Published in final edited form as:

Am Heart J. 2010 November ; 160(5): 979–986.e1. doi:10.1016/j.ahj.2010.07.007.

Differential effects of post-dilation after stent deployment in patients presenting with and without acute myocardial infarction

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Abstract

Background—In the practice of percutaneous coronary intervention (PCI), post-dilatation often is performed after stent deployment to improve stent expansion. However, aggressive mechanical expansion is a risk factor of distal embolization and microvascular injury, especially for patients with acute myocardial infarction (AMI). Few studies have investigated the effects of post-dilatation on medium-term clinical outcomes.

Methods and Results—Patients enrolled in the multicenter NHLBI Dynamic Registry between 2001 and 2006 were evaluated. Patients who were treated with ≥ 1 stent were studied. Patients with cardiogenic shock or history of coronary artery bypass graft surgery were excluded. Patients were followed up to 1 year. Because of the significant statistical interaction ($P = .02$) between post-dilatation and AMI status on the hazard of death/myocardial infarction (MI), post-dilatation effects were estimated separately for patients who did and did not present with an AMI. Among the 1,358 patients who presented with an AMI, post-dilatation was associated with a significantly higher risk of death/MI (hazard ratio [HR] = 1.78, 95% CI 1.12–2.83, $P = .01$), not associated with the risk of repeat revascularization (HR = 1.15, 95% CI 0.81–1.62, $P = .43$). Among the 2,699 patients who did not present with AMI, post-dilatation was not associated with risks of death/MI (HR = 1.08, 95% CI 0.77–1.50, $P = .67$) or repeat revascularization (HR = 1.17, 95% CI 0.93–1.47, $P = .19$). Similar effects were observed for the restricted analysis with additional adjustment for lesion characteristics among the 1,039 AMI patients and 2,179 non-AMI patients with a single lesion treated.

Conclusions—Stent post-dilatation is associated with an increased risk of death/MI in AMI patients but not in non-AMI patients. Further investigation is warranted.

Post-dilation with noncompliant balloons after stent deployment has been shown to enhance stent expansion.^{1–3} Given that stent underexpansion is an important predictor of stent thrombosis^{4,5} and restenosis after percutaneous coronary intervention (PCI),^{6,7} post-dilation is widely recommended. Aggressive mechanical intervention, however, may cause distal embolization, particularly in patients presenting with acute myocardial infarction

(AMI).^{8,9} A relatively large proportion of AMI patients are at risk for distal embolization despite having normal flow after PCI.¹⁰⁻¹² We hypothesized that post-dilation would have differential effect on death or myocardial infarction (MI) in AMI and non-AMI patients. To test this hypothesis, we used data from the National Heart, Lung, and Blood Institute (NHLBI)-sponsored Dynamic Registry to examine the associations between post-dilation and clinical outcomes among AMI patients and non-AMI patients.

Methods

Study design and patient selection

The NHLBI Dynamic Registry is a multicenter, prospective, observational study of consecutive patients undergoing PCI during 5 prespecified “periods” or “waves,” as described previously.^{13,14} For the present analysis, we used patients enrolled in the 3 most recent recruitments (2001-2006) because the stent delivery systems of these PCI procedures and the performance of post-dilation most represent current clinical practice. Patients were included if they were treated with at least one stent. Patients presenting with cardiogenic shock or a history of coronary artery bypass graft surgery (CABG) were excluded. The selection process is shown in Figure 1. We also conducted a subgroup analysis by restricting the analysis to patients who had only one lesion treated to better control the influence from lesion characteristics. The incidence of death, MI, repeat PCI, or CABG was recorded during the 1-year follow-up period.

Definitions and follow-up

Death is defined as all-cause mortality. Myocardial infarction is diagnosed based on evidence of at least 2 of the following: typical chest pain not relieved by nitroglycerin; serial electrocardiogram showing changes from baseline or serially in ST and T waves, Q waves, or both in more than 2 contiguous leads; a rise in creatine kinase (CK) level to more than twice the upper limit of the reference range with an increase in CK-MB of more than 5% of the total value, or troponin level elevation to more than twice the upper limit of normal. Acute myocardial infarction is defined as the onset of MI within 10 days of this procedure, including both ST-elevation MI (STEMI) and non-ST-elevation MI (NSTEMI). Repeat PCI is nonstaged, and includes both target and non-target vessel interventions. Repeat revascularization includes both repeat PCI and CABG.

