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Clinical and Anatomical Correlates of Out-of-Hospital Cardiac Arrest in Patients with ST-Elevation Myocardial Infarction

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Abstract

Background: Out-of-hospital cardiac arrest (OHCA) is a severe manifestation of ST-elevation myocardial infarction (STEMI). This study evaluates clinical and angiographic factors associated with OHCA in STEMI patients undergoing primary percutaneous coronary intervention (PCI). **Methods:** This retrospective study included consecutive STEMI patients undergoing primary PCI at Rambam Health Care Campus between 2010 and 2024. Clinical and angiographic characteristics were compared by pre-hospital OHCA status using multivariable logistic regression. **Results:** Among 2176 STEMI patients undergoing primary PCI, 135 (6.2%) experienced pre-hospital OHCA. OHCA patients were younger and had higher admission serum creatinine levels. Infarct-related artery distribution differed significantly ($p = 0.005$), with more frequent LMCA and LAD and less frequent RCA involvement among OHCA patients. Factors independently associated with OHCA were LMCA (OR 6.03, 95% CI 1.16–31.2), LAD (OR 1.81, 95% CI 1.16–2.8), and higher creatinine (OR 1.44, 95% CI 1.17–1.77). Increasing age (OR 0.97 per year, 95% CI 0.95–0.98) and smoking (OR 0.62, 95% CI 0.42–0.90) were inversely associated with OHCA, potentially reflecting survivor bias and residual confounding. Culprit-lesion location, initial TIMI flow, and number of diseased vessels (one, two, or three), did not differ significantly between groups. 30-day mortality was higher with OHCA (21.5% vs. 5.2%, $p < 0.001$; adjusted HR 6.00, 95% CI 3.86–9.35). **Conclusions:** Among STEMI patients undergoing primary PCI, resuscitated OHCA was associated with younger age, higher serum creatinine, and more frequent LMCA or LAD involvementm, as well as higher 30-day mortality. Findings may not be generalizable beyond patients who survived to coronary intervention.

Keywords: ventricular fibrillation; primary PCI; ST elevation MI; out-of-hospital cardiac arrest (OHCA)

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1. Introduction

Coronary artery disease (CAD) and myocardial infarction (MI) are believed to underlie 75% of deaths in patients with sudden cardiac death (SCD) [1]. In many cases of OHCA associated with acute myocardial infarction, the cardiac arrest is caused by ventricular fibrillation, occurring during the first hours after the onset of symptoms [2].

An acute ST-elevation myocardial infarction (STEMI) occurs due to occlusion of one or more coronary arteries, causing transmural myocardial ischemia, which in turn results in myocardial injury or necrosis [3].

Ventricular fibrillation (VF) complicates STEMI in approximately 10% of patients. More than half of the deaths associated with acute MI occur out of the hospital and within 1 h of symptom onset [4].

While a small minority of all patients with acute coronary syndrome (ACS) present as out-of-hospital cardiac arrest (OHCA), ACS is the most common cause of OHCA [5]. Overall, the majority of ventricular arrhythmias (VAs) occur early, within 24–48 h of symptom onset.

Despite advances in reperfusion therapies, mortality remains high in STEMI patients who experience OHCA, even when percutaneous coronary intervention (PCI) is performed successfully [6,7].

Factors associated with the occurrence of OHCA in setting of STEMI are still poorly understood. Early studies examining the risk factors for early VF or out-of-hospital VF in patients with acute MI derived inconsistent results. Gheeraert et al. showed that acute myocardial infarction due to occlusion in the left coronary system is associated with greater risk for out-of-hospital VF compared to the right coronary artery (RCA), while the location of occlusion within the left coronary system and extent of CAD are not related to out-of-hospital VF [8]. On the other hand, Velders et al. showed that risk for OHCA was highest for proximal left coronary lesions compared with non-proximal ones [9]. Jabbari et al. identified multiple risk factors for VF, including age younger than 60, anterior infarction, lack of flow in the artery, atrial fibrillation, alcohol consumption, and family history of sudden cardiac death [10]. This study aims to identify and systematically characterize specific clinical and angiographic characteristics associated with OHCA among STEMI patients undergoing primary PCI. As a secondary objective, we evaluated the association between OHCA and 30-day mortality to provide additional insight into the short-term prognosis of this patient population.

