

Long-term all-cause mortality rate after ST-elevation myocardial infarction and its predictors: ST Elevation Myocardial Infarction Cohort in Isfahan Study

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Background: ST-elevation myocardial infarction (STEMI) remains a significant global health concern, especially in low- and middle-income regions. This study aimed to identify long-term prognostic factors among STEMI patients, offering insights into improving patient outcomes. **Materials and Methods:** This study represents the 5-year follow-up of STEMI patients in the SEMI Cohort in Isfahan registry, a clinical-based registry of STEMI patients in Isfahan, Iran, from October 2015. All patients with STEMI within 24 h of symptom onset underwent a comprehensive evaluation. The dataset included demographic information, laboratory data, medical history, and clinical in-hospital data. Over 5 years, annual follow-ups were conducted to track hospitalization and patient all-cause mortality. Utilizing univariate and multivariate Cox regression proportional hazard modeling, we aimed to identify predictors of death. **Results:** In this study, involving 759 patients (621 men and 138 women) with a mean age of 58.92 ± 11.79 years, 158 deaths (21%) with a mean age of 70.33 ± 12.66 years occurred after STEMI. In the multiple model our analysis revealed that the following variables significantly increased all-cause mortality independently: Older age (hazard ratio [HR]: 1.070, $P < 0.001$), lower body mass index (HR: 0.890, $P < 0.001$), hypertension status (HR: 2.441, $P < 0.001$), lower systolic blood pressure at initial presentation (HR: 0.983, $P < 0.001$), number of affected epicardial territories (HR: 2.979, $P < 0.001$), lower last ejection fraction before discharge (HR: 0.951, $P < 0.001$), lower hemoglobin level (HR: 0.747, <0.001), higher plasma glucose level (HR: 1.005, $P < 0.001$), and in-hospital complications (HR: 7.646, $P < 0.001$). **Conclusion:** This study identified a range of factors that predict STEMI-related mortality. These findings are pivotal for future planning and decision-making regarding appropriate diagnostic and therapeutic strategies during patient follow-up, contributing to improved outcomes in STEMI care.

Key words: Cardiovascular disease, mortality, risk factor, secondary prevention, ST-elevation myocardial infarction

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INTRODUCTION

Acute coronary syndrome (ACS) is a leading cause of both mortality and disability worldwide.^[1,2] Among its manifestations, ST-elevation myocardial infarction (STEMI) represents the most lethal subtype of ACS, typically presenting with chest pain or similar

symptoms and confirmed by elevated ST segment and serum troponin levels.^[3] Despite advances in the treatment and management of STEMI, it continues to have high mortality and morbidity rates, particularly in low- and middle-income countries.^[4] In the initial month post-STEMI, patients face a heightened risk of death, often due to cardiogenic shock, cardiopulmonary arrest,

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and mechanical complications. In addition, both noncardiac factors, such as malignancy and infectious diseases, and cardiac factors, such as severe heart failure, contribute to a high long-term mortality rate.^[5,6]

Several factors predict the risk of morbidity and mortality in STEMI patients, including modifiable (lifestyle factors such as body mass index, smoking status, and blood pressure) and nonmodifiable (such as sex and age) factors.^[7,8] The impact of these modifiable risk factors has been thoroughly explored in previous studies.^[9-14] With recent lifestyle changes in developing countries, the prevalence of post-MI late major adverse cardiac events, such as high blood pressure, hypercholesterolemia, and reduced daily activity, has varied.^[15] While recent studies have examined the short-term relationship between STEMI and subsequent death, only a limited number have focused on the long-term outcomes of this condition.^[15,16] Given the challenges associated with long-term patient monitoring and follow-up, it is imperative to identify more potent risk factors and develop strategies for monitoring and managing them to enhance patients' quality of life and prognosis.^[17]

Therefore, in this 5-year cohort study, our objective was to shed light on the burden of myocardial infarction (MI) and evaluate the relationship between risk factors such as demographic information, past medical history, laboratory data, and clinical information and the occurrence of 5-year mortality in patients with STEMI.

