ARYA Atherosclerosis J

Original Article Open Access

Impact of Smoking Status on Major Adverse Cardiovascular Events in STEMI Patients: A Retrospective Cohort Study in Western Iran

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Received: 2025-08-27
Accepted: 2026-04-06

How to cite this article:
Janjani P, Salehi N, Motevaseli S, Asadmobini A. **Impact of Smoking Status on Major Adverse Cardiovascular Events in STEMI Patients: A Retrospective Cohort Study in Western Iran.** ARYA Atheroscler. 2026; 22(3): 27-35.

DOI:
https://doi.org/10.48305/arya.2026.45612.3094

Abstract

BACKGROUND: ST-elevation myocardial infarction (STEMI) remains a major cause of mortality worldwide. Whether smoking status and cumulative exposure influence prognosis in the contemporary era of timely reperfusion and intensive secondary prevention remains uncertain, particularly in populations with very high smoking rates. This study aimed to evaluate the association between smoking status and major adverse cardiovascular events (MACE) among patients with STEMI in western Iran.

METHODS: A retrospective cohort study was conducted involving 645 consecutive patients with STEMI at a tertiary center in Western Iran. Patients were stratified by smoking status: never-smokers (n = 356), moderate smokers (<15 pack-years, n = 124), and heavy smokers (≥15 pack-years, n = 165). Outcomes included 1-year all-cause mortality and MACE over 365 days. Cox regression models adjusted for confounders were used to estimate hazard ratio with 95% confidence intervals (HR, 95% CI).

RESULTS: In crude analyses, heavy smokers had lower 1-year mortality (HR = 0.44, 95% CI: 0.22–0.87) and MACE (HR = 0.60, 95% CI: 0.40–0.91) compared to never-smokers. After full adjustment, these associations became non-significant (mortality HR = 0.92, 95% CI: 0.28–3.02; MACE HR = 1.51, 95% CI: 0.81–2.81). Never-smokers were older, more likely female, and had higher comorbidity rates, explaining crude differences. Socioeconomic factors, particularly education, influenced outcomes.

CONCLUSION: The smoker's paradox is driven by confounders, not a protective effect of smoking. Smoking cessation and addressing socioeconomic disparities are critical for improving STEMI outcomes in high-risk populations.

Keywords: Myocardial Infarction; Smoking; Mortality; Cardiovascular Diseases

Introduction

ST-elevation myocardial infarction (STEMI) remains a major global contributor to cardiovascular morbidity and mortality, requiring urgent interventions like primary percutaneous coronary intervention (PPCI) to improve outcomes[1,2]. Cigarette smoking, a key modifiable risk factor, accelerates atherosclerosis and precipitates acute coronary events, often at younger ages[3,4]. Historical studies from the thrombolytic era reported a "smoker's paradox," suggesting better short-term outcomes in smokers post-myocardial infarction, potentially due to enhanced thrombolytic responses or ischemic preconditioning[5,6].

Recent PPCI-era research has largely debunked this paradox, attributing apparent benefits to confounders such as younger age, male predominance, and fewer comorbidities in smokers[7,8]. However, gaps remain, particularly in retrospective analyses with limited adjustment for confounders like socioeconomic status and renal function, or in regions with high smoking prevalence like western Iran[9,10]. The impact of smoking intensity (quantified by pack-years) on 1-year mortality and major adverse cardiovascular events (MACE) over 365 days is underexplored in these settings[11].

This retrospective cohort study investigates the impact of smoking intensity, measured in pack-years, on MACE in STEMI patients at a tertiary center in western Iran, a region with high smoking prevalence. We aim to clarify whether the smoker's paradox persists or is driven by confounding factors, providing insights for targeted prevention strategies in high-risk populations.

Methods

Study Design and Population

This retrospective cohort study included all consecutive patients with STEMI at Imam Ali Hospital, a tertiary cardiovascular referral center in western Iran, between December 2019 and August 2020. Patients were eligible if they presented within 12 hours of symptom onset (typical chest pain or anginal equivalent). Patients with cardiogenic shock at admission, a history of myocardial infarction (MI), prior revascularization (PCI/CABG), missing smoking history, or incomplete follow-up data were excluded. The final analysis cohort comprised 645 patients.