2. Materials and Methods

This analytical-observational retrospective cohort study included patients who presented to Rambam Health Care Campus between 1 January 2010 and 31 December 2024 with STEMI and were treated with primary percutaneous coronary intervention, with or without OHCA prior to hospital admission.

Acute STEMI on ECG was defined according to established guideline criteria as new ST-segment elevation at the J-point in at least two anatomically contiguous leads, with an elevation of ≥1 mm in all leads other than V2–V3. In leads V2–V3, the diagnostic thresholds were ≥2.5 mm in men younger than 40 years, ≥2 mm in men aged 40 years or older, and ≥1.5 mm in women, regardless of age.

OHCA was defined as cardiac arrest occuring before hospital arrival and requiring cardiopulmonary resuscitation, irrspective of the initial cardiac rhythm.

All data were retrieved from the electronic medical record system at Rambam Health Care Campus during that period. Clinical characterisitics included age, sex, cardiovascular risk factors (hypertension, diabetes, and smoking), atrial fibrillation, prior stroke, prior coronary artery disease, admission serum creatinine, and baseline beta-blocker use. Angiographic variables included location of the infarction, infarct-related artery (IRA), extent of coronary artery disease, TIMI flow grade pre-reperfusion therapy, chronic coronary occlusions, and proximal versus distal location of culprit lesions.

The culprit lesion was identified at the discretion of the PCI operator; when multiple lesions were present, the lesion corresponding to the territory of ST-segment elevation on the ECG was selected.

Serum creatinine levels were measured upon hospital admission in all patients. In patients presenting with OHCA, these measurements were obtained following cardiac

arrest and resuscitation.

Echocardiography: A transthoracic echocardiogram was performed by cardiac sonographers as part of the routine standard of care during the index admission post-PCI at a median of 2 days from admission (interquartile range 1 to 3 days). LV wall motion score index (WMSI) was calculated according to a 16-segment model according to the American Society of Echocardiography (ASE) guidelines [11]. Individual segments were scored as follows: (1) normal or hyperkinetic, (2) hypokinetic (reduced thickening), (3) akinetic (absent or negligible thickening), and (4) dyskinetic (systolic thinning or stretching). WMSI was calculated by adding the scores of individual segments and dividing this total by the number of segments assessed.

2.1. Statistics

Continuous variables were tested for normality using the Shapiro–Wilk test. Non-normally distributed data were transformed or analyzed using non-parametric methods as appropriate. Categorical variables were analyzed using a chi-square test or two-tailed Fisher's exact test. For continuous variables, *t*-tests or ANOVA were employed, depending on the data distribution. Multivariable logistic regression analysis was conducted to identify factors independently associated with OHCA. The model included age (per year), smoking status, admission serum creatinine (per 1 mg/dL increase), and infarct-related artery (LMCA, LAD, LCx, and side branches, with RCA as the reference category).

As a secondary outcome, 30-day all-cause mortality was compared between patients with and without OHCA. Kaplan–Meier plots were used to assess event-free probability of 30-day mortality associated with OHCA, and the log-rank test was used to compare the curves. 30-day all-cause mortality was analyzed using Cox proportional hazards regression. The unadjusted model included OHCA status as the sole explanatory variable. The multivariable model included OHCA status and covariates selected from age, sex, body mass index, previous stroke, previous myocardial infarction, hypertension, diabetes mellitus, smoking, atrial fibrillation, chronic lung disease, and infarct-related artery. The variables retained in the final model and the stepwise selection criteria were specified according to the original statistical analysis.

A *p*-value of less than 0.05 was considered statistically significant for all analyses. All statistical analyses were performed using STATA software version 18.

