Published by Elsevier Science Inc.

ISSN 0735-1097/03/$30.00
PII S0735-1097(02)02617-7

Postprocedure Chest Pain After Coronary Stenting: Implications on Clinical Restenosis

Annapoorna S. Kini, MD, MRCP, Paul Lee, MD, Cristina A. Mitre, MD, Mary E. Duffy, NP, Samin K. Sharma, MD, FACC

New York, New York

OBJECTIVES The goal of this study was to analyze the incidence and predictors of postprocedure chest pain (PPCP) after percutaneous coronary intervention (PCI) and its correlation with clinical restenosis.

BACKGROUND Chest pain after PCI occurs frequently even in the absence of procedural events and is considered to be due to vasospasm or coronary artery stretch. The short- and long-term significance of PPCP after otherwise successful stenting is not clear.

METHODS We analyzed 1,362 patients undergoing coronary stenting for PPCP, procedural and in-hospital events, 30-day major adverse cardiac events, and target vessel revascularization (TVR) at 6 to 9 months.

RESULTS There were 488 patients with PPCP and, of these, 312 patients were excluded due to procedural events. The remaining 176 patients with PPCP were compared with 874 patients without PPCP. Creatine kinase-MB isoenzyme elevation occurred in 25.6% of the PPCP group versus 9.6% of the no PPCP group ($p < 0.001$). Despite similar reference vessel diameter, the PPCP group had larger postprocedure minimum lumen diameter, higher stent-to-vessel ratio, and higher inflation pressure versus the no PPCP group ($p < 0.01$). At 30 days, the emergency room visits and repeat catheterization (16% vs. 2.7%; $p < 0.001$) were higher in the PPCP group versus the no PPCP group, but repeat intervention was similar. At 6- to 9-month follow-up, the TVR was significantly higher in the PPCP group compared with the no PPCP group (29.5% vs. 16.6%; $p < 0.01$).

CONCLUSIONS Our analysis suggests micromyonecrosis and vessel stretch as causes of PPCP. Postprocedure chest pain is associated with similar short-term outcome as no PPCP, but has higher restenosis, perhaps mediated by deep vessel wall injury. Therefore, PPCP may identify patients at high risk for restenosis. (J Am Coll Cardiol 2003;41:33–8) © 2003 by the American College of Cardiology Foundation

Postprocedure chest pain (PPCP) after percutaneous coronary intervention (PCI) occurs frequently and is usually related to procedural events (1–3). Several reports have suggested that the PPCP is most likely due to various procedural events, such as abrupt vessel closure, coronary vasospasm, side-branch closure, dissection, distal thromboembolism, slow-flow, focal vessel stretch, or adventitial injury, which often occur with newer devices such as atherectomy and stenting (1–8). Many of these patients undergo early angiography (sometimes in-hospital) to rule out acute closure frequently associated with creatine kinase-MB isoenzyme (CK-MB) elevation (1–3). The use of glycoprotein IIb/IIIa inhibitors (GPI) during PCI has been shown to reduce periprocedural enzyme elevation, perhaps by decreasing platelet-mediated distal microthromboembolism, an inherent part of PCI (9,10). However, in the absence of procedural events, PPCP is considered benign and postulated to be due to local vessel stretch and deep adventitial injury, especially in patients undergoing stenting (2,5). There have been reports suggesting a higher incidence of intimal hyperplasia or periprocedural enzyme release after stent implantation using high-pressure deployment and/or higher stent-to-vessel size ratio (11–13). This may be mediated via deep vessel wall (adventitial) injury causing aggressive intimal hyperplasia and subsequent restenosis (12). With the current practice of moderate- to high-pressure stent deployment, the clinical implication of PPCP without any obvious procedural complications is not well understood. We analyzed patients undergoing coronary stenting in relation to the presence or absence of PPCP and correlated with periprocedural enzyme release, short-term outcome, and need for target vessel revascularization (TVR).

