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Preinfarction angina does not affect infarct size in STEMI patients undergoing primary angioplasty

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ABSTRACT

Background: Several clinical studies have demonstrated that anginal attacks shortly before the onset of STEMI limit infarct size and improve short- and long-term outcomes. However, the clinical significance of preinfarction angina in STEMI patients treated by primary PCI is still controversial. Therefore, the aim of the current study was to evaluate the impact of preinfarction angina on scintigraphic infarct size in STEMI patients undergoing primary PCI.
Methods: Our population is represented by 430 STEMI patients undergoing primary PCI. Infarct size was evaluated at 30 days by technetium-99m-sestamibi.
Results: Preinfarction angina was associated with more advanced age, a larger prevalence of family history for CAD, smoking, and longer ischemia time. No difference was observed in other clinical or angiographic characteristics. Preinfarction angina did not affect either the rate of postprocedural TIMI 3 flow or infarct size (19 ± 15.5 vs 16 ± 13.9, $p = 0.18$). Similar results were observed in subanalyses according to infarct location (anterior STEMI: 22.7 ± 14.8 vs 19.2 ± 16.1, $p = 0.36$; non-anterior STEMI: 16.1 ± 15.7 vs 13.8 ± 11.6, $p = 0.36$), gender (female gender: 15.6 ± 14.5 vs 11.5 ± 13.2, $p = 0.30$; male gender 20.4 ± 16 vs 17.2 ± 13.8, $p = 0.3$) or ischemia time (≤ or > 4 h) (17.6 ± 15.6 vs 15.8 ± 14.1, $p = 0.52$; 21.6 ± 15.5 vs 16.7 ± 13.3, $p = 0.18$). The absence of any impact of preinfarction angina on infarct size was confirmed after correction for baseline characteristics, such as age, smoking, family history for CAD and ischemia time (OR [95% CI] = 1.26 [0.66-2.41], $p = 0.48$).
Conclusions: This study shows that among STEMI patients undergoing primary PCI preinfarction angina does not affect infarct size.

Availability of pharmacologic and mechanical reperfusion therapies has significantly reduced cardiac mortality among ST-segment elevation acute myocardial infarction (STEMI) patients [1–8]. However, even though primary angioplasty has been shown to provide larger benefits as compared to thrombolysis, a suboptimal myocardial reperfusion is observed in a still relevant proportion of patients, despite optimal epicardial recanalization [8,9]. In experimental animals, brief episodes of ischemia before sustained coronary occlusion significantly decrease infarct size [9–11]. This phenomenon, known as ischemic preconditioning, also occurs in humans. Several clinical studies have demonstrated that anginal attacks shortly before the onset of STEMI limit infarct size and improve short- and long-term outcomes [12–15]. Among STEMI patients treated with thrombolysis, recanalization has been shown to be more rapid and frequent in patients with than without preinfarction angina [15,16]. However, the clinical significance of preinfarction angina in STEMI patients undergoing primary angioplasty is still controversial. Therefore, the aim of the current study was to evaluate the impact of preinfarction angina on scintigraphic infarct size in STEMI patients undergoing primary angioplasty.

1. Materials and methods

Our population is represented by 430 STEMI patients treated by primary angioplasty, who were included in randomized trials that aimed at the evaluation of infarct size at 30 days after intervention [5,17,18]. All patients were admitted within 12 h from symptom onset, and received at the time of diagnosis aspirin (500 mg intravenously) and heparin (60 IU/Kg intravenously), as much as

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beta-blockers and nitroglycerine intravenously if not contra-indicated, whereas the decision to provide glycoprotein IIb-IIIa inhibitors was left at the discretion of the operator at the time of intervention. All patients were on dual oral antiplatelet therapy (aspirin and clopidogrel or ticlopidine) for at least 4 weeks after stent implantation.

All demographic, clinical, procedural and in-hospital and follow-up data were collected in a database. Particular attention was paid at the time of hospital admission to precisely collect data on the time from symptom onset and patient clinical history, including preinfarction angina, defined as any angina occurring ≤24 h before the onset of STEMI.

1.1. Coronary angiography and mechanical revascularization

Selective coronary angiography was performed in multiple projections before mechanical reperfusion. Immediately after diagnostic angiography, percutaneous coronary intervention with stenting of the infarct-related vessel was performed using standard material. Successful primary percutaneous coronary intervention was defined as Thrombolysis In Myocardial Infarction (TIMI) grade 3 coronary flow in the treated vessel with a residual stenosis <20% [18].

1.2. Infarct size assessment

As previously described [19], gated single-photon emission computed tomography (SPECT) acquisition began 60 min after technetium-99m-sestamibi injection (740 MBq), using a double-head gamma-camera equipped with high-resolution collimators, 180° rotation arc, 32 projections, 60 s/projection, 8 frames/heart cycle and 64 × 64 matrices. The studies were reconstructed using filtered back-projection without attenuation or scatter correction and realigned along the heart axis. Perfusion defects were quantified as percentage of LV wall, with the defect threshold set at 60% of peak uptake [20].

