

# Angiographic and Clinical Outcomes Among Patients With Acute Coronary Syndromes Presenting With Isolated Anterior ST-Segment Depression

## A TRITON–TIMI 38 (Trial to Assess Improvement in Therapeutic Outcomes by Optimizing Platelet Inhibition With Prasugrel–Thrombolysis In Myocardial Infarction 38) Substudy

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**Objectives** This study sought to determine angiographic and clinical outcomes among patients with acute coronary syndrome (ACS) presenting with isolated anterior ST-segment depression on 12-lead electrocardiogram (ECG).

**Background** In patients with ACS, anterior ST-segment depression on 12-lead ECG may represent plaque rupture with: 1) acute thrombotic occlusion with elevation of cardiac biomarkers (+Tn); 2) a patent artery with +Tn; or 3) a patent artery with –Tn.

**Methods** The TRITON–TIMI 38 (Trial to Assess Improvement in Therapeutic Outcomes by Optimizing Platelet Inhibition with Prasugrel–Thrombolysis In Myocardial Infarction 38) enrolled 13,608 ACS patients. Those with isolated anterior (leads V<sub>1</sub> to V<sub>4</sub>) ST-segment depression were analyzed. Angiograms and ECGs were interpreted by local investigators.

**Results** There were 1,198 (8.8%) patients with isolated anterior ST-segment depression. Of those, 314 (26.2%) had an occluded culprit artery (TIMI flow grade 0/1) and +Tn, 641 (53.5%) had a patent culprit artery (TIMI flow grade 2/3) and +Tn, and 243 (20.3%) had TIMI flow grade 2/3 and –Tn. Among patients with an occluded artery, the culprit artery was most often the left circumflex artery (48.4%). The 30-day incidence of the composite of death and MI was significantly higher among patients with an occluded artery (8.6%) than among those with a patent culprit artery and either +Tn (6.3%) or –Tn (2.9%) (3-way  $p = 0.006$ ). Among patients with an occluded artery, the median time from ECG to percutaneous coronary intervention was 29.4 h (interquartile range 26.1 to 44.1 h).

**Conclusions** Among ACS patients presenting with isolated anterior ST-segment depression, over one-quarter had an occluded culprit artery and elevated cardiac biomarkers. These patients had significantly worse clinical outcomes, and few underwent urgent angiography. (J Am Coll Cardiol Intv 2010;3:806–11) © 2010 by the American College of Cardiology Foundation

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Prompt diagnosis and early reperfusion therapy among patients with ST-segment elevation myocardial infarction (STEMI) is associated with a reduction in morbidity and mortality (1–3) and relies on the principle that early reperfusion of an occluded coronary artery improves clinical outcomes. However, an estimated 2% to 12% of STEMI are initially missed at the time of presentation to the emergency department (4,5). Discharge from the emergency department, when compared with hospital admission, for patients with STEMI has been associated with a doubling of mortality (6). Current clinical practice relies heavily on initial electrocardiographic changes for the diagnosis of STEMI, and a significant portion of missed cases might be attributable to the limited sensitivity of the traditional 12-lead surface electrocardiogram (ECG), which samples a relatively small area of the upper anterior thorax for identifying coronary occlusion (7).

The limited sensitivity and specificity of the 12-lead ECG in diagnosing STEMI is exemplified by the clinical scenario of isolated anterior ST-segment depression. This electrocardiographic finding can represent pathophysiology across the spectrum of acute coronary syndrome (ACS): 1) plaque rupture with a patent artery and no elevation of cardiac biomarkers leading to unstable angina; 2) a patent artery supplying the anterior myocardium with elevated cardiac biomarkers (i.e., non-STEMI); or 3) acute thrombotic occlusion of the posterior circulation with elevated cardiac biomarkers (i.e., posterior STEMI). Previous studies have demonstrated that acute thrombotic occlusion of a vessel supplying the posterior wall is particularly challenging to diagnose, both because of its inconsistent presentation on the ECG and the relatively small contribution of the posterior wall to the QRS complex in the traditional anterior pre-cordial leads (8).

The goal of this analysis was to determine the angiographic and clinical outcomes among patients with ACS presenting with isolated anterior ST-segment depression on 12-lead ECG.