Statistical methods

This study focused on the possible interaction between AMI status at presentation and post-dilation treatment. This statistical interaction was evaluated formally using a Cox proportional hazards regression model that included terms for the main effects of post-dilation and AMI and their interaction. To ascertain whether the statistical interaction between post-dilation and AMI was independent of other covariates, additional adjustment was made (see Appendix A). A 2-sided *P* value of .05 or less was considered to be statistically significant in the primary analyses.

Separate propensity score models were fit to AMI and non-AMI patients, using logistic regression to model the predicted probability of post-dilation; this adjusts for the potential selection bias inherent in the decision whether to post-dilate the stent.¹⁵ A candidate list of demographic variables, disease history, and procedural characteristics was considered. A backward selection method was used with a *P* value of .40 to select variables predicting the decision to post-dilate (see Appendix B). Differences in continuous variables between the 2 treatment groups were tested using *t* tests or Wilcoxon nonparametric tests. Differences in proportions of categorical variables were tested by χ^2 tests. The balance of covariates between the 2 treatment groups was assessed after adjustment for the propensity score as a

continuous covariate; we used logistic regression to assess balance on categorical covariates and linear regression on continuous covariates.

For each adverse event end point, the 1-year cumulative incidence proportion was estimated using the Kaplan-Meier method and was compared using a log-rank test between the 2 treatment groups. Patients were censored at the last known date of contact or at 1-year follow-up, whichever was earlier. Cox proportional hazards models were used to estimate 1-year hazard ratios (HRs) for adverse events by post-dilation status in each AMI subgroup, adjusting for the respective propensity scores as continuous covariates, and post-discharge statin, β -blockers, and angiotensin-converting enzyme inhibitors. To better control the influence from lesion characteristics, we restricted our analysis to patients who had treatment of one lesion only. Lesion characteristics were included in the propensity score model. To study the potential underlying mechanisms, we compared the TIMI grade before and after PCI procedure, and the rate of no reflow among the 2 treatment groups by χ^2 test. Statistical analyses were performed using the SAS program, version 9.1 (SAS Institute, Cary, NC). The Dynamic Registry was supported by the National Institutes of Health. The authors are solely responsible for the design and conduct of this study, all study analyses, and the drafting and editing of the paper and its final contents.

Results

Among the 4,359 patients included in the analysis, 1,358 (31%) presented with AMI at the time of their index PCI procedure and the remaining 3,001 (69%) presented for PCI with indications other than AMI.

The statistical interaction between post-dilation and AMI status in terms of the HR of death/MI was significant ($P = .02$ and $P = .03$ before and after adjustment for other covariates), although not significant for repeat revascularization. Therefore, we present the subsequent results for AMI and non-AMI patients separately.

Among the 1,358 AMI patients, 586 (43%) patients received post-dilation. The proportion of STEMI was similar among post-dilation group and non-post-dilation group (53.9% vs 53.5%, $P = 0.87$). Patients receiving post-dilation were more likely to have severe noncardiac disease (including cerebrovascular disease, peripheral vascular disease, pulmonary disease, cancer, renal failure, or other severe disease); history of congestive heart failure; double- or triple-vessel narrowing; more likely to receive drug-eluting stents; and more likely to have calcified, ulcerated, ostial, or type C lesions (Table I).

Among the 3,001 non-AMI patients, 1,281 (43%) received post-dilation. Patients receiving post-dilation were less likely to have history of hypertension, history of hypercholesterolemia, more likely to receive drug-eluting stent, more likely to have lesions of thrombus, calcification, plaque ulceration, ostial and type C lesions (Table I).

The distributions of the propensity scores in the 2 treatment groups for AMI and non-AMI patients are shown in Figure 2. These graphs demonstrate considerable overlap in the propensity score distributions between the 2 treatment groups, indicating a good comparability between the 2 treatment groups other than the treatment itself. After propensity score adjustment, none of the differences in demographics, disease history, and procedural characteristics between post-dilation group and non-post-dilation group shown in Table I were statistically significant for patients presenting with or without AMI.