METHODS

Patients. All consecutive patients undergoing coronary stenting of native coronary artery at Mount Sinai Hospital, New York, from July 1999 to July 2000, were analyzed. Patients with acute myocardial infarction (MI) ($n = 162$) and elevated preprocedural CK-MB, troponin I (TnI), or ongoing chest pain ($n = 306$) were excluded. Also, patients with planned staged intervention ($n = 312$) and lost for follow-up ($n = 22$) were excluded. A total of 1,362 consecutive nonacute MI patients undergoing stenting were analyzed, of which 488 (35.8%) had PPCP or some procedural event, and 874 (64.2%) had no PPCP with no procedural event. In the PPCP or procedural event group,

From the Cardiac Catheterization Laboratory of the Zena & Michael A. Wiener Cardiovascular Institute, Mount Sinai Hospital, New York, New York. This study was presented, in part, at the 48th Annual Scientific Session of the American College of Cardiology in New Orleans, Louisiana, in March 2001.

Manuscript received March 27, 2002; revised manuscript received July 18, 2002, accepted September 20, 2002.

Abbreviations and Acronyms

ACC/AHA	= American College of Cardiology/American Heart Association
CI	= confidence interval
CK-MB	= creatine kinase-MB isoenzyme
ECG	= electrocardiogram
GPI	= glycoprotein IIb/IIIa inhibitors
LAD	= left anterior descending coronary artery
MACE	= major adverse cardiac events
MI	= myocardial infarction
MLD	= minimum lumen diameter
OR	= odds ratio
PCI	= percutaneous coronary intervention
PPCP	= postprocedure chest pain
TnI	= troponin I
TVR	= target vessel revascularization

312 (63.9%) patients had some procedural events (94% also had PPCP) and were analyzed as the procedural event registry patients, while the remaining 176 patients with PPCP and no procedural events (12.9% from entire group) comprised the study population. These 176 patients with PPCP and no procedural complications were compared with 874 patients with no PPCP and no procedural event. A 12-lead electrocardiogram (ECG) was done on all patients pre-, post-, and morning after procedure, and as frequently as needed clinically in the presence of PPCP. Cardiac enzymes (CK-MB, TnI) were measured at baseline, preprocedure, 6 to 8 h, and 12 to 24 h after procedure, and thereafter if still rising. All patients were routinely questioned for presence or absence of chest pain after procedure and the next morning by an independent nurse practitioner (14). Chest pain severity was graded from 1 to 10, 10 being the worst. Patients were followed for in-hospital major adverse cardiac events (MACE). All procedural details, postprocedural events, and in-hospital events were captured on an angioplasty worksheet and transferred to the interventional database. All angiograms were carefully analyzed to identify any procedural events, especially side-branch closure of >1.0-mm size. All patients had 30-day and 6- to 9-month clinical follow-up by a telephone call to the patient or private physician. All interventions were done using a conventional technique, using aspirin 325 mg daily pre- and postprocedure indefinitely, clopidogrel 300 mg loading preprocedure and 75 mg daily for one month after PCI, and low-dose heparin (70 IU/kg) to keep the activated clotting time at approximately 250 s. The types of stent used were Multilink (Guidant Corp., Indianapolis, Indiana), Duet (Guidant Corp.), or Tetra (Guidant Corp.) in 55% of cases, NIR (Boston Scientific/Scimed, Maple Grove, Minnesota) in 35%, and S660 or S670 (Medtronic Inc., Minneapolis, Minnesota) in 20% of cases. A single experienced individual who was unaware of the purpose and outcome of the study independently performed quantitative angiographic analysis on the CMS Medis system (The Netherlands).

Definitions. Postprocedure chest pain was defined as varying degrees of typical or atypical chest pain starting after PCI. Creatine kinase-MB isoenzyme measured by the Mass technique was considered normal if <16 U/l and elevated if ≥16 U/l further subdivided in 1 to 3 × (16 to 48 U/l), 3 to 5 × (49 to 80 U/l), and >5 (>81 U/l) normal. Troponin I of ≥2.0 ng/ml was considered elevated. Significant ECG changes were ST-segment depression ≥1.0 mm or any ST-segment elevation. Nonspecific ECG changes were ST-segment depression <1.0 mm or T-wave changes. Procedural events were defined as transient or persistent acute closure, coronary vasospasm, side-branch occlusion of >1.0-mm size, coronary dissection type C, thromboembolism or air embolism, slow-flow or no flow, perforation, and/or prolonged hypotension. Stent-to-vessel ratio was calculated by dividing the nominal size of the maximum final stent or postdilation balloon to the reference vessel size. Angiographic lesion morphology was classified in the usual manner, including complex/thrombotic (irregular, ulcerated, haziness, or visible filling defect), calcified, and American College of Cardiology/American Heart Association (ACC/AHA) classification. Major adverse cardiac events were defined as death, Q-wave MI or large non–Q-wave MI with CK-MB >8× normal, or urgent revascularization.

