Original Article

Correlations between Cardiovascular Risk Factors and Ventricular Arrhythmias Following Primary Percutaneous Coronary Intervention in Patients with ST-Elevation Myocardial Infarction

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Abstract

Background: *Ventricular arrhythmias (VAs), which result from acute myocardial infarction and revascularization, are preventable causes of sudden cardiac death. This study aimed to determine the incidence, types, and risk factors of VAs in patients with ST-elevation myocardial infarction undergoing primary percutaneous coronary intervention (PCI).*

Methods: *This cross-sectional study was conducted at the cardiology department of a tertiary care cardiac center in Zanjan, Iran. All the patients were monitored during hospitalization, and the incidence of cardiac arrhythmias and the outcomes were recorded.*

Results: *Among 315 patients, the mean age was 62.14±10.11 years, and 76.2% were male. Male gender was significantly associated with VA occurrence (P=0.038). Among the patients, 50.5% had VAs, of which 26.4% were sustained ventricular tachycardia (sustained VT) and ventricular fibrillation (VF). Sustained VT and VF, but not total arrhythmias, were more common in anterior infarctions. Most arrhythmias occurred during the first 12 hours, and frequent premature ventricular contractions (43.3%) and idioventricular rhythm (20.1%) were the most common. A history of PCI and coronary artery bypass grafting (CABG) was associated with substantially reduced arrhythmias (P=0.017 and P=0.013, respectively). However, cardiovascular risk factors exerted no statistically significant effects on the VA type.*

Conclusion: *Approximately half of our patients experienced reperfusion-induced VAs. Overall, gender and a history of PCI and CABG were significantly associated with VA occurrence. Therefore, males and patients without a positive history of PCI and CABG should receive antiarrhythmic drugs as a precaution.*

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Keywords: *Cardiac arrhythmias; Percutaneous coronary intervention; Heart disease risk factors; Outcome assessment; ST-elevation myocardial infarction*

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Introduction

Coronary artery disease (CAD) is one of the most common causes of morbidity and mortality worldwide, with its incidence and complications rising in developing countries. The most life-threatening disease in this category is acute myocardial infarction (AMI) and its associated complications.[1-3] The majority of deaths in AMI are due to arrhythmias, and most arrhythmias are seen in the first 48 to 72 hours after the onset of symptoms. Some rhythm disturbances in patients with AMI may be related to coronary artery reperfusion.[4]

In patients with ST-elevation myocardial infarction (STEMI), timely primary percutaneous coronary intervention (PPCI) is considered the most acceptable treatment method for coronary artery reperfusion and myocardial salvage. Despite the clear advantages of reperfusion in terms of improved patient outcomes, there are risks, such as reperfusion injury and reperfusion-induced ventricular arrhythmias (VAs).[5, 6] The most frequently observed reperfusion VAs are premature ventricular contractions (PVCs), sustained or non-sustained ventricular tachycardia (sustained VT or NSVT), accelerated idioventricular rhythm (AIVR), and ventricular fibrillation (VF).[7, 8]

The critical mechanism of arrhythmias in the acute phase of coronary occlusion is due to the lack of coordination of electrophysiological and biomechanical properties of ischemic myocardium with healthy non-ischemic areas. Following ischemia, adenosine triphosphate deficiency, anaerobic glycolysis causing cellular acidosis, elevated extracellular potassium ion concentrations, and intracellular calcium accumulation happen. They will contribute to variations in the action potential duration and reentrant arrhythmias, resulting in widened QRS complexes and PVCs or VT and VF.[9] Frequent PVCs occur following coronary artery reperfusion in patients with STEMI and indicate premature depolarization followed by premature contractions of myocytes. This rhythm can sometimes lead to malignant VAs, VF, and VT.[10]

The present study aimed to determine the incidence, types, and risk factors of reperfusion-induced VAs in patients with STEMI undergoing PPCI during hospitalization at a tertiary care hospital in Zanjan, Iran.