1.3. Statistical analysis

Statistical analysis was performed with the SPSS 17.0 statistical package. Continuous data were expressed as mean ± SD and categorical data as percentage. The ANOVA test was appropriately used for continuous variables. The chi-square test or the Fisher's exact test was used for categorical variables. Patients were divided according to the presence of absence of preinfarction angina (defined as any angina occurring within 24 h from symptoms onset). Multiple logistic regression analysis was used to evaluate the impact of preinfarction angina on infarct size after adjustment for significant ($p < 0.05$) confounding baseline characteristics.

2. Results

Patients characteristics are shown in Tables 1 and 2. Preinfarction angina was associated with more advanced age, a larger prevalence of family history for CAD, smoking, and longer ischemia time. No difference was observed in other clinical or angiographic characteristics. Preinfarction angina did not affect the rate of postprocedural TIMI 3 flow.

As shown in Fig. 1, preinfarction angina did not affect infarct size (19 ± 15.5 vs 16 ± 13.9, $p = 0.18$). Similar results were observed in subanalyses according to infarct location (anterior STEMI: 22.7 ± 14.8 vs 19.2 ± 16.1, $p = 0.36$; non-anterior STEMI: 16.1 ± 15.7 vs 13.8 ± 11.6, $p = 0.36$), gender (female gender: 15.6 ± 14.5 vs 11.5 ± 13.2, $p = 0.30$; male gender 20.4 ± 16 vs 17.2 ± 13.8, $p = 0.3$) and ischemia time (≤ or > 4 h) (17.6 ± 15.6 vs 15.8 ± 14.1, $p = 0.52$; 21.6 ± 15.5 vs 16.7 ± 13.3, $p = 0.18$) (Fig. 2).

The absence of any impact of preinfarction angina on infarct size was confirmed after correction for baseline characteristics, such as age, smoking, family history for CAD and ischemia time (OR [95% CI] = 1.26 [0.66–2.41], $p = 0.48$).

Table 1
Demographic and clinical characteristics according to preinfarction angina.

Variable	Preinfarction angina ($n = 47$)	Control ($n = 383$)	p value
Age	60.7 ± 11.5	64.5 ± 12.1	0.041
Female gender (%)	29.8	20.4	0.14
Smoking (%)	53.2	25.2	0.016
Hypertension (%)	31.9	42.3	0.17
Dyslipidemia (%)	44.7	36.8	0.29
Diabetes (%)	12.8	13.3	0.92
Family history of CAD (%)	14.9	6.5	0.039
Previous MI (%)	0	3,7	0.18
Previous CABG (%)	0	1.3	0.43
Previous PTCA (%)	4.3	2.4	0.44
Ischemia time (minutes)	229 ± 95	203 ± 76	0.031
Killip class (%)			0.45
1	89.4	85.6	
2	4.3	7	
3	4.3	1.8	
4	2.1	5.5	
Therapy at discharge			
Clopidogrel or ticlopidine (%)	100	100	1.0
ACE-inhibitors (%)	46.8	46.4	0.958
Beta-blockers (%)	6.3	8.6	0.582
Statins (%)	44.6	39.1	0.516

3. Discussion

This is one of the largest study conducted so far to evaluate the impact of preinfarction angina on scintigraphic infarct size in STEMI patients undergoing primary PCI. We found that preinfarction angina does not affect infarct size.

Table 2
Angiographic and procedural characteristics according to preinfarction angina.

Variable	Preinfarction angina ($n = 47$)	Control ($n = 383$)	p value
Chronic occlusion (%)	4.3	6.5	0.54
Collateral circulation			0.41
0 (%)	89.4	89.3	
1 (%)	4.3	7.6	
2 (%)	6.4	2.6	
3 (%)	0	0.5	
Preprocedural TIMI flow			0.59
0–1 (%)	79.6	74.5	
2 (%)	12.8	12	
3 (%)	12.8	8.4	
IRA			0.91
RCA (%)	46.8	45.4	
CX (%)	8.5	12.8	
Graft (%)	0	0.3	
LAD (%)	44.7	41.3	
LM (%)	0	0.3	
Multivessel disease (%)	40.4	39.9	0.95
N. lesions			0.16
1 (%)	68.1	80.4	
2 (%)	29.8	16.4	
3 (%)	2.1	2.9	
Abciximab (%)	74.5	83	0.15
Stenting (%)	97.9	99	0.51
IABP (%)	4.3	2.8	0.21
Postprocedural TIMI 3 flow (%)	100	99.5	0.98