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Research Article

Association of End-of-Procedural Angiographic Response with 1-Year Mortality after Primary Percutaneous Coronary Intervention in ST-Segment Elevation Myocardial Infarction: A Retrospective Cohort Study

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Abstract

Objectives: We evaluated whether the final angiographic result after primary percutaneous coronary intervention (PCI) was associated with 1-year all-cause mortality in patients with ST-segment elevation myocardial infarction (STEMI).

Methods: This retrospective single-center cohort study included 222 consecutive patients with STEMI who underwent primary PCI between January 2013 and December 2015. Final thrombolysis in myocardial infarction (TIMI) flow, residual TIMI thrombus grade, and myocardial blush grade (MBG) were recorded at the end of the procedure. An end-of-procedural angiographic response phenotype was defined before survival modeling. Optimal response was defined as final TIMI flow 3, residual TIMI thrombus grade 0–1, and MBG 2–3. Suboptimal response was defined as any of the following: final TIMI flow <3, residual TIMI thrombus grade ≥2, or MBG ≤1. Vital status at 1 year was obtained from the national death registry (e-Nabız). Kaplan–Meier analysis and Cox proportional hazards regression were used.

Results: The mean age was 57.9±13.6 years, and 173 patients (77.9%) were male. The median overall follow-up was 620 days (interquartile range, 443–813 days), whereas survival analyses were restricted to the first 365 days after index PCI. In-hospital mortality occurred in 23 patients (10.4%), and 1-year all-cause mortality occurred in 33 (14.9%). Compared with survivors, patients who died by 1 year had lower final TIMI flow, higher residual TIMI thrombus grade, and lower MBG (all p<0.001). The suboptimal angiographic response phenotype was present in 50 patients (22.5%) and was associated with markedly higher 1-year mortality than optimal response (46.0% vs. 5.8%; log-rank p<0.001). In multivariable Cox analysis adjusted for age, diabetes mellitus, Killip class, and left ventricular ejection fraction (LVEF), suboptimal angiographic response remained independently associated with 1-year mortality (hazard ratio 8.50, 95% confidence interval 3.81–18.94; p<0.001).

Conclusion: Simple end-of-procedural angiographic markers were strongly associated with 1-year mortality after STEMI. A suboptimal angiographic response phenotype may support early post-PCI risk stratification in routine practice.

Keywords: Coronary Angiography, Mortality, Myocardial Infarction, No-reflow Phenomenon, Percutaneous Coronary Intervention, ST-elevation

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Primary percutaneous coronary intervention (PCI) is the standard reperfusion strategy for ST-segment elevation myocardial infarction (STEMI) when it can be delivered without delay.[1] Even after successful opening of the infarct-related artery, however, downstream tissue reperfusion may remain incomplete because of distal embolization, persistent thrombus burden, endothelial injury, and microvascular dysfunction.[2] These phenomena are clinically relevant because they are linked to larger infarct size, impaired myocardial recovery, and worse survival.[3]

The angiographic variables most often used to describe final reperfusion are the thrombolysis in myocardial infarction (TIMI) flow grade,[4] residual TIMI thrombus grade,[5] and myocardial blush grade (MBG).[6] These measures are available immediately at the end of primary PCI and are already familiar to interventional cardiologists. In daily practice, they may offer a practical way to distinguish patients with an apparently successful procedure from those who remain at high residual risk despite epicardial patency.

Recent work on no-reflow in STEMI has focused largely on pre-procedural or procedural prediction models.[7] Less attention has been given to the prognostic value of the final angiographic result as an integrated post-procedural phenotype. We therefore examined whether end-of-procedural angiographic response, defined by final TIMI flow, residual thrombus burden, and MBG, was associated with 1-year all-cause mortality after primary PCI in STEMI.

Methods

Study design and population

This retrospective observational cohort study included consecutive patients with STEMI who underwent primary PCI at Bagcilar Training and Research Hospital between January 2013 and December 2015. Patients with unavailable core angiographic data or unavailable survival status were excluded. The final study population consisted of 222 patients. The manuscript was prepared in accordance with the STROBE statement.[8] The approval for this study was obtained from the Bağcılar Training and Research Hospital Non-Invasive Clinical Research Ethics Committee (decision no: 2015-423, dated 26.11.2015, 38th board meeting). The study was conducted in accordance with the Declaration of Helsinki. Written informed consent was waived because of the retrospective study design.

The diagnosis of STEMI, the decision to perform primary PCI, and intraprocedural pharmacologic management were made by the treating cardiology team according to the routine standards of the study period. Baseline demographic, clinical, laboratory, procedural, and angiographic variables were obtained from the institutional database.