Methods

The current cross-sectional study was conducted at the adult cardiology department of a tertiary care cardiac center in Zanjan, Iran, from July 2018 through April 2020. Patients 18 years of age or older with a STEMI diagnosis who underwent PPCI and had symptom onset within 12 hours were eligible for enrolment. Patients with non-STEMI; unstable angina; cardiac conduction diseases, including any atrioventricular block and fascicular block; permanent pacemakers; mechanical ventilation support; cardiogenic shock; dilated cardiomyopathy; chronic kidney disease; and thyroid disorders were excluded from the study. The investigation commenced after approval was granted by the Ethics Review Committee of Zanjan University of Medical Sciences (ethics code: IR.ZUMS.REC.1396.173). The primary investigator clearly explained the purpose of the study and potential advantages to each participant before they were enrolled in the study and obtained verbal informed consent from each participant.

Demographic characteristics and risk factors, such as age, gender, body mass index, current smoking, diabetes mellitus, a family history of CAD, a prior PCI, and hypertension, were recorded. The number of vessels involved and culprit coronary vessels were extracted from angiographic reports. STEMI and cardiac arrhythmias were diagnosed based on electrocardiography. Infarct localizations were categorized according to the place of ST-segment–elevation lead in electrocardiographic derivations as anterior, inferior, right ventricular inferior, and lateral AMI.

All the patients were monitored through electrocardiographic telemetry monitoring systems during their hospitalization. VAs, such as AIVR, frequent PVCs, NSVT, sustained VT, and VF, were recorded as per the operational definition by the principal investigator. Frequent PVCs were defined as PVC counts more than 6 per minute, NSVT was defined as more than 3 ventricular ectopic beats at a rate of 120 bpm or more lasting less than 30 seconds, and sustained VT was defined as a duration exceeding 30 consecutive seconds. AIVR was defined as more than 3 successive ventricular rhythm beats at a rate below 120 bpm.[11]

The type of STEMI was defined based on the ST-segment elevation in different leads: ST-segment elevation in leads V_1-V_4 as anteroseptal; V_3 and V4 as anterior; V_1-V_6 as extensive anterior; V_1-V_6, I, and aVL as anterolateral; I and aVL as lateral; II, III, and aVF as inferior; I, II, III, aVF, and aVL as inferolateral; II, III, aVF, V_8, and V_9 as inferoposterior; II, III, aVF, aVL, V_8, and V_9 as inferoposterolateral; and II, III, aVF, and V_3-V_6 right as inferior + right ventricular STEMI.[1-3]

Data were analyzed using the Statistical Package for the Social Sciences (SPSS), version 23. In descriptive statistics, continuous quantitative data were reported as averages and standard deviations and nominal and qualitative data as percentages and frequencies. The χ^2 test was employed to determine the relationship between nominal and qualitative variables. Multivariable and binary logistic regression analyses were performed to evaluate the impact of cardiovascular risk factors on VAs. A P value of less than 0.05 was considered statistically significant for all the analyses.

Results

The study population consisted of 315 patients (240 males, 76.2%) at a mean age of 62.14±10.11 years. Smoking was reported in 41% of the patients, hypertension in 49.5%, body mass index ≥25 in 46.7%, diabetes mellitus in 22.2%, and a family history of CAD in 31.1%. The majority of the patients had single-vessel disease (45.1%), and the most frequent culprit vessels were the left anterior descending artery (57.8%) and the right coronary artery (25.4%). During hospitalization, 159 patients (50.5%), comprising 30 female patients (9.5%) and 129 male patients (41.0%), experienced VAs. A history of PCI was reported in 35 patients (11.1%), 11 of whom (31.4%) had VAs during hospitalization. Furthermore, 6 patients (1.9%) had a positive history of CABG, and 1 of them suffered VAs during hospitalization. The occurrence of VAs was increased by male gender and decreased by a history of PCI and CABG (Table 1).

Frequent PVCs and AIVR were the most prevalent VAs in both genders. Lethal arrhythmias, including sustained VT and VF, occurred among 26 (8.3%) and 16 (5.1%) patients, respectively. No statistically significant relationship existed between the type of VA and gender, age, a history of PCI and CABG, other cardiovascular risk factors, and angiographic features (Table 2).