MATERIALS AND METHODS

Study population

This observational, prospective longitudinal cohort study represents the 5th-year follow-up of STEMI patients in the ST Elevation Myocardial Infarction Cohort in Isfahan (SEMI-CI) study. The SEMI-CI registry has been comprehensively described previously.^[18] In summary, this study constructed a clinical-based registry of STEMI patients. From October 2015 to October 2016, all consecutive patients who presented with STEMI and were referred to three university-affiliated hospitals in Isfahan, Iran, were prospectively enrolled. The inclusion criteria included all patients aged >18 years who presented with chest pain or equivalent symptoms lasting more than 20 min within 24 h before admission, along with ST-segment elevations or left bundle branch block evident on the diagnostic electrocardiogram (ECG). The exclusion criteria consisted of patients who did not meet the diagnostic criteria for STEMI, those who declined to participate, patients who died during hospitalization, patients who refused follow-up, or whose follow-up information was unavailable or incomplete.

Data collection

At the onset of the registry, trained nurses meticulously reviewed patients' admission records in the emergency room. To reduce information bias, consultation with the quality control committee, comprising epidemiologists, statisticians, specialized physicians, and information technology personnel, was conducted before enrolling the STEMI patients. To explore long-term mortality risk factors, we leveraged a subset of data collected during the baseline phase of the SEMI-CI study. This dataset included demographic information such as age and sex; laboratory data encompassing glucose plasma level, creatinine, and hemoglobin (Hb) levels; and medical history, covering hypertension, diabetes mellitus (DM), congestive heart failure (CHF), prior cardiovascular disease (CVD), and cigarette smoking.

Clinical in-hospital data included initial ECG findings, site of acute MI (AMI), Killip class, systolic blood pressure (SBP), pulse rate (PR), and body mass index (BMI). Furthermore, details about reperfusion therapy, including angiographic findings, angioplasty details, the number of affected epicardial territories, the type of reperfusion therapy, and the ejection fraction (EF) before discharge, were meticulously recorded for each patient. In-hospital complications were also assessed.

Data collection procedures, such as medical interviews, physical examinations, and paraclinical tests, were conducted by trained personnel utilizing calibrated instruments and a standardized protocol to minimize measurement bias. Blood samples were drawn from each patient's cubital vein after a minimum overnight fasting of ≥12 h during hospitalization, and the serum levels of glucose, creatinine, and Hb were assayed. DM was defined as a fasting blood glucose level ≥126 mg/dL or the use of antidiabetic agents.^[19] Hypertension was diagnosed in participants with a history of blood pressure ≥140/90 mmHg or who were taking antihypertensive medications.^[20] Congestive heart failure was determined according to the Framingham criteria. Killip classification was defined as: Class I, patients with no clinical signs of heart failure; Class II, patients with rales or crackles in the lungs, an S3 gallop, and elevated jugular venous pressure; Class III, patients with acute pulmonary edema; Class IV, patients with cardiogenic shock, SBP lower than 90 mmHg, and evidence of low cardiac output.^[21]

Patients with a history of MI, percutaneous cardiac intervention (PCI), coronary artery bypass grafting (CABG), confirmed stable angina, peripheral vascular disease, or stroke were classified as having CVD. Current smoking status was defined as regular smoking within the month preceding admission. In-hospital complications included

cerebrovascular accidents, reinfarction, stent thrombosis, tamponade, ventricular septal defect, heart failure, and atrial fibrillation.

SBP and PR were recorded at the initial presentation by trained nurses. BMI was defined as body weight (kg) divided by the square of body height (m^2) measured during hospitalization.

The 5th-year follow-up was conducted with the approval of the Ethics Committee of the National Institute for Medical Research Development (NIMAD) under the reference number "IR.NIMAD.REC.1399.252." Informed consent was obtained from all patients before study inclusion.

Patient follow-up occurred annually for five years. Patients who remained in the study were contacted by trained nurses, and information regarding hospitalization and patient deaths was gathered through telephone interviews using a standardized checklist. The outcome of the study was the occurrence of all-cause mortality. For patients who had passed away during the previous years of follow-up, data concerning the cause and date of death were obtained from the patient's family and recorded in the patient's file. In addition, all hospital records for in-hospital deaths were collected.

Statistical analysis

Descriptive statistics were employed to provide a concise summary of the data. Continuous variables are presented as the mean \pm standard deviation, while categorical variables are summarized using frequencies and percentages.

