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RESEARCH ARTICLE

Assessment of the Influence of Coronary Artery Stenting on the Frequency and Severity of Ventricular Ectopic Activity in Patients with Prior Myocardial Infarction with Preserved and Mildly Reduced Left Ventricular Ejection Fraction

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Abstract

Background: Ventricular ectopic activity (VEA) is a common complication in post-myocardial infarction (MI) patients and is associated with increased risk of sudden cardiac death (SCD). The effect of coronary revascularization by stenting on the burden and severity of VEA in patients with preserved and mildly reduced left ventricular ejection fraction (LVEF) remains insufficiently studied.

Objective: To evaluate the influence of complete versus incomplete coronary revascularization on the frequency and severity of VEA in post-MI patients with preserved and mildly reduced LVEF.

Methods: A total of 270 patients (age 38–75 years) with prior MI were enrolled and divided into three groups: complete revascularization (CR, n=115), incomplete revascularization (IR, n=94), and no revascularization (NR, n=61). All patients underwent baseline coronary angiography and 24-hour Holter ECG monitoring, which was repeated at 1-year follow-up. VEA was assessed by daily burden, Bigger's criteria, and Lown-Wolf classification. Clinical outcomes, including arrhythmic and non-arrhythmic adverse events, were also recorded.

Results: At baseline, VEA was detected in 74–85% of patients across groups, without significant differences. After 1 year, the CR group demonstrated a significant reduction in VEA burden (36% decrease, p=0.048), fewer patients with >10 ectopic beats/hour (26.5% vs. 35.4%, p<0.001), and lower incidence of non-sustained ventricular tachycardia (NSVT, p=0.018) compared with IR and NR groups. Favorable shifts in Lown-Wolf classes were also observed, with an increased proportion of patients without VEA. In contrast, IR and NR groups showed persistent or worsening VEA, including higher rates of frequent ectopy and NSVT. The NR group exhibited a significant 1.5-fold increase in daily ectopy burden (p=0.015).

Conclusion: Complete coronary revascularization by stenting is associated with significant reduction in VEA frequency and severity in post-MI patients with preserved and mildly reduced LVEF. Incomplete or absent revascularization fails to provide such benefits and may be linked to progression of arrhythmogenic activity. Achieving complete revascularization should be considered an important strategy for arrhythmia prevention in this patient population.

Keywords: ventricular ectopic activity, myocardial infarction, percutaneous coronary intervention, revascularization, sudden cardiac death, Holter monitoring, coronary artery disease.

Introduction

Despite significant advancements in medicine, myocardial infarction (MI) remains one of the leading causes of morbidity and mortality among non-communicable diseases. One of the most dangerous complications in the post-infarction period is ventricular arrhythmias (VA) due to scar tissue formation [1,2]. In these patients, VA may present as life-threatening arrhythmias such as frequent and polymorphic ventricular ectopic activities (VEAs), non-sustained ventricular tachycardia (NSVT), sustained ventricular tachycardia (VT), or ventricular fibrillation (VF). These arrhythmias are often linked to increased ventricular ectopic activity, which is considered a manifestation of myocardial electrical instability and an early predictor of sudden cardiac death (SCD) [1,3].

The development of SCD in the post-MI period results from complex interplay between scar-related fibrosis, myocardial remodeling, autonomic nervous system imbalance, and ongoing silent ischemia [4,5]. Fibrotic changes in the myocardium disrupt uniform electrical conduction, facilitating re-entry mechanisms and focal ectopic activity. Simultaneously, silent ischemia without clinical symptoms increases myocardial arrhythmogenicity [6].

In recent years, percutaneous coronary intervention (PCI) with stent implantation has revolutionized the treatment of ischemic heart disease, especially in ST-elevation (STEMI) and non-ST-elevation (NSTEMI) myocardial infarction. Beyond restoring perfusion, stenting may help stabilize the myocardial electrical substrate, thereby reducing electrical instability [7,8]. Large randomized clinical trials such as COMPLETE [9], SYNTAX [10], and REVIVED-BCIS2 [11] have shown that complete revascularization improves outcomes by reducing overall mortality, recurrent infarction, and heart failure progression.

