ARAŞTIRMA MAKALESİ / *RESEARCH ARTICLE*

The Relationship Between Primary Ventricular Arrhythmias and In-hospital Mortality and Resuscitated Cardiopulmonary Arrest in Patients with Acute ST Segment Elevation Myocardial Infarction

ST Segment Yükselmeli Miyokard Enfarktüsü ile Gelen Hastalarda Gelişen Primer Ventriküler Aritmilerin Hastane İçi Ölüm ve Resüsitasyon Yapılmış Kardiyopulmoner Arrest ile İlişkisi

Evliya Akdeniz[1], Baris Simsek[1], Veysel Ozan Tanik[2], Mehmet Saygi[1], Levent Pay[1], Kemal Emrecan Parsova[1], Furkan Durak[1], Ahmet Cagdas Yumurtas[1], Osman Uzman[3], Duygu Inan[1], Duygu Genc[1], Can Yucel Karabay[1].

[1]Department of Cardiology, Dr. Siyami Ersek Thoracic and Cardiovascular Surgery Training and Research Hospital, İstanbul; [2]Department of Cardiology, Dışkapı Yıldırım Beyazıt Training and Research Hospital, Ankara; [3]Department of Cardiology, Şehit Prof. Dr. İlhan Varank Sancaktepe Training and Research Hospital, İstanbul, Türkiye

ABSTRACT

Aim: *Arrhythmic complications in patients with acute coronary syndromes during hospitalization are not uncommon in cardiology practice. In our retrospective study, we investigated the effect of primary ventricular arrhythmias occurring within the first 48 hours from the time of hospitalization on cardiovascular mortality and resuscitated cardiopulmonary arrest in patients with ST-segment elevation myocardial infarction.*

Introduction: *Although the rate of myocardial infarction has decreased over the years, it is responsible for hundreds of thousands of deaths worldwide. Arrhythmic complications take an essential place for morbidity and mortality in the course of myocardial infarction. Ventricular arrhythmias play a pivotal role in terms of mortality in acute terms of myocardial infarction.*

Material and Method: *In our single center and retrospective study, 18-year-old and older patients who presented to Sağlık Bilimleri University Dr. Siyami Ersek Thoracic and Cardiovascular Surgery Training and Research Hospital emergency department with ischemic symptoms and were admitted to hospital with the diagnose of ST-segment elevation myocardial infarction were enrolled. The primary endpoint was in-hospital mortality and resuscitated cardiopulmonary arrest.*

Results: *1,137 patients were included in the study. Primary VT/VF which occurred within the first 48 hours from the time of hospitalization was observed in 8.2% of patients (n: 93). Previous MI history (3.47 OR, 95% CI 1.41–8.55 p=0.0068, LASSO: 0.388), KILLIP class at the time of admission (1.52 OR, 95% CI 0.94–2.45 p=0.0813, LASSO: 0.627), Primary VT/VF (4.02 OR, 95% CI 1.44–11.21 p=0.0077, LASSO: 0.440), left ventricle ejection fraction calculated by transthoracic echocardiography (0.88 OR, 95% CI 0.78–0.99 p=0.0444, LASSO: -0.032) and serum CRP level (1.81 OR, 95% CI 0.22–14.53 p=0.57, LASSO: 0.036) were independent predictors of MACE including in-hospital mortality and resuscitated cardiopulmonary arrest.*

Conclusion: *Primary VT and VF are independent predictors of in-hospital mortality and resuscitated cardiopulmonary arrest events in patients with ST-segment elevation acute MI.*

Key words: *myocardial infarction, STEMI, ventricular arrhythmias, in-hospital mortality*

ÖZET

Amaç: *Akut koroner sendrom hastalarının hastanede yatış sürecinde gelişen aritmik komplikasyonlar kardiyoloji pratiğinde nadir karşılaşılan durumlar değildir. Retrospektif çalışmamızda ST yükselmeli miyokard infarktüsü ile gelen hastaların hastaneye yatışı sonrası ilk 48 saatte gelişen primer ventriküler aritmilerin kardiyovasküler mortalite ve resussite kardiyopulmoner arrest üzerine etkilerini araştırdık.*

Materyal ve Metot: *18 yaş ve üzeri iskemik semptomlarla Sağlık Bilimleri Üniversitesi Dr. Siyami Ersek Göğüs Kalp ve Damar Cerrahisi Eğitim ve Araştırma Hastanesi'ne başvuran ve ST yükselmeli miyokard enfarktüsü tanısı ile hastaneye yatışı yapılan hastalar tek merkezli ve retrospektif çalışmamıza alındı. Birincil sonlanım noktası hastane içi ölüm ve resussitasyon yapılmış kardiyopulmoner arrest olarak belirlendi.*

