folia medica

Folia Medica 65(2):243-250
DOI: 10.3897/folmed.65.e80189

Original Article

Smoker Pseudo-Paradox in ST-segment Elevation Myocardial Infarction Patients

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Received: 7 Jan 2022 ♦ **Accepted:** 5 Apr 2022 ♦ **Published:** 30 Apr 2023

Citation: Janjani P, Salehi N, Asadmobini A, Siabani S, Nalini M. Smoker pseudo-paradox in ST-segment elevation myocardial infarction patients. Folia Med (Plovdiv) 2023;65(2):243-250. doi: 10.3897/folmed.65.e80189.

Abstract

Introduction: Cigarette smoking is a preventable cause of cardiovascular morbidity and mortality. Despite the adverse effects of smoking, some studies have reported the term "smoker's paradox', meaning better outcomes in smokers following acute myocardial infarction.

Aim: The aim of the present study was to evaluate the relationship between smoking status and one-year mortality in patients with ST-segment elevation myocardial infarction (STEMI).

Materials and methods: This was a registry-based cohort study of STEMI patients from Imam-Ali hospital, Kermanshah, Iran. Consecutive STEMI patients (July 2016-October 2018) were stratified by smoking status and followed for one year. Cox proportional models were used to estimate crude, age-adjusted, and full-adjusted hazard ratios with 95% confidence intervals (HR, 95%CI).

Results: Of 1975 patients (mean age 60.1 years, 76.6% male) included in the study, 48.1% (n=951) were smokers (mean age 57.7 years, 94.7% male). Crude and age-adjusted HR (95% CI) for the associations of smoking and mortality were 0.67 (0.50-0.92) and 0.89 (0.65-1.22), respectively. After adjusting for age, sex, hypertension, diabetes, body-mass index, anterior wall myocardial infarction, creatine kinase-MB, glomerular filtration rate, left ventricular ejection fraction, low-density lipoprotein cholesterol, and hemoglobin, smoking was associated with increased risk of mortality: HR (95% CI: 1.56 (1.04-2.35).

Conclusions: In our study, smoking was associated with an increased risk of mortality. Although the smokers had a better outcome, this would be reversed after controlling for age and the other STEMI associated factors.

Keywords

smoker's paradox, cigarette smoking, ST-elevation myocardial infarction, primary percutaneous coronary intervention, thrombolytic therapy

INTRODUCTION

Cigarette smoking is one of the most important preventable causes of morbidity and mortality in the world and the second most common cause of disability-adjusted life years.[1] Despite the adverse effects of smoking on the cardiovascular system, some studies have reported the term "smoker's paradox", meaning better outcomes in smokers following acute myocardial infarction.[2-5]

Over the last few decades, there has been a lot of interest in the mechanisms underlying this paradoxical association. Some suggested that the smoker's paradox was probably due to the more 'thrombotic' nature of myocardial infractions in smokers as opposed to atherosclerotic nature in non-smokers and hence better reperfusion response after thrombolysis.[6] Other studies reported the smoker's paradox in various reperfusion strategies, including thrombolysis and primary percutaneous coronary intervention.[7,8]

The researchers revealed that this paradox is not because of the benefit that arises from smoking, but just because smokers undergo such conditions at a very young age with low levels of comorbidities.[9-12]

Against this background, it seems that the smoker's paradox and related mechanisms in myocardial infarction constitute an important and debatable topic for researchers.

AIM

We aimed to evaluate the association between smoking status and one-year mortality in patients with ST-segment elevation myocardial infarction (STEMI).

MATERIALS AND METHODS

Study design, setting, and participants

This is a registry-based prospective cohort study at Imam Ali Hospital affiliated to Kermanshah University of Medical Sciences, Kermanshah, Iran. This hospital is the main tertiary cardiovascular center in the Kermanshah province, in the west of Iran. It is also the only hospital in the province with 24 hours a day, 7 days a week primary percutaneous coronary intervention (PPCI) capability. Therefore, patients may be directly admitted to Imam Ali hospital or be referred from other non-PPCI capable hospitals in the province. All eligible adult patients (≥18 years) with STEMI, diagnosed by current guidelines[13], were enrolled in the registry (July 2016-October 2018). Patients who were hospitalized more than 24 hours before referring to Imam Ali hospital were excluded from the registry. In the present study, we also excluded patients with previous cardiovascular events (myocardial infarction or stroke) and interventions (percutaneous coronary intervention, or coronary artery bypass graft surgery) and those with out-of-hospital cardiac arrest.