Given that ventricular arrhythmias play a central role in the pathogenesis of SCD, the direct effect of complete versus incomplete stenting on VEA burden has not been sufficiently studied. The current literature contains limited data on this relationship. Modern clinical guidelines emphasize an individualized approach when assessing the risk of SCD in post-infarction patients. In patients with severely reduced left ventricular ejection fraction (LVEF <35%), diagnostic and treatment algorithms are well established [12]. However, in those with mildly reduced (40-49%) or preserved LVEF (>50%), echocardiographic parameters have limited predictive value. In these groups, VEAs and NSVT are considered sensitive electrophysiological markers [13].

Nevertheless, whether VEAs are independent, prognostically significant, and modifiable risk factors remains controversial. There is a lack of robust evidence about how mechanical interventions like stenting affect VEA frequency. Although the aforementioned major clinical trials demonstrated that complete revascularization improves cardiovascular outcomes, most did not evaluate arrhythmias as a specific endpoint. The impact of revascularization on changes in VEA frequency remains unclear. Based on pathophysiological mechanisms, it is hypothesized that eliminating ischemia could restore electrical stability, reduce ectopic focus activity, normalize repolarization gradients, and suppress arrhythmogenic mechanisms [14].

This study aims to fill this clinical and scientific gap by evaluating the impact of stent-based revascularization on ventricular ectopic activity in post-myocardial infarction patients with preserved or mildly reduced LVEF.

Materials and Methods

Study Design and Participants

A total of 270 patients aged 38 to 75 years with a history of myocardial infarction were enrolled in this prospective study. In the first stage (in-hospital phase), within the initial 24 hours, patients underwent collection of anamnesis and physical examination, general clinical, laboratory, and instrumental assessments, including 24-hour Holter ECG monitoring (HECGM) to evaluate ventricular ectopic activity.

Ventricular Ectopic Activity Assessment

Ventricular ectopic activity was assessed based on: (1) the total number of VEAs recorded per day, (2) the VEA burden, defined as the ratio of VEAs to total heartbeats in 24 hours, (3) the maximum Lown-Wolf class observed in each patient, (4) the presence of sustained or non-sustained ventricular tachycardia, and (5) life-threatening ventricular arrhythmias.

Sustained VT was defined as episodes lasting longer than 30 seconds with a heart rate >120 bpm, comprising at least 3 consecutive ventricular complexes, RR interval <600 ms, QRS duration >120 ms. Episodes with spontaneous termination within 30 seconds without intervention were considered non-sustained VT [15].

According to the Lown-Wolf classification, VEAs were categorized as follows: Class 0: no VEAs during 24-hour monitoring; Class 1: <30 VEAs per hour; Class 2: >30 VEAs per hour, monomorphic; Class 3: polymorphic isolated VEAs; Class 4A: paired VEAs; Class 4B: non-sustained or sustained VT; Class 5: R-on-T phenomenon.

Patient Grouping and Revascularization

Based on clinical findings, coronary angiography (CAG) was performed to assess the extent of coronary artery disease. According to the number of stenosed coronary vessels and whether coronary stenting was performed, patients were divided into three groups: Complete Revascularization (CR) Group (n=115), Incomplete Revascularization (IR) Group (n=94), and No Revascularization (NR) Group (n=61).

Ethical Considerations

Written informed consent was obtained from all patients prior to intervention. The study was conducted in accordance with the Declaration of Helsinki and approved by the institutional ethics committee. Diagnostic CAG and PCI with stenting were performed under standard therapeutic conditions [16]. After discharge, patients were provided with standard medical therapy.

Follow-up

One year after diagnostic CAG and/or PCI, patients underwent follow-up clinical evaluation and repeat 24-hour HECGM in outpatient settings. Primary and secondary endpoints such as sudden cardiac death/arrhythmic death, death from other causes, cardiovascular mortality, non-fatal myocardial infarction, coronary artery bypass grafting, and emergency stenting were assessed as adverse events at 1-year follow-up.