Bulgular: *Çalışmamıza 1.137 hasta dâhil edildi. Hastaneye yatış sonrası ilk 48 saatte gelişen Primer VT/VF oranı %8,2 (93 hasta) idi. Miyokard enfarktüsü öyküsü (3,47 RR, %95 GA 1,41–8,55 p=0,0068, LASSO: 0,388), başvuru esnasındaki KILLIP sınıflaması (1,52 RR, %95 GA 0,94–2,45 p=0,0813, LASSO: 0,627), Primer VT/VF (4,02 RR, %95 GA 1,44–11,21 p=0,0077, LASSO: 0,440), transtorasik ekokardiyografide hesaplanan sol ventrikül ejeksiyon fraksiyonu (0,88 RR, %95 GA 0,78–0,99 p=0,0444, LASSO: -0,032) ve serum CRP düzeyi (1,81 RR, %95 GA 0,22–14,53 p=0,57, LASSO: 0,036) hastane içi ölüm ve resussitasyon yapılmış kardiyopulmoner arrestti de içeren majör kardiyovasküler olumsuz olayların bağımsız öngördürücüleri olarak belirlendi.*

Sonuç: *ST yükselmeli miyokard enfarktüsü hastalarında Primer VT/VF, hastane içi ölüm ve resussitasyon yapılmış kardiyovasküler arrest olaylarının bağımsız öngördürücüleridir.*

Anahtar kelimeler: *miyokard enfarktüsü, STYME, ventriküler aritmiler, hastane içi ölüm*

İletişim/Contact: *Evliya Akdeniz, Dr. Siyami Ersek Thoracic and Cardiovascular Surgery Training and Research Hospital, Department of Cardiology, İstanbul, Türkiye* • ***Tel:*** *0538 749 55 35* • ***E-mail:*** *evliyakdeniz@gmail.com* • ***Geliş/Received:*** *21.01.2024* • ***Kabul/Accepted:*** *08.06.2024*

ORCID: *Evliya Akdeniz: 0000-0002-4688-7992 • Barış Şimşek: 0000-0001-9412-0035 • Veysel Ozan Tanık: 0000-0002-7193-4324 • Mehmet Saygı: 0000-0002-7258-2797 • Levent Pay: 0000-0002-7491-8119 • Kemal Emrecan Parsova: 0000-0002-2436-0241 • Furkan Durak: 0000-0001-8211-8253 • Ahmet Çağdaş Yumurtaş: 0000-0002-7311-2826 • Osman Uzman: 0000-0002-7386-2432 • Duygu İnan: 0000-0003-3921-6469 • Duygu Genç: 0000-0001-7430-8276 • Can Yücel Karabay: 0000-0002-9653-9048*

Introduction

Although myocardial infarction (MI) and its mortality rates showed a trend of decline in the last decades, myocardial infarction with ST-segment elevation (STEMI) has similar mortality rates when compared with rates of the early 2000 s[1]. Similarly, although the rates of life-threatening ventricular arrhythmias (VAs) seem to decline over time, ventricular tachycardia (VT) and ventricular fibrillation (VF) can complicate the course of MI with relatively high rates of up to 5–7.5%[2–5].

Whether VAs adversely impact both short- and long-term outcomes in patients suffering from MI has long been investigated[3,5–8]. Data from the literature reveals that the vast majority of VAs tend to occur within the first 48 hours of the index event[5,8,9]. As understood from the study results, the prognostic significance of VAs developing in the early stage of MI on in-hospital, short- and long-term survival is still unclear[3,7,8,10,11].

This study aims to investigate whether primary VF and VT in the setting of STEMI impact in-hospital mortality in patients undergoing primary percutaneous coronary intervention (pPCI).

Material and Method

Study Population

Our study was designed as a retrospective and single-center study. Between January 2017 and July 2018, 1,137 patients admitted to tertiary centers with acute onset chest pain accompanied by ≥1 mm persistent ST-segment elevation at least two consecutive leads on surface electrocardiogram (ECG) were included in the study. Patients with structural heart disease or channelopathies, patients with a history of VT/VF and implantable cardioverter defibrillator (ICD) implantation, patients with previously known left ventricular ejection fraction (LVEF) 30% or less and patients under 18 years of age were excluded from the study population. The local ethical committee of the hospital approved the study protocol.

Data Ascertainment

Peripheral venous blood samples were obtained on admission for complete blood count and biochemical parameters. Hematologic parameters were studied with Cell-Dyn 37000 Hematology Analyzer (Abbott Diagnostic Division, Wiesbaden, Germany) and biochemical parameters such as serum creatinine and C-reactive protein (CRP) levels were studied with Architect Plusci 4100 (Abbott Laboratories, Abbott Park, Illinois).