2.2. Ethical Aspects

The Helsinki Committee at Rambam Health Care Campus approved the retrospective data analysis of STEMI patients, and the retrospective data acquisition followed approval on 5 March 2025, approval code: RMB-D-0471-24. No informed consent forms were required as the study was conducted as a retrospective data analysis of patients without the use of any identifying details.

3. Results

The study cohort included 2176 patients who presented with ST-elevation myocardial infarction between January 2010 and December 2024, and were treated with primary percutaneous coronary intervention. Of these, 135 patients (6.2%) experienced out-of-hospital cardiopulmonary resuscitation prior to hospital admission. The clinical characteristics of patients with and without OHCA are presented in Table 1. Patients with OHCA were younger and with worse renal function. The prevalence of prior coronary artery disease did not differ significantly between patients with and without OHCA (14.1% vs. 14.7%, $p = 0.842$). Similarly, beta-blocker use and other cardiovascular risk factors were broadly comparable between groups (Table 1).

Table 1. Baseline Clinical Characteristics.

Baseline Characteristics	No OHCA $n = 2041$	OHCA $n = 135$	p-Value
Age (years)	61 ± 12	58 ± 11	0.001
Male gender	1657 (81)	109 (81)	0.90
Atrial fibrillation	94 (5)	4 (3)	0.37
Smoking	1227 (60)	71 (53)	0.08
Prior stroke	37 (2)	5 (4)	0.18
Prior coronary artery disease	300 (15)	19 (14)	0.84
Hypertension	1011 (50)	57 (42)	0.10
Diabetes mellitus	562 (28)	32 (24)	0.33
Serum creatinine (mg/dL)	1.0 ± 0.6	1.2 ± 0.5	0.001
Beta blockers	1233 (60)	81 (60)	0.93

Data are numbers (%) or mean ± SD. OHCA, out-of-hospital cardiac arrest.

Echocardiographic comparison between groups demonstrated that patients with OHCA had a higher wall motion score index (WMSI) compared with those without OHCA (mean WMSI 1.72 ± 0.4 vs. 1.58 ± 0.39; $p = 0.009$) (Figure 1). Furthermore, for each individual myocardial segment, the score was higher in the OHCA group (Figure 2).

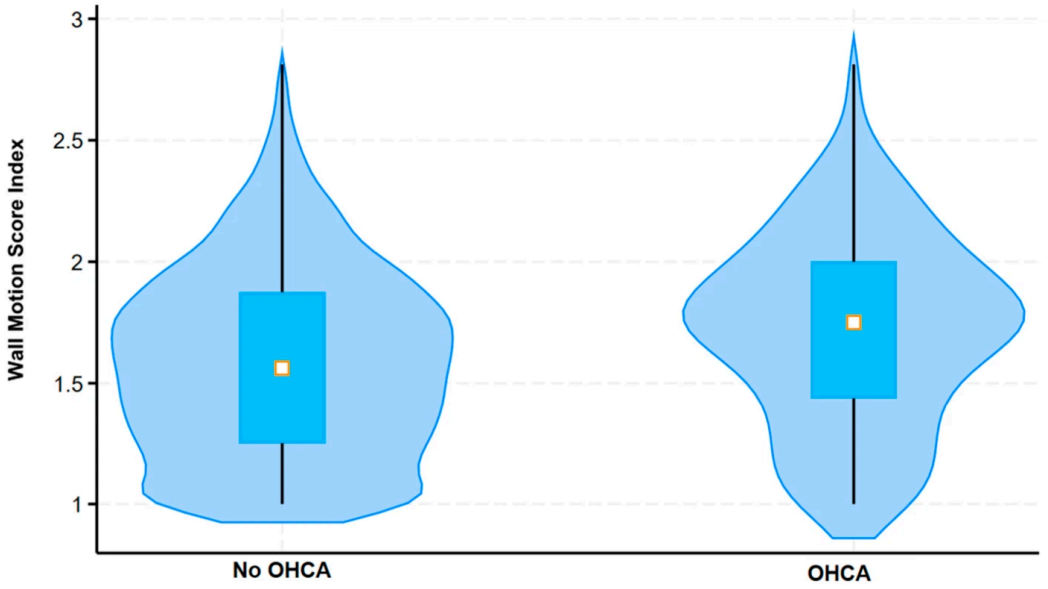


Figure 1. Box and violin plot comparison of wall motion score index.