## Methods

The TRITON-TIMI 38 (Trial to Assess Improvement in Therapeutic Outcomes by Optimizing Platelet Inhibition with Prasugrel-Thrombolysis In Myocardial Infarction 38) enrolled patients with moderate- to high-risk ACS scheduled to undergo percutaneous coronary intervention (PCI) (9). The patient population has been previously described (10). Briefly, the inclusion criteria for patients with unstable

angina or non-STEMI were ischemic symptoms lasting 10 min or more and occurring within 72 h before randomization, a TIMI risk score of 3 or more (11), and either ST-segment deviation of 1 mm or more or elevated levels of a cardiac biomarker of necrosis. Patients with STEMI could be enrolled within 12 h after the onset of symptoms if primary PCI was planned or within 14 days after receiving medical treatment for STEMI.

For the present analysis, only patients with isolated anterior ST-segment depression in leads V<sub>1</sub> to V<sub>4</sub> were included. Patients with ST-segment elevation in other leads were excluded. Electrocardiograms and angiograms were interpreted locally by the enrolling physician. Flow in the culprit artery undergoing PCI was graded according to the TIMI flow grade (TFG) criteria (1). Patients were grouped into 3 categories based on initial TFG in the culprit artery and initial serum cardiac biomarkers: 1) TFG 0/1 and elevated cardiac biomarkers (+Tn); 2) TFG 2/3 and +Tn; and 3) TFG 2/3 and no elevation of cardiac biomarkers (–Tn). As an estimate of infarct size, the ratio of the peak serum concentration of creatine kinase-myocardial band fraction to the upper limit of normal was calculated among the groups.

As an internal control, the subset of patients with isolated inferior ST-segment depression was also evaluated for the presence or absence of elevated cardiac biomarkers and an occluded or patent culprit artery at the time of angiography.

All continuous variable values are reported as the mean ± SD.

The chi-square test was used for the analysis of categorical variables. The Kruskal-Wallis test was performed for the analysis of continuous variables. Bonferroni adjustments were made when appropriate. Hazard ratios were calculated using the Cox proportional hazards model. Multivariable logistic regression was used to adjust for differences in baseline characteristics. A p value of <0.05 was considered significant.

## Results

**Baseline characteristics.** There were 13,608 patients enrolled in the study, of whom 1,198 (8.8%) had isolated

### Abbreviations and Acronyms

- ACS** = acute coronary syndrome
- ECG** = electrocardiogram
- PCI** = percutaneous coronary intervention
- STEMI** = ST-segment elevation myocardial infarction
- TFG** = Thrombolysis In Myocardial Infarction flow grade
- TIMI** = Thrombolysis In Myocardial Infarction
- Tn** = cardiac biomarkers

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	TFG 0/1, +Tn (n = 314)	TFG 2/3, +Tn (n = 641)	TFG 2/3, -Tn (n = 243)	p Value
Age, yrs	62 (11.28)	63 (10.7)	63 (10.7)	0.517
Male sex	71%	66%	61%	0.066
White	97%	95%	97%	0.270
BMI, kg/m <sup>2</sup>	28 (4.6)	28 (5.0)	28 (4.2)	0.581
Medical history				
Diabetes mellitus	18%	23%	20%	0.210
Hypertension	60%	70%	72%	0.002
Hypercholesterolemia	50%	54%	60%	0.047
Prior MI	15%	15%	21%	0.092
Current smoker	33%	31%	30%	0.668
Prior CABG	3%	5%	7%	0.070
Multivessel disease	15%	13%	14%	0.361
Creatinine clearance, ml/min	101 (37.8)	100 (39.0)	97 (31.5)	0.440
Glycoprotein IIb/IIIa inhibitor use	56%	52%	25%	<0.001

Values presented as mean (SD) or % unless otherwise noted.  
BMI = body mass index; CABG = coronary artery bypass graft surgery; MI = myocardial infarction; TFG = Thrombolysis In Myocardial Infarction flow grade; Tn = cardiac biomarkers.

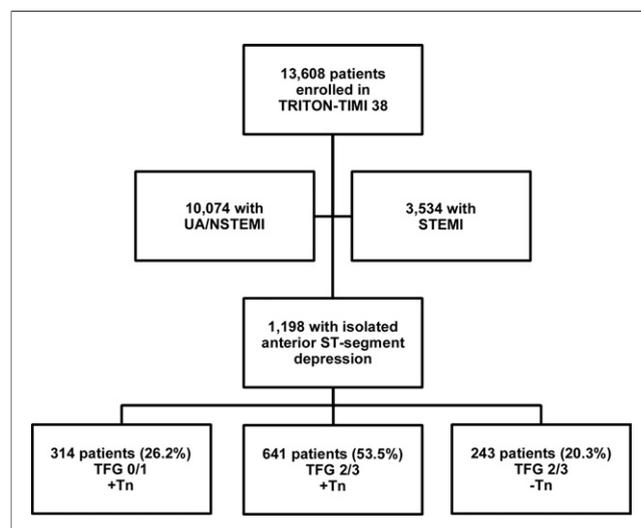
anterior ST-segment depression at presentation. Patients presenting with isolated anterior ST-segment depression and a patent culprit artery at the time of angiography had a higher incidence of hypertension and hypercholesterolemia than patients with an occluded artery did at the time of angiography (Table 1). Otherwise, the baseline characteristics were similar between the groups.