Survival rates were estimated using the Kaplan–Meier method, and *P* values for comparing survival curves were calculated through the log-rank test. All the statistical tests were two-tailed, and significance was defined as *P* < 0.05. To identify predictors of all-cause mortality, the conditional forward stepwise procedure was used. Cox proportional hazard modeling was also employed to ascertain predictors of the composite outcome of all-cause mortality. Data analysis was performed using SPSS software (version 22.0, IBM Corporation, Armonk, NY, USA).

RESULTS

A total of 759 patients, comprising 621 men and 138 women, were included in our analyses. The mean age of the surviving patients was 58.92 ± 11.79 years (*P* < 0.01). Over 5 years following STEMI, 158 deaths were recorded, equating to a mortality rate of 20.8%. The mortality rate of women was 36.23% while it was 17.39% among men. The mean age of the deceased patients was 70.33 ± 12.66 years, with complete data available for all individuals [Figure 1].

Demographic data, laboratory data, past medical history, and clinical data were compared between the two groups of surviving and deceased individuals during the 5-year follow-up, as shown in Table 1. In this comparison, all variables except the use or non-use of PCI for initial reperfusion showed a significant difference between the two groups. In addition, Kaplan–Meier survival analysis was employed to estimate all-cause mortality rates for patients at various time points based on age and sex [Figures 2–4]. The mean of survival time is 1534 days.

We further constructed both crude and adjusted Cox regression models. The crude hazard ratios (HRs) for various variables in relation to death due to STEMI are presented in Table 2. Female sex, each year of age increase, a history of previous CVD, history of DM, history of hypertension, history of previous CHF, each unit increase in PR, a higher Killip class, each unit increase in creatinine, each unit increase in glucose plasma level, number of affected epicardial territories, transmural MI of the anterior wall, and any complications that occurred during hospitalization, all of which exhibited significant relationships with an elevated risk of death due to STEMI (*P* < 0.05). Conversely, higher BMI, history of past smoking, each unit increase in SBP at initial presentation, higher EF at discharge, initial reperfusion by thrombolysis, and each unit increase in Hb were significantly related to a decreased risk of death due to STEMI (*P* < 0.05).

A multivariate Cox proportional hazard analysis was performed for the significant variables to adjust the effects of the variables on each other and to independently assess the effects of each risk factor on STEMI patient mortality. The adjusted hazard ratios are shown in Table 3. The proportional hazards assumption was assessed with the

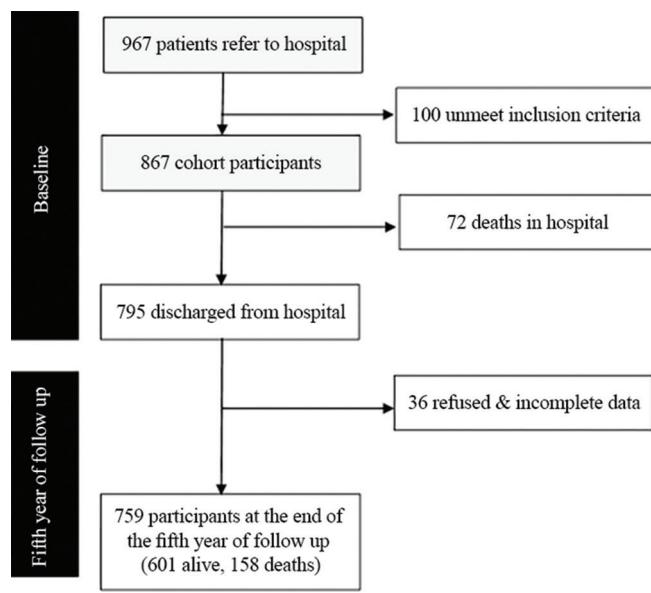


Figure 1: Flow-chart of patient selection

Table 1: Comparison of all variables between surviving and deceased ST-elevation myocardial infarction patients after 5 years of follow-up