Statistical Analysis

Statistical analysis was conducted using IBM SPSS Statistics 29.0 software, with data collected and processed in Microsoft Excel 2010. Median (Me), lower quartile (Q1), and upper quartile (Q3) values were calculated. The distribution of variables within groups was assessed using the Kolmogorov-Smirnov test. For comparison of three or more groups with non-normal distribution, the Kruskal-Wallis test was applied. The Chi-squared (χ^2) test was used to evaluate qualitative variables between groups, and McNemar's test was used for within-group dynamic changes. Differences were considered statistically significant at $p<0.05$.

Results

Baseline Clinical Characteristics

When comparing clinical characteristics across the three groups (Table 1), no significant differences were found in terms of age, sex, body mass index, infarction localization, time since acute MI, indexed left ventricular mass, systolic and diastolic function, and in-hospital pharmacotherapy. Notably, one- and two-vessel disease was more prevalent in the CR group, while three-vessel disease was more common in the IR and NR groups, resulting in significant differences in SYNTAX scores.

Table 1: Clinical Characteristics of Patients

Parameter	*CR Group (n=115)*	*IR Group (n=94)*	*NR Group (n=61)*	*χ^2/F*	*p-value*
Age, years	60 [54; 65]	62 [58; 69.8]	64 [58; 68]	0.20	0.658
Sex				3.147	0.207
Male	96 (83.5%)	74 (78.7%)	44 (72.2%)		
Female	19 (16.5%)	20 (21.3%)	17 (27.8%)		
BMI, kg/m^2	28.3 [25; 31]	28.4 [26.6; 31.3]	29.7 [28; 33.7]	0.15	0.694
Normal weight, n (%)	12 (10.5%)	13 (13.8%)	8 (13.1%)	1.610	0.447
Period from acute MI to CAG, days	145 [67.5; 566]	245 [45; 700]	414 [379; 432]	5.488	0.055
Amount of atherosclerotic lesion vessels				66.883	<0.001
1 vessel	58 (50.4%)	30 (31.9%)	22 (36.1%)		
2 vessels	42 (36.5%)	64 (68.1%)	17 (27.8%)		
3 and more	15 (13.1%)	0 (0%)	22 (36.1%)		
Total number of atherosclerotic lesion vessels	1.63±0.70	2.68±0.46	1.96±0.89	86.423	<0.001
Rate revascularization, %	100%	50 [33.3; 66.7]	0%	-	-
SYNTAX Score	13 [8; 18]	19.5 [14; 25.3]	16.8 [9; 25.9]	7.112	0.032
LVEF, %	54 [47; 59.7]	53.9 [43.7; 56.4]	50.7 [45; 56.3]	0.544	0.661
iLVMM, g	109.3 [95; 127.4]	116.1 [100; 141.5]	115.7 [100.2; 154.5]	1.022	0.422
Medications					
Beta blocker	112 (97.5%)	94 (100%)	57 (93.4%)	4.982	0.083
ACE/ARB	56 (59.6%)	62 (66.0%)	37 (60.7%)	0.323	0.851
Calcium antagonists	70 (61.0%)	50 (53.2%)	31 (50.8%)	1.234	0.540
ARNI	21 (18.3%)	20 (21.3%)	19 (31.2%)	1.883	0.390
Nitrates	29 (25.2%)	28 (28.5%)	21 (34.4%)	0.587	0.746
Amiodarone	11 (9.5%)	8 (8.5%)	7 (11.5%)	0.148	0.929
AMR	53 (46.0%)	43 (45.7%)	36 (59.1%)	0.453	0.656
Statin	112 (97.5%)	94 (95.9%)	60 (98.3%)	2.528	0.283
Aspirin	115 (100%)	94 (100%)	59 (96.7%)	0.941	0.625
Clopidogrel	111 (96.6%)	90 (95.8%)	59 (96.7%)	8.508	0.014

BMI: body mass index; MI: myocardial infarction; CAG: coronary angiography; LVEF: left ventricular ejection fraction; iLVMM: indexed left ventricular myocardial mass; ACE: angiotensin-converting enzyme; ARB: angiotensin receptor blocker; ARNI: angiotensin receptor-neprilysin inhibitor; AMR: antimineralocorticoid receptor

Table 2: Frequency and Structure of Initial Ventricular Ectopic Activity Based on Holter Monitoring