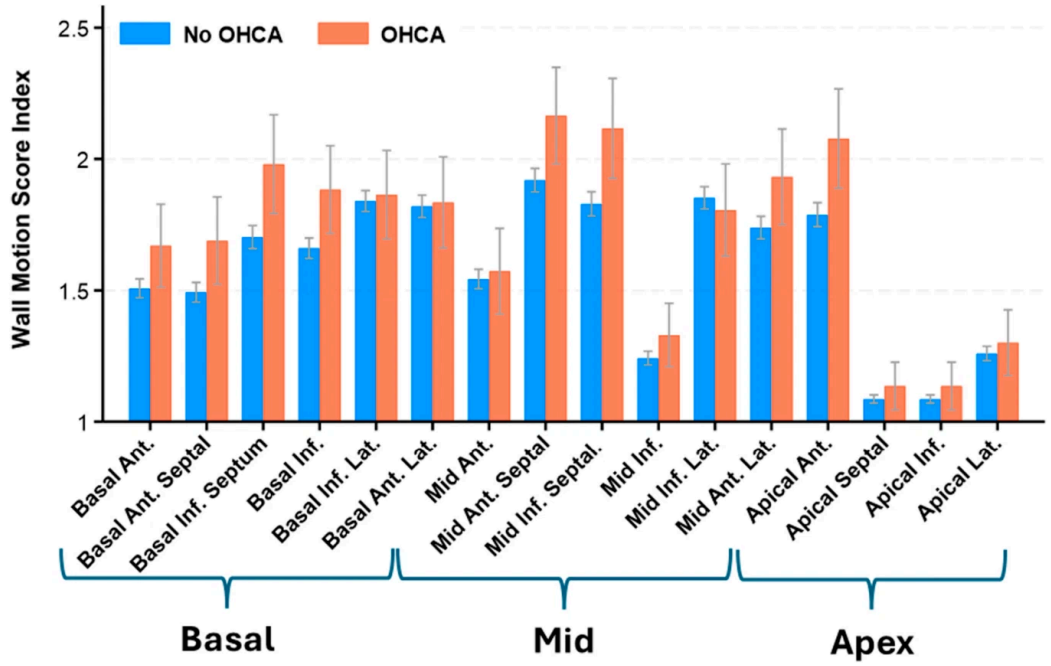


Figure 2. Wall motion score index of 16 myocardial segments in patients with and without OHCA.

Angiographic findings showed significant differences in IRA distribution ($p = 0.005$): left main coronary artery (LMCA) (2.2% vs. 0.5%) and left anterior descending artery (LAD) lesions (52.6% vs. 43.1%) were more frequent among OHCA patients, while right coronary artery (RCA) involvement was less common (23% vs. 35.3%). Lesion location (proximal/ostial vs. Mid/Distal), initial TIMI flow, and extent of coronary artery disease did not differ significantly (Table 2).

Table 2. Angiographic Data.

	No OHCA ***n* = 2041**	**OHCA** ***n* = 135**	***p*-Value**
IRA			0.005
LMCA	10 (0.5%)	3 (2.2%)	
LAD	880 (43.1%)	71 (52.6%)	
LCx	195 (9.6%)	15 (11.1%)	
RCA	721 (35.3%)	31 (23%)	
Side branch	235 (11.5%)	15 (11.1%)	
Location			
Ostial/proximal	1019 (49.9%)	77 (57.0%)	0.11
TIMI Flow			
TIMI 0	1277 (62.6%)	91 (67.4%)	0.26
Number of diseased vessels			0.14
One vessel	958 (46.9%)	64 (47.4%)	
Two vessels	648 (31.7%)	35 (25.9%)	
Three vessels	412 (20.2%)	36 (26.7%)	

Data are numbers (%). IRA, infarct-related artery; LAD, left anterior descending; LCX, left circumflex; LMCA, left main coronary artery; OHCA, out-of-hospital cardiac arrest; RCA, right coronary artery; TIMI, thrombolysis in myocardial infarction. Data on the number of diseased vessels were missing for 23 patients (1.1%) in the non-OHCA group; percentages reflect total group size.

