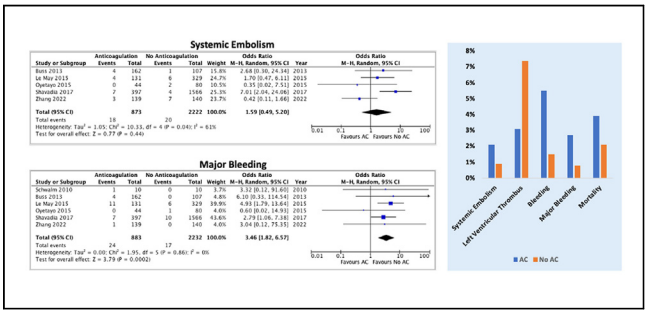

CRT-100.32

A Double-Blinded Randomized Controlled Trial Comparing Eptifibatide Bolus Only Versus Bolus Plus Infusion in Patients Undergoing Primary Percutaneous Coronary Intervention for St-Elevation Myocardial Infarction Revealed Similar Outcome

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BACKGROUNDS The use of eptifibatide combined with heparin during percutaneous coronary intervention (PCI) in patients presenting with ST-elevation myocardial infarction (STEMI) is recommended to be followed by continuous infusion. Recently, there are some suggestions that using bolus only may be sufficient and cost-effective but randomized trials are lacking.

AIMS The goal of this study was to evaluate these two approaches in a double-blinded randomized control trial.

METHODS The primary PCI patients who received bolus eptifibatide were randomized to 75mg IV eptifibatide infusion (short-term protocol = group A) or placebo (group B) blindly. The patients were followed up for the primary outcome of vascular or bleeding complications and secondary outcome of ischemic complications.

RESULTS 330 patients (165 from each group) completed the study. The mean age of our patients was 57.67 ± 11.53 years, and 77.3% were male. Major bleeding was seen in 2 patients (1 patient in each group). Hematoma occurred in 28 patients (8.5%). The relative risks of hematoma and ecchymosis in group A and group B were 0.988 (95% CI: 0486 - 2.006) and 1.032 (95% CI: 0.729 - 1.459). Multivariate analysis confirmed no significant differences in the occurrence of ecchymosis. Furthermore, there was no significant difference in in-hospital death or ischemic events.

CONCLUSION There were no differences in the risk of access site ecchymosis, hematoma or major bleeding. Ischemic events and stent thrombosis rates were also similar. Our study suggests that using eptifibatide bolus only during PCI of patients with STEMI is safe and can be cost-saving.

	All patients	Group A: Bolus eptifibatide + eptifibatide Inf	Group B: Bolus eptifibatide + placebo Inf	P value
Major bleeding (All GIB)	2 (0.6%)	1 (0.6%)	1 (0.6%)	0.993*
Hematoma	28 (8.5%)	14 (8.5%)	14 (8.6%)	0.973**
Hematoma surface area in those with hematoma (cm^2)	9 [4, 16] 35.0 ± 83.4	9 [7.5, 16]	9 [4, 25]	0.901***
Ecchymosis	92 (27.2%)	47 (28.5%)	45 (27.6%)	0.860**
Ecchymosis <5 cm	41 (12.5%)	18 (10.9%)	23 (14.1%)	0.381**
Ecchymosis = 5cm	58 (17.7%)	33 (20%)	25 (15.3%)	0.268**
Ecchymosis surface area in those with ecchymosis (cm^2)	25 [9, 100] 77.9 ± 182.5	25 [9, 100]	25 [9, 39.5]	0.143***
Hospitalization time (Days)	4 [3, 5] 4.17 ± 2.52	4 [3, 5] 4.22 ± 2.07	4 [3, 5] 4.13 ± 2.91	0.277***
Suspected stent thrombosis	2 (0.6)	1 (0.6)	1 (0.6)	0.993*
In hospital death	16 (4.8%)	6 (3.6%)	10 (6.1%)	0.305 **

CRT-100.49

Diaphoresis, but Not Dyspnea, Nausea, or Dizziness, Is Associated With Finding a Culprit Lesion in Patients With Suspected ST Elevation Infarction

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BACKGROUND False positive (FP) invasive laboratory activation for suspected ST elevation infarction (STEMI), with no culprit lesion (Cul) found, remains common. Non-chest pain (NCP) symptoms (sx), including dyspnea (Dysp), nausea/vomiting (N/V), diaphoresis (Diaph), or dizziness/syncope (Diz/Syn) are regarded as supporting the diagnosis of STEMI, but this assumption has never been tested against angiography. We reviewed patients with ST elevation (STE) who had urgent catheterization (cath) and assessed whether NCP sx increased the likelihood that a Cul was present.

METHODS 1,394 consecutive patients undergoing urgent cath for suspected STEMI at an 11 hospital network, with ≥1mm STE in ≥2 leads. Records were reviewed for NCP sx and for the presence (Cul+) or absence (FP) of a culprit lesion.

RESULTS Only Diaph was associated with increased likelihood of a Culprit lesion. Results were similar when analyzed by STE location (anterior, inferior, lateral); or by the 3 epicardial arteries (vessel analysis limited to Cul+ patients). Among Cul+, 809 had total occlusion (TO); 429 had incomplete occlusion (IO). There was no difference in the prevalence of NCP sx between Tot vs IO except for Diaph, which was more prevalent in TO (57.1%) vs IO (47.3%) or FP (30.1%; both p<0.001 vs TO). Among a subset of patients with STE but no chest pain (CP), NCP sx did not predict Cul+. In patients with no CP, there was a trend toward higher prevalence of Dysp in FP (45.9%) vs. Cul+ (32.2%) patients, suggesting that Dysp without CP triggered FP cath activation.

CONCLUSIONS 1) In patients with suspected STEMI, Dysp, N/V, and Diz/Syn do not increase the likelihood of Cul+. 2) In contrast, Diaph significantly increases the likelihood of Cul+, as well as the likelihood that the Cul is TO. 3) In patients with STE but no chest pain, NCP sx should not be regarded as "chest pain equivalents."

	Culprit Present (n=1,238)	No Culprit (FP) (n=156)	odds ratio	p value
Dyspnea	630 (51%)	78 (50%)	1.02	p=0.83
Nausea/Vomiting	461 (37%)	48 (31%)	1.19	p=0.11
Diaphoresis	666 (54%)	47 (30%)	1.80	p<0.001
Dizziness/Syncope	200 (16%)	34 (22%)	0.73	p=0.08