Factors influencing survival of patient with in-hospital ventricular fibrillation: experience of a single center

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Objective To evaluate the factors influencing the outcome of patients who suffered in-hospital ventricular fibrillation (IHVF).

Methods Data of patients with IHVF in a single center were collected. Clinical characteristics of patients were compared between those survived (n=112) and those died (n=94), and those with IHVF occurred in inpatient ward and in emergency center. Multiple logistic regression analysis was used to identify factors associated with survival. Results There were 206 events in the analysis. The most common underlying disease was coronary artery disease (CAD), especially acute myocardial infarction (AMI). On multiple logistic regression analysis, independent predictors for failure to survive were higher NYHA class (odds ratio 1.7, 95% CI, 1.3-2.2, P < 0.001), lower serum potassium concentration ([K+] ) (odds ratio, 2.9, 95% CI, 1.9-4.3, P=0.007) and adrenaline usage (odds ratio, 25, 95% CI 11.5-55.1, P < 0.001). Emergency group have better NYHA class (P = 0.012), lower [K+] (P < 0.001) than in inpatient ward group. Hypokalemia (serum potassium level <4.5 mmol/L) was found in all patients with AMI in emergency group. In AMI sub-group, 56.9% of IHVF events occurred within the first day after AMI, and decreasing within 2 weeks. Patients with right coronary artery as infarction related artery (IRA) often (8/9, 88.9%) had bradycardia (R-R interval > 1s) before the occurrence of IHVF, while those with left anterior descending artery as IRA often showed tachycardia (R-R interval < 0.6s) (8/12, 66.7%). Conclusion The most common disease causing IHVF is CAD. Keeping [K+] above 4.5mmol/l could prevent on-setting IHVF, especially to AMI patients. The worse heart function is associated with higher rate of IHVF and worse prognosis (J Geriatr Cardiol 2010; 7:21-24).

Key words in-hospital ventricular fibrillation; heart function; hypokalemia

Introduction

Sudden cardiac arrest remains a major public health problem. Despite advances in emergency medical systems, only 3%-10% of patients who have cardiac arrest are successfully resuscitated. The majority of sudden cardiac arrest are caused by ventricular fibrillation (VF). Even in hospital, VF is an type of arrhythmia with high mortality rate. The data of VF occurred out of hospital had been reported by many studies, but few concerning VF happened in hospital. The purpose of this article is to analyze the data of patients suffered in-hospital VF (IHVF), in order to evaluate the relationship between the clinic characteristics and the outcomes.

Methods

Study population Case subjects with diagnosed IHVF were enrolled at our hospital. Clinic data were recorded, such as gender, age, main disease, complicating disease. NYHA class, left ventricular ejection fraction (LVEF), left ventricular ending diastolic diameter (LVEDD), serum potassium concentration ([K+] ) and lipoprotein concentration, smoke history, IHVF on-setting time, adrenaline usage, ECG, and coronary angiography. Those subjects with VF occurred prior to hospital admission were excluded.

Definition and groups IHVF means VF onset in inpatient ward or in the emergency room. The successful cardioversion was defined as absence of reoccurred VF within 24 hours of the resuscitation. All subjects were divided into successful resuscitated group (successful group) and failure resuscitated group (failure group). In the acute myocardial infarction (AMI) subgroup, those subjects with VF during AMI were divided into left anterior descending (LAD) group and right coronary artery (RCA) group, according to infarction related artery (IRA).

Statistical analysis Mean values were expressed as mean ± SEM. Compari-
son of discrete variables between groups were made by Fisher exact analysis, and continuous variables by Student’s t test. Stepwise Logistic regression analysis was used to determine the prognostic factors of the defibrillation. All tests presented in this report were two-tailed; a P value less than 0.05 was defined as statistical significance (SPSS version 10.0, SPSS Inc.).

Results

A total of 201 patients (132 males, mean age 58.1±14.2 years) were recruited in the analysis, with 206 VF events were recorded, in which 5 patients had other 5 VF events in different admission.