Based on the results of the binary logistic regression for cardiovascular risk factors, the occurrence of VAs after PPCI was statistically significantly associated with male gender (P=0.038). Additionally, a history of PCI and CABG was associated with substantially reduced arrhythmias (P=0.017 and P=0.013, respectively). Men were 2.2 times more likely to have an arrhythmia than women, and patients without a history of PCI and CABG were 2.7 times and 3.6 times, respectively, more likely to have an arrhythmia (Figure 1).

The first episode of arrhythmias occurred 35 minutes after PPCI, and the latest episode was 202 hours after PPCI. The average onset time of arrhythmias was 23.32±17.74 hours. The first 12 hours after PPCI was the most likely time for arrhythmias to develop, with 91 cases (57.2%) of arrhythmias. Only 6.9% of VAs occurred after 48 hours. Furthermore, lethal arrhythmias occurred more frequently in the first 12 hours than in subsequent periods. Although no statistically significant relationship existed between the type and the onset time of arrhythmias (P=0.398), the results revealed that the chance of arrhythmia occurrence declined after PPCI with time (Figure 2).

The most prevalent types of STEMI were anteroseptal (23.2%) and extensive anterior (18.1%), followed by inferior (14.6%), inferior and posterior (12.7%), anterior and lateral (10.2%), and anterior (9.8%). The incidence of VAs was

Table 1. Ventricular arrhythmias based on demographic, anamnestic, and angiographic features

Variable n (%)	Total (n=315)	VAs (n=159)	No VAs (n=156)	*P*
Sex,				0.045
Male	240 (76.2)	129 (81.1)	111 (71.2)	
Age (y)	62.14±10.11	62.17±11.32	61.91±12.10	0.745
Smoking	129 (41.0)	64 (40.3)	65 (41.7)	0.456
HTN	156 (49.5)	80 (50.3)	76 (48.7)	0.621
DM	70 (22.2)	31 (19.5)	39 (25.0)	0.123
Previous PCI	35 (11.1)	11 (6.9)	24 (15.4)	0.023
Previous CABG	6 (1.9)	1 (0.6)	5 (3.2)	0.001
BMI ≥25	147 (46.7)	69 (43.4)	78 (50.0)	0.093
Positive FH	98 (31.1)	51 (32.1)	47 (30.1)	0.586
Number of vessels involved				0.214
SVD	142 (45.1)	77 (48.4)	65 (41.7)	
2VD	85 (27.0)	42 (26.4)	43 (27.6)	
3VD	88 (27.9)	40 (25.2)	48 (30.7)	
Culprit coronary vessel				0.412
RCA	80 (25.4)	48 (30.2)	32 (20.5)	
LAD	182 (57.8)	85 (53.5)	97 (62.2)	
LCX	33 (10.5)	19 (12.0)	14 (9.0)	
OM	8 (2.5)	3 (1.9)	5 (3.2)	
Ramus	1 (0.3)	0 (0)	1 (0.6)	
PDA	5 (1.6)	1 (0.6)	4 (2.6)	
Major D1	3 (1.0)	1 (0.6)	2 (1.3)	
Major D2	1 (0.3)	1 (0.6)	0 (0)	
LMCA	2 (0.6)	1 (0.6)	1 (0.6)	

The χ^2 test was used for demographic and anamnestic features and the Fisher exact test for angiographic features.
VA, Ventricular arrhythmia; HTN, Hypertension; DM, Diabetes mellitus; PCI, Percutaneous coronary intervention; CABG, Coronary artery bypass graft; BMI, Body mass index; FH, Family history of coronary artery disease; SVD, Single-vessel disease; 2VD, Two-vessel disease; 3VD, Three-vessel disease; RCA, Right coronary artery; LAD, Left anterior descending artery; LCX, Left circumflex artery; OM 1, First obtuse marginal artery; PDA, Posterior descending artery; Major D1, First diagonal artery; Major D2, Second diagonal artery; LMCA, Left main coronary artery