Among AMI patients, the no reflow rate was nonsignificantly higher in post-dilation group than in non-post-dilation group (1.7% vs 1.3%, $P = .53$). The 1-year cumulative incidence of death, MI, and death/MI were significantly higher in AMI patients who underwent post-

dilation compared with AMI patients not receiving post-dilation (Table II). Kaplan-Meier survival curves are shown in Figure 2 for death/MI. There were no significant differences in repeat revascularization rates between the 2 treatment groups. After propensity score adjustment in Cox model, post-dilation was associated with a significantly higher risk of death/MI among AMI patients (HR = 1.78, 95% CI 1.12-2.83, $P = .01$) (Table III). Nonsignificantly higher risks for each of the other adverse events except for a nonsignificantly lower risk of CABG were observed among patients receiving post-dilation relative to patients not receiving post-dilation.

The restricted analysis on 1,039 patients with a single lesion treated showed similar effect estimates after additional adjustment for lesion characteristics. In addition, results were consistent when the analysis was stratified by STEMI vs non-STEMI patients (data not shown). The no reflow rate was similar in post-dilation group and non-post-dilation group (2.1% vs 1.5%, $P = .45$). The TIMI grades were improved after PCI procedure when compared with before PCI procedure regardless of post-dilation status (Table IV).

Among non-AMI patients, the no reflow rate was similar in post-dilation group and non-post-dilation group (0.5% vs 0.5%, $P = .84$). There were no significant differences in cumulative incidence of death, MI, repeat PCI, CABG, death/MI, or repeat revascularization by post-dilation status (Table II). Kaplan-Meier survival curves are shown in Figure 3 for death/MI. The propensity score-adjusted risks for adverse cardiac events by 1 year also were not significantly different between 2 treatment groups (Table III).

The restricted analysis on 2,179 patients with a single lesion treated showed similar effect estimates after additional adjustment for lesion characteristics. The no reflow rate was similar in post-dilation group and non-post-dilation group (0.7% vs 0.6%, $P = .76$). The TIMI grades were improved after PCI procedure when compared with before PCI procedure regardless of post-dilation status (Table IV).

Discussion

The major finding of the present study is that the risk of death/MI associated with post-dilation during PCI differs between patients presenting with AMI and those with indications other than AMI. Post-dilation was associated with a nearly 2-fold propensity score-adjusted risk of death/MI in AMI patients, whereas no such relationship was apparent in non-AMI patients. No appreciable association was observed between post-dilation and other cardiovascular outcomes, including repeat revascularization, in either the AMI or the non-AMI patients.

Although this study cannot identify a mechanism for the apparent differential association between post-dilation and outcomes among AMI patients and non-AMI patients, distal embolization due to aggressive mechanical expansion from post-dilation may be a possible underlying mechanism. Patients presenting with AMI have plaques that are more thrombotic and ulcerated than those seen in patients who present for PCI with indications other than AMI. Studies from the TIMI group suggest that stent expansion to diameters greater than the reference segment is associated with higher risk of mortality and lower myocardial perfusion.¹⁶ And direct stenting in selected AMI patients has been suggested as a strategy to avoid microvascular injury because of predilation.^{17,18} This phenomenon, consistent with results in our study, suggests that greater stent expansion, whether through post-dilation or with aggressive sizing during direct stenting, may result in distal embolization, which in turn can predispose to adverse clinical outcomes. Consistent with this premise is that previous studies have shown that aggressive stent expansion is associated with higher periprocedural CK-MB release¹⁹ and angiographic no-reflow.²⁰ On the other hand, the separation in 1-year

death/MI curves in the AMI group tended to occur between 90 and 180 days after PCI. This is only partially consistent with a very early increase in MI events possibly related to release of enzymes and slow flow. Again, our study cannot identify the precise mechanism(s) associated with the higher rate of death/MI among AMI patients who received post-dilation.