Statistics. Data were entered in the interventional database (Access-based program), and the required data retrieved in Microsoft Excel format and transferred to the statistical program for analysis. Pearson's chi-squared test was used for analysis of cross-tabulation of the categorical variables. Fisher exact test was used for 2 × 2 cross-table analysis. Student *t* test was used for continuous variables. Continuous variables were presented as mean ± SD. Stepwise logistic regression technique (forward logistic regression method) was employed to obtain multivariate predictors for restenosis (entry probability 0.05; removal probability 0.20). All analysis was performed using SAS/JMP (SAS Institute, Cary, North Carolina) and SPSS 10.0 (SPSS Inc., Chicago, Illinois). A p value of <0.05 was considered statistically significant.

RESULTS

Baseline characteristics. Baseline clinical and procedural characteristics are shown in Table 1. There was a significantly higher incidence of females, patients with rest angina or post-MI, and GPI use in the PPCP versus no PPCP groups. Angiographic and quantitative coronary angiographic data are shown in Table 2. Patients with PPCP were more likely to have left anterior descending coronary artery (LAD), complex thrombotic lesions, and more severe stenosis. The PPCP group had similar reference vessel size, but higher postprocedure minimum lumen diameter (MLD), need for postdilation, less debulking, and higher inflation pressure (>16 atm) versus the no PPCP group.

Nature and evaluation of PPCP. In 92% of patients, PPCP was predominantly at rest. In 68% of patients, PPCP

Table 1. Baseline Clinical and Short-Term Characteristics in the PPCP Versus No PPCP Groups

Variables	PPCP Group (n = 176)	No PPCP Group (n = 874)	p Value
Age (yrs)	62.3 ± 9.4	64.2 ± 9.1	NS
Female gender (%)	27.3	20.0	0.03
Rest angina (%)	32.3	21.3	0.001
Post-MI (%)	40.9	12.8	0.001
Hypertension (%)	40.1	42.2	0.65
Diabetes mellitus (%)	22.2	23.3	0.56
Hyperlipidemia (%)	52.3	51.6	0.55
LVEF (%)	45 ± 12	46 ± 12	0.72
GP IIb/IIIa use (%)	81.8	74.8	0.05
Multivessel intervention (%)	8.5	10.2	0.62
Multilesion intervention (%)	21.3	19.4	0.55
Activated clotting time (s)	252 ± 62	248 ± 48	0.33
In-hospital MACE (%)	0.6	0.4	0.52
In-hospital repeat angiography (%)	9.1	0.6	< 0.001
30-day events			
Repeat angiography (%)	16.0	2.7	< 0.001
Repeat intervention (%)	2.3	1.9	0.44
MACE (%)	2.8	2.2	0.43

GP = glycoprotein; LVEF = left ventricular ejection fraction; MACE = major adverse cardiac events (death, Q-wave or large non–Q-wave, MI, or urgent revascularization); MI = myocardial infarction; PPCP = postprocedure chest pain.

was not similar to preprocedure anginal pain. In 54% of patients, PPCP was continuous dull aching with fluctuation in severity. The PPCP severity grades varied from 1 to 7 (average 4). In 96% of patients, PPCP started within 24 h of PCI. In 47% of the patients who had PPCP, the pain lasted for nearly 24 h (mean, 14.12 ± 10.21 h) after procedure, 35% had pain up to 72 h, and minority had pain up to 14 days (Fig. 1). Nonspecific ECG changes were seen in 11.4% of the PPCP patients, but no patient had significant ECG changes. The average length of stay was 3.1 ± 1.2 days versus 2.1 ± 1.6 days in the PPCP versus no PPCP groups (p = 0.07).