Angiographic assessment

Epicardial flow was graded with the TIMI flow scale.[4] Thrombus burden was assessed with the TIMI thrombus grade classification.[5] Myocardial perfusion was graded with MBG.[6] The angiographic no-reflow variable was retained as recorded in the institutional dataset.

For the primary analysis, an exploratory end-of-procedural angiographic response phenotype was defined before survival modeling. Optimal angiographic response was defined as final TIMI flow 3, residual TIMI thrombus grade 0 to 1, and MBG 2 to 3. Suboptimal angiographic response was defined as any of the following: final TIMI flow below 3, residual TIMI thrombus grade 2 or higher, or MBG 1 or lower.

Follow-up and outcomes

The primary outcome was 1-year all-cause mortality. Survival status was ascertained through the national death registry (e-Nabız). Follow-up time was calculated from the index PCI date to the date of death or the date of the last confirmed status. Secondary outcomes were in-hospital mortality, cardiovascular death during follow-up, recurrent myocardial infarction, repeat revascularization, stroke, and heart failure hospitalization.

Because the study used retrospectively collected clinical and angiographic data, no additional intervention was performed for research purposes.

Statistical Analysis

Continuous variables are presented as mean ± standard deviation or median (interquartile range), as appropriate, and categorical variables are presented as number (percentage). Group comparisons were performed with the Mann–Whitney U test for continuous variables and the chi-square test or Fisher's exact test for categorical variables, as appropriate.

Kaplan–Meier curves were compared with the log-rank test. Cox proportional hazards regression was used to estimate hazard ratios (HRs) with 95% confidence intervals (CIs). Given the limited number of events, the multivariable model was kept parsimonious and prespecified to include the angiographic response phenotype together with age, diabetes mellitus, Killip class, and LVEF. Survival analyses were restricted to the first 365 days after index PCI, although longer follow-up data were available for censoring. Statistical analyses were performed with Python 3.13, and a two-sided *p*-value $p<0.05$ was considered statistically significant.

Results

Cohort characteristics

The study cohort included 222 patients. The mean age was 57.9±13.6 years, and 173 patients (77.9%) were male. The

median overall follow-up duration was 620 days (interquartile range, 443–813 days). Thirty-three patients (14.9%) died within 365 days, and all observed deaths occurred during the first year of follow-up. Baseline clinical characteristics according to 1-year vital status are summarized in Table 1.

Patients who died by 1 year had more frequent cardiogenic shock at presentation (36.4% vs. 2.6%; p<0.001), higher Killip class (2.61±1.32 vs. 1.59±0.89; p<0.001), lower systolic blood pressure (109.09±25.07 vs. 125.07±19.98 mmHg; p<0.001), and lower LVEF (35.97±13.43% vs. 43.96±10.44%; p=0.001). Age, sex, diabetes mellitus, hypertension, previous myocardial infarction, previous PCI, chronic kidney disease, and heart rate were not significantly different between groups.

Procedural and angiographic findings

Procedural and angiographic findings are shown in Table 2. In-hospital mortality occurred in 23 patients (10.4%), and angiographic no-reflow occurred in 14 (6.3%). Pre-PCI TIMI flow and pre-PCI thrombus grade were similar between groups, whereas final angiographic metrics showed clear separation.

Compared with survivors, patients who died by 1 year had lower final TIMI flow (2.55±0.90 vs. 2.97±0.18; p<0.001), higher residual TIMI thrombus grade (2.03±1.40 vs. 0.46±0.70; p<0.001), lower MBG (1.42±1.06 vs. 2.71±0.45; p<0.001), more frequent angiographic no-reflow (24.2% vs. 3.2%; p<0.001), and more frequent suboptimal angiographic response (69.7% vs. 14.3%; p<0.001).

Clinical outcomes according to angiographic response phenotype

The exploratory suboptimal angiographic response phenotype was present in 50 patients (22.5%). Clinical outcomes according to angiographic response phenotype are presented in Table 3.

Patients with suboptimal response had substantially higher in-hospital mortality (46.0% vs. 0.0%; p<0.001) and 1-year all-cause mortality (46.0% vs. 5.8%; p<0.001) than those with optimal response. Cardiovascular death during follow-up was also more frequent in the suboptimal group (46.0% vs. 4.7%; p<0.001). Recurrent myocardial infarction, repeat revascularization, and stroke were numerically similar between groups, whereas heart failure hospitalization occurred more often in the suboptimal response group (22.0% vs. 9.3%; p=0.025).

Survival analysis

Kaplan–Meier analysis demonstrated early and persistent separation of the survival curves according to angiographic response phenotype (Fig. 1), with significantly lower 1-year survival in the suboptimal response group (log-rank p<0.001).