Baseline assessment

Trained nurses collected data of demographic, lifestyle, and clinical characteristics from personal interviews with patients and/or their attendants. Study participants were determined to have a history of tobacco smoking based on self-report. Previous cardiovascular events, coronary intervention, diabetes, and hypertension were recorded based on physician-confirmed self-reports. Information about vital signs, early reperfusion therapy, electrocardiography, medical treatment, and laboratory tests was obtained from hospital medical records. Early reperfusion therapy included PPCI, thrombolytic therapy, and none (no reperfusion). Body-mass index (BMI) – weight in kilograms divided by the square of height in meters – was measured using standard protocols. Lipid profile and creatinine and hemoglobin (Hb) levels were measured at the first day of admission. Glomerular filtration rate (GFR) was estimated using the CKD-EPI equation. The highest levels of creatine kinase (CK-MB) after STEMI were recorded. The echocardiography results were used to record left ventricular ejection fraction (LVEF). All recorded data were quality controlled by trained physicians.

Study outcome and follow-up

The outcome was all-cause mortality one year from STEMI events – during index hospitalization or after discharge. In-hospital mortality was recorded using hospital documents. Upon hospital admission, contact information of patients, family members or attendants were recorded. Patients were followed after 1 year by phone call. If a death was reported, all clinical or hospital records and the cause of death were collected and evaluated by the research team. Follow-up time extended from the date of STEMI diagnosis to the date of death, loss-to-follow up, or 365 days after STEMI, whichever came first.

Ethical approval and consent for study

All patients signed a written informed consent before enrolling in the study. The Research Ethics Committee at Deputy of Research of the Kermanshah University of Medical Sciences has approved the study protocol (Ethics registration code: IR.KUMS.REC.1400.252).

Statistical analysis

Continuous variables were presented as mean ± standard deviation (SD) and categorized variables as absolute value and percentages. Chi-squared and Student's *t*-test were used to compare the baseline characteristics between ever-smokers and never-smokers. Cox proportional hazard regression analysis was performed to determine hazard ratio and 95% confidence interval (HR, 95% CI) for the association between smoking and all-cause death. We reported three HRs (95% CIs) using crude, age-adjusted, and full-adjusted Cox models. In the full-adjusted model, we evaluated the association of smoking with mortality after adjusting for age (continuous), sex, hypertension (yes/no), diabetes (yes/no), CK-MB (tertile), BMI (continuous), GFR (continuous), anterior wall MI/LBBB (yes/no), LVEF (<40, 40-49, ≥50%) and reperfusion therapy (PPCI, thrombolytic, no reperfusion). In subgroup analyses, we analyzed the association of smoking with all-cause mortality based on sex, reperfusion therapy, and death time (at index hospitalization or after discharge). In this study, the number of missing values for the covariates were relatively small (diabetes, 36; hypertension, 17; BMI, 21; LVEF, 45; GFR, 2; Hb, 2; LDL-cholesterol, 89). We performed all analyses on complete case data. Seventeen patients were lost to follow-up. All analyses were performed using a standard software package (Stata, version. 14.0; Stata Corp). A *p*-value

<0.05 or 95% CIs not including one was considered statistically significant. This study followed the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines.[14]

RESULTS

A total of 2467 patients were enrolled in the registry, 443 patients had a history of cardiovascular events, 41 patients had out-of-hospital cardiac arrest, and 8 patients had the unknown smoking condition, so they were excluded from the analysis, leaving 1975 patients comprising the study population.

Of the 1975 patients, 951 (48.1%) were ever-smokers and 1024 (51.6%) never-smokers. Baseline characteristics of ever-smokers and never-smokers are presented in **Table 1**. Ever-smokers were significantly younger than never-smokers and more frequently male. Diabetes and hypertension were much higher among never-smokers compared to ever-smokers (25.45% vs. 12.06% and 47.84% vs. 27.55%, respectively). The means for Hb and GFR were higher and for LDL-cholesterol and BMI were lower in ever-smokers compared with never-smokers **(Table 1)**.