Clinical data, laboratory parameters, electrocardiography and transthoracic echocardiographic measurements were retrospectively obtained from the hospital information system.

Definitions

Ventricular tachycardia was defined as a tachycardia (rate >100 beats per minute) with three or more consecutive beats originating from ventricles independent of atrial or atrioventricular nodal conduction. If the QRS complexes were similar in the configuration from beat to beat, they were classified as monomorphic; if the QRS configurations changed from beat to beat, they were classified as polymorphic VT[12].

Ventricular fibrillation was defined as a chaotic rhythm on the surface ECG characterized by irregular undulations in both timing and morphology without discrete QRS complexes[12].

Hypertension (HTN) was defined as the use of antihypertensive medication or systolic blood pressure (SBP) values ≥140 mmHg and/or diastolic blood pressure (DBP) values ≥90 mmHg[13].

Primary VT/VF was defined as VT/VF occurring within the first 48 hours after admission to the hospital.

Diabetes mellitus (DM) was defined as random plasma glucose ≥200 mg/dl with symptoms or glycated hemoglobin level ≥6.5% or using antidiabetic medications[14].

KILLIP classification at admission assessed the presence and severity of heart failure signs and symptoms[15].

Statistical Analysis

Mean and standard deviation, median and interquartile range were used to present numerical parameters, and the percentage and number of patients were used to represent categorical parameters.

Endpoint

The primary endpoint was in-hospital major adverse cardiovascular events (MACE), which was a composite of in-hospital mortality and aborted cardiopulmonary arrest (aCPA).

Candidate Predictors

Predictors were identified a priori and informed by clinical expertise and literature review. The candidate predictors must be moderately/strongly associated with in-hospital MACE. We pre-specified candidate predictors according to these principles. As candidate predictors, age, gender, HTN, DM, previous MI, smoking history, KILLIP classification at the time of admission, SBP, heart rate, VT and VF, in-hospital atrial fibrillation (AF), in-hospital supraventricular tachycardia (SVT), atrioventricular block (AVB), MI related artery (LAD versus non-LAD), LVEF, CRP, hemoglobin level, serum creatinine and potassium level have been determined. Our model did not include Variables with very low or very high frequencies. Finally, we included 19 candidate predictors in our study model.

Sample Size Calculation

A clinical prediction model requires a sufficiently large sample size and a sufficiently conservative number of predators. Specifically, at least ten patients should have the primary endpoint for each candidate predictor (endpoint/variable >10). In our clinical model, 119 patients had the primary outcome. However, 19 candidate predictors were identified (119/19=6.3). Therefore, conventional logistic regression models and penalized maximum likelihood estimation methods were used to minimize overfitting.

Statistical Modeling

We used the minor absolute shrinkage and selection operator (LASSO) along with the 10-fold cross-validation penalty parameter to avoid the risk of overfitting. Numeric variables such as age, KILLIP classification, SBP, pulse rate, LVEF, hemoglobin, serum CRP, creatinine and potassium level were included in our clinical model as flexible smooth parameters using restricted cubic spline. LASSO is a proposed shrinkage regression technique for regression analysis of models with a low incidence of candidate predictors. LASSO coefficients were feasible for prediction but not easily understandable and were presented with multivariable logistic regression models with reported results as odds ratio (OR) and 95% confidence interval (CI).

The Propensity Score (PS) was calculated using the multivariable logistic regression model with the dependent result (with VT/VF versus without). We used the spline function of the logit propensity score to evaluate the relationship between the groups (VT / VF with / without) and the primary outcome. Furthermore, the inverse probability weighting (IPW) method was used to adjust the differences between the two groups. All statistical analyses were performed by R-software v. 3.5.1 (R statistical software, Institute for Statistics and Mathematics, Vienna, Austria).

Results

One thousand one hundred thirty-seven patients admitted to the emergency department with chest pain accompanied by ST-segment elevation on surface ECG were included in the study. The mean age of the study population was 59.8±12.9 years, and male patients were 78.6% of the study population. Primary VT/VF was observed in 8.2% of the patients (n=93). During the hospital stay, the rate of new-onset AF was 7.3% (n=83), and at least second-degree AVB emerged at 3.1% (n=35). The baseline clinical characteristics of patients with and without VT/VF are summarized in Table 1.

The in-hospital mortality rate was 8.5% (n=96), and the rate of in-hospital MACE was 10.5% (n=119) in the whole study population. Additionally, the adverse clinical endpoints, cerebrovascular accident and stent thrombosis, were 1.1% (n=12) and 1.5% (n=17), respectively (Table 2).