PPCP and enzyme elevation. In the PPCP group, any CK-MB elevation (Fig. 2) was seen in 25.6% versus 9.6% in the no PPCP group (p < 0.001). Similarly, TnI elevation was seen in 55.7% versus 15.4% in the PPCP and no PPCP groups, respectively (p < 0.001). Multivariate predictors of CK-MB elevation, excluding PPCP, were acute coronary syndrome (odds ratio [OR], 4.2; 95% confidence interval [CI], 2.5 to 6.6), stent-to-vessel ratio >1.1 (OR, 1.5; 95% CI, 1.1 to 2.2), inflation pressure >16 atm (OR, 3.5; 95% CI, 1.3 to 6.2), thrombotic lesion (OR, 7.5; 95% CI, 2.5 to 14.2), age (OR, 1.8; 95% CI, 1.3 to 2.2), and ACC/AHA type C lesion (OR, 3.9; 95% CI, 2.2 to 5.8). In the PPCP group, there were no differences in inflation pressure and stent-to-vessel ratio between patients with or without CK-MB elevation.

Multivariate predictors of PPCP were acute coronary syndrome (OR, 4.6; 95% CI, 3.1 to 6.3), stent-to-vessel ratio >1.1 (OR, 3.3; 95% CI, 2.4 to 5.1), inflation pressure

Table 2. Angiographic Characteristics and Quantitative Coronary Angiography Results in the PPCP Versus no PPCP Groups

Variables	PPCP Group (n = 176)	No PPCP Group (n = 874)	p Value
LAD lesion (%)	52.3	43.1	0.03
ACC/AHA type B_2/C (%)	58/30	65/24	0.32
Lesion length (mm)	12.3 ± 6.4	11.8 ± 6.3	0.23
Moderate-heavy calcification (%)	28.4	22.5	0.09
Complex/thrombotic lesion (%)	14.8	6.2	< 0.001
Collateral grade ≥2 (%)	8.5	6.8	0.44
Direct stenting (%)	6.2	5.3	0.64
Postdilation (%)	18.7	8.6	< 0.01
Debulking (%)	15.3	24.9	< 0.01
Inflation pressure (%)	16 ± 3	13 ± 2	< 0.01
Stent length (mm)	16.2 ± 8.2	16.8 ± 7.6	0.24
Stent:vessel ratio	1.21 ± 0.12	1.08 ± 0.08	0.02
Reference vessel size (mm)	2.83 ± 0.38	2.82 ± 0.41	0.43
MLD, pre (mm)	0.68 ± 0.21	0.82 ± 0.23	0.05
MLD, post (mm)	2.73 ± 0.42	2.45 ± 0.32	0.01

ACC/AHA = American College of Cardiology/American Heart Association; LAD = left anterior descending coronary artery; MLD = minimum lumen diameter; PPCP = postprocedure chest pain.

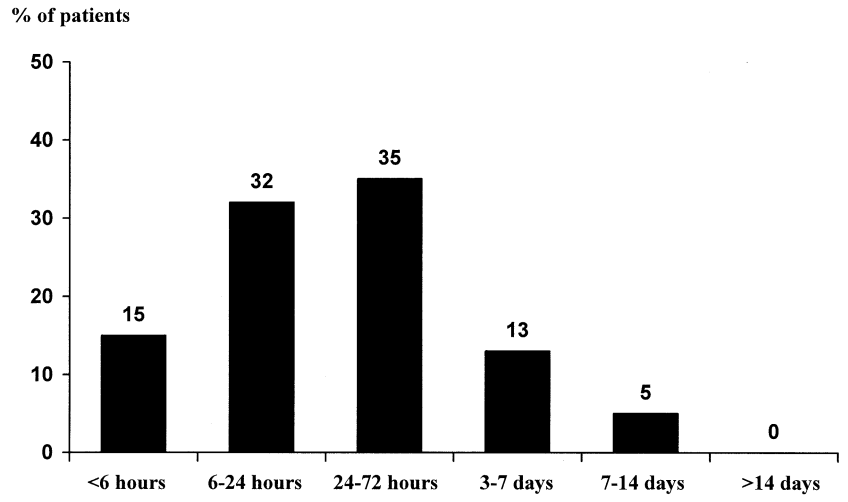

Figure 1. Duration of postprocedure chest pain after percutaneous coronary intervention.

>16 atm (OR, 2.8; 95% CI, 1.4 to 4.7), and LAD lesion (OR, 1.8; 95% CI, 1.2 to 2.6).