Variable	Alive (n=601), n (%)	Dead (n=158), n (%)	P*
Demographic data			
Gender			
Female	88 (14.64)	50 (31.65)	<0.001
Male	513 (85.36)	108 (68.35)	
Age	58.92±11.79	70.33±12.66	<0.001
Laboratory data			
Earliest Hb level	14.49±1.70	13.41±2.03	<0.001
Earliest creatinine	1.17±0.30	1.49±0.68	<0.001
Glucose plasma level	163.39±73.96	208.54±94.21	<0.001
Past medical history			
Previous cardiovascular disease [†]	198 (32.95)	61 (38.60)	<0.001
Heart failure	87 (14.48)	25 (15.82)	<0.001
Smoke	256 (42.60)	42 (26.58)	<0.001
Diabetes	211 (35.11)	59 (37.34)	<0.001
Hypertension	210 (34.94)	71 (44.93)	<0.001
Clinical data			
BMI	26.52±3.73	24.94±2.90	<0.001
PR	77.81±20.32	87.01±28.80	<0.001
SBP at first presentation	129.20±26.04	117.55±30.44	<0.001
Last EF before discharge	38.83±11.16	32.25±9.77	<0.001
Location of MI			
Anterior STEMI	314 (52.25)	97 (61.39)	<0.001
Killip class			
Class I	572 (95.17)	123 (77.85)	<0.001
Class II	27 (4.49)	17 (10.76)	
Class III	1 (0.17)	4 (2.53)	
Class IV	1 (0.17)	14 (8.86)	
Initial reperfusion – primary PCI			
No	325 (54.08)	80 (50.63)	0.474
Yes	276 (45.92)	78 (49.37)	
Initial reperfusion – thrombolysis			
No	311 (51.75)	106 (67.09)	0.001
Yes	290 (48.25)	52 (32.91)	
Number of epicardial territories			
0	10 (1.66)	0	<0.001
1	244 (40.60)	34 (21.52)	
2	171 (28.45)	41 (25.95)	
3	176 (29.28)	83 (52.53)	
In hospital complications [†]			
No	558 (92.85)	85 (53.80)	<0.001
Yes	43 (7.15)	73 (46.20)	

*P<0.05 was considered statistically significant; [†]Including MI, angina, stroke, PCI, CABG, PVD; [‡]Including cerebrovascular accident, Reinfarction, stent thrombosis, mechanical complications, heart failure, atrial fibrillation during PCI. Hb=Hemoglobin; BMI=Body mass index; STEMI=ST-elevation myocardial infarction; PCI=Percutaneous cardiac intervention; MI=Myocardial infarction; EF=Ejection fraction; PR=Pulse rate; SBP=Systolic blood pressure; CABG=Coronary artery bypass grafting; PVD=Peripheral vascular disease

Schoenfeld residuals, and we did not see any significant result. Each year of age increase, history of hypertension, each unit increase in glucose plasma level, number of

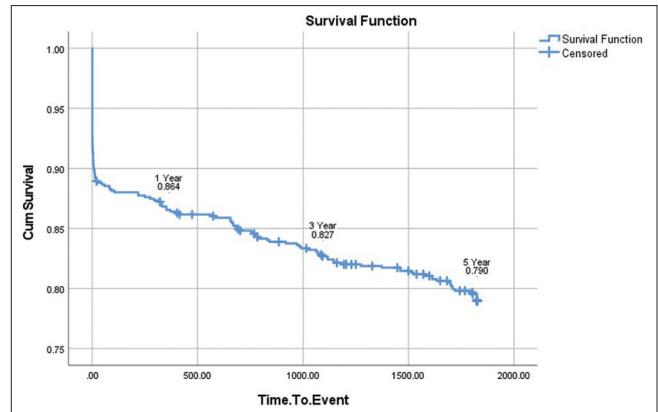


Figure 2: Kaplan-Meier survival curves following STEMI

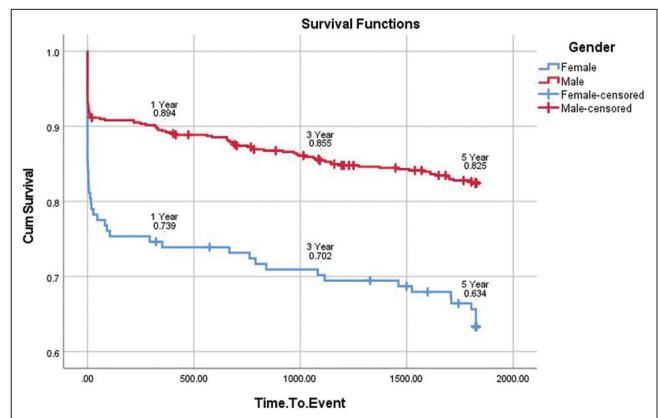


Figure 3: Kaplan-Meier survival curves stratified by sex (the P value of the log-rank test was <0.001)