KILLIP classification at the time of admission, prior MI, age, sex, DM and smoking history, SBP, pulse rate, VT/VF, in-hospital AF and non-AF SVT (atrial flutter, atrial tachycardia and other narrow QRS complex tachycardias), AVB, LVEF, serum CRP, hemoglobin and potassium level were associated with in-hospital mortality and aCPA in univariable analysis (Table 3). In univariate analysis, there might appear to be a positive relationship between female gender and MACE rates. Still, in multivariate analysis, a statistically significant relationship between gender and MACE couldn't have been established.

In multivariable regression analysis, the LASSO method was used to avoid the risk of overfitting. Accordingly, **MI history** (3.47 OR, 95% CI 1.41–8.55 p=0.007, LASSO regression coefficient: 0.388), **KILLIP classification** (1.52 OR, 95% CI 0.94–2.45 p=0.081, LASSO regression coefficient: 0.627), **primary VT/VF** (4.02 OR, 95% CI 1.44–11.2 p=0.008, LASSO regression coefficient: 0.440), **LVEF** (0.88 OR, 95% CI 0.78–0.99 p=0.0444, LASSO regression coefficient: -0, 032)

Table 1. *Baseline characteristics*

VARIABLE	OVERALL (n=1137)	VT/VF PRESENT (n=93)	VT/VF ABSENT (n=1044)
AGE (years)	59.8±12.9	60.1±14.1	59.7±12.7
MALE GENDER	%78.6	%76.1	%78.9
HYPERTENSION	%47.2	%40.9	%47.8
DIABETES MELLITUS			
INSULIN-DEPENDENT	11%, 7	9%, 7	11%, 8
OAD	15%, 7	14%	15%, 8
HISTORY of MI	%19.5	%23.7	%19.1
SMOKING HISTORY			
SMOKERS	53%, 1	65%, 1	52%
NON-SMOKERS	46%, 9	34%, 9	48%
KILLIP CLASSIFICATION			
1	89%	64%, 5	91%, 4
2	1%, 4	2%, 2	1%, 4
3	3%, 5	5%, 4	3%, 3
4	6%	28%	4%, 0
SYSTOLIC BLOOD PRESSURE (mmHg)	135.3±30.8	123.9±37.3	136.5±29.8
PULSE RATE (beat per minute)	83.1±23.1	90.1±26.1	82.5±20.6
IN-HOSPITAL AF	%7.3	%12.9	%6.8
IN-HOSPITAL non-AF SVT	%0.4	%2.2	%0.3
AVB	%3.1	%7.5	%2.7
INFARCT RELATED ARTERY			
LAD	39%, 9	58%, 4	38%, 3
NON-LAD	60%, 1	41%, 6	61%, 7
EJECTION FRACTION (%)	45.4±11.3	40.3±12.2	45.9±11.1
CRP (mg/dL)	2.7±4.2	3.4±5.1	2.7±4.1
HEMOGLOBIN (g/dL)	13.6±2.0	13.7±2.3	13.6±1.9
SERUM CREATININE (mg/dl)	1.08±2.5	1.25±1.15	1.06±2.6
SERUM POTASSIUM (mEq/L)	4.15±0.56	4.16±0.65	4.15±0.55

AF: atrial fibrillation, AVB: atrioventricular block, CPA: cardiopulmonary arrest, CRP: C-reactive protein, g/dL: grams per deciliter, LAD: left anterior descending, mEq/L: milliequivalents per liter, mg/dL: milligrams per deciliter, MI: myocardial infarction, mmHg: millimeter of mercury, OAD: oral antidiabetic, SVT: supraventricular tachycardia, VF: ventricular fibrillation, VT: ventricular tachycardia.

Table 2. *In-hospital adverse clinic endpoint rates in the overall group, patients with and without VT/VF*

VARIABLE	OVERALL (n=1137)	VT/VF PRESENT (n=93)	VT/VF ABSENT (n=1044)
IN-HOSPITAL MORTALITY	%8.5	%32.3	%6.3
IN-HOSPITAL CVA	%1.1	%1.1	%1.1
IN-HOSPITAL STENT THROMBOSIS	%1.5	%6.5	%1.1
IN-HOSPITAL MACE	%10.5	%38.7	%7.9

CPA: cardiopulmonary arrest, CVA: cerebrovascular accident, MACE: major adverse cardiovascular event, VF: ventricular fibrillation, VT: ventricular tachycardia.

and **serum CRP level** (1.81 OR, 95% CI 0.22–14.53 p=0.57, LASSO regression coefficient: 0, 036) were determined as independent predictors of in-hospital mortality and/or aCPA (Table-3). The relationship between independent predictors and in-hospital mortality/aCPA log odds is indicated in Fig. 1.

