International Journal of Biomedicine 5(4) (2015) 195-197
doi: 10.21103/Article5(4)_OA5

ORIGINAL ARTICLE

Cardiology

INTERNATIONAL JOURNAL OF BIOMEDICINE

Main Predictors of Sudden Cardiac Death in Patients with Q-Wave Myocardial Infarction

Ravshanbek D Kurbanov, PhD, ScD; Guzal U. Mullabaeva, PhD; Anvar A Kilichev

The Republican Specialized Center of Cardiology, Tashkent, Uzbekistan

Abstract

The study included 131 patients (mean age 51.9±9.13 year) with Q-wave myocardial infarction (Q-MI). All patients underwent echocardiography and 24-hour ECG monitoring on the 10th through the 14th days of MI. Treatment included thrombolytic therapy, early administration of beta-blockers, antiplatelet agents, anticoagulants, statins, ACE inhibitors, if needed - antiarrhythmics and aldosterone antagonists. Follow-up was 24 months. During the observation period, of the 131 study patients 17(13.0%) died suddenly. Our study suggests that the high risk of sudden cardiac death (in the first 2 years after MI) in patients with Q-MI is associated with anterior localization, early pathological left ventricular remodeling, low myocardial contractility, and development of AHF high Killip classes in the early period of MI, as well as the identification of high heart rate at rest, frequent premature ventricular contractions (mainly polymorphic), systolic dysfunction in the early stages of observation (on the 10th through the 14th days), and older age of patients. **(Int J Biomed. 2015;5(4):195-197.)**

Keywords: *sudden cardiac death; myocardial infarction; premature ventricular contractions; 24-hour ECG monitoring.*

Introduction

The problem of sudden cardiac death (SCD) is particularly relevant in patients after a myocardial infarction (post-MI patients), since this patient population is particularly vulnerable to the development of fatal ventricular rhythm disorders [1-3]. During the first year after MI, 3% to 6% of patients die, the majority of them suddenly [4]. Because this event develops very quickly, within an hour of the onset of symptoms, and treatment interventions are often too late, the main approach to solving this problem is the prevention of SCD. Premature ventricular contractions (PVCs) are one of the predictors of SCD [5,6].

The aim of the study was to evaluate the incidence of PVCs at the 10th through the 14th days after Q-wave myocardial infarction (Q-MI) and its relationship with the SCD development.

Materials and Methods

We examined 131 (mean age 51.9±9.13 years) male patients with primary Q-MI. The study was approved by the Republican Specialized Center of Cardiology Ethics Committee. Written *i*nformed consent was obtained from each patient. The treatment of acute MI was carried out in accordance with recommendations for the Management of Patients with ST-Elevation Myocardial Infarction and included thrombolytic therapy, early administration of beta-blockers, antiplatelet agents, anticoagulants, nitrates, statins, ACE inhibitors, if needed - antiarrhythmics and aldosterone antagonists.

All patients underwent echocardiography and 24-hour ECG monitoring on the 10th through the 14th days of MI. To characterize the premature ventricular contractions (PVCs), the B. Lown and M. Wolf classification (1971) and the prognostic classification of J. Bigger (1984) were used. Hourly qualitative and quantitative assessment of PVCs was performed in accordance with the Lown-Wolf gradation: Class 0 – absence of PVCs, Class I – rare monomorphic PVCs; Class II – frequent single PVCs; Class III – polymorphic (polytopic) PVCs; Class IVA – paired PVCs; Class IVB – group PVCs; Class V – early PVCs, R/T phenomena. After MI, according to the J. Bigger classification, PVCs>10 per hour, pair and group PVCs are potentially hazardous ventricular arrhythmias. Follow-up was 24 months.

The obtained data were processed using computer software Microsoft Excel, STATISTICA 6 and Biostat. *T*he mean (M) and standard deviation (SD) were calculated. The odds ratio (OR) and 95% confidence interval (95% CI) were calculated using logistic regression. Group comparisons with

respect to categorical variables are performed using chi-square tests or, alternatively, Fisher's exact test when expected cell counts were less than 5. Correlations were examined using regression analysis and Spearman rank correlation coefficient. A probability value of $P<0.05$ was considered statistically significant.

Results and Discussion

During the observation period, of the 131 study *patients* 17/13.0% died suddenly. In particular, 8/47.1% patients died in the first 6 months, 5/29.4% during the first year, and 4/23.5% patients died after 1 year from the onset of the disease. Our data are consistent with the view of S.H. Hohnloser [7] that 10% to 20% of patients die within one year, while more than 50% of deaths occur within the first 3 to 6 months after acute MI. Clinical characteristics of the dead (Group 1) and surviving (Group 2) patients are presented in Table 1.

Table 1.