The underlying diseases were, in descending order, coronary artery disease (CAD) (137/206, 66.5%), valvular heart disease (23/206, 11.2%), myocardiopathy (19/206, 9.2%), congenital heart disease (6/206, 2.9%), isolated ventricular arrhythmia (6/206, 2.9%), pulmonary artery hypertension (4/206, 1.9%), acute myocarditis (3/206, 1.5%), and other heart disease (7/206, 3.4%). There were 28 subjects (28/206, 13.6%) with heart function NYHA class I, 23 subjects (23/28, 82.1%) were defibrillated successfully; 28 subjects (28/206, 13.6%) with NYHA class II, 19 subjects (19/28, 67.9%) defibrillated successfully; 27 subjects (27/206, 13.1%) with NYHA class III, 14 subjects (14/27, 51.9%) defibrillated successfully; 123 subjects (123/206, 59.7%) with NYHA class IV, 56 subjects (56/123, 45.5%) defibrillated successfully.

Of the 201 patients, 43 subjects (43/201, 21.4%) were complicated with diabetes mellitus, 77 (77/201, 38.3%) with hypertension, and 28 (28/201, 13.9%) with both. One hundred and two subjects (102/201, 50.7%) had smoke history.

In 206 episodes of IHVF, 112 were defibrillated successfully (successful group) and 94 failed (failure group). The subjects in the successful group had better NYHA class than those in the failure group; Mean [K+] was higher in successful group than those in failure group. There were more patients with hypokalemia and using adrenaline in resuscitation in successful group than those in failure group. Other values didn’t show any significant difference (see Table 1).

Multiple logistic regression analysis showed higher NYHA class (OR 1.7, 95% CI 1.3-2.2, P<0.001), without hypokalaisim before IHVF on-setting (OR 2.9, 95% CI 1.9-4.3, P=0.007), and using adrenaline (OR 25, 95% CI 11.5-55.1, P<0.001) are the predicatable factors for failure to defibrillation.

There were 47 IHVF episodes in emergency group. In inpatient ward group, there were 159 episodes. The majority of underlining diseases were CAD and AMI in emergency group, and more than in inpatient ward group (82.6% vs 62.6%, P=0.008; 73.9% vs 46.5%, P=0.001).

There were more subjects with NYHA class IV in inpatient ward group than those in emergency group (63.5% vs 46.8%, P=0.05). The patients in emergency group had better NYHA class than in inpatient ward group (II-III vs III-IV, P = 0.012). The subjects in emergency group had significant lower mean [K+] than that in inpatient ward group (3.49 vs 4.35 mmol/ L, P<0.01). And the proportion of subjects with abnormal [K+] before VF episode (< 3.5 mmol/ L or > 5.5 mmol/ L) was higher in emergency group than in inpatient ward group, (59.6% vs 27.7%, P< 0.01). Other data didn’t show any difference.

Of the 106 subjects in the AMI subgroup, there were 74 males and the average age was 63.1±11.9 years.. Mean NYHA class was III-IV and LVEF 44.4±12.4%. Total 109 IHVF episodes were recorded within 30 days after AMI; 35 happened in emergency room (group 1), and 74 happened in inpatient ward (group 2). The proportion of IHVF episodes with hypokalaisim ([K+] <3.5 mmol/ L) in group 1 was significantly higher than that in group 2 (65.7% vs 16.2%, P< 0.01). All 35 episodes occurred in emergency room were devided into 4 groups according to mean [K+] before IHVF on-setting: group A (<3.5mmol/L, n = 21), group B (3.5-4.0 mmol/ L, n = 10), group C (4.0-4.5 mmol/ L, n = 4), and group D (>4.5 mmol/ L, n = 0).

There were 65 episodes onset in first day after AMI, 21 within 7 days, 13 within 7-14 days, and 10 more than 2 weeks.