**Angiographic outcomes.** Of the 1,198 patients with isolated anterior ST-segment depression, 314 (26.2%) had an occluded culprit artery (TFG 0/1) at the time of angiography and +Tn at presentation, 641 (53.5%) had a patent culprit artery (TFG 2/3) at the time of angiography and +Tn at presentation, and 243 (20.3%) had a patent culprit artery at the time of angiography and -Tn at presentation (Fig. 1). None of the patients with an occluded artery had negative cardiac biomarkers. Among the 314 patients with an occluded culprit artery, the occluded artery was most often the left circumflex artery (n = 152, 48.4%), followed by the left anterior descending artery (n = 106, 33.8%) and the right coronary artery (n = 56, 17.8%) (Fig. 2).

Among the 270 patients who presented with isolated inferior ST-segment depression, 9 (3.3%) had an occluded culprit artery and +Tn, 226 (83.7%) had a patent culprit artery and +Tn, and 35 (13.0%) had a patent culprit artery and -Tn.

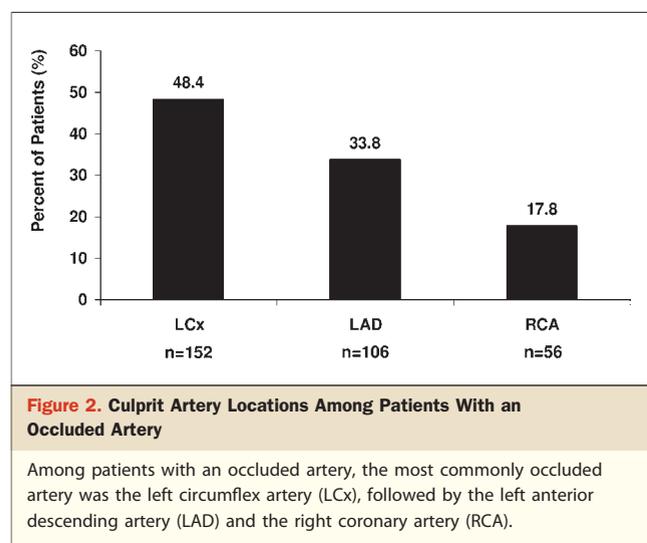
**Clinical outcomes.** The 30-day incidence of the composite of death and MI was significantly higher among patients with an occluded artery than among those with a patent culprit artery with either +Tn or -Tn (8.6%, 6.3%, and 2.9%, respectively; 3-way p = 0.006) (Fig. 3). Patients with an occluded artery and +Tn were significantly more likely to have death or MI than those with a patent artery and -Tn (hazard ratio [HR]: 3.06, 95% confidence interval [CI]:

1.33 to 7.03, p = 0.008) even after adjusting for differences in baseline characteristics (HR<sub>adjusted</sub>: 3.21, 95% CI: 1.37 to 7.49, p = 0.007). Patients with an occluded culprit artery and elevated cardiac biomarkers were more likely to have death and MI than those with a patent artery and elevated biomarkers (HR: 1.39, 95% CI: 0.85 to 2.26, p = 0.187), although the difference was not statistically significant. However, after adjusting for differences in baseline charac-



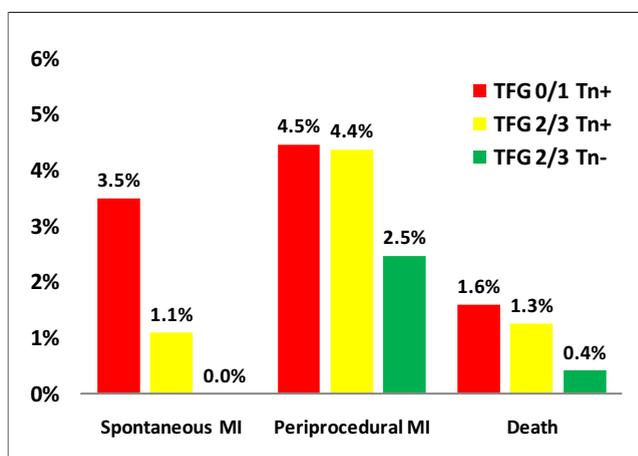
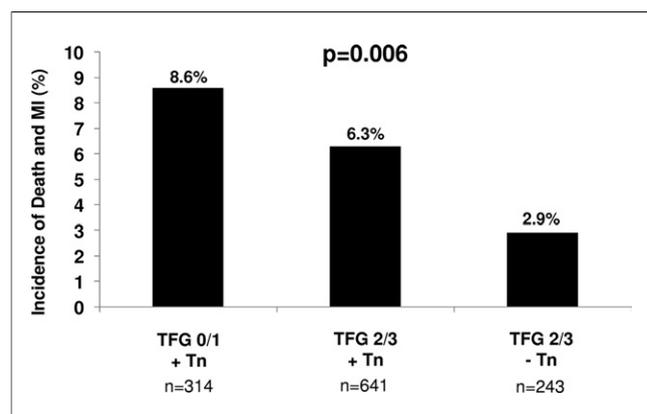
**Figure 1. Flow of Patients**