Baseline clinical characteristics of patients

Variable	Patients (n)	Group 1 (n=17)	Group 2 (n=114)	OR	95% CI	*P*
Age, year		52.6±9.2	51.7±0.14			*0.71*
Anterior MI	72	15/88.2%	67/58.8%	5.26	1.15-24.1	*0.04*
Inferior MI	49	2/11.8%	47/41.2%			
DM	10	3/17.6%	7/6.1%	3.28	0.76-14.1	*0.24*
EPIA	52	8/47.1%	44/38.6%	1.41	0.51-3.94	*0.69*
AH	111	12/70.6%	99/86.8%	0.36	0.11-1.18	*0.17*
Aneurism	43	11/64.7%	32/28.1%	4.70	1.60-13.8	*0.006*
Thrombolytic	16	2/11.8%	14/12.3%	0.95	0.20-4.67	*0.74*
AHF Killip I-II	77	13/76.5%	64/56.1%	2.54	0.78-8.26	*0.16*
AHF Killip III-IV	10	4/23.5%	6/5.3%	5.54	1.38-22.2	*0.03*
LVEF,%		39.6±8.8	49.2±12.3			*0.002*
LVEF<50%	77	15/88.2	62/54.4	6.29	1.38-28.8	*0.02*

Polymorphic (polytopic) PVCs and frequent monomorphic PVCs were detected more frequently in Group 1 compared to Group 2: 58.8% vs. 13.2% (OR=9.43; 95% CI: 3.11-28.6; *P<0.0001*) and 35.3% vs. 12.3% (OR=3.90; 95% CI: 1.25-12.2; *P=0.04*), respectively, (Fig. 1).

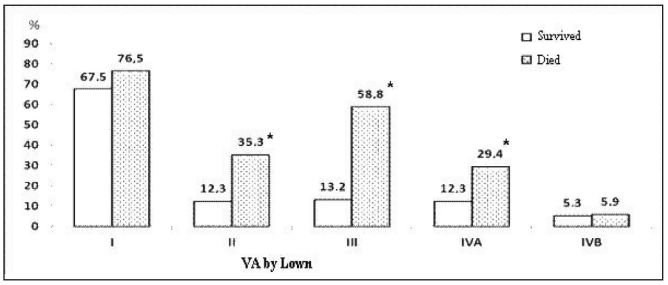

Fig. 1. *The frequency of PVCs in the two groups*

It should be noted that other high-grade PVCs were found more frequently in Group 1 than in Group 2 (OR=2.56; 95% CI 0.85-7.75; *P=0.17*), but the differences did not reach a statistically significant level. In Group 1, we revealed a positive correlation between age and PVCs (*r=0.74; P=0.001*), heart rate (HR) per min (*r=0.84; P=0.0001*), PVCs>10/hour (*r=0.34; P=0.04*), and frequent PVCs (*r=0.82; P=0.0001*). A negative correlation was found between the left ventricular ejection fraction (LVEF) and PVCs (*r=-0.51; P=0.04*), HR per min (*r=-0.70; P=0.002*), PVCs>10/h (*r=-0.57; P=0.04*), and frequent PVCs (*r=-0.46; P=0.05*) (Fig. 2). Significant correlations in Group 2 were not found.

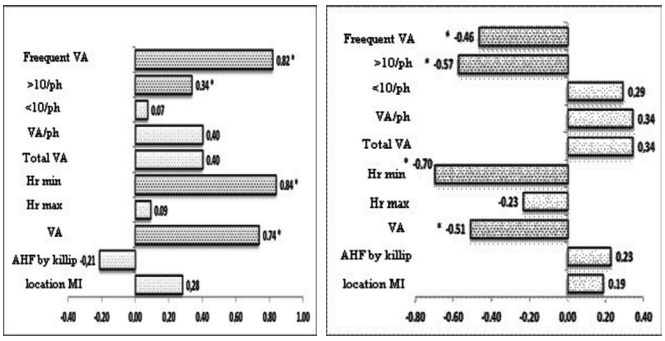

Fig. 2. *Correlation analysis between age, LVEF and indicators of VAs in Group 1*

Many studies have shown that the main electrophysiological causes of SCD are malignant rhythm disorders [8,9]. H.J. Trappe, P. Brugada et al. [10], based on the results of observations of 200 patients who underwent MI complicated with VT or VF, concluded that these types of arrhythmias can be considered as prognostically life-threatening only in cases when they are accompanied by loss of consciousness and have occurred within the first 2 months after MI onset. The mortality among these patients reaches 83% [9]. However, some experts are showing less confidence in assessing the value of PVCS as an independent factor determining the prognosis of patients with MI, considering that the survival of patients is also dependent on the state of LV and condition of the coronary arteries. Based on the above data, the aim of this study was to assess the detectability of PVCS on the 10th through the 14th days of Q-MI and analyze the survival of these patients during 24 months of follow-up. So, by the end of the observation period, out of 131 patients 114 (87.0%) survived and SCD appeared in 13.0% of cases, with most of the deaths occurring in the first year (76%). So far, there is no consensus about the prognostic significance of localization of MI. Currently, it is known that anterior localization of MI makes a significant contribution to CHF development and the frequency of re-infarction [11], but the issue about SCD is still debated. In our study, anterior Q-MI and complications such as heart failure and LV aneurysm were found significantly more often in the Group 1 patients with SCD. Absence of differences in the incidence of early post-infarction angina in the two groups was not consistent with the opinion of other researchers that the presence of residual ischemia often predicts the risk of re-infarction [4,6]. In our study, we found that resting HR was significantly higher in Group 1 compared to Group 2 (72.6±8.30 vs. 64.5±12.1, *P=0.009*). The analysis of the detectability of PVCS in the two groups showed that groups