In univariable Cox analysis, suboptimal angiographic response was associated with 1-year mortality (HR 11.31, 95% CI 5.36–23.84; p<0.001). Killip class and systolic blood pressure were also associated with mortality, whereas higher LVEF was associated with lower risk. In the prespecified multivariable model, suboptimal angiographic response remained independently associated with 1-year all-cause mortality (adjusted HR 8.50, 95% CI 3.81–18.94; p<0.001). Killip class remained independently associated with higher risk (adjusted HR 1.48, 95% CI 1.11–1.98; p=0.007), and higher LVEF remained independently associated with lower risk (adjusted HR 0.97, 95% CI 0.94–1.00; p=0.043) (Table 4).

Table 1. Baseline clinical characteristics according to one-year vital status

Variable	Alive at 1 year (n=189)	Dead by 1 year (n=33)	p
Age, years	57.41±13.39	60.61±14.81	0.152
Male sex	146 (77.2)	27 (81.8)	0.654
Diabetes mellitus	55 (29.1)	10 (30.3)	1.000
Hypertension	104 (55.0)	14 (42.4)	0.192
Previous myocardial infarction	22 (11.6)	5 (15.2)	0.566
Previous PCI	22 (11.6)	5 (15.2)	0.566
Chronic kidney disease	18 (9.5)	2 (6.1)	0.745
Cardiogenic shock at presentation	5 (2.6)	12 (36.4)	<0.001
Killip class	1.59±0.89	2.61±1.32	<0.001
Systolic blood pressure, mmHg	125.07±19.98	109.09±25.07	<0.001
Heart rate, bpm	80.44±18.93	82.48±20.27	0.511
LVEF, %	43.96±10.44	35.97±13.43	0.001

Values are mean±standard deviation or number (percentage). LVEF: Left ventricular ejection fraction; PCI: Percutaneous coronary intervention.

Table 2. Procedural and angiographic findings according to one-year vital status

Variable	Alive at 1 year (n=189)	Dead by 1 year (n=33)	p
Multivessel disease	67 (35.4)	16 (48.5)	0.174
Thrombus aspiration	58 (30.7)	12 (36.4)	0.545
Glycoprotein IIb/IIIa inhibitor use	75 (39.7)	10 (30.3)	0.338
Pre-PCI TIMI flow	0.59±0.88	0.48±0.80	0.585
Final TIMI flow	2.97±0.18	2.55±0.90	<0.001
Pre-PCI TIMI thrombus grade	3.96±0.93	3.76±0.90	0.189
Residual TIMI thrombus grade	0.46±0.70	2.03±1.40	<0.001
Myocardial blush grade	2.71±0.45	1.42±1.06	<0.001
Angiographic no-reflow	6 (3.2)	8 (24.2)	<0.001
Suboptimal angiographic response*	27 (14.3)	23 (69.7)	<0.001

*Suboptimal angiographic response was defined as final TIMI flow <3, residual TIMI thrombus grade ≥2, or myocardial blush grade ≤1. TIMI: Thrombolysis in myocardial infarction; PCI: Percutaneous coronary intervention.

Table 3. Clinical outcomes according to end-of-procedural angiographic response phenotype

Outcome	Optimal response (n=172)	Suboptimal response (n=50)	p
In-hospital death	0 (0.0)	23 (46.0)	<0.001
1-year all-cause mortality	10 (5.8)	23 (46.0)	<0.001
Cardiovascular death during follow-up	8 (4.7)	23 (46.0)	<0.001
Recurrent myocardial infarction	7 (4.1)	3 (6.0)	0.698
Repeat revascularization	12 (7.0)	3 (6.0)	1.000
Stroke	5 (2.9)	2 (4.0)	0.656
Heart failure hospitalization	16 (9.3)	11 (22.0)	0.025

Suboptimal angiographic response was defined as final TIMI flow <3, residual TIMI thrombus grade ≥2, or myocardial blush grade ≤1.