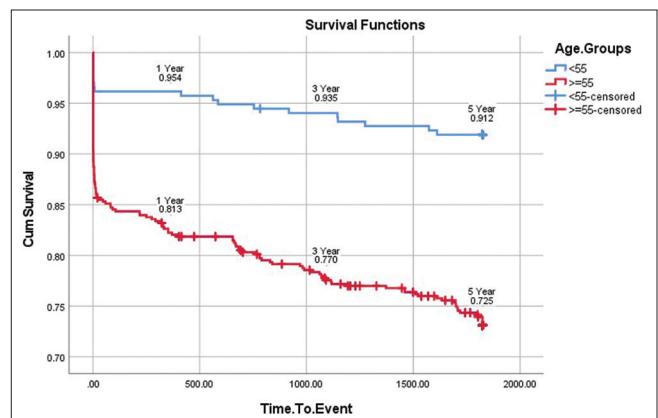


Figure 4: Kaplan-Meier survival curves stratified by age groups (the P value of the log-rank test was <0.001)

affected epicardial territories, and any complications that occurred during hospitalization independently increased the risk of death after STEMI ($P < 0.05$). On the other hand, higher BMI, each unit increase in SBP at initial presentation, higher EF at discharge, and each unit increase in Hb were associated with a lower risk of death ($P < 0.05$).

Table 2: Crude hazard ratios (95% confidence intervals) for all risk factors

Variable	B	HR	95% CI	P*
Sex (female/male)	0.844	2.325	1.662–3.251	<0.001
Age	0.068	1.070	1.056–1.084	<0.001
Earliest Hb level	−0.291	0.747	0.689–0.811	<0.001
Earliest creatinine	0.735	2.085	1.772–2.453	<0.001
Glucose plasma level	0.005	1.005	1.003–1.006	<0.001
Previous cardiovascular disease [†]	0.725	2.064	1.457–2.924	<0.001
CHF	0.556	1.743	1.125–2.702	0.013
Smoke	−0.491	0.612	0.426–0.878	0.008
Diabetes	0.651	1.918	1.355–2.716	<0.001
Hypertension	0.893	2.441	1.710–3.485	<0.001
BMI	−0.117	0.890	0.849–0.932	<0.001
PR	0.014	1.014	1.008–1.019	<0.001
SBP at initial presentation	−0.017	0.983	0.977–0.989	<0.001
Last EF before discharge	−0.050	0.951	0.937–0.965	<0.001
Location of MI (anterior STEMI/other STEMI)	0.331	1.392	1.011–1.918	0.043
Killip class (class II/class I)	0.978	2.658	1.600–4.418	<0.001
Killip class (class III/class I)	1.947	7.010	2.585–19.011	<0.001
Killip class (class IV/class I)	2.461	11.722	6.643–20.686	<0.001
Initial reperfusion primary PCI (yes/no)	0.116	1.123	0.822–1.535	0.464
Initial reperfusion thrombolysis (yes/no)	−0.573	0.564	0.405–0.786	0.001
Number of epicardial territories (2/1)	0.503	1.653	1.049–2.605	0.030
Number of epicardial territories (3/1)	1.092	2.979	1.998–4.442	<0.001
In Hospital complications [‡]	2.034	7.646	5.561–10.512	<0.001

*P<0.05 was considered statistically significant; [†]Including MI, Angina, Stroke, PCI, CABG, PVD; [‡]Including cerebrovascular accident, reinfarction, stent thrombosis, mechanical complications, heart failure, atrial fibrillation during PCI. CHF=Congestive heart failure; BMI=Body mass index; PR=Pulse rate; SBP=Systolic blood pressure; CI=Confidence interval; STEMI=ST-elevation myocardial infarction; MI=Myocardial infarction; CABG=Coronary artery bypass grafting; PVD=Peripheral vascular disease; PCI=Percutaneous cardiac intervention; HR=Hazard ratio; EF=Ejection fraction; Hb=Hemoglobin