Table 2. The distribution of ventricular arrhythmias by demographic, anamnestic, and angiographic features

Variable n (%)	Total (n=159)	Frequent PVCs (n=71)	NSVT (n=14)	Sustained VT (n=23)	VF (n=16)	AIVR (n=30)	NSVT + AIVR (n=2)	NSVT + Sustained VT (n=2)	AIVR + Sustained VT (n=1)	*P*
Sex										0.543
Male	129 (81.1)	54 (76.1)	12 (85.7)	19 (82.6)	14 (87.5)	25 (83.3)	2 (100.0)	2 (100.0)	1 (100.0)	
Age (y)	62.17±11.32	63.41±4.62	62.22±9.01	63.11±10.12	62.04±11.12	60.31±10.90	61.21±10.04	63.14±6.83	62.00±0	0.622
Smoking	64 (40.3)	27 (38.0)	6 (42.9)	9 (39.1)	7 (43.8)	13 (43.3)	0 (0)	2 (100.0)	0 (0)	0.712
HTN	80 (50.3)	37 (52.1)	9 (64.3)	10 (43.5)	4 (25.0)	17(56.7)	2(100.0)	0(0)	1 (100.0)	0.187
DM	31 (19.5)	13 (18.3)	4 (28.6)	4 (17.4)	2 (12.5)	7(23.3)	1(50.0)	0(0)	0(0)	0.798
Previous PCI	11 (6.9)	4 (5.6)	0 (0)	2 (8.7)	2 (12.5)	3 (10.0)	0 (0)	0 (0)	0 (0)	0.471
Previous CABG	1 (0.6)	1 (1.4)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0.875
BMI ≥ 25	69 (43.4)	40 (56.3)	5 (35.7)	9 (39.1)	3 (18.8)	12 (40.0)	0 (0)	0 (0)	0 (0)	0.700
Positive FH	51 (32.1)	22 (31.0)	4 (28.6)	9 (39.1)	5 (31.2)	8 (26.7)	2 (50.0)	1 (50.0)	0 (0)	0.614
Number of vessels involved										0.907
SVD	77 (48.4)	31 (43.7)	8 (57.1)	15 (65.2)	9 (56.2)	11 (36.7)	2 (100.0)	0 (0)	1 (100.0)	
2VD	42 (26.4)	19 (26.7)	4 (28.6)	5 (21.7)	3 (18.8)	10 (33.3)	0 (0)	1 (50.0)	0 (0)	
3VD	40 (25.2)	21 (29.6)	2 (14.3)	3 (13.1)	4 (25.0)	9 (30.0)	0 (0)	1 (50.0)	0 (0)	
Culprit coronary vessel										0.988
RCA	48 (30.2)	25 (35.2)	3 (21.4)	4 (17.4)	4 (25.0)	10 (33.3)	0 (0)	1 (50.0)	1 (100.0)	
LAD	85 (53.5)	28 (39.5)	9 (64.3)	18 (78.3)	9 (56.3)	18 (60.0)	2 (100.0)	1 (50.0)	0 (0)	
LCX	19 (12.0)	14 (19.7)	2 (14.3)	0 (0)	2 (12.5)	1 (3.3)	0 (0)	0 (0)	0 (0)	
OM	3 (1.9)	1 (1.4)	0 (0)	1 (4.3)	1 (6.2)	0 (0)	0 (0)	0 (0)	0 (0)	
Ramus	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	
PDA	1 (0.6)	1 (1.4)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	
Major D1	1 (0.6)	0 (0)	0 (0)	0 (0)	0 (0)	1 (3.3)	0 (0)	0 (0)	0 (0)	
Major D2	1 (0.6)	1 (1.4)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	
LMCA	1 (0.6)	1 (1.4)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	