Clinical follow-up. At 30 days, the number of emergency room visits (9.1% vs. 1.4%; $p < 0.001$), readmissions (23.9% vs. 3.0%; $p < 0.001$), and repeat angiography (16.0% vs. 2.7%; $p < 0.001$) were higher in the PPCP group versus the no PPCP group (Table 1). However, repeat intervention, subacute thrombosis, death, re-MI, and 30-day MACE were not different between the two groups. The incidence of 30-day MACE was 2.5% in the atypical PPCP versus 3.0% in the typical anginal PPCP ($p = 0.16$). Clinically driven coronary angiography after 30 days occurred in 36% of the PPCP patients and 32% of no PPCP patients (p = NS). At 7.4 ± 1.2 months clinical follow-up, TVR was 29.5% in the PPCP group versus 16.6% in the no PPCP group ($p < 0.01$). Among the PPCP patients, TVR was 22.2% in the CK-MB elevation group compared with 32% in the no CK-MB elevation group ($p = 0.20$). For the entire group (both PPCP and no PPCP), the incidence of TVR was 17.8% in the CK-MB elevation group versus 18.9% in the no CK-MB elevation group (p = NS). The incidence of MI and mortality was not different between the two groups (2.2% vs. 1.5%; $p = 0.20$).

On multivariate analysis the predictors of TVR after stenting were PPCP (OR, 3.2; 95% CI, 1.9 to 4.6), diabetes mellitus (OR, 2.6; 95% CI, 1.5 to 3.8), and lesion length >10 mm (OR, 1.9; 95% CI, 1.2 to 2.8). Postprocedure MLD was not predictive of TVR once PPCP was entered in the multivariate model.

Procedural events registry. In 312 patients with some procedural events, 94% also had PPCP with CK-MB elevation of 69.9% (1 to 3 × normal = 37.8%, 3 to 5 × normal = 22.8%, and >5 × normal = 9.3%) and TnI elevation of 77.9%. In-hospital MACE and 30-day MACE were 2.2% and 4.8%, respectively; $p < 0.02$ versus the PPCP or the no PPCP group. The incidence of repeat angiography in-hospital or at 30 days was 5.1% and 11.5% (lower than PPCP group, but higher than no PPCP group). At 8.2 ± 1.8 months clinical follow-up, the incidence of death and MI was 6.7% ($p < 0.01$ vs. PPCP or no PPCP

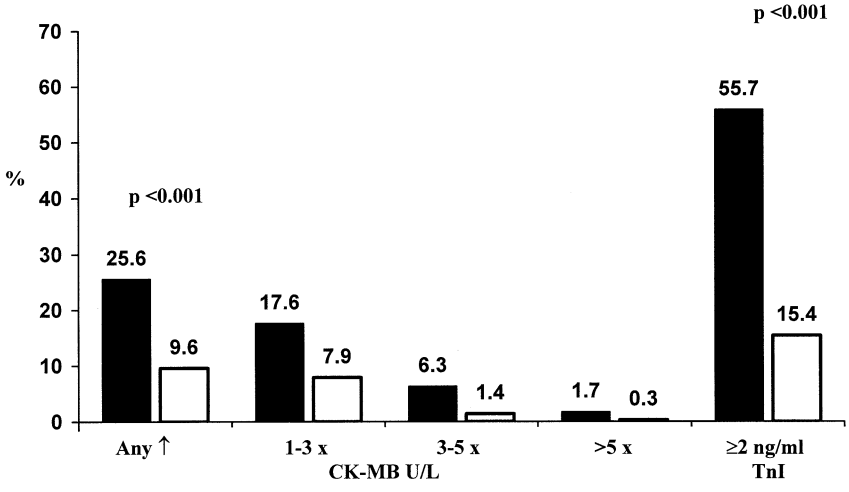

Figure 2. Incidence and magnitude of creatine kinase-MB isoenzyme (CK-MB) and troponin I (TnI) elevation in the two groups. **Solid bar** = postprocedure chest pain (PCPP) (n = 176); **open bar** = no PCPP (n = 874).

group), and TVR was 18.9% ($p = 0.007$ vs. PPCP and $p = 0.32$ vs. no PPCP).

DISCUSSION

This study reports for the first time the clinical implication of PPCP, which is seen in 36% of the patients undergoing PCI, and, in two-thirds of the cases, a procedural complication/event could be identified. In the remaining one-third of patients with PPCP who had no identifiable procedural events, the most likely cause could be vessel stretch and/or micromyonecrosis. Previous studies suggested vessel stretch/adventitial injury as a possible mechanism of PPCP, which was also observed in our study as suggested by higher stent-to-vessel ratio and postprocedure MLD in the PPCP group (1–4).

