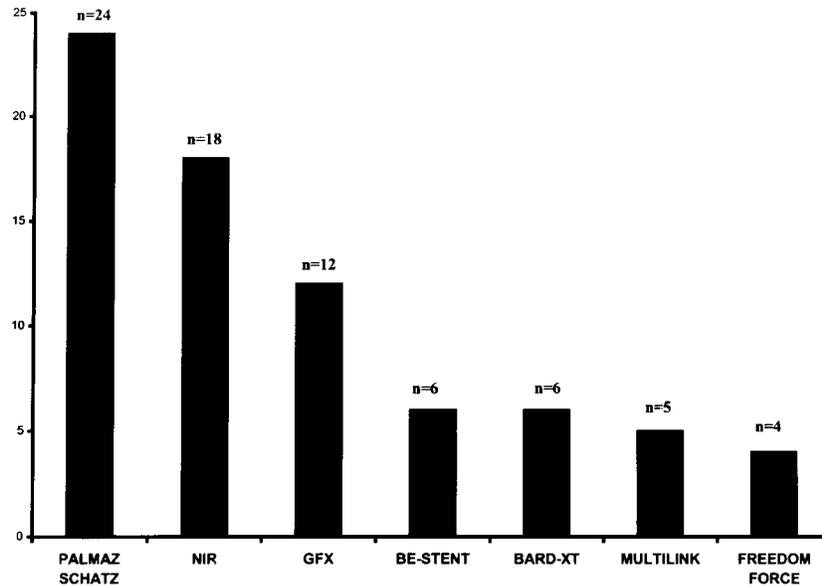
Fig. 4.   Bar diagram showing types and numbers of various stents used.

TABLE I. Baseline Clinical and Angiographic Features in 68 Patients*

	Stent n = 57 (%)	PTCA only n = 11 (%)	P value
Age (years)	57 ± 13	56 ± 12	.89
Males	45 (79)	8 (73)	.48
Diabetes mellitus	12 (21)	3 (27)	.76
Hypertension	26 (46)	5 (45)	.92
Current cigarette smoking	21 (37)	4 (36)	.95
Prior MI	9 (16)	2 (18)	.83
Prior stroke	2 (3.5)	1 (9)	.59
ECG infarct location			
Anterior	34 (60)	5 (46)	.19
Inferior	18 (32)	4 (36)	.55
Posterior	3 (5)	1 (9)	.62
Lateral	2 (3)	1 (9)	.59
Admission Killip class >II	14 (25)	2 (18)	.61
Symptom onset to ER arrival (min.)	165 ± 178	172 ± 168	.83

*MI, myocardial infarction; ER, emergency room.

TABLE II. Angiographic Characteristics of Patients*

	Stent: n = 57 (%)	PTCA only: n = 11 (%)	P value
No. of diseased vessel			
One	35 (62)	3 (27)	.006
Two	15 (26)	3 (27)	.85
Three	7 (12)	5 (46)	.02
Infarct-related artery distribution			
LMCA	0	1 (9)	.91
LAD	34 (59.6)	4 (36.3)	.005
LCX	8 (14)	2 (18.1)	.42
RCA	15 (26.3)	4 (36.3)	.38
Infarct-related artery lesion location			
Proximal vessel	31 (54.3)	4 (36.3)	.01
Mid-vessel	23 (40.3)	4 (36.3)	.28
Distal vessel	3 (5.2)	3 (27.2)	<.0001
Ref. segment diameter (mm)	3.21 ± 0.48	2.42 ± 0.68	<.0001
Lesion MLD (mm)	0.25 ± 0.42	0.31 ± 0.58	.15
Lesion DS (%)	91.5 ± 18.2	86.0 ± 20.5	.07
LVEF (%)	42 ± 16	49 ± 12	.006

*Abbreviation: LAD = left anterior descending; LCX = left circumflex artery; LMCA = left main coronary artery; RCA = right coronary artery; DS = diameter stenosis; LVEF = left ventricular ejection fraction.

≤2.5 mm in five (46%), heavy calcification in two (18%), diffuse distal disease in two (18%), and planned pre-discharge elective CABG in two (18%) patients.

The baseline demographic, admission, and angiographic features of patients in whom stenting was or was not attempted appear in Tables I and II. The lesions which were not attempted for stenting were located more distally and had smaller reference diameters. In addition, stenting was performed more often for single-vessel disease as compared to PTCA.