Values are expressed as the number of patients (%).
The χ^2 test was used for demographic and anamnestic features and the Fisher exact test for angiographic features.
VA, Ventricular arrhythmia; PVC, Premature ventricular contraction; NSVT, Non-sustained ventricular tachycardia; Sustained VT, Sustained ventricular tachycardia; VF, Ventricular fibrillation; AIVR, Accelerated idioventricular rhythm; HTN, Hypertension; DM, Diabetes mellitus; PCI, Percutaneous coronary intervention; CABG, Coronary artery bypass grafting; BMI, Body mass index; FH, Family history of coronary artery disease; SVD, Single-vessel disease; 2VD, Two-vessel disease; 3VD, Three-vessel disease; RCA, Right coronary artery; LAD, Left anterior descending artery; LCX, Left circumflex artery; OM 1, First obtuse marginal artery; PDA, Posterior descending artery; Major D1, First diagonal artery; Major D2, Second diagonal artery; LMCA, Left main coronary artery.

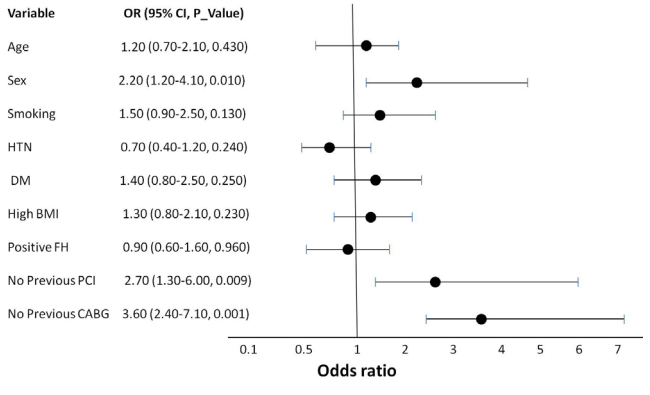

Figure 1. The image depicts the multivariable analysis (adjusted odds ratio) for the evaluation of the impact of cardiovascular risk factors on ventricular arrhythmias.
HTN, Hypertension; DM, Diabetes mellitus; BMI, Body mass index; FH, Familial history; PCI, Percutaneous coronary intervention; CABG, Coronary artery bypass grafting

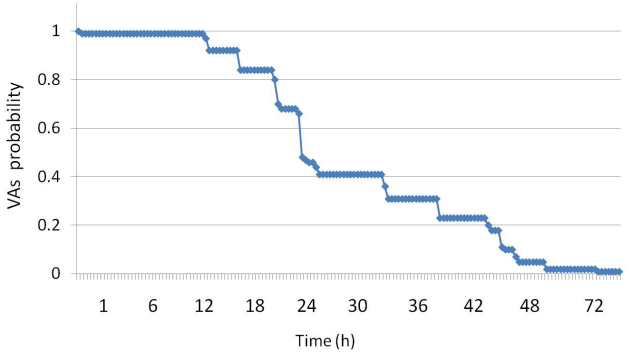

Figure 2. The image illustrates the probability of ventricular arrhythmias during hospitalization based on time.
VAs, Ventricular arrhythmia