Table 1 The clinical features of survival group vs. failure group

<table>
<thead>
<tr>
<th></th>
<th>Survival group(n=112)</th>
<th>Failure group(n=94)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>56.8±13.5</td>
<td>59.7±14.9</td>
<td>0.142</td>
</tr>
<tr>
<td>Male (n, %)</td>
<td>75 (67)</td>
<td>58 (61.7)</td>
<td>0.467</td>
</tr>
<tr>
<td>Smoke history (n, %)</td>
<td>57(50.9)</td>
<td>77(50)</td>
<td>0.51</td>
</tr>
<tr>
<td>NYHA class</td>
<td>II-III</td>
<td>III-IV</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Diabetes (n, %)</td>
<td>29 (25.9)</td>
<td>14 (14.9)</td>
<td>0.038</td>
</tr>
<tr>
<td>Hypertension (n, %)</td>
<td>39(34.8)</td>
<td>38(40.4)</td>
<td>0.247</td>
</tr>
<tr>
<td>LDL-C (mmol/L)</td>
<td>2.58±0.75</td>
<td>2.60±0.73</td>
<td>0.877</td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>46.2±13.4</td>
<td>43±13.9</td>
<td>0.104</td>
</tr>
<tr>
<td>LVEDD (mm)</td>
<td>55.6±9.2</td>
<td>57.6±13.2</td>
<td>0.239</td>
</tr>
<tr>
<td>serum <a href="mmol/L">K+</a></td>
<td>3.83±0.73</td>
<td>4.53±0.91</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>[K+]&lt;3.5mmol/L (n, %)</td>
<td>36 (34.6)</td>
<td>13 (16.5)</td>
<td>0.007</td>
</tr>
<tr>
<td>Adrenaline use (n, %)</td>
<td>28 (25)</td>
<td>84 (89.4)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>
seventy-one percent episodes happened in the first day were defibrillated successfully, 42.9% within 7 day, 23.1% within 7-14 days, and 60% after 2 weeks. The defibrillation rate descended with time within the first 2 weeks after initial symptom of AMI.

In the AMI subgroup, there were 50 subjects undergone coronary angiography, 22 subjects had left LAD artery as IRA, 24 had RCA as IRA, and last 4 subjects had normal coronary artery when they suffered AMI. Comparing the clinic data between the LAD and RCA groups, the average R-R intervals before IHVF on-setting was statistically shorter in LAD group than that in RCA group (0.75±0.23 s vs 1.01±0.37 s, P=0.07). Other variables didn’t show significant differences. Total 46 patients (LAD group and RCA group) were divided into 3 groups: bradycardia group (R-R interval > 1.2 sec.), normal rate group (0.6sec < R-R interval < 1.2 sec), and tachycardia group(R-R interval < 0.6 sec). Eight of nine patients (88.9%) in the bradycardia group had RCA as IRA, while 66.7% (8/12) in the tachycardia group had LAD as IRA. The numbers of patients in normal rate group with LAD or RCA as IRA were 13 and 12, respectively.

Discussion

β-blocker, β-amiodarone, and ICD were known as effective methods in treating ventricular arrhythmia. But because of the high mortality rate of VF, so many patients died in their first episode. So it would be the most effective method to recognize and correct the risk factors before VF happening. Some studies aimed to the prognosis of in-hospital SCD, but their data didn’t include only IHVF but pulseless ventricular tachycardia and cardiac arrest. As we know different arrhythmias mean different mechanisms and prognosis, so our study was designed to analyze the data of IHVF, in order to assess predictable factors.

Same as former study, the main diseases causing VF is CAD and AMI. One mechanism is electrical heterogeneity developing reentry between necrotic, ischemia and normal myocardium tissues. Some other mechanisms include: ischemia causes abnormal cell ion current, dysfunctional cell metabolism, catecholamine releasing, and electrolyte imbalance, those factors increase autorhythmicity of myocardial cells, and then cause VF. Those mechanisms make the highest VF rate within the first 24 hours after AMI. Fortunately, the successful defibrillation rate is also the highest in this period, because the ischemia and the electrolyte imbalance are easier to be relieved. When the necrotic area scarred, the reentry around the necrotic areas, heart dysfunction, and abnormal neuroendocrine become the main mechanisms. In this stage, although VF rate decreases but the defibrillation rate decreases too, because the abnormalities are hardly to be corrected.