Assessment of smoking exposure

Smoking status was determined through patient self-report at admission and confirmed by review of medical records. Never-smokers were defined as individuals who had never smoked cigarettes. Ever-smokers were categorized into two groups based on cumulative smoking exposure, expressed in pack-years: moderate smokers (<15 pack-years) and heavy smokers (≥15 pack-years). The cut-off of 15 pack-years was chosen based on prior literature[12] and the distribution of smoking intensity in our cohort. We categorized smoking burden using a 15 pack-year cut-off (<15 vs ≥15 pack-years). This threshold was selected a priori based on prior cardiovascular and respiratory literature that commonly uses 15 pack-years to distinguish lighter from heavier lifetime smoking exposure, and because it corresponded to the distribution of smoking intensity in our cohort[12].

Clinical and laboratory variables

Baseline demographic data, cardiovascular risk factors (hypertension, diabetes, dyslipidemia), and clinical characteristics at admission were collected from a standardized checklist. Laboratory parameters, including serum creatinine, blood glucose, and lipid profile, were obtained on admission.

Outcomes

The primary endpoint was all-cause 1-year mortality followed after hospital discharge and continued for 12 months following STEMI diagnosis. The secondary endpoint was MACE, defined as a composite of cardiac mortality, myocardial infarction, PCI, CABG, or stroke occurring within 365 days post-STEMI.

Follow-up

Contact information for patients or their families was recorded at admission to facilitate follow-up. Patients were contacted by phone one year after AMI diagnosis. For reported deaths, clinical and hospital records, including cause of death, were reviewed. The follow-up period was defined as the time from AMI diagnosis to death, loss to follow-up, or 365 days post-STEMI.

Ethical Approval and Consent

All patients provided written informed consent. The study protocol was approved by the Research Ethics Committee at Kermanshah University of Medical Sciences (Ethics code: IR.KUMS.REC.1400.252).

Statistical Analysis

Continuous variables were presented as mean ± SD and categorical variables as counts and percentages. Baseline characteristics were compared using chi-square tests and analysis of variance (ANOVA) as appropriate. Cox proportional hazards regression models estimated hazard ratios (HRs) and 95% confidence intervals (CIs) for all-cause mortality and MACE. Three models were assessed: crude, age-adjusted, and fully adjusted including age, gender, diabetes, hypertension, high-density lipoprotein-cholesterol (HDL-C), body mass index (BMI), estimated glomerular filtration rate (eGFR), systolic blood pressure (SBP), left ventricular ejection fraction (LVEF), and Killip class. The proportionality of hazards was verified by plotting the Schoenfeld residuals versus time and the Schoenfeld global test (P = 0.511). The study adhered to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines[13].

Results

Demographic and Clinical Characteristics

As shown in Table 1, the study included never-smokers (n = 356), moderate smokers (< 15 pack-years, n = 124), and heavy smokers (≥ 15 pack-years, n = 165). Over 213,840 person-days of follow-up, 66 all-cause deaths (10.23%) occurred. Table 1 summarizes demographic and clinical characteristics.

Never-smokers were significantly older (62.38 ± 13.33 years) compared to moderate (55.53 ± 11.81 years) and heavy smokers (57.63 ± 9.89 years; P < 0.001). Smokers were predominantly male (< 15 pack-years: 95.97%; ≥ 15 pack-years: 96.63%) compared to never-smokers (63.20%; P < 0.001). Heavy smokers had lower BMI (25.50 ± 4.28 vs. 26.81 ± 4.68 kg/m^2; P = 0.034), lower prevalence of hypertension (22.42% vs. 47.89%; P < 0.001) and diabetes (10.91% vs. 29.49%; P < 0.001), and higher eGFR (78.83 ± 17.66 vs. 67.58 ± 22.67 mL/min; P < 0.001) compared to never-smokers. LVEF differed slightly across groups (P = 0.004). 1-year mortality was higher in never-smokers (13.20%) than in moderate (7.26%) and heavy smokers (6.06%; P = 0.021). MACE rates, assessed over 365 days, varied significantly (P = 0.033), with heavy smokers having a higher proportion of patients alive (81.82%) compared to never-smokers (72.19%).