Table 3. Distribution of ventricular arrhythmias by type of STEMI

Variable	Total	Type of STEMI										*P*
		Ant	Inf	Lat	Anteroseptal	Ext Ant	Ant + Lat	Inf + Post	Inf + RV	Inf + Post + RV	Inf + Lat	
Total	315	31	46	4	73	57	32	40	8	12	12	
VAs	159 (50.5)	14 (45.2)	31 (67.4)	2 (50.0)	38 (52.1)	20 (35.1)	13 (40.6)	26 (65.0)	4 (50.0)	5 (41.7)	6 (50.0)	
No VAs	156 (49.5)	17 (54.8)	15 (32.6)	2 (50.0)	35 (47.9)	37 (64.9)	19 (59.4)	14 (35.0)	4 (50.0)	7 (58.3)	6 (50.0)	0.163
Frequent PVCs	71 (22.5)	6 (19.4)	17 (37.0)	0 (0)	11 (15.1)	8 (14.0)	6 (18.8)	15 (37.5)	2 (25.0)	2 (16.7)	4 (33.3)	0.163
NSVT	14 (4.4)	2 (6.4)	2 (4.3)	0 (0)	3 (4.1)	1 (1.8)	1 (3.1)	2 (5.0)	1 (12.5)	1 (8.3)	1 (8.3)	0.163
Sustained VT	23 (7.3)	1 (3.2)	2 (4.3)	1 (25.0)	11 (15.1)	4 (7.0)	1 (3.1)	3 (7.5)	0 (0)	0 (0)	0 (0)	0.163
VF	16 (5.1)	2 (6.4)	4 (8.7)	0 (0)	2 (2.7)	5 (8.8)	1 (3.1)	1 (2.5)	0 (0)	1 (8.3)	0 (0)	0.163
AIVR	30 (9.5)	2 (6.4)	5 (10.9)	1 (25.0)	9 (12.3)	2 (3.5)	4 (12.5)	5 (12.5)	1 (12.5)	0 (0)	1 (8.3)	0.163
NSVT+AIVR	2 (0.6)	1 (3.2)	0 (0)	0 (0)	1 (1.4)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0.163
NSVT+ Sustained VT	2 (0.6)	0 (0)	1 (2.2)	0 (0)	1 (1.4)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0.163
AIVR+ Sustained VT	1 (0.3)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	1 (8.3)	0 (0)	0.163

Values are expressed as the number of patients (%).
The Fisher exact test was used for the evaluation of the distribution of ventricular arrhythmias by type of STEMI.
VA, Ventricular arrhythmia; STEMI, ST-elevation myocardial infarction; PVC, Premature ventricular contraction; NSVT, Non-sustained ventricular tachycardia; sustained VT, Sustained ventricular tachycardia; VF, Ventricular fibrillation; AIVR, Accelerated idioventricular rhythm; Ant, Anterior; Inf, Inferior; Lat, Lateral; Ext, Extensive; Post, Posterior; RV, Right ventricular

significantly higher in inferior wall involvement than in anterior and lateral wall involvement (P=0.045) such that the ratio of patients with arrhythmias to patients without arrhythmias in inferior AMI and then inferoposterior AMI was the highest and in extensive anterior and anterolateral types was the lowest. Of all kinds of STEMI, frequent PVCs were the most common VAs, and there was no significant association between types of MI and reperfusion VAs (P=0.163). The distribution of arrhythmias by type of STEMI is presented in Table 3.

Two hundred seventy patients (85.7%) underwent PPCI within less than 90 minutes of entering the hospital, and 50% had VAs. Although the ratio of patients with arrhythmias to those without arrhythmias in patients who underwent PPCI within more than 90 minutes of entering the hospital was higher than that in the other group, no statistically significant relationship was observed between the incidence and type of reperfusion-induced VAs and the door-to-balloon time.

In 228 cases (72.4%), the patients were hospitalized for a maximum of 7 days. Patients who developed VAs had a lengthier hospital stay than those without VAs. However, no statistically significant association existed between the occurrence and type of reperfusion VAs and the hospitalization length. When the length of hospitalization was more than 15 days, the ratio of patients with arrhythmias to those without arrhythmias was more remarkable than when the extent of hospitalization was less than 15 days. Furthermore, the incidence of lethal arrhythmias increased with extended hospitalization.

Discussion

VAs resulting from AMI and revascularization are preventable causes of sudden cardiac death. They are now considered treatable thanks to the development of intensive cardiac care units and novel treatments.[12] Our study investigated the incidence, types, and risk factors of reperfusion-induced VAs during hospitalization in patients with STEMI.

The most remarkable result to emerge from the data was that gender, previous CABG, and a history of PCI were significantly associated with the incidence of VAs. Nonetheless, we found no statistically significant relationships between VA types and gender, age, smoking, hypertension, diabetes mellitus, a history of PCI, a positive family history, body mass index, and numbers and types of culprit coronary vessels.