Mechanism of PPCP and short-term events. Incidence of periprocedural CK-MB elevation after PCI varies (10% to 30%), mostly due to various procedural events including distal embolization of plaque and thrombus, and side-branch closure especially after stenting (6–8). To evaluate the exact mechanism of periprocedural CK-MB elevation, a recent report of contrast-enhanced magnetic resonance imaging, before and after PCI, in nine patients revealed side-branch closure in five of them (55%) and distal thromboembolism in four (45%) despite very high use of GPI (8). Several studies have shown that chest pain after coronary intervention correlates with procedural complications or ongoing ischemia as reflected by elevated CK-MB (2,3,7). In our study, the incidence of CK-MB and TnI elevation after PCI in the PPCP group with no identifiable procedural events was 25.6% and 55.7%, respectively, which we hypothesized to be due to micromyonecrosis caused by distal microthromboembolism. This hypothesis can be further substantiated by the fact that more patients with acute coronary syndrome and thrombotic lesions were seen in the PPCP group and were likely to be at high-risk for distal thromboembolism, despite a higher use of GPI. Markers of vessel stretch (high inflation pressure, high stent-to-vessel ratio) were higher in both CK-MB elevation and no CK-MB elevation patients in the PPCP group. This may implicate vessel stretch contributing to distal microembolization (13).

Stent implantation results in a larger postprocedure MLD, causing a higher degree of circumferential stretching resulting in irritation of sensory nerves located in adventitia and, thereby, can cause PPCP (5). This adventitial irritation can be a plausible explanation of PPCP in our study in patients without any postprocedure enzyme elevation. Both micromyonecrosis and vessel stretch contributed to the development of PPCP, but the exact extent of contribution of individual pathophysiologic process could not be established with certainty in the present study. In the current study, occurrence of PPCP in the absence of any obvious procedural complications was benign at short-term (30 days), not associated with higher revascularization or ischemic complications, but at the cost of higher health care resource utilization. Therefore, many times PPCP can be distinguished from ongoing ischemic pain by its nature, which is often atypical, continuous, and dull aching with fluctuation in severity.

PPCP as a predictor of clinical restenosis. Larger final MLD after various interventional devices has correlated with lower restenosis based on the "bigger is better" hypothesis (15). However, deep vessel wall injury, which may occur in a subset of patients due to overexpansion of stent causing adventitial irritation with resultant PPCP, has been shown to subsequently cause intense inflammation and exaggerated intimal hyperplasia by stent struts (11,12). This process of aggressive intimal hyperplasia after stenting may explain the higher incidence of TVR in the PPCP group in our study and even offset the advantage of a larger final MLD, as the final MLD was not associated with lower restenosis on multivariate analysis in this study. Also, PPCP was the strongest independent predictor of clinical restenosis (in addition to diabetes and lesion length), while other traditional risk factors like rest angina, thrombotic lesion, LAD location, and debulking were not predictors on multivariate analysis. Another interesting observation is that the majority of patients requiring follow-up angiography had clinical restenosis in the PPCP group versus the no PPCP group, perhaps due to more symptom recurrence.

Therefore, PPCP identifies a group of patients who are at risk of developing periprocedural enzyme elevation despite GPI use and no obvious procedural complications, but have a high incidence of clinical restenosis. As previously believed, patients developing PPCP without procedural events or significant ECG changes can be safely monitored without a need for repeat coronary angiography during the initial index hospitalization. Nevertheless, these patients need to be closely followed for symptoms of early restenosis.

Procedural events registry analysis. Similar to earlier reported observation, patients undergoing PCI complicated by procedural events are associated with high short- and long-term clinical events of death or MI compared with patients without procedural events, but not associated with a higher incidence of clinical restenosis (16). This could be explained by restenosis being clinically silent in these patients, the majority of whom have suffered periprocedural MI.

Study limitations. This is a nonrandomized study with clinical follow-up, but no routine follow-up angiography. Intravascular ultrasound was not routinely performed, which could have added in understanding the mechanism and probably confirmed the theory of "vessel stretch" in causing PPCP.