Association with All-Cause Mortality

Table 2 presents unadjusted and adjusted HRs for all-cause 1-year mortality. In the crude model, heavy smokers had a lower mortality risk (HR: 0.44, 95% CI: 0.22–0.87) compared to never-smokers. After age adjustment, this association attenuated (HR: 0.55, 95% CI: 0.27–1.10). In the fully adjusted model, no significant associations were observed (< 15 pack-years: HR: 0.27, 95% CI: 0.33–2.19; ≥ 15 pack-years: HR: 0.92, 95% CI: 0.28–3.02).

Association with MACE

Table 3 shows associations with MACE over 365 days. In the crude model, heavy smokers had a reduced MACE risk (HR: 0.60, 95% CI: 0.40–0.91). This weakened after age adjustment (HR: 0.66, 95% CI: 0.44–1.00) and became non-significant in the fully adjusted model (< 15 pack-years: HR: 1.68, 95% CI: 0.91–3.11; ≥ 15 pack-years: HR: 1.51, 95% CI: 0.81–2.81).

Survival Curves

Using the fully adjusted Cox models from Tables 2 and 3, the corresponding survival curves for

Table 1. Demographic and clinical characteristics of study patients

Characteristics	All (n=645)	Never-smoker (n=356)	≤15 Pack-Years (n=124)	>15 Pack-Years (n=165)	P-value
Age (years)	59.58±12.57	62.38±13.33	55.53±11.81	57.63±9.89	<0.001[a]
Gender, n (%)					
Male	503(77.98%)	225(63.20%)	119(95.97%)	159(96.63%)	<0.001[b]
Female	142(22.02%)	131(36.80%)	5(4.03%)	6(3.64%)	
Education					
Illiterate	206(32.04%)	147(41.29%)	26(21.31%)	33(20%)	
Primary school	146(22.71%)	71(19.94%)	23(18.85%)	52(31.52%)	<0.001
Secondary school	121(18.82%)	51(14.33%)	35(28.69%)	32(21.21%)	
Diploma	106(16.49%)	51(14.33%)	24(19.67%)	31(18.79%)	
Post-diploma and upper	64(9.95%)	36(10.11%)	14(11.48%)	14(8.48%)	
Income					
Very low	327(51.01%)	182(51.27%)	61(50.41%)	84(50.91%)	
Low	137(21.37%)	74(20.85%)	30(24.79%)	33(20%)	
Moderate	117(18.25%)	64(18.03%)	17(14.05%)	36(21.82%)	0.265
Good	55(8.58%)	34(9.58%)	10(8.26%)	11(6.67%)	
Very good	5(0.78%)	1(0.28%)	3(2.48%)	1(0.61%)	
BMI (kg/m2)	26.35±4.53	26.81±4.68	26.31±4.34	25.50±4.28	0.034
Systolic pressure (mmHg)	135.37±26.84	138.54±28.57	130.71±24.21	132.15±24.06	0.002
Diastolic pressure (mmHg)	83.30±14.82	85±16.02	80.40±12.90	81.90±13.00	0.002
Hypertension,n (%)	246(38.20%)	170(47.89%)	39(31.45%)	37(22.42%)	<0.001
Diabetes, n (%)	143(22.17%)	105(29.49%)	20(16.13%)	18(10.91%)	<0.001
Total cholesterol, mmol/l	167.50±41.55	168.41±42.75	171±44.66	162.90±36.01	0.228
HDL-C, mg/dL	38.48±14.65	38.56±12.53	40.78±20.84	36.55±12.87	0.105
eGFR, ml/min	72.23±21.95	67.58±22.67	76.90±21.92	78.83±17.66	<0.001
LVEF	38.71±8.44	38.41±8.83	38.83±8.20	39.27±7.72	0.004
Killip class					
I	411(66.72%)	235(69.12%)	72(61.02%)	104(65.82%)	
II	149(24.19%)	76(22.35%)	36(30.51%)	37(23.42%)	0.065[c]
III	46(7.47%)	22(6.47%)	9(7.63%)	15(9.49%)	
IV	10(1.62%)	7(2.06%)	1(0.85%)	2(1.27%)	
Reperfusion	531(82.33%)	283(79.49%)	105(84.68%)	143(86.67%)	0.102
One year mortality	66(10.23%)	47(13.20%)	9(7.26%)	10(6.06%)	0.021
Yes					
MACE					
MI	9(1.40%)	6(1.69%)	1(0.81%)	2(1.21%)	
PCI	81(12.56%)	42(11.80%)	21(16.94%)	18(10.91%)	
CABG	24(3.72%)	15(4.21%)	6(4.84%)	3(1.82%)	0.033
Cardiac death	51(7.91%)	36(10.11%)	8(6.45%)	7(4.24%)	
Stroke	1(0.16%)	----------	1(0.81%)	----------	