Among patients presenting with indications other than AMI, no significant associations between post-dilation and risk of adverse cardiac events were identified. Non-AMI patients undergoing post-dilation had slightly lower risk of death and slightly higher risk of MI than comparable nondilated patients. Post-dilation may improve stent expansion within a certain period,^{1,2} but the risk of repeat PCI and CABG was not reduced.

Few studies have been designed to assess the effects of post-dilation on long-term clinical outcomes. To our knowledge, no published studies examine post-dilation among AMI and non-AMI patients separately. A randomized, multicenter clinical trial carried out between 1995 and 1996 in Austria evaluated whether high-pressure post-dilation of Wiktor stents provided short- and long-term benefits compared to the conventional low-pressure implantation without post-dilation. Their results showed that initial lumen gain by high-pressure post-dilation did not translate into improved long-term clinical outcomes (ie, death, MI, or target lesion revascularization).²¹ This trial indirectly supports our results of a lack of association with reduced need for repeat revascularization with post-dilation. This trial, however, does not support our findings of an increased hazard with post-dilation. One possible explanation for this is that patients with evidence of intracoronary thrombus were excluded from their study. Another possible explanation is that the trial did not examine post-dilation effect in patients presenting with AMI and non-AMI separately.

Study limitations

Our study has limitations. First, the Dynamic Registry was not designed specifically to address the question of differential associations involving post-dilation in AMI and non-AMI patients. Although we considered an extensive array of factors potentially associated with the decision to post-dilate, post-dilation may be still a marker for a more complex patient, whose prognosis will be worse than that of a patient who does not require post-dilation. In light of this, the adverse association between post-dilation and death/MI that we observed among AMI patients could be systematically tested through the conduct of a large randomized clinical trial. In the absence of clinical trial data, the present observational study found an adverse effect associated with the use of post-dilation among AMI patients, and essentially no evidence of benefit in these patients. Second, the absolute number of hard clinical events was relatively low in our study (ie, suboptimal statistical power), which tempers the extent to which firm conclusions can be made. Third, although we postulated a mechanism to explain the higher risk of death/MI, some important angiographic and ultrasonic characteristics are missing; we did not observe significant difference in final TIMI flow between post-dilation group and non-post-dilation group, as would be expected if the observed harmful effect was related to distal embolization; this has to be interpreted with caution, given that in our study TIMI flow rates were operator-reported and not obtained from core laboratory analysis. Nevertheless, further research to identify the underlying mechanism(s) is warranted but not possible using the data available in our registry. Lastly, although the difference in lesion characteristics between the 2 treatment groups could explain some of the observed differences, it is unlikely that this is the case, as we made adjustments in a multivariable model restricting the analysis to patients with a single lesion treated.

Conclusions

Our study shows that in patients undergoing PCI, stent post-dilation in patients presenting with AMI is associated with an increased hazard of death/MI at 1 year, which is not seen in those presenting with conditions other than AMI. Furthermore, stent post-dilation was not associated with an appreciable benefit in reducing the need for repeat revascularization in either AMI or non-AMI patients. Further investigation is warranted on this topic.

Acknowledgments

Disclosures

This work was supported by grants HL-33292-14 and HL-33292-16 from the National Heart, Lung, and Blood Institute.

Appendix A. Variables used in Cox model for adjustment to test the significance of the interaction term

Gender, age, race, enrollment wave, body mass index (BMI), insurance, prior history of PCI, MI, diabetes, congestive heart failure (CHF), and severe noncardiac diseases (including cerebrovascular disease, peripheral vascular disease, pulmonary disease, cancer, renal failure or other severe disease), CHF at admission, ad hoc procedure, periprocedural IIb/IIIa antagonist, vessel disease, statin, or other cholesterol-lowering drugs at discharge.

Appendix B. Variables used in the propensity score models

AMI Patients

Gender, enrollment wave, history of PCI, history of severe noncardiac diseases, history of CHF, CHF at admission, vessel disease, ad hoc procedure, drug-eluting stent use, periprocedural IIb/IIIa receptor antagonist, angiographic success.

Non-AMI Patients

Gender, Hispanic ethnicity, age, enrollment wave, insurance, body mass index, procedure circumstance, ad hoc procedure, history of PCI, MI, diabetes, hypertension, CHF, hypercholesterolemia, vessel disease, drug-eluting stent use, procedure indication, periprocedural aspirin, clopidogrel, IIb/IIIa receptor antagonist.