Study implications. Although PPCP in the absence of procedural events is not associated with short-term clinical events, the present study identifies these patients as high-risk for periprocedural enzyme elevation and clinical restenosis. While full stent expansion and optimum stent deployment have been shown to be important for late outcome

(15), our data suggest that oversizing stents and very high inflation pressures could be detrimental because of deep wall injury, causing aggressive intimal hyperplasia and restenosis. Therefore, modifying procedural factors likely to cause PPCP such as high inflation pressure, oversize stents, and using appropriate debulking may result in lower clinical restenosis. This practice is now being routinely applied in our cath lab.

Reprint requests and correspondence: Dr. Samin K. Sharma, Mount Sinai Hospital, Box 1030, One Gustave Levy Place, New York, New York 10029-6574. E-mail: samin.sharma@msnyuhealth.org.

REFERENCES

1. Mansour M, Carrozza J, Kuntz R, et al. Frequency and outcome of chest pain after two new coronary interventions (atherectomy and stenting). Am J Cardiol 1992;69:1379–82.
2. Jeremias A, Kutscher S, Haude M, et al. Nonischemic chest pain induced by coronary interventions: a prospective study comparing coronary angioplasty and stent implantation. Circulation 1998;98:2656–8.
3. Schuepp M, Ullmer E, Weinbacher M, et al. Chest pain after percutaneous coronary intervention: incidence and relation to ECG changes, cardiac enzymes and follow-up events. J Invasive Cardiol 2001;13:211–6.
4. Warth D, Leon M, O'Neill W, et al. Rotational atherectomy multicenter registry: acute results, complications and 6-month angiographic follow-up in 709 patients. J Am Coll Cardiol 1994;24:641–8.
5. Gulbenkian S, Saetrum-Opgaard O, Ekman R, et al. Peptidergic innervation of human epicardial coronary arteries. Circ Res 1993;73:579–88.
6. Kini A, Marmur J, Kini S, et al. Creatine kinase-MB elevation after coronary intervention correlates with diffuse atherosclerosis, and low-to-medium level elevation has a benign clinical course. J Am Coll Cardiol 1999;34:663–71.
7. Kini A, Kini S, Marmur J, et al. Incidence and mechanism of creatine kinase-MB enzyme elevation after coronary intervention with different devices. Cathet Cardiovasc Intervent 1999;48:123–9.
8. Ricciardi M, Wu E, Davidson C, et al. Visualization of discrete microinfarction after percutaneous coronary intervention associated with mild creatine kinase-MB elevation. Circulation 2001;103:2780–3.
9. EPISTENT Investigators. Randomized placebo-controlled and balloon angioplasty-controlled trial to assess safety of coronary stenting with use of platelet glycoprotein IIb/IIIa blockade. Lancet 1998;352:87–92.
10. The ESPRIT Investigators. Novel dosing regimen of eptifibatide in planned coronary stent implantation (ESPRIT): a randomized, placebo-controlled trial. Lancet 2000;356:2037–44.
11. Farb A, Sangiorgi G, Carter A, et al. Pathology of acute and chronic coronary stenting in humans. Circulation 1999;99:44–52.
12. Hoffmann R, Mintz G, Mehran R, et al. Tissue proliferation within and surrounding Palmaz-Schatz stents is dependent on the aggressiveness of stent implantation technique. Am J Cardiol 1999;83:1170–4.
13. Dirschinger J, Kastrati A, Neumann F, et al. Influence of balloon pressure during stent placement in native coronary arteries on early and late angiographic and clinical outcome: a randomized evaluation of high-pressure inflation. Circulation 1999;100:918–23.
14. Spertus J, Winder J, Dewhurst T, et al. Development and evaluation of the Seattle Angina Questionnaire: a new functional status measure for coronary artery disease. J Am Coll Cardiol 1995;25:333–41.
15. Kuntz R, Gibson C, Nobuyoshi M, et al. Generalized model of restenosis following conventional balloon angioplasty, stenting, and directional atherectomy. J Am Coll Cardiol 1993;21:15–25.
16. Narins C, Miller D, Califf R, Topol E. The relationship between periprocedural myocardial infarction and subsequent target vessel revascularization following percutaneous coronary revascularization: insights from the EPIC trial. J Am Coll Cardiol 1999;33:647–53.