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Smoking and infarct size among STEMI patients undergoing primary angioplasty

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ABSTRACT

Background: Prior studies have found that smokers with STEMI have lower mortality rates and a more favorable response to fibrinolytic therapy than nonsmokers, phenomenon defined as "the smoker's paradox". Still poorly explored is the impact of cigarette smoking in patients undergoing primary percutaneous coronary intervention. Aim of the current study was to evaluate the impact of cigarette smoking on scintigraphic infarct size in STEMI patients undergoing primary PCI.
Methods: Our population is represented by 830 STEMI patients undergoing primary PCI. Infarct size was evaluated at 30 days by technetium-99m-sestamibi.
Results: Smoking was associated with younger age ($p < 0.001$), a lower prevalence of female gender ($p < 0.001$), hypertension ($p < 0.001$), diabetes ($p = 0.003$), shorter ischemia time ($p = 0.037$), but higher rates of previous PCI ($p = 0.016$). No differences were observed in other clinical or angiographic characteristics. In particular, smoking did not affect the rate of postprocedural TIMI 3 flow. As shown in Fig. 1, smoking did not affect infarct size (12.5% [3.3%–23.7%] vs 12.7% [4.9%–25.9%], $p = 0.12$). Similar results were observed in subanalyses according to infarct location (anterior STEMI, p int $= 0.33$), gender (p int $= 0.95$) age, (p Int $= 0.96$), diabetes (p int $= 0.85$). The absence of any impact of smoking on infarct size was confirmed after correction for baseline characteristics, such as age, gender, hypertension, diabetes, previous PCI, ischemia time (OR [95% CI] $= 0.80$ [0.59–1.09], $p = 0.15$).
Conclusions: This study shows that among STEMI patients undergoing primary PCI smoking status does not affect infarct size.

1. Introduction

It is well know that smoking is a major risk factors for coronary artery disease (CAD), with significantly higher rates of ST-segment elevation myocardial infarction (STEMI) and death [1–3]. Paradoxically, despite the increased occurrence of STEMI in active smokers, prior studies have found that the mortality rate of smokers after STEMI is lower than in nonsmokers [4–7], especially after fibrinolytic therapy [8–12]. This phenomenon, defined as "the smoker's paradox", has been partly explained by fewer coexisting high-risk features in patients with STEMI who are current smokers [6–12]. In addition, is has been supposed a difference in lesion composition with a greater thrombotic component and relatively less atherosclerotic plaque burden in smokers [10], thereby contributing to the more benign long-term prognosis in these patients.

The impact of cigarette smoking on clinical outcome in patients undergoing primary percutaneous coronary intervention has not been largely investigated. In particular, the supposed higher thrombotic component may be associated with impaired reperfusion and larger infarct size when a mechanical reperfusion is performed. Therefore, the aim of the current study was to evaluate whether cigarette smoking does affect scintigraphic infarct size among STEMI patients undergoing primary angioplasty.

2. Materials and methods

Our population is represented by 830 STEMI patients treated by primary angioplasty, who were included in randomized trials

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conducted between 2001 and 2009, that aimed at the evaluation of infarct size at 30 days after intervention [13–15]. 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 beta-blockers and nitroglycerine intravenously if not contraindicated, 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.

2.1. 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%.

2.2. Infarct size assessment

As previously described [15], 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 [16].

2.3. Statistical analysis

Statistical analysis was performed with the SPSS 17.0 statistical package. Continuous data were expressed as median [25–75th percentiles] and categorical data as percentage. The analysis of variance test (ANOVA) or Mann–Whitney *U* test was appropriately used for continuous variables, according to the normality of distribution, as evaluated by the Shapiro–Wilk test. The chi-square test or the Fisher's exact test was used for categorical variables. Multiple logistic regression analysis was used to evaluate the impact of smoking on infarct size after adjustment for significant ($p < 0.05$) confounding baseline characteristics. A propensity score analysis was performed in order to evaluate the impact of smoking on infarct size in homogeneous subgroups of patients.