Table 1. Baseline characteristics of the study population according to smoking status

	All (n=1975)	**Ever-smoker (n=951)**	**Never-smoker (n=1024)**	***P* value**
Age (years)	60.10±12.52	57.67 ±11.79	62.36 ±12.77	<0.001
Sex				<0.001
Male	1512 (76.56%)	901 (94.74%)	611 (59.67%)	
Female	463 (23.44%)	50 (5.26%)	413 (40.33%)	
Diabetes mellitus				<0.001
Yes	368 (18.98%)	113 (12.06%)	255 (25.45%)	
No	1571 (81.02%)	824 (87.94%)	747 (74.55%)	
Hypertension				<0.001
Yes	746 (38.10%)	259 (27.55%)	487 (47.84%)	
No	1212 (61.90%)	681 (72.45%)	531 (52.16%)	
BMI (kg/m^2)	26.16±4.07	25.70±4.11	26.56±3.99	<0.001
LDL-cholesterol (mg/dL)	106.60±31	104.46±29.37	108.65±32.37	0.016
Hemoglobin (g/dL)	14.73±1.81	15.15±1.74	14.33±1.79	<0.001
GFR (mL/min/1.73 m^2)	69.20±17.99	74.19±17.42	64.57±17.26	<0.001
Anterior wall MI/LBBB				0.810
Yes	332 (16.81%)	162 (17.03%)	170 (16.60%)	
No	1643 (83.19%)	789 (82.97%)	854 (83.40%)	
CK-MB (U/L)				0.283
1st tertile	623 (31.54%)	290 (30.49%)	333 (32.52%)	
2nd tertile	670 (33.92%)	316 (33.23%)	354 (34.57%)	
3rd tertile	682 (34.53%)	345 (36.28%)	337 (32.91%)	
Early reperfusion therapy				0.001
PPCI	1157 (58.58%)	562 (59.10%)	595 (58.11%)	
Thrombolytic	510 (25.82%)	268 (28.18%)	242 (23.63%)	
No reperfusion	308 (15.59%)	121 (12.72%)	187 (18.26%)	
LVEF				0.059
<40%	338 (17.51%)	164 (17.63%)	174 (17.40%)	
40-49%	780 (40.41%)	399 (42.90%)	381 (38.10%)	
≥50%	812 (42.07%)	367 (39.46%)	445 (44.50%)	

BMI: body mass index; LDL-cholesterol: low-density lipoproteins cholesterol; GFR: *g*lomerular filtration rate; MI: myocardial infarction; LBBB: left bundle branch block; CK-MB: creatine kinase-MB; PPCI: primary percutaneous coronary intervention; LVEF: left ventricular ejection fraction. Values are mean ± standard deviation (SD) or %.

Eighty-six (4.35%) patients died in hospital, most of whom were never-smokers (n=59, p=0.001). During the follow up, 83 patients died (39 ever-smokers and 44 never-smokers, p=0.725).

As illustrated in **Table 2**, smoking was a protective factor of mortality with an unadjusted HR (95% CI) of 0.67 (0.50-0.92, p=0.01). After adjusting for age, this association was attenuated with a HR (95% CI) of 0.89 (0.65-1.22, p=0.41). Interestingly, when the model was fully adjusted for all the variables (age, sex, BMI, diabetes, hypertension, LDL-cholesterol, hemoglobin, CK-MB, GFR, anterior wall MI/LBBB, LVEF, and reperfusion therapy), the mortality risk of smoking became obvious: HR (95% CI): 1.56 (1.04-2.35; p=0.042). Based on the full-adjusted model, smokers had 56% higher risk of one-year mortality compared with non-smokers. Other independent risk factors of mortality were age, high CK-MB (3rd tertile), no reperfusion therapy, and low EF (<40%), while GFR was an independent protective factor.

Fig. 1 shows the smoker's paradox in all-cause mortality during a 1-year follow-up. The protective association of smoking with mortality in the crude model was changed, qualitatively, and the risk effect of smoking was revealed in the full-adjusted model. **Fig. 2** shows the survival curves for ever-smokers versus never-smokers based on the full-adjusted Cox regression model.

Subgroup analyses are reported in **Table 3**. In sex subgroup analyses, females were at higher risk than males. Although 94.74% of the smokers were men, HR was higher in female smokers. In females, smoking was significantly associated with mortality in both crude and full-adjusted models.

Likewise, in patients without reperfusion therapy, smoking was associated with the increased risk of mortality in both crude and full-adjusted models, although these associations were not statistically significant. Subgroup patients based on in-hospital and out-of-hospital mortality showed that while there was a reduction in HR in-hospital mortality, there was no difference after adjustment. Among all the other defined subgroups, the protective trends of associations between smoking and mortality in crude models disappeared in full-adjusted models.