Multivariable logistic regression identified independent associations of OHCA that included LMCA (OR 6.03, 95% CI 1.16–31.2, $p = 0.032$), LAD (OR 1.81, 95% CI 1.16–2.8, $p = 0.008$), and higher creatinine (OR 1.44, 95% CI 1.17–1.77, $p = 0.001$). In contrast, older age (OR 0.97 per year, 95% CI 0.95–0.98, $p = 0.008$) and smoking (OR 0.62, 95% CI 0.42–0.9, $p = 0.011$) were associated with lower odds of OHCA (Table 3).

Table 3. Multivariate logistic regression model.

Parameter	Odds Ratio	95% CI	*p* -Value
IRA			
RCA	1.0	-	-
LCx	1.82	0.96–3.46	0.07
Side branch	1.47	0.78–2.80	0.24
LMCA	6.03	1.16–31.20	0.032
LAD	1.81	1.16–2.80	0.008
Clinical characteristics			
Age (per 1 year increase)	0.97	0.95–0.98	0.008
Smoking	0.62	0.42–0.90	0.011
Creatinine	1.44	1.17–1.77	0.001

IRA, infarct-related artery; LAD, left anterior descending; LCX, left circumflex; LMCA, left main coronary artery; RCA, right coronary artery.

Thirty-day mortality was higher in OHCA patients compared to patients without OHCA (21.5% vs. 5.2%, $p < 0.001$). Figure 3 displays the Kaplan–Meier plots of the 2 study groups. The unadjusted hazard ratio (HR) for 30-day mortality in the OHCA group vs. no OHCA was 4.39 (95% CI 2.9 to 6.62, $p < 0.0001$). After adjustments for other risk factors for mortality, the adjusted hazard ratio was 6.00 (95% CI 3.86 to 9.35, $p < 0.0001$).

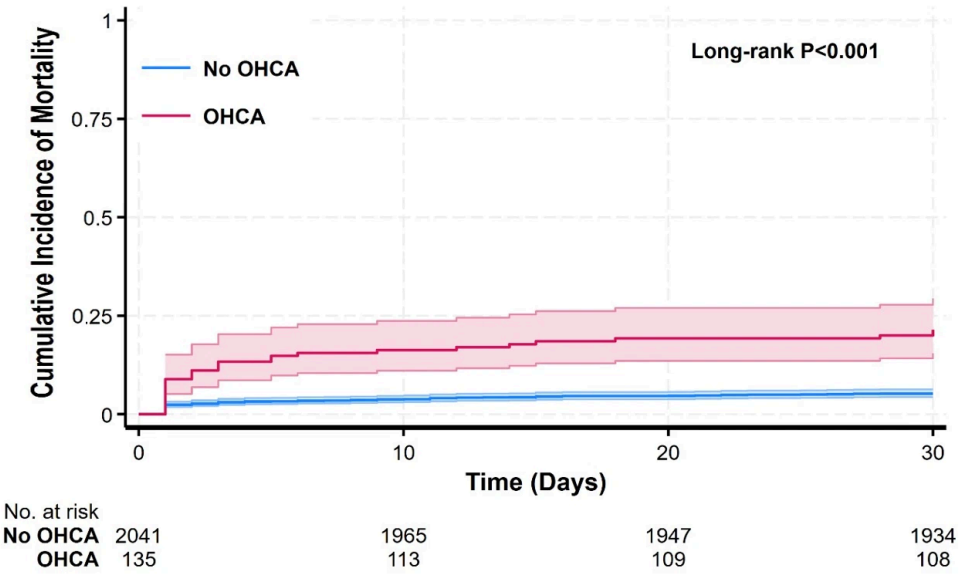


Figure 3. Kaplan–Meier plot of 30-day mortality.