were initially different in detection of PVCs. This corresponds to the data of GISSI-2 [12], namely, the presence of more than 10 PVBs per hour or of complex ventricular arrhythmias was significantly associated with a higher mortality risk regardless of the presence of LV dysfunction. Meanwhile, in our study the initially more expressed systolic dysfunction was detected in Group 1. In particular, LVEF was 39.6±8.81% in Group 1 vs. 49.2±12.3% in Group 2 (P=0.002). The number of patients with LVEF<50% was 88.2% in Group 1 vs. 54.4% in Group 2 (*P=0.02*). The correlation analysis also showed a strong negative relationship between LVEF and PVC frequency. According to many researchers, older age is one of the factors that determine SCD risk in post-MI patients [11]. In our study, we did not observe significant differences between the study groups, but the correlation analysis revealed a positive relationship between age and the frequency of PVCs and HR only in Group 1.

In conclusion: Based on our data it can be argued that PVCS cannot be regarded as an independent predictor of SCD. Our study suggests that the high risk of SCD (in the first 2 years after MI) in patients with Q-MI is associated with anterior localization, early pathological LV remodeling, low myocardial contractility, and development of AHF high Killip classes in the early period of MI, as well as the identification of high HR at rest, frequent PVCs (mainly polymorphic), systolic dysfunction in the early stages of observation (on the 10^{th} through the 14th days), and older age of patients.

Competing interests

The authors declare that they have no competing interests.

References

1. Bokeria OL, Akhobekov AA. Sudden cardiac death: mechanisms and risk stratification. Ann Aritm 2012; 3:5-13. [Article in Russian].
2. Bokeria LA, Bokeria OL, Kirtbaya LN. Heart failure and sudden cardiac death. Ann Aritm 2009; 4:7-20. [Article in Russian].
3. Bokeria LA, Revishvili AS. Sudden cardiac death. M.: GEO-TAP-Media; 2011. [Manual in Russian].
4. Boldueva SA, Shabrov AV, Lebedev DS, Burak Tatyana Leonova IA, Samohvalova MV, Beetle VS, Bykov EG. Prediction and prevention of sudden cardiac death in patients with myocardial infarction. Kardiov Ter Prof 2008; 7(3):56-62. [Article in Russian].
5. Kurbanov RD. Ventricular arrhythmias. Tashkent; 2012. [Monograph in Russian].
6. Kurbanov RD, Kiyakbaev GK. Myocardial infarction. Life expectancy. Tashkent; 2001. [Monograph in Russian].
7. Goldstein S, Medendorp SV, Landis JR, Wolfe RA, LeigttonR, Ritter G, et al. Analysis of cardiac symptoms preceding cardiac arrest. Am J Cardiol 1986; 58(13):1195-8.
8. Epstein AE, DiMarco JP, Ellenbogen KA, Estes NA 3rd, Freedman RA, Gettes LS, et al. ACC/AHA/HRS 2008 Guidelines for Device-Based Therapy of Cardiac Rhythm Abnormalities: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the ACC/AHA/NASPE 2002 Guideline Update for Implantation of Cardiac Pacemakers and Antiarrhythmia Devices): developed in collaboration with the American Association for Thoracic Surgery and Society of Thoracic Surgeons. Circulation 2008; 117(21):e350-408.
9. Goldberger JJ, Buxton AE, Cain M, Costantini O, Exner DV, Knight BP, et al. Risk stratification for arrhythmic sudden cardiac death: identifying the roadblocks. Circulation 2011; 123(21): 2423-30.
10. Trappe HJ, Brugada P, Talajic M, Lezaun R, Wellens HJ. Ventricular tachycardia and ventricular fibrillation following myocardial infarct: determinants of prognosis and disease course. Z Kardiol 1988; 77(11):687-95. [Article in German].
11. Stecker EC, Vickers C, Waltz J, Socoteanu C, John BT, Mariani R, et al. Population-based analysis of sudden cardiac death with and without left ventricular systolic dysfunction: two-year findings from the Oregon Sudden Unexpected Death Study. J Am Coll Cardiol 2006; 47(6):1161-6.
12. Maggioni AP, Zuanetti G, Franzosi MG, Rovelli F, Santoro E, Staszewsky L, et al. Prevalence and prognostic significance of ventricular arrhythmias after acute myocardial infarction in the fibrinolytic era. GISSI-2 results. Circulation 1993; 87:312–22.