References

1. Blackman DJ, Porto I, Shirodaria C, et al. Usefulness of high-pressure post-dilatation to optimize deployment of drug-eluting stents for the treatment of diffuse in-stent coronary restenosis. *Am J Cardiol* 2004;7:922–5. [PubMed: 15464678]
2. Johansson B, Allared M, Borgencrantz B, et al. Standardized angiographically guided over-dilatation of stents using high pressure technique optimize results without increasing risks. *J Invasive Cardiol* 2002;5:221–6. [PubMed: 11983940]
3. Brodie BR, Cooper C, Jones M, et al. Is adjunctive balloon postdilatation necessary after coronary stent deployment? Final results from the POSTIT trial [see comment]. *Catheter Cardiovasc Interv* 2003;2:184–92. [PubMed: 12772236]
4. Cheneau E, Leborgne L, Mintz GS, et al. Predictors of subacute stent thrombosis: results of a systematic intravascular ultrasound study. *Circulation* 2003;1:43–7. [PubMed: 12821553]

5. Fujii K, Carlier SG, Mintz GS, et al. Stent underexpansion and residual reference segment stenosis are related to stent thrombosis after sirolimus-eluting stent implantation: an intravascular ultrasound study. *J Am Coll Cardiol* 2005;7:995–8. [PubMed: 15808753]
6. Sonoda S, Morino Y, Ako J, et al. Impact of final stent dimensions on long-term results following sirolimus-eluting stent implantation: serial intravascular ultrasound analysis from the sirius trial. *J Am Coll Cardiol* 2004;11:1959–63. [PubMed: 15172398]
7. Fitzgerald PJ, Oshima A, Hayase M, et al. Final results of the Can Routine Ultrasound Influence Stent Expansion (CRUISE) study. *Circulation* 2000;5:523–30. [PubMed: 10920064]
8. Stone GW, Webb J, Cox DA, et al. Distal microcirculatory protection during percutaneous coronary intervention in acute ST-segment elevation myocardial infarction: a randomized controlled trial. *JAMA* 2005;9:1063–72. [PubMed: 15741528]
9. Limbruno U, De Carlo M, Pistoletti S, et al. Distal embolization during primary angioplasty: histopathologic features and predictability. *Am Heart J* 2005;1:102–8. [PubMed: 16084155]
10. van 't Hof AW, Liem A, Suryapranata H, et al. Angiographic assessment of myocardial reperfusion in patients treated with primary angioplasty for acute myocardial infarction: myocardial blush grade. Zwolle Myocardial Infarction Study Group. *Circulation* 1998;23:2302–6. [PubMed: 9639373]
11. Stone GW, Peterson MA, Lansky AJ, et al. Impact of normalized myocardial perfusion after successful angioplasty in acute myocardial infarction. *J Am Coll Cardiol* 2002;4:591–7. [PubMed: 11849856]
12. De Luca G, van 't Hof AW, Ottervanger JP, et al. Unsuccessful reperfusion in patients with ST-segment elevation myocardial infarction treated by primary angioplasty. *Am Heart J* 2005;3:557–62. [PubMed: 16169340]
13. Marroquin OC, Selzer F, Mulukutla SR, et al. A comparison of bare-metal and drug-eluting stents for off-label indications. *N Engl J Med* 2008;4:342–52. [PubMed: 18216354]
14. Zhang ZJ, Marroquin OC, Weissfeld JL, et al. Beneficial effects of statins after percutaneous coronary intervention. *Eur J Cardiovasc Prev Rehabil* 2009;4:445–50. [PubMed: 19369875]
15. Rosenbaum PR, Rubin DB. The central role of the propensity score in observational studies for causal effects. *Biometrika* 1983;70:41–55.
16. Gibson CM, Kirtane AJ, Boundy K, et al. Association of a negative residual stenosis following rescue/adjunctive percutaneous coronary intervention with impaired myocardial perfusion and adverse outcomes among ST-segment elevation myocardial infarction patients. *J Am Coll Cardiol* 2005;3:357–62. [PubMed: 15680712]
17. Loubeyre C, Morice MC, Lefevre T, et al. A randomized comparison of direct stenting with conventional stent implantation in selected patients with acute myocardial infarction. *J Am Coll Cardiol* 2002;1:15–21. [PubMed: 11755281]
18. Ozdemir R, Sezgin AT, Barutcu I, et al. Comparison of direct stenting versus conventional stent implantation on blood flow in patients with ST-segment elevation myocardial infarction. *Angiology* 2006;4:453–8. [PubMed: 17022381]
19. Iakovou I, Mintz GS, Dangas G, et al. Increased CK-MB release is a “trade-off” for optimal stent implantation: an intravascular ultrasound study. *J Am Coll Cardiol* 2003;11:1900–5. [PubMed: 14662249]
20. Maekawa Y, Asakura Y, Anzai T, et al. Relation of stent overexpansion to the angiographic no-reflow phenomenon in intravascular ultrasound-guided stent implantation for acute myocardial infarction. *Heart Vessels* 2005;1:13–8. [PubMed: 15700197]
21. Yang P, Gyongyosi M, Hassan A, et al. Short- and long-term outcomes of Wiktor stent implantation at low versus high pressures. Austrian Wiktor Stent Study Group. *Am J Cardiol* 1999;6:644–9. [PubMed: 10498132]