Table 2. Unadjusted and adjusted associations between smoking and mortality

	Crude HRs (95% CIs)	**Age-adjusted HRs (95% CIs)**	**Full-adjusted HRs (95% CIs)**
Smoking	0.67 (0.50-0.92)	0.89 (0.65-1.22)	1.56 (1.04-2.35)
Age (years)	1.06 (1.05-1.07)		1.02 (1.00-1.04)
Sex (female vs. male)	2.42 (1.79-3.29)	1.74 (1.27-2.38)	1.48 (0.95-2.32)
Diabetes	1.83 (1.31-2.55)	1.75 (1.26-2.45)	1.34 (0.90-1.99)
Hypertension	2.37 (1.74-3.22)	1.63 (1.18-2.24)	1.34 (0.90-1.98)
BMI (kg/m^2)	0.94 (0.90-0.98)	0.98 (0.94-1.02)	0.97 (0.92-1.01)
LDL-cholesterol	0.997 (0.991-1.002)	0.999 (0.994-1.005)	0.997 (0.99-1.003)
Hemoglobin (g/dL)	0.76 (0.71-0.83)	0.84 (0.77-0.92)	0.93 (0.84-1.04)
GFR (mL/min/1.73 m^2)	0.95 (0.94-0.96)	0.96 (0.95-0.97)	0.97 (0.96-0.98)
Anterior wall MI/LBBB	1.38 (0.96-2.00)	1.36 (0.94-1.97)	0.96 (0.59-1.55)
CK-MB (IU/L)			
1st tertile	Reference	Reference	Reference
2nd tertile	0.70 (0.47-1.03)	0.72 (0.49-1.06)	1.11 (0.69-1.78)
3rd tertile	1.06 (0.74-1.50)	1.15 (0.81-1.64)	1.89 (1.19-3.01)
Reperfusion therapy			
PPCI	Reference	Reference	Reference
Thrombolytic	1.34 (0.92-1.95)	1.36 (0.93-1.98)	1.44 (0.95-2.20)
No reperfusion	2.93 (2.06-4.17)	2.19 (1.52-3.15)	2.17 (1.38-3.42)
LVEF			
≥50%	Reference	Reference	Reference
40-49%	1.52 (0.78-2.98)	1.47 (0.75-2.88)	1.23 (0.62-2.45)
<40%	4.39 (2.36-8.17)	3.55 (1.90-6.62)	2.73 (1.42-5.23)

BMI: body mass index; LDL-cholesterol: low-density lipoproteins cholesterol; GFR: glomerular filtration rate; MI: myocardial infarction; LBBB: left bundle branch block; CK-MB: creatine kinase-MB; PPCI: primary percutaneous coronary intervention; LVEF: left ventricular ejection fraction. Data are hazard ratios (HRs) with 95% confidence intervals (95% CIs).

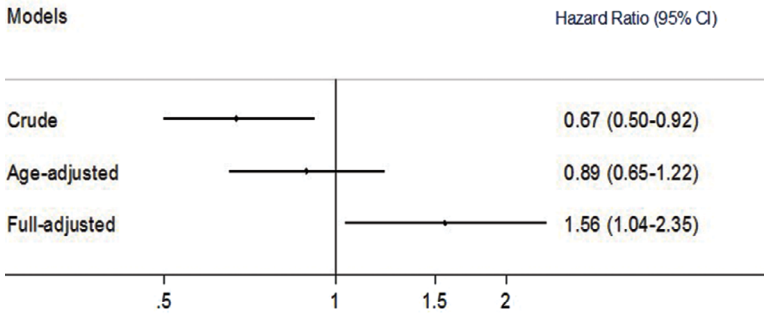

Figure 1. Smoker's paradox: the crude and adjusted associations between smoking and mortality. The point estimate and 95% confidence interval for the hazard ratio (HR) associated with smoking is presented for an unadjusted, age-adjusted, and full-adjusted for smoking, age, sex, diabetes, hypertension, BMI, GFR, CK-MB, anterior wall MI/LBBB, LDL-cholesterol, Hemoglobin, EF, reperfusion therapy [PPCI, thrombolytic, no reperfusion].