Stent Deployment

Stents were successfully delivered to the lesion site and deployed successfully in all the patients. Figure 4 shows the different types of stents used. Among the stent group patients a mean (±SD) of 1.3 ± 0.5 stents (range 1–3) were implanted per patient. The mean stent diameter was 3.3 ± 0.5 mm (Median 3.4), and 15.2 ± 2.3 atmospheres pressure was used. Long stents (>20 mm length) were used in 15 patients (26.3%), and the mean length of the stent in this group was 26 ± 5.2 mms. By quantitative coronary analysis the final measured mean balloon artery ratio was 1.08 ± 0.14.

The angiographic measurements and the TIMI flows are depicted in Table III. The mean post-procedural

TABLE III. TIMI Flows and Quantitative Coronary Angiographic (QCA) Measurements*

	Stent: n = 57 (%)	PTCA: n = 11 (%)
Initial TIMI flow grade		
0/1	46 (80.7)	9 (81.8)
2	8 (14)	2 (18.2)
3	3 (5.3)	0
Final TIMI flow grade		
0/1	—	—
2	6 (10.5)	1 (9)
3	51 (89.5)	10 (91)
QCA measurements		
Ref. segment	3.21 ± 0.48	2.42 ± 0.68
Initial MLD (mm)	0.25 ± 0.42	0.31 ± 0.58
Final MLD (mm)	2.81 ± 0.54	1.78 ± 0.58
Initial % DS	91.5 ± 18.2	86.6 ± 20.5
Final % DS	14 ± 17	35 ± 18

*Ref. Segment, reference segment; MLD, minimum lumen diameter; DS, diameter stenosis.

TABLE IV. Predischage Angiography QCA Parameters and TIMI-Grade Flow

	Stent: n = 57	PTCA: n = 11
Days after AMI onset	5.7 ± 3.2	6.2 ± 2.9
Ref. vessel diameter	3.1 ± 0.46	2.41 ± 0.56
MLD (mm)	2.75 ± 0.52	1.36 ± 0.59
Diameter stenosis %	15 ± 14	44 ± 19
TIMI 0/1	0	0
TIMI 2	5	0
TIMI 3	52	11

residual stenosis in the stent group was 14 ± 17% vs. 35 ± 18% in the PTCA only group. Thus, a TIMI grade 3 flow was obtained in 90% patients in this group of patients.

Clinical Outcomes

One-month follow-up data was complete for 67 patients. Repeat pre-discharge angiography was performed in all patients as a part of the protocol (Figs. 1–3; Table IV). Among the stent group, stent occlusion was not seen in any patient. However, persistent slow flow (TIMI grade 2) was documented in five of the six patients in whom slow flow had persisted after successful stenting. The clinical outcomes of these five patients with persistent slow flow as compared to those with TIMI 3 flow are shown in Table V. Of the five patients with persistent slow flow at pre-discharge angiographic study, four patients had required intra-aortic balloon pump support with inotropes for more than 72 hours and had a significantly lower left ventricular ejection fraction at discharge as compared to the patients with TIMI-3 grade flow (27.5 ± 10.2% vs. 42 ± 15.2; $P < .001$). Two of these five patients died within 1 month of their primary stenting as a result of progressive low output state. One additional patient who had TIMI-3 flow at repeat in hospital

TABLE V. Comparative Clinical Outcomes Based on TIMI Flow at Discharge in Patients With Primary Stenting

	TIMI-3: n = 52 (%)	TIMI-2: n = 5 (%)	P Value
Mean hospital stay (days)	4.8 ± 2.1	7.1 ± 3.4	<.01
LVEF (%) at discharge	42 ± 15.2	27.5 ± 10.2	<.001
Death	1 (1.9)	2 (40)	<.001
Repeat PTCA/CABG	None	None	
Symptomatic at 30 days >NYHA class II	2 (3.8)	2 (40)	<.001
Readmission for			
Heart failure	None	1 (20)	
Angina	None	None	

angiography died suddenly a few hours before planned discharge, presumably owing to a malignant arrhythmia. Symptom onset to emergency room arrival time in patients with persistent slow flow was not different from that in the remaining patients with TIMI-3 flow (151 ± 161 vs. 171 ± 180 min, P = ns). Only one patient with persistent slow flow had received a long stent. The mean hospital stay was 4.8 ± 2.1 days among patients with TIMI-3 flow vs. 7.1 ± 3.4 days in patients with persistent slow flow.

Three patients in the PTCA-only group were sent for surgery (one emergent because of a dissection extending to left main; this patients also had three-vessel disease). The other two patients underwent elective CABG because of additional disease in other vessels.