Figure 1.
The selection process from Dynamic Registry waves 3, 4, and 5.

Figure 2.

Probability density functions of the propensity scores for patients receiving and not receiving post-dilation. The solid line depicts the propensity score among patients receiving post-dilation; the dashed line depicts the propensity score among patients not receiving post-dilation. The similar shape and the large overlapping area between the 2 curves indicate good comparability between the 2 treatment groups in terms of observed covariates. Panel A shows the subgroup of 1,358 AMI patients and panel B shows the subgroup of 3,001 non-AMI patients.

Figure 3. The Kaplan-Meier survival curves for death/MI among (A) AMI patients and (B) non-AMI patients. The solid line represents the survival of patients receiving post-dilation and the dashed line represents the survival of patients not receiving post-dilation.

Patient demographics, disease history, and lesion and procedural characteristics of 1220 AMI patients and 2608 non-AMI patients, by post-dilation status

Table 1

Characteristic	AMI patients			Non-AMI patients		
	No post-dilation (n = 772)	Post-dilation (n = 586)	P*	No post-dilation (n = 1720)	Post-dilation (n = 1281)	P*
Female	37.0	37.2	.96	35.7	35.8	.98
Mean age (y)	60.1(12.7)	61.7(12.9)	.02	64.2(11.6)	63.5(11.5)	.08
Prior intervention	13.5	15.5	.33	31.3	34.5	.07
Prior MI	14.0	15.1	.59	23.3	23.2	.96
Severe noncardiac disease	26.8	33.9	<.01	37.1	34.4	.12
History of diabetes	23.0	26.0	.21	31.8	34.0	.21
History of chest pain	26.7	29.3	.30	61.5	59.7	.32
Chest pain at admission	90.8	89.9	.59	62.0	61.3	.69
History of congestive heart failure	4.4	8.1	.01	9.5	8.4	.27
Congestive heart failure at admission	8.3	6.7	.26	4.6	5.6	.24
History of hypertension	63.9	67.2	.21	79.9	75.7	.01
History of hypercholesterolemia	60.9	59.6	.64	76.6	73.3	.04
No. of coronary artery narrowing >50%			.03			<.01
1	45.3	38.1		45.6	40.3	
2	32.1	35.3		35.6	36.1	
3	22.6	26.6		18.9	23.6	
Procedure indication			.87			.92
ST-elevation MI	53.5	53.9		—	—	
Non-ST-elevation MI	46.5	46.1		—	—	
Unstable angina	—	—		48.6	48.3	
Stable angina	—	—		28.5	28.1	
Asymptomatic coronary artery disease or others	—	—		23.0	23.6	
Procedure circumstance			.93			.06
Elective	21.6	21.1		67.9	71.0	
Urgent	43.8	43.3		32.2	29.0	
Emergent	34.7	35.6		0.0	0.0	