Table 3: Adjusted hazard ratios (95% confidence intervals) for significant variables in multivariate analysis

Variable	B	HR	95% CI	P*
Age	0.043	1.044	1.028–1.060	<0.001
BMI	−0.083	0.920	0.873–0.970	0.002
Hypertension	0.484	1.622	1.115–2.360	0.011
SBP at first presentation	−0.006	0.994	0.988–1.000	0.042
Number of epicardial territories (2/1)	0.491	1.635	1.027–2.602	0.038
Number of epicardial territories (3/1)	0.683	1.980	1.313–2.985	0.001
Last EF before discharge	−0.020	0.981	0.966–0.996	0.012
Earliest Hb level	−0.100	0.905	0.830–0.986	0.022
Glucose plasma level	0.002	1.002	1.001–1.004	0.005
In hospital complications [†]	1.298	3.661	2.515–5.329	<0.001

*P<0.05 was considered statistically significant; [†]Including cerebrovascular accident, reinfarction, stent thrombosis, mechanical complications, heart failure, atrial fibrillation during PCI. SBP=Systolic blood pressure; BMI=Body mass index; HR=Hazard ratio; EF=Ejection fraction; Hb=Hemoglobin; BMI=Body mass index; CI=Confidence interval

DISCUSSION

This study estimated the 5-year mortality rate among STEMI patients and identified several significant risk factors affecting long-term patient mortality. These factors included age, BMI, hypertension, SBP, the number of affected epicardial territories, EF, Hb levels, plasma glucose levels, and in-hospital complications.

Notably, global studies on the long-term mortality rates of STEMI patients have varied. For instance, a large-scale Japanese cohort study reported a cumulative 5-year incidence of all-cause death of 21.4%, consistent with our findings.^[22]

Conversely, an American Academic Hospital study with a 9.5-year follow-up of ACS patients noted an unadjusted mortality rate of 44% among STEMI patients.^[23] Furthermore, a comparative analysis of 2-year mortality rates in STEMI patients between two cohorts ($n = 1479$ and $n = 22,432$) revealed a significantly greater mortality rate in the latter cohort, at 14.5%, compared to 11.4%.^[24]

Studies investigating the long-term mortality rates of STEMI patients in Iran are relatively scarce. However, a recent study reported survival rates of 0.95, 0.88, and 0.82 at 28 days, 1 year, and 3 years post-MI, respectively.^[25] The observed trend of increasing mortality rates during

the follow-up period can be attributed to various factors, including disparities in quality of life, obesity, education level, access to appropriate medical care, patient treatment adherence, secondary prevention strategies, utilization of cardiac rehabilitation, and the increase in noncardiovascular diseases. These factors significantly influence the prognosis of STEMI patients during long-term follow-up.^[26-29] For instance, in our study, only 46.64% of patients received PCI, a rate considerably lower than those reported in high-income countries where PCI access often exceeds 70%.^[30]

Patient characteristics are pivotal in determining outcomes, and numerous factors have been identified in the literature. A systematic review and meta-analysis suggested greater short-term mortality in women but not long-term.^[31] However, our analysis, consistent with recent research, revealed a greater long-term mortality rate in women than in men.^[32] Hormonal differences, older age at the onset of MI, and the presence of more comorbidities at that age in women may help explain these findings.^[33,34]

In prior studies, the age at which MI occurs has consistently been identified as a primary determinant of long-term mortality.^[16,35] The association between older age and increased long-term mortality can be attributed to factors such as reduced life expectancy, a greater prevalence of comorbidities, and atypical symptoms that may hinder treatment and diagnosis.^[36]