At 30 days follow-up, 54 of the stented group were alive. Thirty-eight (71%) patients were asymptomatic, 12 (22%) patients were symptomatic with dyspnoea on exertion NYHA class II, and four (7%) patients had NYHA class III dyspnea on exertion. Two out of these four patients with NYHA class III dyspnea were those with persistent slow flow at discharge. One of them required repeat admission for management of heart failure. None of the patients complained of angina on exertion.

DISCUSSION

The present study reaffirms that a primary stent strategy for the management of AMI is safe and feasible in a large majority of patients undergoing direct or primary mechanical revascularization. The routine use of high-pressure stent deployment and concomitant peri-procedural medication consisting of aspirin, ticlopidine, and low-molecular-weight heparin for 72 hours makes acute stent occlusion a rare phenomenon. Conventional guidelines consider thrombus-containing lesions, as is the case in AMI, a contraindication for stenting. However, there are now sufficient data to suggest that optimally deployed stents in a thrombogenic milieu and in the presence of

angiographically demonstrable thrombus have a very low rate of acute and subacute occlusion [11,12]. Several possible explanations may underlie the apparent safety of stenting in an obvious thrombotic environment. After primary PTCA for AMI the presence of dissection and a residual stenosis >30% have been shown to be major predictors of recurrent ischemia and infarct-related artery acute or subacute reocclusion [2–5]; both of these limitations of plain balloon angioplasty are routinely overcome by stenting. The establishment by the stent of a wide lumen channel with no recoil as seen in the pre-discharge angiograms with brisk antegrade flow in the majority of patients may facilitate natural clot resolution by endogenous fibrinolysis with aspirin, ticlopidine, and heparin.

The favorable outcomes of primary coronary plain balloon angioplasty is also reduced by restenosis, which occurs in 31–45% of patients [13,14] as in the case of balloon angioplasty for chronic stable angina pectoris. It has been reported that intracoronary stent implantation reduces the restenosis rate in denovo coronary artery lesions [15,16]. These beneficial effects of stenting have also been demonstrated in patients with AMI [8], though not in a prospective randomized study.

Intracoronary ultrasound, which is recommended to confirm the full expansion of the stent [17], was not used in this study. However, high-pressure deployment ≥ 12 atmospheres was used in all the instances (usually ≥ 14 atmospheres), irrespective of the design of the stent. This study also shows that choice of the stent is not an important issue as long as the stent can be successfully deployed at the target site in patients undergoing primary stenting for AMI. This has also been shown in several equivalancy studies performed in matched patients populations [18–20] though not in a setting of AMI. However, the previously reported studies of primary stenting in AMI have used the Palmaz-Schatz stent in most instances [8,9]. Brisk TIMI-3 flow immediately after coronary intervention in AMI is the desired result to minimize the effects of ischemic insult on myocardium [21]. The pre-discharge angiograms in the stent group show no loss in MLD, indicating no recoil as compared to the balloon group where there was a significant recoil $(1.36 \pm 0.59$ mm vs. 1.78 ± 0.58, $P < .05)$. This favorable property of the stents which eliminates recoil has been clearly demonstrated by intravascular ultrasound studies [22,23].

One of the disturbing aspects of our study has been the observation that six of the 57 patients (10.5%) in the stent group did not resume TIMI-grade 3 flow despite a successful deployment of the stent. This feature has not been reported or highlighted in the recent studies on this subject [7,9]. There was no disproportionate representation of any particular stent type or length in patients who had persistence of slow flow. Follow-up pre-discharge angiograms done in all the patients in this study showed that in 83% of these patients, the slow flow seen at the end of the procedure persisted distal to the fully patent stent. These patients had an unfavorable outcome during the follow-up with lower LVEF and higher mortality due to pump failure. Such an outcome is expected on the basis of GUSTO study findings, where failure to resume TIMI-3 flow soon after revascularization was found to be associated with a poorer clinical outcome [21].

The mechanism of slow-flow phenomenon is not clearly understood, although the postulated mechanisms include distal microembolization and microvascular spasm [24–26]. In all our cases, the slow flow could not be eliminated despite using intracoronary nitroglycerine and verapamil. Pretreatment by abciximab or other platelet glycoprotein IIb/IIIa inhibitors could possibly have changed the outcome favorably [27–29]. Abciximab has also been recently reported to improve the outcome after coronary stenting [30]. There are no previous reports in which persistence of slow flow for several days has been reported.

