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

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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 informed 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. The 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.**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group 1 (n=17)</th>
<th>Group 2 (n=114)</th>
<th>OR</th>
<th>95% CI</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, year</td>
<td>52.6±9.2</td>
<td>51.7±0.14</td>
<td></td>
<td></td>
<td>0.71</td>
</tr>
<tr>
<td>Anterior MI</td>
<td>72/1588.2%</td>
<td>67/58.8%</td>
<td>5.26</td>
<td>1.15-24.1</td>
<td>0.04</td>
</tr>
<tr>
<td>Inferior MI</td>
<td>49/2/11.8%</td>
<td>47/41.2%</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DM</td>
<td>10/3/17.6%</td>
<td>7/6.1%</td>
<td>3.28</td>
<td>0.76-14.1</td>
<td>0.24</td>
</tr>
<tr>
<td>EPIA</td>
<td>8/47.1%</td>
<td>44/38.6%</td>
<td>1.41</td>
<td>0.51-3.94</td>
<td>0.69</td>
</tr>
<tr>
<td>AH</td>
<td>111/1270.6%</td>
<td>99/86.8%</td>
<td>0.36</td>
<td>0.11-1.18</td>
<td>0.17</td>
</tr>
<tr>
<td>Aneurism</td>
<td>43/1164.7%</td>
<td>32/28.1%</td>
<td>4.70</td>
<td>1.60-13.8</td>
<td>0.006</td>
</tr>
<tr>
<td>Thrombotic</td>
<td>16/2/11.8%</td>
<td>14/12.3%</td>
<td>0.95</td>
<td>0.20-4.67</td>
<td>0.74</td>
</tr>
<tr>
<td>AHF Killip I-II</td>
<td>77/1376.5%</td>
<td>64/56.1%</td>
<td>2.54</td>
<td>0.78-8.26</td>
<td>0.16</td>
</tr>
<tr>
<td>AHF Killip III-IV</td>
<td>10/423.5%</td>
<td>6/5.3%</td>
<td>5.54</td>
<td>1.38-22.2</td>
<td>0.03</td>
</tr>
<tr>
<td>LVEF,%</td>
<td>39.6±8.8</td>
<td>49.2±12.3</td>
<td></td>
<td></td>
<td>0.002</td>
</tr>
<tr>
<td>LVEF&lt;50%</td>
<td>77/1588.2%</td>
<td>62/54.4%</td>
<td>6.29</td>
<td>1.38-28.8</td>
<td>0.02</td>
</tr>
</tbody>
</table>

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).

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 10th through the 14th days), and older age of patients.

**Competing interests**

The authors declare that they have no competing interests.

**References**