Parameter	*CR Group (n=115)*	*IR Group (n=94)*	*NR Group (n=61)*	χ^2	*p-value*
Density of VEA, %	0.22 [0.03; 7.6]	0.22 [0.05; 5.6]	0.23 [0.04; 3.85]	2.305	0.305
Frequently (>10/hour) VEA (Bigger), n (%)	43 (37.4%)	47 (50.0%)	25 (41.0%)	2.109	0.348
Maximal registered VEA class in 1 patient (Lown-Wolf), n (%)					
Class 0	26 (22.8%)	16 (18.1%)	10 (16.4%)	1.750	0.417
Class I	88 (77.2%)	78 (81.9%)	47 (83.6%)		
Class II	24 (20.8%)	23 (24.4%)	18 (29.5%)	1.316	0.518
Class III	21 (18.3%)	18 (19.2%)	11 (18.0%)	0.199	0.905
Class IVA	31 (27.0%)	26 (27.6%)	20 (32.9%)	0.236	0.889
Class IVB (NSVT)	9 (7.8%)	11 (11.7%)	11 (18.0%)	5.304	0.071

VEA: ventricular ectopic activity; NSVT: non-sustained ventricular tachycardia. Values represent maximum class observed per patient during 24-hour monitoring.

Baseline Ventricular Ectopic Activity

The structural distribution of ventricular ectopic activity recorded during 24-hour Holter monitoring showed a high prevalence of VEA across all groups (Table 2). In the CR group, VEA was detected in 88 patients (77.2%), including Lown-Wolf class III in 21 patients (18.3%), class IVA in 31 (27.0%), class IVB (NSVT) in 9 (7.8%), and frequent polymorphic and/or paired PVCs without NSVT in 61 patients (53.1%). Quantitatively, using the Bigger criteria, frequent VEA (>10/hour) was identified in 43 patients (37.4%), and >30/hour based on Lown-Wolf class II in 24 patients (20.8%).

In the IR group, VEA was observed in 78 patients (81.9%), including Lown-Wolf class III in 18 (19.2%), class IVA in 26 (27.6%), NSVT in 11 (11.7%), and frequent PVCs without NSVT in 55 (58.3%). According to the Bigger criteria, >10/hour VEA was found in 47 patients (50.0%), and >30/hour in 23 patients (24.4%).

In the NR group, VEA was present in 47 patients (83.6%), including Lown-Wolf class III in 11 (18.0%), class IVA in 20 (32.9%), NSVT in 11 (18.0%), and frequent PVCs without NSVT in 40 patients (65.5%). Based on the Bigger criteria, >10/hour VEA was seen in 25 patients (41.0%), and >30/hour in 18 patients (29.5%).

Despite observable differences in VEA indicators among the groups, these differences did not reach statistical significance.

One-Year Follow-up Results

At one-year follow-up, 235 patients completed evaluation (CR: n=110, IR: n=80, NR: n=45). Significant intergroup differences were observed in quantitative indicators of ventricular ectopic activity (Table 3).

In the CR group, the proportion of patients without ventricular ectopic activity was 30 (28.2%), with daily VE burden of 0.14 [0.03; 4.9]. High-grade Lown-Wolf classifications were recorded as: class III in 23 patients (20.9%), class IVA in 19 patients (17.3%), and NSVT in 4 patients (3.6%).

In the IR group, the proportion of patients without ventricular arrhythmias was 8 (10.0%), with daily VE burden of 0.26 [0.07; 4.8]. Lown-Wolf classifications were: class III in 14 patients (17.5%), class IVA in 24 patients (30.0%), and NSVT in 10 patients (12.5%).

In the NR group, the number of patients without VE activity was 5 (11.1%), with daily VE burden of 0.35 [0.03; 5.3], and the Lown-Wolf distribution was: class III in 10 patients (22.2%), class IVA in 9 patients (20.0%), and NSVT in 7 patients (15.6%).