CONCLUSIONS

Our findings suggest that coronary stenting constitutes a safe and effective strategy for managing patients with AMI with good short-term results in the majority of cases. This is especially important since recent data have demonstrated that thrombus-containing lesions, especially in the AMI setting, not only have a lower initial success rate after plain balloon angioplasty but also have significantly higher recurrent ischemic events. However, one of the problems encountered with primary stenting was the phenomenon of persistent slow flow in a significant number of patients. These patients had an adverse outcome. Optimal treatment strategy to minimize this problem needs to be defined.

REFERENCES

1. Hartzler GO, Rutherford BD, McConahay DR, Johnson WL, Jr, McCallister BD, Gura GM Jr, Conn RC, Crockett JE. Percutaneous transluminal coronary angioplasty with and without thrombolytic therapy for treatment of acute myocardial infarction. Am Heart J 1983;106:965–973.

2. Brodie BR, Weintraub RA, Stuckey TD, LeBauer EJ, Katz JD, Kelly TA, Hansen CT. Outcomes of direct coronary angioplasty for acute myocardial infarction in candidates and non-candidates for thrombolytic therapy. Am J Cardiol 1991;67:7–12.

3. Gibbons RJ, Holmes DR, Reeder GS, Bailey KR, Hopfenspirger MR, Gersh BJ, Phil D, for the Mayo Coronary Care Unit and Catheterization Laboratory Groups. Immediately angioplasty compared with the administration of a thrombolytic agent followed by conservative treatment for myocardial infarction. N Engl J Med 1993;328:685–691.

4. Zijlstra F, DeBoer MJ, Hoorntje JCA, Reiffers S, Reiber JHC, Suryapranata H. A comparison of immediate coronary angioplasty with intravenous streptokinase in acute myocardial infarction. N Engl J Med 1993;328:680–684.

5. Grines CL, Browne KR, Marco J, Rothbaum D, Stone GW, O'Keefe J, Overlie P, Donohue B, Chelliah N, Timmis GC, et al, for the Primary Angioplasty in Myocardial Infarction Study Group. A comparison of primary angioplasty with thrombolytic therapy for acute myocardial infarction. N Engl J Med 1993;328:673–679.

6. Ellis SG, Roubin GS, King SB III, Douglas JS, Jr, Weintraub WS, Thomas RG, Cox WR. Angiographic and clinical predictors of acute closure after native vessel coronary angioplasty. Circulation 1988;77:372–379.

7. Rodriguez AE, Fernandez M, Santaere U, Larriban M, Bernards V, Castano H, Palacios IF. Coronary stenting in patients undergoing percutaneous transluminal coronary angioplasty during acute myocardial infarction. Am J Cardiol 1996;77:685–689.

8. Saito S, Hosakawe G, Kim K, Tanake S, Miyake S. Primary stent implantation without coumadin in acute myocardial infarction. J Am Coll Cardiol 1996;28:74–81.

9. Stone GW, Brodie BR, Griffin JJ, Morie MC, Constantini C, Goar FG, Overlie PA, Popma JJ, Mc Donnel J, Jones D, Neill WW, Grines CL for PAMI Stent Pilot Investigators. Prospective, multicenter study of the safety and feasibility of primary stenting in acute myocardial infarction: in hospital and 30 days results of the PAMI stent pilot trial. J Am Coll Cardiol 1998;31:23–30.

10. The TIMI Study Group. The thrombolysis in myocardial infarction (TIMI) trial: phase I findings. N Engl J Med 1985;312:932–936.

11. Kaul U, Aggarwal R, Jain P, Wasir HS. Safety and efficacy of intracoronary stenting for thrombus containing lesions. Am J Cardiol 1996;77:425–427.

12. Alfonso F, Rodriguez P, Phillips PD, Goicolea J, Hernandez R, Perez-Vizcayno MJ, Fernandex-ortiz A, Segovia J, Banuelos C, Aragoncillo P, Macaya C. Clinical and angiographic implications of coronary stenting in thrombus containing lesion. J Am Coll Cardiol 1997;29:725–733.

13. Brodie BR, Weintrab RA, Stuckey TD, Le Baner EJ, Katz JD, Kelly TA, Hansen CT. Outcomes of direct coronary angioplasty for acute myocardial infarction in candidates and non candidates for thrombolytic therapy. Am J Cardiol 1991;67:7–12.

14. Rothbaum DA, Linnemeir TJ, Landlin RJ, Brown KF, Vliestra RE, Browne KF. Emergency percutaneous coronary angioplasty in acute myocardial infarction, a 3 years experience. J Am Coll Cardiol 1987;10:264–272.