4. Discussion

Our results suggest that in STEMI patients treated with primary PCI, specific clinical and angiographic features are associated with presenting with OHCA. These find-

ings directly address the knowledge gaps highlighted in the introduction, where early studies by Gheeraert et al. [8], Velders et al. [9], and Jabbari et al. [10] reported conflicting results regarding culprit vessel location (left vs. right coronary system, proximal vs. distal lesions) and clinical predictors of out-of-hospital VF/OHCA in acute MI. While those studies suggested varying associations with left coronary involvement, younger age, and impaired flow, our analysis provides a unified perspective from a large contemporary PCI-treated cohort, confirming left main/LAD predominance and younger age as independent predictors while clarifying the lack of association with lesion location, TIMI flow, or multivessel disease.

An important consideration in interpreting our findings is the selection of the study population. Our cohort included only patients who survived long enough to undergo primary PCI and therefore does not represent the entire population of patients experiencing STEMI. In particular, patients who died before hospital admission or coronary angiography were not included. Consequently, the observed associations may be influenced by survivor and selection bias and should not be interpreted as risk factors for OHCA in the overall STEMI population. Rather, our findings characterize differences between patients with and without OHCA within a selected population undergoing primary PCI.

The overall incidence of OHCA in the cohort was 6.2%, indicating that a substantial minority of STEMI patients arrive after resuscitated cardiac arrest. This group was younger, had worse renal function, and showed a different distribution of infarct-related artery compared with patients without OHCA, highlighting a distinct clinical and angiographic profile.

Patients with OHCA were significantly younger. Previous studies have also demonstrated that younger age is a risk of OHCA. Younger individuals are more susceptible to sudden cardiac arrest during acute myocardial infarction, presumably due to a relative lack of ischemic preconditioning and collateral coronary circulation [12]. Notably, the prevalence of prior coronary artery disease did not differ significantly between the groups (14.1% vs. 14.7%, $p = 0.842$), and therefore does not provide additional support for this proposed mechanism. Elevated serum creatinine was also independently associated with OHCA, possibly reflecting worse hemodynamic status and acute physiological changes associated with cardiac arrest and resuscitation. On the other had, impaired renal clearance of cardiotoxic metabolites, or microvascular dysfunction heightens the vulnerability to malignant arrhythmias and poor tolerance to acute ischemia. In contrast, most traditional cardiovascular risk factors, including hypertension, diabetes, prior stroke, and atrial fibrillation, did not differ significantly between the groups. The angiographic findings further support the concept that greater ischemic burden and territory at risk play a central role in the pathogenesis of OHCA. Lesions in the LMCA and LAD were more frequent among OHCA patients, whereas RCA involvement was less frequent in this group. Since LMCA and LAD supply large myocardial territories including the anterior wall and, in the case of LMCA, a substantial portion of the left ventricle, their acute occlusion is expected to cause more extensive ischemia and a higher likelihood of rapid hemodynamic collapse and ventricular fibrillation. Nevertheless, the association between LMCA involvement and OHCA should be interpreted with caution, as only three patients in the OHCA group had LMCA involvement. The small number of observations raises concerns regarding sparse-data bias and the stability of the estimated association, as reflected by the wide confidence interval. Therefore, although the observed association is clinically plausible, its magnitude remains uncertain and requires confirmation in larger cohorts.

The higher WMSI observed in patients with OHCA indicates more extensive my-

ocardial dysfunction following PCI. However, because echocardiography was performed after cardiac arrest and resuscitation, these differences may reflect both ischemic myocardial injury and post-resuscitation myocardial dysfunction. Therefore, WMSI cannot reliably distinguish the extent of the initial ischemic insult from myocardial dysfunction associated with cardiac arrest itself.