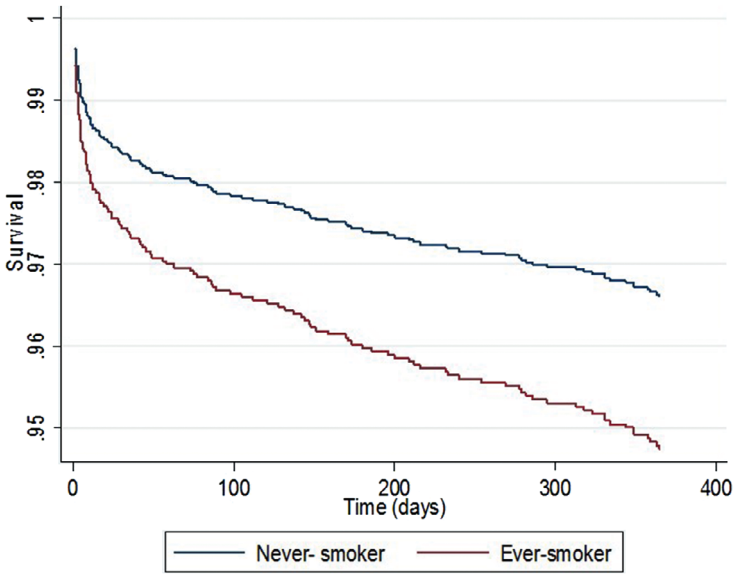

Figure 2. The full-adjusted Cox regression survival curves for ever-smokers and never-smokers.

Table 3. Sub-group analyses according to sex, reperfusion therapy, and death time

Subgroups	Crude HRs (95% CIs)	Age-adjusted HRs (95% CIs)	Full-adjusted HRs (95% CIs)
Sex			
Male	0.80 (0.54-1.19)	1.02 (0.68-1.53)	1.40 (0.87-2.26)
Female	1.86 (1.00-3.46)	1.78 (0.96-3.32)	3.01(1.42-6.39)
Reperfusion therapy			
PPCI	0.57 (0.35-0.93)	0.76 (0.46-1.24)	1.22 (0.65-2.29)
Thrombolytic therapy	0.56 (0.31-1.04)	0.71 (0.38-1.31)	1.03 (0.48-2.19)
No reperfusion	1.23 (0.71-2.11)	1.41 (0.82-2.45)	3.14 (1.39-7.11)
Death time			
In hospital	0.49 (0.31-0.77)	0.61 (0.38-0.97)	1.07 (0.56-2.05)
After discharge	0.92 (0.60-1.42)	1.28 (0.83-2.00)	1.96 (1.15-3.32)

Data are hazard ratios (HRs) with 95% confidence intervals (95% CIs). PPCI: primary percutaneous coronary intervention.

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DISCUSSION

In the present study, smokers had a better prognosis in unadjusted models; however, after adjustment for age and other covariates, smokers had a higher risk for mortality after a one-year follow-up. The results of this registry-based study indicated that the smoker's paradox was a false impression of the effect of smoking in the STEMI patients undergoing reperfusion therapy.

The favorable results obtained in our study between ever-smokers and never-smokers could be related to a significant difference between ages in ever- and never-smokers. So, the smoker paradox was probably due to lower ages of smokers versus non-smokers at the time of STEMI which was consistent with previous reports.[10,15-17] The fact that smokers developed STEMI a few years earlier than non-smokers might be related to acceleration atherosclerosis, increased blood coagulability, and greater platelet reactivity in smokers.[18] Smokers under PCI were younger, male, and had lower comorbidities.

Although ever-smoker patients had a lower mortality rate than never-smokers, this protection was not present after adjustment for other variables, suggesting the existence of a smoker's pseudo-paradox on mortality for the STEMI patients. Our findings were consistent with other studies that challenged the smoker's paradox in the STEMI patients.[6,19,20]

Ever-smokers had significantly fewer coronary risk factors compared with never-smokers. Significantly less prevalence of hypertension, diabetes, BMI, and LDL-cholesterol in our study among ever-smokers was in accordance with the previous study and the reason could be the younger mean age of the ever-smokers.[19,21,22] These results can predict a better outcome and put the ever-smoker at an advantage when compared to never-smokers. These findings underscored the fact that smokers were prone to CAD even with a lower prevalence of risk factors.

As illustrated in **Table 3**, females were at higher risk than males. It may be due to the fact that smoking instigates severe stress responses in females.[23] On the other hand, there was no smoker paradox in females. It may be because women started smoking at later ages.[24] Women are more vulnerable to the side effects of smoking. In our study, smoking increased the risk of death more than 3 times in women and by 40% in men in a fully adjusted model. Women were older and had higher risk factors due to their older age, which confirms the age hypothesis about the paradox.