a, one-way analysis of variance (ANOVA)
b, Chi-square
c, Fisher's exact test
Data are mean ± SD or n (%). Abbreviations: BMI: body mass index, HDL: high density lipoprotein, eGFR: estimated glomerular filtration rate. LVEF, left ventricular ejection fraction. MACE: major adverse cardiovascular events. MI: myocardial infraction. PCI: Percutaneous coronary intervention. CABG: coronary artery bypass graft.

Table 2. Unadjusted and adjusted association between smoking and all-cause mortality

Variables	Crude model, HRs (95%CIs)	P-value	Age-adjusted HRs (95%CIs)	P-value	Full-adjusted HRs (95%CIs)	P-value
Never-smoker	Reference		Reference		Reference	
< 15 Pack-Years	0.55(0.26-1.11)	0.097	0.64(0.30-1.38)	0.259	0.27(0.33-2.19)	0.221
>5 Pack-Years	0.44(0.22-0.87)	0.019	0.55(0.27-1.10)	0.093	0.92(0.28-3.02)	0.896

Fully adjusted for age, gender, diabetes, hypertension, high-density lipoprotein cholesterol, body mass index, estimated glomerular filtration rate, systolic blood pressure, left ventricular ejection fraction, Killip class.

Table 3. Unadjusted and adjusted association between smoking and MACE

Variables	Crude model, HRs (95%CIs)	P-value	Age-adjusted HRs (95%CIs)	P-value	Full-adjusted HRs (95%CIs)	P-value
Never-smoker	Reference		Reference		Reference	
< 15 Pack-Years	1.07(0.73-1.56)	0.732	1.23(0.83-1.81)	0.299	1.68(0.91-3.11)	0.096
>15 Pack-Years	0.60(0.40-0.91)	0.016	0.66(0.44-1.00)	0.052	1.51(0.81-2.81)	0.190

Fully adjusted for age, gender, diabetes, hypertension, high-density lipoprotein cholesterol, body mass index, estimated glomerular filtration rate, systolic blood pressure, left ventricular ejection fraction, Killip class.