As depicted in Fig. 1a and Fig. 1b, having a history of MI and developing VT/VF within the first 48 hours are significantly associated with the primary outcome of in-hospital aCPA and in-hospital mortality. As graphically illustrated in Fig. 1c, an increase in KILLIP class at admission significantly affects the primary outcome. Fig. 1d shows the relationship between LVEF and in-hospital mortality/aCPA. Left ventricular ejection fraction is a parameter that considerably affects the primary outcome statistically, indicating that a decrease below 40% in LVEF could lead to poor outcomes. Finally, Fig. 1e demonstrates a significant relationship between CRP and MACE rates.

Besides, the relation between VT/VF and in-hospital mortality/aCPA remained statistically significant when the propensity score-adjusted multivariable model (**2.76 OR, 95% CI 1.17–6.35 p=0, 0168**) and inverse probability weighting model (IPW) (**6.87 OR, 95% CI 4.39–10.77 p<0, 001**) were performed.

Table 3. *Regression table*

VARIABLE	UNIVARIABLE ODDS RATIO (OR), 95% CONFIDENCE INTERVAL (CI)	p VALUE	MULTIVARIABLE ODDS RATIO (OR), 95% CONFIDENCE INTERVAL (CI)	LASSO	MULTIVARIABLE P VALUE
AGE (for an increase of each one year)	1.05 (1.03–1.06)	<0.001	0.92 (0.81–1.05)	-	0.26
GENDER (female vs. male)	0.46 (0.31–0.70)	0.02	0.51 (0.17–1.49)	-	0.22
HTN (presence vs. absence)	1.26 (0.86–1.85)	0.22	0.45 (0.17–1.18)	-	0.1
DM (presence vs. absence)	1.27 (1.01–1.61)	0.04	1.04 (0.59–1.80)	-	0.88
HISTORY of MI (presence vs. absence)	2.46 (1.63–3.72)	<0.001	3.47 (1.41–8.55)	0.388	0.0068
SMOKING (presence vs. absence)	0.53 (0.39–0.73)	<0.001	0.70 (0.37–1.33)	-	0.28
KILLIP CLASS (for increase of each 1 class)	3.64 (3.02–4.38)	<0.001	1.52 (0.94–2.45)	0.627	0.0813
SBP (for an increase of each one mmHg)	0.97 (0.96–0.98)	<0.001	0.94 (0.89–1.00)	-	0.0525
PULSE RATE (for increase of each one b. p. m.)	1.02 (1.01–1.03)	<0.001	0.98 (0.89–1.07)	-	0.7
VT/VF (presence vs. absence)	7.37 (4.58–11.84)	<0.001	4.02 (1.44–11.21)	0.44	0.0077
IN-HOSPITAL AF (presence vs. absence)	2.25 (1.25–4.03)	0.006	1.23 (0.32–4.64)	-	0.75
IN-HOSPITAL non-AF SVT (presence vs. absence)	6.09 (1.00–36.84)	0.04	2.57 (0.10–61.67)	-	0.55
AVB (presence vs. absence)	3.64 (1.70–7.78)	0.001	0.44 (0.03–6.22)	-	0.55
INFARCT RELATED ARTERY (LAD vs. NON-LAD)	1.48 (0.94–2.33)	0.08	0.52 (0.20–1.38)	-	0.19
EF (for increase of each 1%)	0.91 (0.89–0.93)	<0.001	0.88 (0.78–0.99)	-0.032	0.0444
CRP (for increase of each 1 mg/dl)	1.12 (1.08–1.16)	<0.001	1.81 (0.22–14.53)	0.036	0.57
ADMISSION HGB (for an increase of each one g/dl)	0.75 (0.68–0.82)	<0.001	0.47 (0.28–0.79)	-	0.0042
SERUM CREATININE (for an increase of each 1 mg/dl)	1.05 (0.98–1.12)	0.13	5.07 (0.0003–82909.2)	-	0.74
ADMISSION POTASSIUM (for an increase of each one mEq/L)	1.90 (1.37–2.61)	<0.001	5.76 (0.25–130.95)	-	0.27

AF: atrial fibrillation, AVB: atrioventricular block, b. p. m.: beat per minute, CI: confidence interval, CPA: cardiopulmonary arrest, CRP: C-reactive protein, DM: diabetes mellitus, EF: ejection fraction, g/dl: grams per deciliter, HTN: hypertension, LAD: left anterior descending, LASSO: least absolute shrinkage and selection operator, mEq/L: milliequivalents per liter, mg/dl: milligrams per deciliter, MI: myocardial infarction, mmHg: millimeter of mercury, OR: Odds Ratio, SVT: supraventricular tachycardia, VF: ventricular fibrillation, vs.: versus, VT: ventricular tachycardia.