The impact of BMI on patients' long-term mortality rates has yielded contradictory results in recent studies, often referred to as the "obesity paradox."^[37,38] An analysis of more than 5 million entries from Japanese Circulation Society medical facilities spanning from 2012 to 2020 revealed that while a low BMI is associated with increased mortality rates, the impact of obesity on in-hospital mortality varies depending on the type of CVD.^[39] In our study, a higher BMI was associated with a better patient prognosis. One potential explanation is that overweight and obese patients may exhibit earlier AMI symptoms, possibly due to increased awareness of their greater risk or more severe symptoms resulting from increased myocardial muscle demand in overweight individuals.^[40] Another study described several hypotheses that might clarify the observed beneficial clinical outcomes in patients with higher BMI. One explanation could be that individuals in the lower BMI categories often exhibit compromised metabolic health, including cachexia. In addition, patients with elevated BMI tend to receive more consistent medical therapy. This systematic pharmacological management, coupled with diligent post-PCI follow-up, may contribute positively to their long-term prognosis.^[41] However, A single-center retrospective study of 6496 patients showed

uniform aggressive treatment across all patients; findings demonstrate that the obesity paradox persists even after extended long-term follow-up in MI patients undergoing PCI.^[42] Nonetheless, it is crucial to consider that BMI is an index that does not differentiate between fat, muscle, and skeletal mass. The association between a higher BMI and a better prognosis may be attributed to a greater muscle volume in these patients.^[38]

Consistent with previous studies, our findings demonstrated that a higher heart rate at admission was associated with a worse long-term prognosis.^[43] This can be justified by the increased myocardial oxygen consumption in ischemic conditions and the increased likelihood of arrhythmias.^[44]

Elevated plasma glucose levels at admission were identified as a predictor of adverse future outcomes in our patients. While previous studies generally agree on this effect,^[45,46] Conflicting views remain regarding which markers of glucose dysregulation – such as fasting glucose, 2-h post-load glucose from an oral glucose tolerance test, or glycated hemoglobin A1c – serve as the most reliable predictors of patient prognosis.^[47-49]

Interestingly, our study reported greater long-term survival rates in smokers than in nonsmokers. Although smoking is a recognized risk factor for AMI, studies have shown conflicting results regarding its effect on the long-term mortality rate of AMI patients.^[25,50] A study conducted in Korea involving more than 20,000 AMI patients revealed that current smokers were younger, more likely to be male, and had a lower incidence of hypertension, diabetes, dyslipidemia, and a previous history of ischemic heart disease than nonsmokers. In addition, smokers had less severe initial manifestations of hemodynamic imbalance.^[51] Another possible explanation is that smokers may benefit more from clopidogrel treatment. A trial involving more than 3400 patients with STEMI assessed the clinical efficacy of clopidogrel and revealed that current smokers experienced greater benefits from the medication than nonsmokers.^[52] It is important to note that the relationship between smoking and long-term mortality in AMI patients is complex, and further research is required to fully understand the mechanisms and factors contributing to these outcomes. Smoking cessation remains an essential goal in overall cardiovascular risk reduction despite the potential complexities in the association between smoking and long-term prognosis in AMI patients.

Patients' admission creatinine levels were found to have an adverse prognostic effect on the disease course. This effect can be attributed to its long-term impact on reducing cardiac output and causing cardiac diastolic dysfunction.^[53,54] A recent study focusing on the impact of acute kidney injury

and recovery patterns on the outcome of STEMI patients undergoing PCI demonstrated that even patients who returned to normal renal function before discharge were at a greater risk of adverse outcomes.^[55,56] Moreover, two studies from European regions showed that patients with impaired renal function are less likely to receive optimal therapies, such as invasive procedures like PCI and tend to receive more conservative medication regimens due to concerns about bleeding risk and drug accumulation.^[57,58] Therefore, these findings underscore the importance of renal function as a prognostic factor.

In our study, anterior STEMI was associated with increased mortality. This finding is consistent with the results of previous studies.^[59,60] It is associated with increased in-hospital mortality, a more pronounced reduction in left ventricular EF, and a higher incidence of congestive heart failure compared to infarctions in other cardiac regions.^[61] A recent study in Italy has suggested that a larger infarct size and increased cardiac enzymes contribute to this outcome.^[59]

Patients with a history of DM were found to have a greater risk of mortality in this study. DM is one of the most common comorbidities in patients with MI, affecting approximately 30% of patients according to one study.^[62] This result is consistent with a study reported by Burgess.^[63]

The Killip class, a widely used risk stratification tool for cardiac death mortality, has been found to have a relatively high hazard ratio, indicating an increased risk of adverse outcomes in patients with ACS.^[64] In addition, a greater EF of the left ventricle is associated with decreased mortality risk in ACS patients, as supported by previous literature. Notably, patients with EFs >35% demonstrate higher survival rates than those with EFs <35%, particularly those with EFs <25%.^[65] Furthermore, our study revealed that a history of CHF is accompanied by a higher rate of mortality.