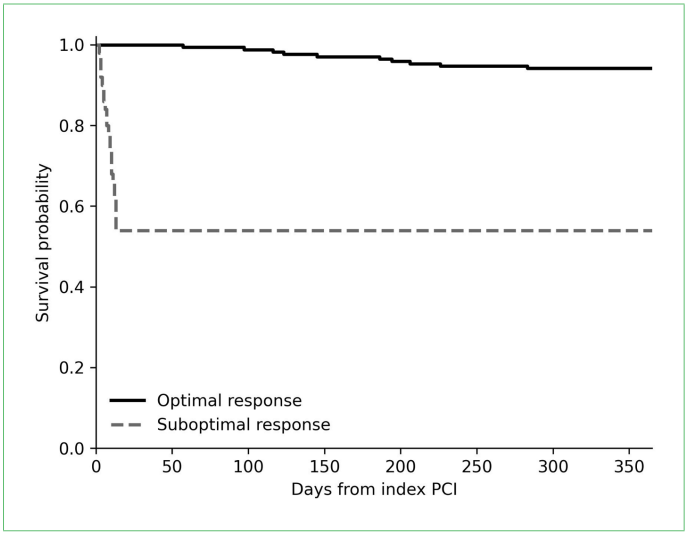

Figure 1. Kaplan-Meier estimate of 1-year survival according to end-of-procedural angiographic response phenotype. Suboptimal response was defined as final TIMI flow <3, residual TIMI thrombus grade ≥2, or myocardial blush grade ≤1.

Discussion

In this retrospective STEMI cohort treated with primary PCI, the final angiographic result was strongly associated with subsequent mortality. Patients with lower final TIMI flow, higher residual thrombus burden, and lower myocardial blush had worse outcomes, and the composite suboptimal angiographic response phenotype remained independently associated with 1-year all-cause mortality after adjustment for age, diabetes mellitus, Killip class, and LVEF.

These findings are biologically plausible. Final TIMI flow reflects epicardial reperfusion.[4] Residual thrombus grade reflects the remaining thrombotic burden after intervention.[5] MBG reflects tissue-level myocardial perfusion.[6] Persistent abnormalities in any of these domains may indicate incomplete reperfusion despite technically successful PCI, with ongoing distal embolization, microvascular obstruction, or impaired downstream flow. From a clinical perspective, the final angiographic picture therefore captures more than a procedural endpoint; it may summarize the residual

Table 4. Cox regression analysis for one-year all-cause mortality

Predictor	Univariable HR (95% CI)	p	Adjusted HR (95% CI)	p
Suboptimal angiographic response	11.31 (5.36-23.84)	<0.001	8.50 (3.81-18.94)	<0.001
Killip class	2.12 (1.60-2.80)	<0.001	1.48 (1.11-1.98)	0.007
Systolic blood pressure (per mmHg)	0.97 (0.95-0.98)	<0.001		
LVEF (per %)	0.94 (0.91-0.97)	<0.001	0.97 (0.94-1.00)	0.043
Age (per year)			1.02 (1.00-1.05)	0.097
Diabetes mellitus			1.32 (0.62-2.84)	0.473

HR: Hazard ratio; CI: Confidence interval.

ischemic and thrombotic risk carried by the patient after revascularization.

Our results are consistent with prior work showing adverse prognostic implications of residual thrombus burden in STEMI.[9] Adverse associations between no-reflow and clinical outcomes have also been reported in meta-analytic data.[10] The present study adds a practical post-procedural perspective by integrating these familiar angiographic markers into a simple phenotype that can be recognized immediately in the catheterization laboratory. Unlike more elaborate prediction models, this approach does not require additional testing or delayed risk assessment.

This has potential implications for early clinical decision-making. A patient with suboptimal angiographic response after primary PCI may merit closer hemodynamic surveillance, more cautious discharge planning, and more intensive follow-up after the index hospitalization. The phenotype could also be useful in future studies that aim to enrich high-risk post-PCI populations or evaluate tailored secondary prevention strategies.

Conclusion

The study has several limitations. It was retrospective and single-center, which introduces the possibility of selection bias and limits generalizability. The cohort was relatively modest in size, and the number of deaths constrained the complexity of multivariable modeling. Angiographic variables were obtained from the institutional dataset rather than from a blinded core laboratory. The cohort reflects practice from 2013 to 2015, so contemporary interventional techniques and adjunctive pharmacotherapy may differ. In addition, the composite angiographic response phenotype should be viewed as exploratory and requires external validation before broader adoption.

Despite these limitations, the signal observed in this cohort was large, clinically coherent, and based on variables that are readily available in routine care. In conclusion, end-of-procedural angiographic response was strongly associated with 1-year mortality after primary PCI in STEMI. A simple phenotype based on final TIMI flow, residual thrombus grade, and myocardial blush may help identify patients at increased residual risk immediately after the procedure.

Disclosures

Ethics Committee Approval: The approval for this study was obtained from the Bağcılar Training and Research Hospital Non-Invasive Clinical Research Ethics Committee (decision no: 2015-423, dated 26.11.2015, 38th board meeting). The study was conducted in accordance with the Declaration of Helsinki.

Informed Consent: Written informed consent was waived because of the retrospective study design.

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