15. Serruys PW, de Jaegere P, Kiemeneij F, Macaya L, Rutsch W, Heyndrickx G, Emanuelsson H, Marco J, Legrand V, Materne P, Belardi J, Sigwart U, Colombo A, Goy JJ, Van-den Huevel P, Delcan J, Morel MH for the BENESTENT Study Group. A comparison of balloon expandable stent implantation with balloon angioplasty in patients with coronary artery disease. N Engl J Med 1994;331:489–495.

16. Fischman DL, Leon MB, Baim D, Schatz RA, Penn I, Detre K, Savage MD, Veltri L, Ricci D, Nobuyoshi M, Cleman M, Heuser R, Almond D, Teirstein P, Fish D, Colombo A, Brinker S, Moses J, Hirschfeld J, Bailey S, Ellis S, Rake R, Goldberg S. A randomised comparison of coronary stent placement and balloon angioplasty in the treatment of coronary artery disease. N Engl J Med 1994;331:496–501.

17. Colombo A, Hall P, Nakamura S, Almagor Y, Maiello L, Martini G, Gaglione A, Goldberg SL, Tobis JM. Intracoronary stenting without anticoagulation accomplished with intravascular ultrasound guidance. Circulation 1995;91:1676–1688.

18. Heuser RR, Kuntz R, Lansky A, Whitlow D, Safian R, Yeung A, Senerchia C, Cutlip D, Pino-Mauch R, Pedan A. The SMART Trial. Acute outcome indicates superior efficacy with the AVE stent (Abstract). Circulation 1997;96:I–597.

19. Hausleiter J, Dirschinger J, Schuhlen H, Giehrl W, Walter H, Pache J, Elezi S, Wehinger A, Boekstegers P, Steinbeck G, Schomig A. A multicenter randomized trial comparing five different types of slotted-tube stents (Abstract). J Am Coll Cardiol 1998;31:80A.

20. Lansky AJ, Popma JJ, Mehran R, Saucedo JF, Abizaid AS, O'Shaughnessy C, Hermiller JB, Cutlip D, Kuntz RE, Baim DS, Leon MB. Late quantitative angiographic results after NIR stent use: results from the NIRVANA randomized trial and registries (Abstract). J Am Coll Cardiol 1998;31:80A–81A.

21. Morton JK, Joseph AM, Frank VA, Richard GB, Eugene AC, Thomas W, Alexander FK, Carol Mechem RN, Thomas JD. Determination of angiographic (TIMIGRADE) blood flow by intra coronary doppler flow velocity during acute myocardial infarction. Circulation 1996;94:1545–1552.

22. Mintz GS, Popma JJ, Pichard AD, Kent KM, Satler LF, Chaung C, Griffin J, Leon MB. Intravascular ultrasound predictors of restenosis after percutaneous transcatheter coronary revascularization. J Am Coll Cardiol 1996;27:1678–1687.

23. Mintz GS, Popma JJ, Pichard AD, Kent KM, Satler LF, Wong SC, Hong MK, Korach JA, Leon MB. Arterial remodelling after coronary angioplasty—a serial intravascular ultrasound study. Circulation 1996;94:35–43.

24. Baim DS, Carrozza JP, Jr. Understanding the ''No-Reflow'' problem. Cathet Cardiovasc Diagn 1996;39:7–8.

25. Kaplan BM, Benzuly KH, Kinn JW, Bowers TR, Tilli FV, Grines CL, O'Neill WW, Safian RD. Treatment of no reflow in degenerated saphenous vein graft interventions. Comparison of intracoronary verapamil and nitroglycerin. Cathet Cardiovasc Diagn 1996;39:113–118.

26. Abbo KM, Dooris M, Glazier S, O'Neill WW, Byrd D, Grines CG, Safian RD. Features and outcome of no reflow after percutaneous coronary intervention. Am J Cardiol 1995;75:778–782.

27. EPIC Investigators. Use of a monoclonal antibody directed against the platelet glycoprotein IIb/IIIa receptors in high risk coronary angioplasty. N Engl J Med 1994;330:956–961.

28. The IMPACT II Investigators. Effect of competitive platelet glycoprotein IIb/IIIa inhibitors with Integrilin in reducing complications of percutaneous coronary interventions. Lancet 1998;349:1422–1424.

29. Topol EJ. Toward a New Frontier in myocardial reperfusion therapy—emerging platelet preeminence. Circulation 1998;97:211–218.

30. Kereiakes DJ, Lincoff AM, Miller DP, Tcheng JE, Cabot CF, Anderson KM, Weisman HF, Califf RM, Topol EJ for the EPILOG Trial investigators. Abciximab therapy and unplanned coronary stent deployment: favourable effects on stent use, clinical outcomes, and bleeding complications. Circulation 1998;97:857–864.