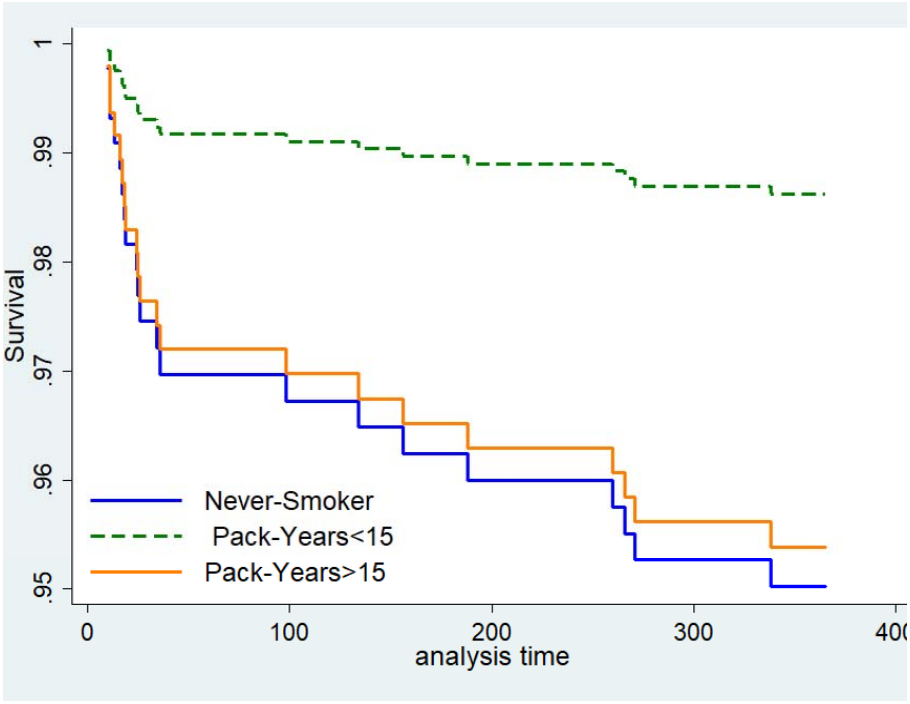

Figure 1. Fully adjusted Cox regression survival curves for all-cause mortality for heavy, moderate, and never-smokers in STEMI patients.

heavy and moderate smokers versus non-smokers are illustrated in Figure 1 (for all-cause mortality) and Figure 2 (for MACE).

Discussion

In this retrospective cohort study of 645 STEMI patients undergoing PPCI, we evaluated the association between smoking status (never-smokers [n = 356], < 15 pack-years [n = 124], ≥ 15 pack-years [n = 165]) and outcomes, including all-cause 1-year mortality and MACE, over a 12-month follow-up. Unadjusted analyses suggested a "smoker's paradox," with heavy smokers showing lower mortality (HR: 0.44, 95% CI: 0.22–0.87) and MACE (HR: 0.60, 95% CI: 0.40–0.91) compared to never-smokers. However, these associations attenuated after age adjustment (mortality HR: 0.55, 95% CI:

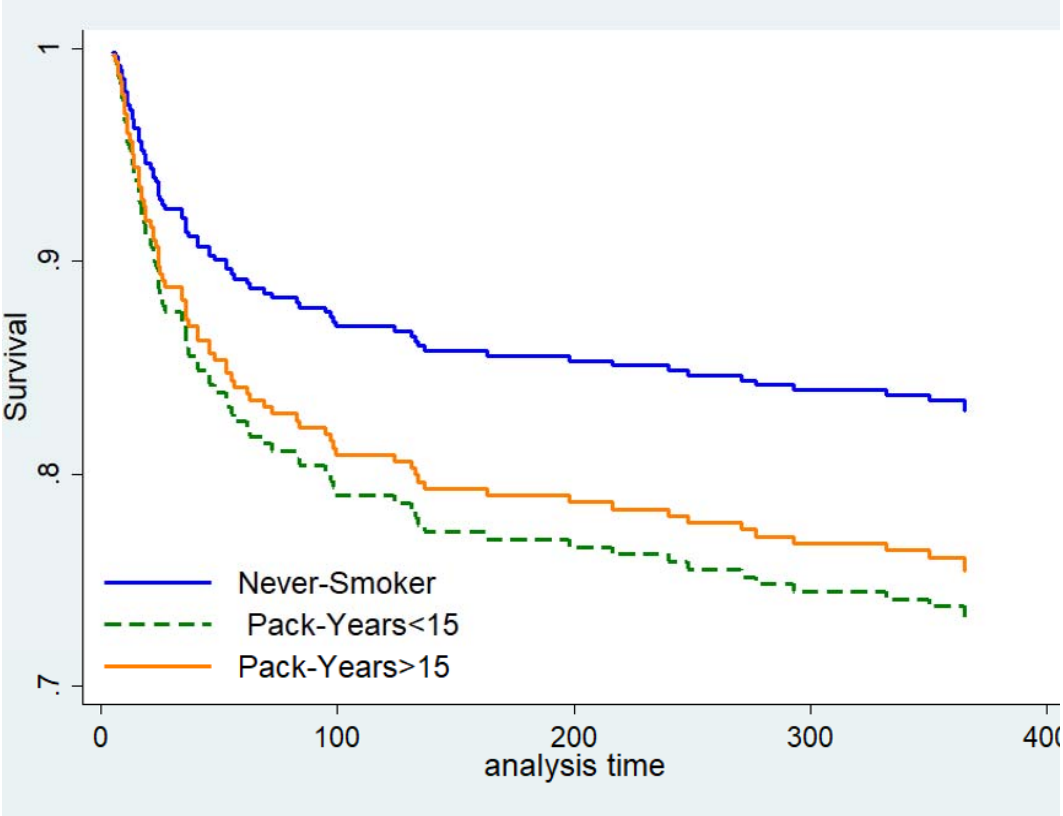

Figure 2. Fully adjusted Cox regression survival curves for MACE for heavy, moderate, and never-smokers in STEMI patients.

0.27–1.10; MACE HR: 0.66, 95% CI: 0.44–1.00) and became non-significant in fully adjusted models (mortality HR: 0.92, 95% CI: 0.28–3.02; MACE HR: 1.51, 95% CI: 0.81–2.81).

The smoker's paradox, initially reported in the thrombolytic era, suggested improved short-term outcomes in smokers post-acute myocardial infarction (AMI), potentially due to enhanced fibrinolytic activity or ischemic precond-itioning[5,6]. Proposed mechanisms included smoking-induced thrombotic occlusions responding better to reperfusion or nicotine and carbon monoxide mimicking preconditioning effects[14,15]. However, PPCI-era studies have largely refuted this, attributing crude benefits to younger age, male predominance, and lower comorbidity burden in smokers[7,16]. Our cohort aligns with this, with never-smokers being older (mean age 62.38 ± 13.33 vs. 57.63 ± 9.89 years in heavy smokers; P < 0.001), more likely female (36.80% vs. 3.64%; P < 0.001), and having higher rates of hypertension (47.89% vs. 22.42%; P < 0.001) and diabetes (29.49% vs. 10.91%; P < 0.001).

Recent studies corroborate the absence of a true paradox. A 2023 study of 1,612 AMI patients reported lower crude mortality in smokers but higher adjusted long-term mortality (HR: 1.56, 95% CI: 1.14–2.14), driven by confounders like age and NT-proBNP levels[8]. A South Asian cohort showed a crude protective HR (0.67) reversing to an increased risk (HR: 1.56) after adjustment, mirroring our findings[9]. A pooled analysis of 2,564 STEMI patients found no mortality benefit for smokers post-adjustment but noted higher risks of heart failure and recurrent infarction[11]. These findings collectively suggest smoking accelerates AMI onset without conferring survival advantages[3,4].

Biological and clinical factors may explain the crude paradox. Smokers often present with thrombotic occlusions, more amenable to PPCI, and earlier symptom onset due to tobacco-induced platelet activation and fibrinogen

elevation[3,17]. Our study showed higher reperfusion rates in heavy smokers (86.67% vs. 79.49% in never-smokers; P = 0.102), potentially contributing to crude benefits. Heavy smokers also had lower BMI (25.50 ± 4.28 vs. 26.81 ± 4.68 kg/m²; P = 0.034) and higher eGFR (78.83 ± 17.66 vs. 67.58 ± 22.67 mL/min; P < 0.001), suggesting short-term resilience. However, chronic endothelial dysfunction and oxidative stress from smoking likely negate these effects long-term[7,18].