As reported in **Table 3**, smoking was a protective factor in PPCI and thrombolytic therapy, and was a risk factor in patients who had no reperfusion. In our study, the patients that do not receive treatment were older, so they were more prone to damage. For patients with STEMI, PPCI is an optimal strategy of treatment. On the other hand, many patients could not have PPCI at its optimal time due to geographical or logistical issues. In such cases, thrombolytic therapy was followed by immediate transfer to a PPCI capable center. Therefore, it was necessary to use reperfusion to save patients' lives.

Some studies reported the survival benefit of smokers in the setting of STEMI, ranging from the in-hospital mortality to three-year mortality[24,25] consistent with previous studies[10,26], this paradox was observed in in-hospital mortality. In our study, in contrast to other studies, the smoker's paradox after a one-year follow-up was not observed. It was reported that smokers suffer more out-of-hospital death, thus creating a selection bias when assessing in-hospital mortality.[27]

The strengths of our study were prospective design, one-year follow-up, low rate of loss to follow-up.

The limitations of our study were the use of a single-center experience and self-reported data such as hypertension. We do not have any information about patients who had an out-of-hospital cardiac arrest because smoking increases the risk of sudden cardiac death.

CONCLUSIONS

Our study showed that there is no survival advantage for ever-smokers in patients with STEMI. However, smokers had better clinical outcomes (in-hospital mortalities) after STEMI, but upon adjustment, the seemingly beneficial effects of smoking on mortality disappeared. So, in our population, there was no actual smoker's paradox and the evidence of better outcomes may be related to younger age and fewer risk factors at the time of presentation with STEMI.

Acknowledgements

The authors have no support to report.

Funding

The authors have no funding to report.

Competing Interests

The authors have declared that no competing interests exist.

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Псевдопарадокс курильщика у пациентов с инфарктом миокарда с подъёмом сегмента ST

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Дата получения: 7 января 2022 ♦ **Дата приемки:** 5 апреля 2022 ♦ **Дата публикации:** 30 апреля 2023

Образец цитирования: Janjani P, Salehi N, Asadmobini A, Siabani S, Nalini M. Smoker pseudo-paradox in ST-segment elevation myocardial infarction patients. Folia Med (Plovdiv) 2023;65(2):243-250. doi: 10.3897/folmed.65.e80189.

Резюме

Введение: Курение сигарет является предотвратимой причиной сердечно-сосудистых заболеваний и смертности. Несмотря на неблагоприятные последствия курения, в некоторых исследованиях сообщается о термине „парадокс курильщика", означающем лучшие результаты у курильщиков после острого инфаркта миокарда.

Цель: Целью настоящего исследования было оценить взаимосвязь между статусом курения и смертностью в течение одного года у пациентов с инфарктом миокарда с подъёмом сегмента ST (STEMI).

Материалы и методы: Это было основанное на регистре когортное исследование пациентов с STEMI из больницы Имам-Али, Керманшах, Иран. Последовательные пациенты с STEMI (июль 2016 г. – октябрь 2018 г.) были стратифицированы по статусу курения и наблюдались в течение одного года. Пропорциональные модели Кокса использовались для оценки грубых, скорректированных по возрасту и полных соотношений рисков с 95% доверительными интервалами (HR, 95% CI).

Результаты: Из 1975 пациентов (средний возраст 60.1 года, 76.6% мужчины), включенных в исследование, 48.1% (*n*=951) были курильщиками (средний возраст 57.7 года, 94.7% мужчины). Общий и скорректированный по возрасту HR (95% CI) интервал для установления взаимосвязи между курением и смертностью составил 0.67 (0.50–0.92) и 0.89 (0.65–1.22) соответственно. После поправки на возраст, пол, артериальную гипертензию, диабет, индекс массы тела, инфаркт миокарда передней стенки, креатинкиназу-МБ, скорость клубочковой фильтрации, фракцию выброса левого желудочка, холестерин липопротеинов низкой плотности и гемоглобин курение было связано с повышенным риском смертности: HR (95% CI: 1.56 (1.04-2.35).

Заключение: В нашем исследовании курение было связано с повышенным риском смертности. Хотя у курильщиков был лучший результат, это могло бы измениться после учёта возраста и других факторов, связанных с STEMI.

Ключевые слова

парадокс курильщика, курение сигарет, инфаркт миокарда с подъёмом сегмента ST, первичное чрескожное коронарное вмешательство, тромболитическая терапия