Of patients enrolled in the TRITON-TIMI 38 (Trial to Assess Improvement in Therapeutic Outcomes by Optimizing Platelet Inhibition with Prasugrel-Thrombolysis In Myocardial Infarction 38), 8.8% had isolated anterior ST-segment depression. NSTEMI = non-ST-segment elevation myocardial infarction; STEMI = ST-segment elevation myocardial infarction; TFG = Thrombolysis In Myocardial Infarction flow grade; Tn = cardiac biomarkers; UA = unstable angina.



teristics (HR<sub>adjusted</sub>: 1.61, 95% CI: 0.97 to 2.66, p = 0.062), the difference tended toward significance. Patients with a patent culprit artery and +Tn were also more likely to have death or MI than those with a patent artery and -Tn (HR: 2.20, 95% CI: 0.99 to 4.91, p = 0.054), and this difference was mildly attenuated after adjusting for differences in baseline characteristics (HR<sub>adjusted</sub>: 2.06, 95% CI: 0.91 to 4.64, p = 0.082).

There was no statistically significant difference in the incidence of periprocedural MI (3-way p = 0.39), but patients with an occluded culprit artery had a significantly higher 30-day incidence of spontaneous MI (3.5%) than patients with a patent culprit artery and +Tn (1.1%) or -Tn (0.0%, 3-way p = 0.002) (Fig. 4). This was driven predominantly by an increased incidence of definite or probable stent thrombosis, as defined by the Academic Research



**Figure 4. Short-Term Clinical Outcomes**

Patients presenting with an occluded artery and elevated cardiac biomarkers had a significantly higher incidence of spontaneous myocardial infarction but no significant difference in the incidence of periprocedural myocardial infarction or mortality. Abbreviations as in Figures 1 and 3.

Consortium (3.3% vs. 0.1% vs. 0.0%, 3-way p = 0.003) at 30 days.

Peak serum creatine kinase-myocardial band concentrations were 3.3 times the upper limit of normal among patients with an occluded culprit artery and 1.5 times the upper limit of normal among patients with a patent artery irrespective of biomarker positivity (p < 0.001). When stratified by culprit artery, the difference in enzymatic estimate of infarct size was significantly higher among patients with an occluded artery regardless of the artery involved (Table 2).

**Time to treatment.** The median time from baseline ECG to PCI for patients with an occluded artery was 29.4 h (interquartile range 26.1 to 44.1 h). None of the patients with an occluded artery had an ECG to PCI time <6 h.

## Discussion

Among ACS patients with anterior ST-segment depression in this study, over one-quarter had an occluded culprit artery and elevated biomarkers. These patients had significantly worse short-term clinical outcomes and significantly larger infarct size than those with a patent culprit artery. More-

Peak CK-MB (× ULN)	LAD	RCA	LCx	p Value
Occluded culprit artery (TFG 0/1, +Tn)	2.5 (4.2)	3.0 (3.2)	4.1 (4.3)	<0.001
Patent culprit artery (TFG 2/3, -Tn or +Tn)	1.2 (1.6)	1.3 (1.2)	2.3 (3.1)	

Values are mean (SD).

CK-MB = creatine kinase-myocardial band fraction; LAD = left anterior descending artery; LCx = left circumflex artery; RCA = right coronary artery; -Tn = negative cardiac biomarkers; +Tn = elevated cardiac biomarkers; ULN = upper limit of normal; other abbreviations as in Table 1.

over, very few patients with anterior ST-segment depression and an occluded culprit artery were recognized by clinicians as necessitating urgent angiography as demonstrated by the significant median delay from ECG to PCI.