Socioeconomic factors, particularly education, significantly influenced outcomes in our cohort. Never-smokers had higher illiteracy rates (41.29% vs. 20% in heavy smokers; P < 0.001), which likely impacted healthcare access, treatment adherence, and risk factor management. Low educational attainment is associated with a 75% increase in 30-day hospital readmissions post-myocardial infarction due to poor health literacy, limiting understanding of medical instructions and self-care[10]. A longitudinal study reported a three-fold higher 1-year mortality risk (HR: 3.09, 95% CI: 1.69–5.65) in patients with low education, driven by delayed presentation and suboptimal engagement with preventive services[19]. In our western Iran context, where illiteracy is prevalent among older and non-smoking populations, these factors may amplify disparities, contributing to the crude smoker's paradox by disadvantaging never-smokers in timely reperfusion and follow-up care. Although income levels did not differ significantly (P = 0.265), education-related disparities underscore the need for targeted interventions to improve health literacy and access in vulnerable groups.

Environmental exposures, such as air pollution, may mimic a "new smoker's paradox" by associating with lower short-term AMI mortality in polluted areas, possibly due to preconditioning effects[20,21]. This warrants further exploration in our region, given its environmental challenges. Regional gaps in data on smoking and STEMI outcomes in western Iran, where tobacco use is highly prevalent, remain a research challenge. According to the WHO 2023 report, tobacco use prevalence among adults in the Eastern Mediterranean Region (including Iran) is approximately 26.7%, significantly higher than the global average of 20.9%, with notably higher rates in men (44.3%) than women (8.4%)[22]. This elevated prevalence, coupled with limited region-specific data on smoking intensity (e.g., pack-years), underscores the need for more robust studies. Our findings, which refute the smoker's paradox after adjusting for confounders, align with limited regional studies[9], but the lack of biological validation remains a limitation. For future research, we recommend employing biomarkers such as serum cotinine levels (for precise nicotine exposure assessment) and inflammatory markers like C-reactive protein (CRP) or interleukin-6 (IL-6) to validate the biological effects of smoking intensity on cardiovascular outcomes. This approach could mitigate self-report bias and enhance mechanistic understanding of smoking's impact in high-risk populations.

Limitations

This study has several limitations. First, prehospital deaths were not captured, which may underestimate the true short-term mortality burden of STEMI in this population and could introduce survival bias, as patients who died before reaching the hospital were systematically excluded. Second, smoking exposure was based on self-report, which is subject to recall bias and underreporting, particularly among light or intermittent smokers[23]; the absence of biochemical validation (cotinine levels) may have led to misclassification of smoking intensity. Third, we were unable to account for the type of tobacco used (cigarettes, waterpipe, or other forms), the duration of cessation among former smokers, or quantify exposure to other substances such as opium or alcohol, all of which are relatively prevalent in the region and could independently influence cardiovascular outcomes. Additionally, data on secondhand smoke exposure were not available. These factors limit the ability to distinguish the isolated effect of cigarette smoking from other lifestyle

or environmental exposures. Finally, as a single-center retrospective study, residual confounding cannot be fully excluded despite multivariable adjustment. The sample size (n = 645) may limit statistical power for detecting subtle subgroup effects, especially in females (22% of the cohort), where gender-specific smoking effects are underexplored[24].

Conclusions

Our findings refute a genuine smoker's paradox in STEMI patients, showing that crude benefits in smokers are driven by demographic and clinical confounders. Smoking remains a major risk factor that accelerates AMI onset and offers no prognostic advantage post-adjustment. These results highlight the urgent need for smoking cessation interventions, such as pharmacotherapy (e.g., varenicline) and behavioral counseling, alongside efforts to address socioeconomic disparities, particularly low education, to improve long-term outcomes in high-risk populations.

Acknowledgements

The authors appreciate the experts of the Research Development Center of Imam Ali and Ayatollah Taleghani Hospital in Kermanshah.

Conflict of interests

The authors declare no conflict of interest.

Funding

There is no funding in this study.

Author's Contributions

Study Conception or Design: PJ; NS; AA
Data Acquisition: NS; SM; AA
Data Analysis or Interpretation: PJ; SM
Manuscript Drafting: SM; AA
Critical Manuscript Revision: PJ; NS
All authors have approved the final manuscript and are responsible for all aspects of the work.

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