3. Results

Patients' characteristics are shown in Tables 1 and 2. Smoking was associated with younger age ($p < 0.001$), a lower prevalence of female gender ($p < 0.001$), hypertension ($p < 0.001$), diabetes ($p = 0.003$), shorter ischemia time ($p = 0.037$), but higher rates of previous PCI ($p = 0.016$). No differences were observed in other clinical or angiographic characteristics. In particular, smoking did not affect the rate of postprocedural TIMI 3 flow. As shown in Fig. 1, smoking did not affect infarct size (12.5% [3.3%–23.7%] vs 12.7% [4.9%–25.9%], $p = 0.12$).

Similar results were observed according to tertiles of propensity score (Fig. 2) and in subanalyses according to infarct location (anterior STEMI: 9.6% [4.1%–25.1%] vs 10.2% [3.9%–22.7%], $p = 0.34$; non-anterior STEMI: 12.6% [3.2%–23.2%] vs 13.2% [5.3%–26.3%], $p = 0.38$; p int = 0.33), gender (female gender: 11.6% [2%–20.3%] vs 11% [4%–22%], $p = 0.25$; male gender: [14.5% [5.7%–28.6%] vs 16.1% [6.0%–31.0%]], $p = 0.018$; p int = 0.95) age, (>65 years 12.9% [5%–23.8%] vs 15.3% [6.5%–28%], $p = 0.18$; <65 years: 3.6% [0–18.7%] vs 8.6% [0–19%], $p = 0.28$; p int = 0.96), diabetes (yes: 11.9% [1.8%–23.8%] vs 12.4% [4.6%–23.8%], $p = 0.78$; no: 12.6% [3.7%–23.6%] vs 15.0% [5.2%–27.4%], $p = 0.067$; p int = 0.85), without any significant interaction for each variable.

The absence of any impact of smoking on infarct size was confirmed when the analysis was conducted according to the percentage of patients with infarct size above the median (Fig. 3), even after correction for baseline characteristics, such as age, gender, hypertension, diabetes, previous PCI, ischemia time (OR [95% CI] = 0.80 [0.59–1.09], $p = 0.15$).

Table 1
Demographic and clinical characteristics according to history of smoking.

Variable	Smoking ($n = 401$)	Control ($n = 429$)	p value
Age	61 [53–69]	67 [59–75]	<0.001
Age >75 ys (%)	7.2	32.2	<0.001
Female gender (%)	14	27.3	<0.001
Hypertension (%)	33.7	52.9	<0.001
Dyslipidemia (%)	33.4	34.5	0.74
Diabetes (%)	10.2	17.2	0.003
Previous ACS (%)	5	3.3	0.21
Previous CABG (%)	0.7	0.9	1.0
Previous PTCA (%)	5.2	2.1	0.016
Ischemia time (minutes)	195 [140–269]	210 [155–280]	0.037
Ischemia time >3 h (%)	55.4	62.9	0.03
Anterior MI (%)	36.9	43.1	0.068
Cardiogenic shock (%)	3.0	4.9	0.16

4. Discussion

This is the largest prospective study to date evaluating the impact of cigarette smoking on infarct size among STEMI patients undergoing mechanical reperfusion. We did not find any impact of smoking on myocardial perfusion and scintigraphic infarct size.

The application of reperfusion therapies has largely contributed to the relevant reduction in mortality observed in the last decades in the treatment of STEMI. Primary angioplasty and adjunctive

Table 2
Angiographic and procedural characteristics according to history of smoking.

Variable	Smoking ($n = 401$)	Control ($n = 429$)	p value
Collateral circulation			0.6
RENTROP 0 (%)	89.8	88.8	
RENTROP 1 (%)	6.4	7.8	
RENTROP 2 (%)	3.8	2.7	
RENTROP 3 (%)	0	0.8	
Preprocedural TIMI 3 flow (%)	7.5	8.6	0.56
IRA			0.25
RCA (%)	47.4	43.4	
CX (%)	15.5	13.3	
Graft (%)	0	0.1	
LAD (%)	36.9	43.1	
LM (%)	0	0.2	
Multivessel disease (%)	39.8	43.0	0.36
Abciximab (%)	91.5	88.6	0.16
Stenting (%)	100	98	0.95
DES (%)	5.7	6.2	0.94
Direct Stenting (%)	76	77.2	0.78
Thrombectomy (%)	42.4	41	0.75
IABP (%)	3.0	4.9	0.16
Postprocedural TIMI 3 flow (%)	91.0	93.4	0.19