Dynamic Changes in Ventricular Ectopic Activity

When comparing results one year after intervention, the CR group demonstrated statistically significant improvements in multiple parameters (Table 4). There was a 36% decrease in daily VEA burden ($p=0.048$), a 28.5% reduction in episodes >10/hour based on Bigger's criteria ($p<0.001$), and a significant reduction in NSVT incidence. This positive trend was accompanied by favorable shifts in the Lown-Wolf classification, with reduction in higher-grade ectopy and an increase in the proportion of patients with low-grade VEA. The percentage of patients in whom ectopy completely resolved increased by 4.9% ($p<0.001$).

In the IR group, a non-significant increase in the frequency of ventricular ectopy was observed - daily burden increased by 24%, and both paired/polymorphic VEA and episodes >10/hour per Bigger's criteria showed slight non-significant growth. The proportion of patients with newly developed ventricular ectopy increased by 5.9% ($p<0.001$).

In the NR group, clear and statistically significant deterioration was noted: daily burden of ventricular ectopy increased 1.5-fold ($p=0.015$), frequency of >10/hour episodes increased by 16.7% ($p=0.025$), and paired/polymorphic VEA nearly doubled ($p<0.001$). The proportion of patients with newly developed ectopy increased by 6.6% ($p=0.006$).

Table 3: Frequency and Structure of Ventricular Ectopic Activity Based on 24-hour Holter Monitoring One Year After Intervention

Parameter	*CR Group (n=110)*	*IR Group (n=80)*	*NR Group (n=45)*	χ^2	*p-value*
Density of VEA, %	0.14 [0.03; 4.9]	0.26 [0.07; 4.8]	0.35 [0.03; 5.3]	8.729	0.013
Frequently (>10/hour) VEA (Bigger), n (%)	29 (26.5%)	38 (47.5%)	21 (46.7%)	7.336	0.026
Maximal registered VEA class in 1 patient (Lown-Wolf), n (%)					
Class 0	30 (28.2%)	8 (10.0%)	5 (11.1%)	8.331	0.018
Class I	80 (71.8%)	72 (90.0%)	40 (88.9%)		
Class II	21 (19.1%)	22 (27.5%)	13 (28.9%)	3.282	0.194
Class III	23 (20.9%)	14 (17.5%)	10 (22.2%)	0.231	0.891
Class IVA	19 (17.3%)	24 (30.0%)	9 (20.0%)	5.422	0.066
Class IVB (NSVT)	4 (3.6%)	10 (12.5%)	7 (15.6%)	8.023	0.018
Maximal registered VEA class (Lown-Wolf), n (%)					
Class 0	31 (28.2%)	8 (10.0%)	5 (11.1%)	8.331	0.016
Class I	33 (30.0%)	24 (30.0%)	14 (31.1%)	9.225	0.010
Class III	23 (20.9%)	14 (17.5%)	10 (22.2%)	0.231	0.891
Class IVA	19 (17.3%)	24 (30.0%)	9 (20.0%)	5.422	0.066
Class IVB (NSVT)	4 (3.6%)	10 (12.5%)	7 (15.6%)	8.023	0.018

Table 4: Changes in the Frequency and Structure of Ventricular Ectopic Activity on Holter ECG Monitoring in Patient Groups After 1 Year