The results of our study show that a higher admission SBP is associated with a lower mortality rate, which aligns with prior studies. For example, a study within the China Chest Pain Center system identified admission SBP as an independent protective factor against in-hospital MACE in STEMI patients undergoing primary PCI.^[66] In addition, the ACS-QUIK trial in India observed a U-shaped relationship, with SBP increases up to 159 mmHg correlating with lower 30-day major adverse cardiovascular events.^[67] These studies support our observation that elevated admission SBP is associated with improved short-term outcomes in STEMI patients. Despite our finding that higher admission SBP is associated with reduced 5-year mortality, two studies present nuanced results. In a large Chinese cohort of 7510 STEMI patients, those with

elevated SBP had more comorbidities but, after multivariate adjustment, showed no significant difference in 7- or 30-day mortality, MACE, or bleeding compared to normotensive patients, which does not conflict with our observations.^[68] Conversely, a single-center registry identified admission SBP ≥ 140 mmHg (and DBP < 60 mmHg) as a risk factor for poorer in-hospital outcomes, attributing this to impaired myocardial perfusion; however, this analysis lacked adjustment for confounders, limiting its comparability to our adjusted results.^[69] Higher admission SBP may confer protection through several mechanisms: It supports spontaneous coronary reperfusion, particularly in the 121–150 mm Hg range, leading to improved survival, preserves myocardial oxygen delivery and perfusion pressure during acute ischemia, thereby reducing early MACE, and reflects hemodynamic reserve that correlates with enhanced 30-day and 2-year survival.^[70,71]

Two major treatments and interventions following all types of MI are PCI and thrombolysis. Our study demonstrated that early thrombolysis after STEMI has a significant positive impact on patient survival. A case–control study in Ireland showed that early thrombolysis leads to higher rates of successful reperfusion, which are associated with reduced morbidity and mortality in STEMI patients.^[72] Other studies have shown that in adjusted models, there is no significant difference in post-MI mortality between patients who underwent PCI and those who received thrombolytic therapy.^[73,74] It should be noted that in this study, data on time-to-reperfusion variables, such as door-to-balloon time and door-to-needle time, which are known to influence the success of PCI and thrombolysis, were not available. Further studies are warranted to explore the impact of these time intervals on patient outcomes.

Furthermore, our results are in agreement with those of previous studies, indicating that anemia has an adverse effect on mortality in patients with MI.^[75,76]

Our study has several notable strengths, including its large sample size, prospective design, and long-term follow-up period. Furthermore, by multivariate analysis of twenty risk factors simultaneously, the effects of the risk factors were measured independently of each other. These strengths enhance the reliability and validity of our findings. However, several limitations of our study should be acknowledged. The retrospective cohort design introduces the possibility of selection bias. The generalizability of our study is limited to STEMI patients. Still, the large number of samples and the demographic and clinical diversity among participants allow the results of this study to be extrapolated to all STEMI patients. Moreover, the single-center design of this study, conducted in one city, may limit the generalizability of the findings.

Nevertheless, our study provides valuable insights into MI burden and highlights the importance of targeted interventions for improved outcomes.

CONCLUSIONS

Age, BMI, hypertension, SBP, the number of affected epicardial territories, EF, Hb levels, plasma glucose levels, and in-hospital complications are factors that influence long-term mortality in patients with STEMI. These findings can serve as a valuable guide for identifying STEMI patients at higher mortality risk. They can also play an effective role in determining secondary prevention strategies for diagnostic and therapeutic interventions, such as appropriate cardiac rehabilitation in the follow-up of patients with MI. It can assist health policymakers in developing appropriate secondary prevention strategies by ensuring that high-risk patients receive more rigorous follow-up, optimized pharmacological treatment, and closer monitoring, ultimately aiming to reduce their risk of mortality.

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Conflicts of interest

There are no conflicts of interest.

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