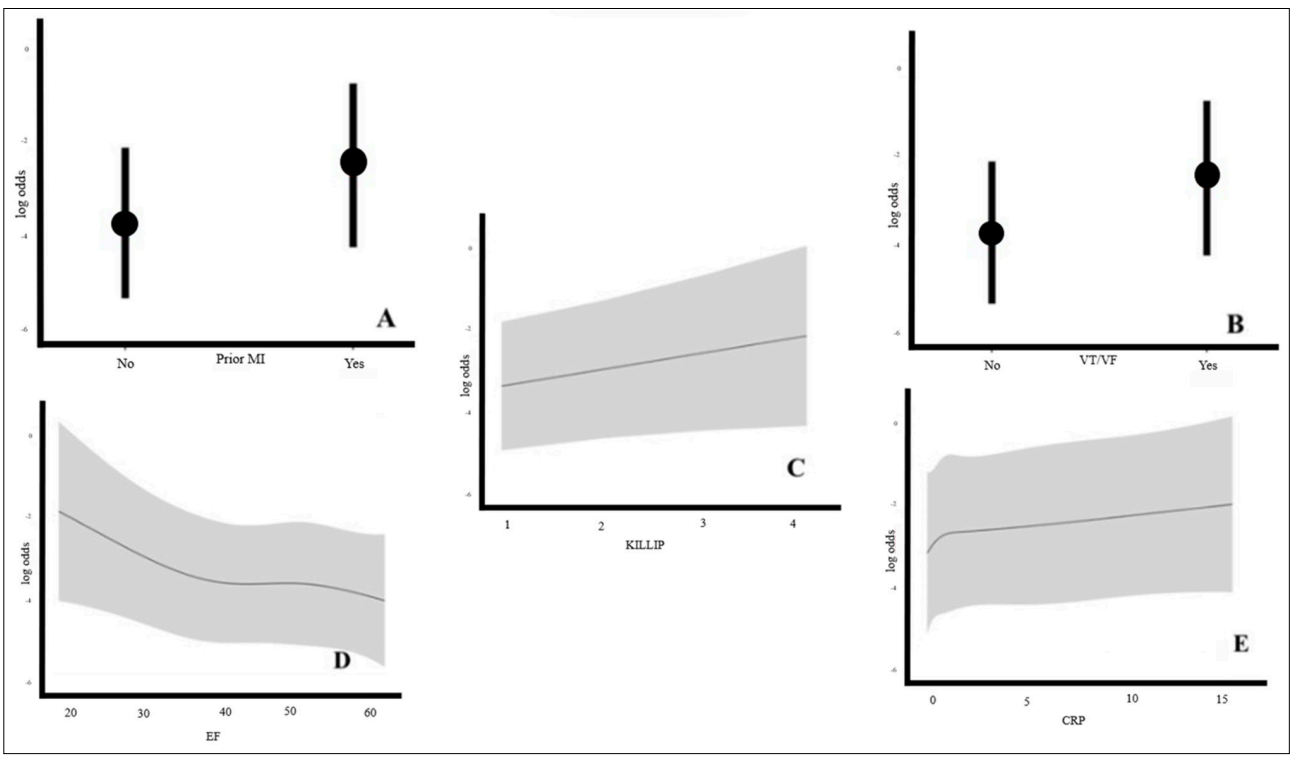

Figure 1. *The relationship between independent predictors and in-hospital mortality/aCPA log odds.*

Discussion

In our study, it was observed that primary VT/VF occurred in 8.2% (n=93) of patients with acute STEMI, and the results demonstrated that primary VT/VF, previous MI, admission KILLIP classification, LVEF and serum CRP level were independent predictors of in-hospital mortality and aCPA in STEMI patients. Among these parameters, primary VT/VF was the predictor with the highest odds ratio in multivariable regression analysis (4.02 OR). We conducted propensity score matching since our study was not a randomized controlled trial. Primary VT/VF occurrence was found to increase the risk of in-hospital mortality and aCPA 2.7 fold after PS adjustment, which was consistent with multivariable regression analysis (2.76 OR, 95% CI 1.17–6.35 p=0.0168 and 4.02 OR, 95% CI 1.44–11.2 p=0.008, respectively).