Other mechanisms can explain the higher rates of OHCA in anterior STEMI. The vagally mediated Bezold-Jarisch reflex is commonly associated with inferior ischemia than anterior ischemia [13,14], and can afford protection from VF in the setting of myocardial ischemia. Receptors with vagal afferents that mediate cardioinhibitory and vasodepressor responses are preferentially located in the inferoposterior wall, and thus, the Bezold-Jarish reflex is seen most commonly in right coronary artery followed by circumflex myocardial infarctions [15].

Interestingly, smoking was inversely associated with OHCA (OR 0.62, 95% CI 0.42–0.9, $p = 0.011$). Although counterintuitive, this finding aligns with the well-described "smoker's paradox" in acute myocardial infarction, where smokers appear to have more favorable short-term outcomes. Paradossi et al. explained this phenomenon by noting that smokers typically present with their first cardiovascular event at a younger age and with fewer atherosclerotic risk factors and comorbidities compared to nonsmokers [16]. Therefore, this result likely reflects residual confounding by younger age and lower comorbidity burden rather than a true protective effect of smoking, and it should be interpreted with caution.

Thirty-day mortality was substantially higher among patients with OHCA compared with those without OHCA (21.5% vs. 5.2%), and this association persisted after multivariable adjustment. This finding highlights the adverse short-term prognosis associated with resuscitated OHCA among STEMI patients undergoing primary PCI.

Taken together, our findings demonstrate that certain clinical and angiographic characteristics are associated with resuscitated OHCA among STEMI patients undergoing primary PCI. In particular, LMCA and LAD involvement were more frequent among patients presenting with OHCA, suggesting a possible association between the territory of myocardial ischemia and cardiac arrest. These findings may have implications for prehospital triage, resource allocation, and early monitoring strategies. However, these findings should be interpreted within the context of the selected study population and potential survivor bias. Further studies involving broader STEMI populations are warranted to validate these associations and determine their potential clinical implications.

Study Limitations

Several limitations should be acknowledged. First, this was a retrospective, single-center observational study, with the inherent potential for residual confounding and incomplete data collection. Most importantly, the study population was restricted to STEMI patients who survived to undergo primary PCI. Patients who died before hospital admission or coronary intervention, as well as those who did not undergo coronary angiography, were not represented. This introduces potential survivor and selection bias, including collider bias, which may influence the observed associations between clinical characteristics, coronary anatomy, and OHCA.

Second, the retrospective design does not allow us to establish causal relationships between the identified variables and the occurrence of cardiac arrest. Certain measurements, including serum creatinine and echocardiographic indices, may have also been influenced by the cardiac arrest itself, limiting their interpretation as pre-existing risk factors.

Third, the relatively small number of patients with LMCA involvement limits the

precision of the corresponding risk estimates. In addition, coronary disease extent was assessed by the number of diseased vessels rather than a comprehensive anatomical scoring system.

Fourth, cardiogenic shock, Killip class, and left ventricular ejection fraction were not included in the adjusted mortality model, and residual prognostic confounding related to these factors cannot be excluded. Importantly, measurements obtained following cardiac arrest may also reflect post-resuscitation physiological changes.

Finally, because the analysis was restricted to patients undergoing primary PCI, our findings cannot establish the incidence of OHCA or identify predictors of its occurrence in the overall STEMI population. Larger, prospective population-based studies incorporating both survivors and non-survivors of pre-hospital cardiac arrest are needed to evaluate the generalizability of these observations.

5. Conclusions

Among STEMI patients who survived to undergo primary PCI, OHCA was associated with younger age, higher serum creatinine levels, and more frequent involvement of the LMCA or LAD as the infarct-related artery. These findings characterize clinical and angiographic differences within a selected population of STEMI patients undergoing coronary intervention. Given the potential for survivor and selection bias, these associations should not be interpreted as causal determinants or generalizable predictors of OHCA in the overall STEMI population. Further studies incorporating broader, population-based cohorts are warranted to validate these observations.

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Informed Consent Statement: No informed consent forms were required as the study was conducted as a retrospective data analysis of patients without the use of any identifying details.

Data Availability Statement: The raw data supporting the conclusions of this article will be made available by the authors on request.

Conflicts of Interest: The authors declare no conflicts of interest.

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