The median time from the diagnostic ECG to PCI in the present study was over 29 h, and it is possible that a significant benefit in clinical outcome would be observed if the presence of an occluded artery had been diagnosed at the time of presentation and emergent revascularization ensued. Current American College of Cardiology/American Heart Association guidelines recommend a door-to-balloon time of 90 min or less for STEMI patients undergoing primary PCI (12). Multiple studies have demonstrated that treatment delays result in greater morbidity and mortality (13–15). In those treated with primary PCI, each 30-min delay from symptom onset to treatment has been estimated to increase the relative risk of 1-year mortality by 7.5% (16). Thus, even small changes in door-to-balloon time may have a large impact on survival.

The results presented here corroborate findings from an analysis of non-STEMI patients enrolled in the PARAGON-B (Platelet IIb/IIIa Antagonism for the Reduction of Acute Coronary Syndrome Events in a Global Organization Network) trial, in which 27% of patients had an occluded culprit artery at the time of angiography (17). Such patients had significantly larger infarct size and higher risk-adjusted 6-month mortality than those with a patent culprit artery. That such patients were more likely to have culprit lesions in the posterior circulation is not surprising, as the difficulty in diagnosing MI involving the posterior circulation is well documented. In the Diltiazem Reinfarction study, 46% of patients initially classified as having anterior non-Q-wave MIs were later found to have posterior STEMI as evidenced by evolution of ECG changes and cardiac biomarkers (8). In patients with left circumflex artery occlusion, the infarct may affect an electrocardiographically silent area of the heart, and the traditional 12-lead ECG may be entirely normal. Although nearly half of patients with an occluded artery in the current analysis had occlusion of the left circumflex coronary artery, fully one-third of patients had left anterior descending coronary artery occlusion. Evidence from a PCI registry suggests that 2% of patients with proximal left anterior descending coronary artery occlusion may present with anterior ST-segment depression (18). Patients with posterior STEMI have been demonstrated to have increased mortality compared with those without evidence of posterior infarction, likely resulting at least in part from a delay in recognition as well as complications such as left ventricular dysfunction, mitral regurgitation, ventricular septal defect, or bradycardia, depending on the affected artery (19,20).

To examine whether arterial occlusion was an epiphenomenon associated with ST-segment depression alone or was specific to the anterior pre-cordial leads, we examined

the angiographic outcomes among patients presenting with isolated inferior ST-segment depression. That less than 5% of patients presenting with inferior ST-segment depression had an occluded culprit artery, as compared with the more than one-quarter with anterior ST-segment depression, suggests that it is electrocardiographic location, rather than ST-segment depression in and of itself, that connotes the possibility of arterial occlusion.

There were no substantial baseline clinical differences between patients with an occluded culprit artery and those with a patent culprit artery at the time of angiography. Therefore, further diagnostic modalities may be warranted in the subset of patients with isolated anterior ST-segment depression. Bedside troponin assays, application of right-sided or posterior ECG leads, or more liberal use of bedside echocardiography may be warranted (8,19,21,22).

**Study limitations.** This analysis is a nonrandomized retrospective analysis, and, as such, it is possible that both identified and unidentified confounders may have influenced the outcomes. Strict enrollment criteria are used in clinical trials, and the results observed here might not be applicable to all patients in clinical practice. In order to be included in the analysis, patients had to survive long enough to undergo angiography, and the distribution of patent and occluded arteries among patients who died prior to angiography is not known. Thus, there is a component of survival bias operative here such that the patients in the present analysis with a closed artery and elevated biomarkers were healthy enough to survive until the time of angiography, a median of 29 h later. Because only those patients who survived until angiography are included in the analysis, it is possible that patients in clinical practice with an occluded artery and elevated biomarkers may face a higher risk of adverse outcomes than observed here. We did not perform ECGs and angiography simultaneously; therefore, a patent artery at the time of ECG may have occluded in the time from ECG to angiography or vice versa. The presence of collaterals was not collected in this study, and collateral circulation to the left circumflex territory may have led to an underappreciation of inferior ST-segment elevation among patients with left circumflex occlusion.

## Conclusions

Among ACS patients presenting with isolated anterior ST-segment depression, more than one-quarter had an occluded artery at the time of angiography. These patients had a significantly higher incidence of death and MI and a greater peak creatine kinase-myocardial band fraction than those with a patent artery. Less than 5% of patients with an occluded artery were recognized by clinicians as having an occluded artery and none underwent urgent angiography. This suggests the need for improved methods to identify

patients with isolated anterior ST-segment depression who have an occluded artery.

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**Key Words:** acute coronary syndrome ■ ST-segment elevation myocardial infarction ■ electrocardiography.