Male gender was predominant for AMI and reperfusion VA incidence, which is analogous to other studies.[13-15] The lower incidence of CAD in women may be due to the cardioprotective effects of the sex hormones estrogen and progesterone, which regulate the lipid profile. In men, testosterone levels decrease gradually with age, and studies suggest that low testosterone levels are associated with higher cardiovascular disease incidence in men.[16, 17]

In our study, VAs were reported in 50.5% of the patients, 26.4% of whom had lethal arrhythmias. Frequent PVCs and AIVR were the most common arrhythmias (63.4%). The incidence rate of VAs after PPCI was approximately 75%.[18,

[19] It has been observed that the frequency of arrhythmias decreases with the increase in the duration of the study. Likewise, our results showed that the chance of arrhythmia incidence declined as time elapsed since PPCI. Terkelsen et al[18] reported that out of 503 patients with a diagnosis of STEMI undergoing PPCI, 3.6% had lethal arrhythmias, and the most prevalent VAs were AIVR at a rate of 42%. The incidence of fatal arrhythmias varies from 3.7% to 42%, depending on the study population.[19-23] Similar to our results, in various recent studies, at least the second most common arrhythmia was AIVR, whose incidence ranged from about 42% to 50%.[18, 19, 21]

In line with 2 sizable observational studies, in the present study, the frequencies of hypertension, diabetes mellitus, and a history of AMI as the risk factors for AMI were about 50%, 20%, and 15%, respectively. In addition, age over 60 is a risk factor for AMI development.[24, 25] Consistent with our research, Paul et al[14] demonstrated that diabetes mellitus, hypertension, obesity, family history, and smoking had no statistically significant relationships with the occurrence of arrhythmias.

In the present study, according to the angiography reports, the most frequent culprit coronary artery was the left anterior descending artery (57.8%), and most patients (45.1%) had more than 50% stenosis in 1 vessel. Consistently, in other studies, single-vessel disease was the most common angiographic diagnosis, with the left anterior descending artery being the most common culprit vessel.[13, 26]

According to the findings of our study, a history of PCI and CABG reduced the incidence of cardiac arrhythmias. These patients consume antiarrhythmic drugs, including β-blockers; consequently, their rate of VAs during hospitalization is lower than that of patients without a history of PCI or CABG.

According to the type of vessel involved in AMI and its blood supply areas, the type of AMI can affect the occurrence of arrhythmias. In this study, the ratio of patients with VAs to those without arrhythmias in the right coronary artery and inferior STEMI was higher than that of patients with other types of vascular involvement and AMI. Still, we found no statistically significant relationship between the occurrence of arrhythmias and the type of involved vessel, unlike the relationship between the occurrence of arrhythmias and the type of AMI (P<0.05). These findings can be justified by the fact that inferior wall STEMI is predominantly caused by the acute occlusion of the right coronary artery (80%) and the left circumflex artery (20%).[27, 28] In addition, the right coronary artery provides both the sinus and atrioventricular node blood supply (or circumflex in the case of left-dominant coronary circulation).[29] Thus, in inferior MI, these nodes are more involved, increasing the probability of arrhythmias.

In the present study, the incidence of VT was 5.3% in anterior wall involvement and 1.5% in inferior wall involvement. The incidence of VF in anterior and inferior wall involvement was 3.1% and 1.9%, respectively. In other studies, the overall frequency of VAs did not relate to MI types, although both VT and VF were more frequent in anterior MI. The higher incidence of lethal VAs in anterior wall infarction may be due to the larger area of myocardium infarct size and sympathetic hyperactivity.[23, 30, 31]

The present investigation had several limitations. Firstly, it was a single-center study with a small sample size; therefore, our results need confirmation by further studies. Secondly, we investigated only patients who underwent PPCI; patients who had not received PPCI were not included in the study. Thirdly, we had no long-term follow-up concerning VA development for our study population.

Conclusion

Approximately half of our study population experienced VAs following PPCI. Most VAs occurred within the first 12 hours of hospitalization. Overall, gender and no history of PCI and CABG were significantly associated with VA occurrence. Therefore, males and patients without a positive history of PCI and CABG, as a precaution, should receive antiarrhythmic drugs, especially within the first 12 hours.

Acknowledgments

This study was approved by the Ethics Committee of Zanjan University of Medical Sciences, Zanjan, Iran.

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