Previous reports on the prognostic importance of VAs in the setting of MI demonstrated different results[2,3,5,7,8,10,11]. According to the pooled data analysis results in The Primary Angioplasty in Myocardial Infarction (PAMI), VT/VF occurrence during pPCI in catheterization laboratory in STEMI patients didn't hurt in-hospital and one-year mortality and MACE. This favorable result might have been driven by excluding some high-risk patients, such as patients with cardiogenic shock or renal failure, from the study population[10].

Global Use of Streptokinase t-PA for Occluded Coronary Arteries (GUSTO-I) and Gruppo Italiano per lo Studio della Sopravvivenza nell'Infarto Miocardico (GISSI-2) trials were conducted in thrombolytic era. Both the GUSTO-I and GISSI-2 trials suggested that patients with ventricular arrhythmias had higher in-hospital mortality rates than patients without16.17. Although pPCI is the contemporary treatment of choice in patients with STEMI and performed widely, our results revealed that the impact of VAs on the in-hospital course of MI can be considered similar to that in GUSTO-I and GISSI-2.

Harmonizing Outcomes with Revascularization and Stents in Acute Myocardial Infarction (HORIZONS-AMI) trial revealed that early VAs didn't show statistical significance on 30-day MACE and mortality rates but had a significant adverse impact on 3-year MACE events[7]. Although in-hospital mortality rates were lacking in this study, short-term mortality rates contradict our results. A larger sample size in the HORIZONS-AMI study might drive this contradiction. Another cohort study, including 9015 patients with acute MI who underwent percutaneous coronary intervention, suggested that early VAs were associated with a 4-fold increase in in-hospital mortality rates. Interestingly, although successful mechanical revascularization decreased mortality rates, the association between in-hospital mortality and early VAs was similar to the study population in patients with successful coronary intervention[3]. Although the relation between early VAs and in-hospital mortality in this study was consistent with our results, it should be emphasized that this study was conducted with all MI patients, not only with STEMI.

Ectopic excitation and reentry circuits should be addressed as the underlying mechanisms of VAs in myocardial ischemia. Firstly, acute ischemia causes metabolic alterations such as adenosine triphosphate depletion and intracellular acidosis in myocytes. Due to metabolic alterations, intracellular calcium mishandling and action potential duration changes cause early and late after-depolarization-induced ventricular ectopics[19,20]. Unidirectional conduction disturbance, such as block or slow conduction in the ischaemic area, might create a re-entry circuit. In addition, myocardial sympathetic innervation abnormalities, innervation/perfusion mismatch and epinephrine retention might create VA substrate in ischemic myocardium[19–22].

Conclusion

Despite the conflicting results of different studies in the literature, our study demonstrated that primary VT/VF in patients with STEMI is significantly associated with in-hospital mortality and aCPA. It can be concluded that VT/VF occurrence within the first 48 hours of hospital admission in patients with STEMI might cause lower in-hospital survival rates than patients without VT/VF. As a matter of course, a high volume of prospective studies is needed on this issue.

Our study's main limitation is its retrospective and single-center nature. The second limitation is that our study results consist of only in-hospital endpoints; long-term follow-up results are still missing. Also, the primary endpoint of our study is in-hospital mortality and aCPA, which prevents the interpretation of whether VT/VF occurrence affects in-hospital mortality rates independently.