Characteristic	AMI patients			Non-AMI patients		
	No post-dilation (n = 772)	Post-dilation (n = 586)	P*	No post-dilation (n = 1720)	Post-dilation (n = 1281)	P*
Any drug-eluting stent	48.6	57.9	.001	54.4	67.2	<.0001
Periprocedural GP IIb/IIIa receptor antagonist	60.5	59.9	.83	34.4	35.5	.51
Any evidence of thrombus	40.9	39.7	.66	5.1	7.1	.02
Any calcification	21.0	25.5	.05	24.2	31.2	<.0001
Any ulceration	20.9	26.0	.03	8.6	11.7	<.01
Any ostial lesion	4.3	8.4	<.01	6.6	8.6	.04
Any type C lesion	25.2	31.7	.01	18.0	27.2	<.0001

Categorical variables are presented as column percentages and continuous variables are presented as mean (SD). GP, Glycoprotein.

* P value of tests for covariate balance between post-dilated and non-post-dilated patients before propensity score adjustment.

† P value of tests for covariate balance between post-dilated and non-post-dilated patients after propensity score adjustment.

Table II
One-year cumulative incident proportions of adverse events in 1220 patients with AML, by post-dilation status

Event	All AMI patients					Non-AMI patients				
	No post-dilation (n = 772)		Post-dilation (n = 586)		P*	No post-dilation (n = 1720)		Post-dilation (n = 1281)		P*
	n	%	n	%		n	%	n	%	
Death	14	2.0	22	4.1	.03	50	3.2	32	2.7	.45
MI	18	2.5	25	4.7	.05	42	2.7	36	3.0	.57
Death/MI	31	4.3	46	8.5	<.01	90	5.7	66	5.5	.87
Repeat percutaneous coronary intervention	60	8.6	56	10.6	.28	139	9.0	119	10.2	.31
Coronary artery bypass graft surgery	12	1.7	8	1.5	.76	32	2.1	30	2.5	.40
Repeat revascularization	70	10.0	65	12.2	.25	165	10.7	142	12.1	.23

* P values are by log-rank test for unadjusted 1-year cumulative incident proportions.

Table III
Propensity score-adjusted hazard ratios of adverse events associated with the use of post-dilation versus no post-dilation among AMI patients

Adverse Event	AMI patients [*]			Non-AMI patients [*]		
	Hazard ratio	95% CI	P	Hazard ratio	95% CI	P
Death	1.80	0.91-3.56	.09	0.94	0.59-1.50	.79
MI	1.71	0.92-3.17	.09	1.28	0.81-2.02	.30
Death/MI	1.78	1.12-2.83	.01	1.08	0.77-1.50	.67
Repeat percutaneous coronary intervention	1.14	0.78-1.65	.50	1.16	0.90-1.49	.24
Coronary artery bypass graft surgery	0.91	0.37-2.27	.84	1.25	0.75-2.08	.39
Repeat revascularization	1.15	0.81-1.62	.43	1.17	0.93-1.47	.19

^{*} Model adjusted for propensity score as a continuous covariate. Refer Appendix A and B for listing of variables in the propensity score.

Table IV
TIMI flow before and after PCI procedure among AMI patients and non-AMI patients with 1 lesion treated

TIMI grade	AMI patients						Non-AMI patients					
	TIMI before PCI			TIMI after PCI			TIMI before PCI			TIMI after PCI		
	No post-dilation		Post-dilation	No post-dilation		Post-dilation	No post-dilation		Post-dilation	No post-dilation		Post-dilation
	n	%	n	n	%	n	n	%	n	n	%	n
Grade 0	179	30.3%	113	26.7%	0	0.0%	41	3.2%	40	4.8%	0	0.0%
Grade 1	29	4.9%	15	3.5%	2	0.3%	36	2.8%	18	2.2%	2	0.2%
Grade 2	82	13.9%	58	13.7%	16	2.7%	105	8.2%	62	7.4%	9	0.7%
Grade 3	300	50.9%	238	56.1%	584	97.9%	1092	85.7%	715	85.6%	1303	92.8%