Parameter	*CR Group (n=110)*			*IR Group (n=80)*			*NR Group (n=45)*		
	Initial	*After 1 year*	*p*	*Initial*	*After 1 year*	*p*	*Initial*	*After 1 year*	*p*
Density of VEA, %	0.22 [0.03; 7.6]	0.14 [0.03; 4.9]	0.048	0.21 [0.05; 5.4]	0.26 [0.07; 4.8]	0.875	0.23 [0.04; 3.82]	0.35 [0.03; 5.3]	0.015
Frequently (>10/hour) VEA (Bigger), n (%)	39 (35.4%)	29 (26.5%)	<0.001	41 (51.3%)	38 (47.5%)	0.683	17 (40.0%)	21 (46.7%)	0.025
Maximal registered VEA class in 1 patient (Lown-Wolf), n (%)									
Class 0	26 (23.6%)	30 (28.2%)	0.063	12 (15.0%)	8 (10.0%)	0.092	8 (17.7%)	5 (11.1%)	0.006
Class I	84 (76.4%)	80 (71.8%)	<0.001	68 (85.0%)	72 (90.0%)	<0.001	37 (82.2%)	40 (88.9%)	0.001
Class II	21 (19.1%)	21 (19.1%)	0.383	19 (23.8%)	22 (27.5%)	0.508	14 (31.1%)	13 (28.9%)	0.727
Class III	49 (44.5%)	29 (26.4%)	<0.001	43 (53.8%)	24 (30.0%)	0.481	21 (46.7%)	17 (37.8%)	0.424
Class IVA	34 (30.9%)	21 (19.1%)	0.090	27 (33.8%)	28 (35.0%)	0.786	17 (37.8%)	16 (35.6%)	0.836
Class IVB (NSVT)	7 (6.4%)	4 (3.6%)	0.109	10 (12.5%)	10 (12.5%)	0.625	4 (8.9%)	7 (15.6%)	<0.001
Maximal registered VEA class (Lown-Wolf), n (%)									
Class 0	26 (23.6%)	31 (28.2%)	0.063	12 (15.0%)	8 (10.0%)	0.092	8 (17.7%)	5 (11.1%)	0.006
Class I	28 (25.5%)	33 (30.0%)	0.072	18 (22.5%)	24 (30.0%)	0.093	7 (15.5%)	14 (31.1%)	0.032
Class III	21 (19.0%)	23 (20.9%)	0.359	18 (22.5%)	14 (17.5%)	0.214	11 (24.5%)	10 (22.2%)	0.827
Class IVA	28 (25.5%)	19 (17.3%)	0.093	22 (27.5%)	24 (30.0%)	0.146	15 (33.3%)	9 (20.0%)	0.058
Class IVB (NSVT)	7 (6.4%)	4 (3.6%)	0.109	10 (12.5%)	10 (12.5%)	0.625	4 (8.9%)	7 (15.6%)	<0.001

VEA: ventricular ectopic activity; NSVT: non-sustained ventricular tachycardia. Statistical analysis performed using McNemar's test for paired comparisons.

Discussion

Cardiac rhythm disturbances, including ventricular ectopic activity (VEA), are among the most frequently observed complications in patients who have experienced myocardial infarction (MI). These arrhythmias are often associated with electrical instability in ischemic zones, neurohormonal imbalance, and impaired repolarization processes. In our study, VEA was recorded in 74% to 85% of patients across the groups. High-grade VEA, according to the Lown-Wolf classification, was observed in 53.1% to 65.5% of patients, and non-sustained ventricular tachycardia (NSVT) was detected in approximately one out of every nine patients—clearly supporting our findings [1,2,3,4].

This study assessed the impact of complete and incomplete revascularization on VEA in the post-MI period. Our results indicate that complete revascularization (CR) had a favorable effect on both the qualitative and quantitative parameters of VEA. This is likely due to full restoration of myocardial perfusion, stabilization of repolarization in ischemic regions, and reduction of structural and morphological factors that contribute to ectopic foci activation. Additionally, this group showed improvements in echocardiographic parameters, including enhanced myocardial systolic function. These findings are consistent with several recent studies that have also shown reduced arrhythmogenic activity following complete revascularization [7,8,17].

In contrast, the incomplete revascularization (IR) and no revascularization (NR) groups continued to exhibit high levels of VEA. In both groups, the persistence of ischemia-prone myocardial tissue and electrical instability were key contributors to the ongoing ectopic activity. Particularly in the IR group, a predominance of triple-vessel disease and high SYNTAX scores indicated more extensive ischemic burden.

In the NR group, the elevated VEA rates were additionally associated with chronic hypoperfusion, progressive remodeling, and fibrosis in ischemic zones.

When compared with existing literature, our findings align with several studies that have demonstrated reduced arrhythmogenic activity in patients undergoing complete revascularization [8,12,15]. While some studies have concluded that incomplete revascularization has no significant clinical impact, many of them did not specifically analyze VEA as an independent outcome [11,18].

Thus, the results of this study confirm the preventive role of complete anatomical revascularization in reducing VEA, which may subsequently improve overall prognosis and quality of life in post-infarction patients by lowering the risk of arrhythmias.

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