References

1. Yeh RW, Sidney S, Chandra M, Sorel M, Selby JV, Go AS -population trends in the incidence and outcomes of acute myocardial infarction. N Engl J Med. 2010;362(23):2155–65.
2. Mehta RH, Starr AZ, Lopes RD, Hochman JS, Widimsky P, Pieper KS, et al. Incidence of and outcomes associated with ventricular tachycardia or fibrillation in patients undergoing primary percutaneous coronary intervention. JAMA. 2009;301(17):1779–89.
3. Piccini JP, Berger JS, Brown DL. Early sustained ventricular arrhythmias complicating acute myocardial infarction. Am J Med. 2008;121(9):797–804.
4. Tran HV, Ash AS, Gore JM, Darling CE, Kiefe CI, Goldberg RJ. Twenty-five-year trends (1986–2011) in hospital incidence and case-fatality rates of ventricular tachycardia and ventricular fibrillation complicating acute myocardial infarction. Am Heart J. 2019;208:1–10.
5. Henkel DM, Witt BJ, Gersh BJ, Jacobsen SJ, Weston SA, Meverden RA, et al. Ventricular arrhythmias after acute myocardial infarction: a 20-year community study. Am Heart J. 2006;151(4):806–12.
6. Vallabhajosyula S, Patlolla SH, Verghese D, Ya'Qoub L, Kumar V, Subramaniam AV, et al. Burden of Arrhythmias in Acute Myocardial Infarction Complicated by Cardiogenic Shock. Am J Cardiol. 2020;125(12):1774–81.
7. Kosmidou I, Embacher M, McAndrew T, Dizon JM, Mehran R, Ben-Yehuda O, et al. Early Ventricular Tachycardia or Fibrillation in Patients With ST Elevation Myocardial Infarction Undergoing Primary Percutaneous Coronary Intervention and Impact on Mortality and Stent Thrombosis (from the Harmonizing Outcomes with Revascularization and Stents in Acute Myocardial Infarction Trial) . Am J Cardiol. 2017;120(10):1755–60.
8. Al-Khatib SM, Stebbins AL, Califf RM, Lee KL, Granger CB, White HD, et al. Sustained ventricular arrhythmias and mortality among patients with acute myocardial infarction: results from the GUSTO-III trial. Am Heart J. 2003;145(3):515–21.
9. Ohlow MA, Geller JC, Richter S, Farah A, Müller S, Fuhrmann JT, et al. Incidence and predictors of ventricular arrhythmias after ST-segment elevation myocardial infarction. Am J Emerg Med. 2012;30(4):580–86.
10. Mehta RH, Harjai KJ, Grines L, Stone GW, Boura J, Cox D, et al. Sustained ventricular tachycardia or fibrillation in the cardiac catheterization laboratory among patients receiving primary percutaneous coronary intervention: incidence, predictors, and outcomes. J Am Coll Cardiol. 2004;43(10):1765–72.
11. García-García C, Oliveras T, Rueda F, Pérez-Fernández S, Ferrer M, et al. Primary Ventricular Fibrillation in the Primary Percutaneous Coronary Intervention ST-Segment Elevation Myocardial Infarction Era (from the "Codi IAM" Multicenter Registry) . Am J Cardiol. 2018;122(4):529–36.
12. Cronin EM, Bogun FM, Maury P, Peichl P, Chen M, Namboodiri N, et al. 2019 HRS/EHRA/APHRS/LAHRS expert consensus statement on catheter ablation of ventricular arrhythmias. Heart Rhythm. 2020;17(1):e2–e154.
13. Williams B, Mancia G, Spiering W, Agabiti Rosei E, Azizi M, Burnier M, et al. 2018 ESC/ESH Guidelines for the management of arterial hypertension [published correction appears in Eur Heart J. 2019;40(5):475]. Eur Heart J. 2018;39(33):3021–104.
14. American Diabetes Association. 2. Classification and Diagnosis of Diabetes: Standards of Medical Care in Diabetes-2021 [published correction appears in Diabetes Care. 2021;44(9):2182]. Diabetes Care. 2021;44(Suppl 1):S15–33.
15. Killip T 3rd, Kimball JT. Treatment of myocardial infarction in a coronary care unit. A two-year experience with 250 patients. Am J Cardiol. 1967;20(4):457–64.
16. Volpi A, Cavalli A, Santoro L, Negri E. Incidence and prognosis of early primary ventricular fibrillation in acute myocardial infarction--results of the Gruppo Italiano per lo Studio della Sopravvivenza nell'Infarto Miocardico (GISSI-2) database. Am J Cardiol. 1998;82(3):265–71.
17. Newby KH, Thompson T, Stebbins A, Topol EJ, Califf RM, Natale A. Sustained ventricular arrhythmias in patients receiving thrombolytic therapy: incidence and outcomes. The GUSTO Investigators. Circulation. 1998;98(23):2567–73.
18. Jabbari R, Risgaard B, Fosbøl EL, Scheike T, Philbert BT, Winkel BG, et al. Factors Associated With and Outcomes After Ventricular Fibrillation Before and During Primary Angioplasty in Patients With ST-Segment Elevation Myocardial Infarction. Am J Cardiol. 2015;116(5):678–85.
19. Bhar-Amato J, Davies W, Agarwal S. Ventricular Arrhythmia after Acute Myocardial Infarction:'The Perfect Storm.' Arrhythm Electrophysiol Rev. 2017;6(3):134–9.
20. Di Diego JM, Antzelevitch C. Ischemic ventricular arrhythmias: experimental models and their clinical relevance. Heart Rhythm. 2011;8(12):1963–68.
21. Sasano T, Abraham MR, Chang KC, Ashikaga H, Mills KJ, Holt DP, et al. Abnormal sympathetic innervation of viable myocardium and the substrate of ventricular tachycardia after myocardial infarction. J Am Coll Cardiol. 2008;51(23):2266–75.
22. Li CY, Li YG. Cardiac sympathetic nerve sprouting and susceptibility to ventricular arrhythmias after myocardial infarction. Cardiol Res Pract. 2015;2015:698368.