We know that the lower LVEF means the higher SCD rate. But some studies showed that the patients with NYHA class III had the highest SCD rate than the other classes. It seems opposite to our results, in which the patients with NYHA class IV had the highest VF rate and the worst survival rate. The possible explain could be that we recruited all IHVF, which included many mortal patients who were excluded by other studies. And those patients with severe heart failure also have the highest mortality because of their worse physical condition than those patients with better NYHA class.

The predictable risks of failure discharge

Adrenaline as a treatment method in resuscitation has been used over 30 years, spatially in those patients with bad conditions and long period of reaccuritation. But by now, there are not valid evidences approving what dose used in resuscitation is the most effective and harmless. Some studies even doubted its efficacy and safety in treating VF. In our study, the results also showed those patients using adrenaline in resuscitation did have the lowest survival rate. So in the relationship of adrenaline and low resuscitation rate, which is the background? Because we can’t get the exact information of the adrenaline usage in the resuscitation, so we think the adrenaline usage just indicates the long time of resuscitation and that means bad conditions of the patients and the bad prognosis. So we think the relationship of the adrenaline usage and the bad prognosis is just a phenomenon but not the causation.

The mean [K+] in failure group is higher than that in successful group. Meanwhile the heart function of failure group is worse than that in successful group. So with those patients failure defibrillation had worse body condition, even some patients were in the final stage with multi-organ failure or even metabolic acidosis, which caused high serum potassium and the high mortality, which cause the patients without hypokalemia as the IHVF-inducing factors had the worse survival rate.

In our study, we found the failure group had higher NYHA class significantly than that in the successful group. It suggested that the worse heart function did not only mean the higher VF occurring rate, but also the lower resuscitation rate.

Patients in emergency room

The patients in emergency group had better NYHA class than those in inpatient ward group, because the emergency group had more AMI patients. Those patients’ heart function was at compensating stage in the early period of AMI. After few days, when those patients entered the wards, the influences to heart function by myocardial infarction were more severe than before.

Madias and colleagues had reported 8% patients had hypokalemia when they suffered the AMI. In our study, the patients in emergency group also had higher rate of hypokalemia, especially in those patients with AMI. Possible explanations are those patients always ate little food or were excessive ventilated because of discomforts caused by AMI; and some patients always had taken diuretic medicine in long time and not monitored electrolyte level. In addition, K+ leaking from ischemia myocardial cell, and adrenaline released from sympathetic nerve activated β blocker enhancing the Na+/K+ exchange, both cause
hypokalemia. We know low [K⁺] would trigger autodepolarization, enhance the autorhythmicity, and reduce the VF threshold. Normalized [K⁺] could reverse the conditions and preventing onset of VF. So normal [K⁺] is an important factor for maintaining the normal heart rhythm. Interestingly, same as the former report, we didn’t find any patient emergency group in suffered AMI and IHVF with [K⁺] higher than 4.5 mmol/L.

Hypokalemia is considered as a transient factor causing VF, therefore it is not an indication for treating patients with ICD or even arrhythmia agents. But a sub-analysis of AVID study reported that those patients who were thought hypokalemia as the VF causing factor, had the worst prognosis than other groups. That means we should think twice before judging the abnormal [K⁺] as the “only” causation of VF attack. But our analysis could not report the long-term outcome of those patients.

The features of VF in AMI
Those patients with LAD as IRA had more rapid heart rate before VF occurring than those with RCA as IRA. It can be explained that the former patients usually have higher reflecting heart rate because of worse heart function due to anterior wall infarction; meanwhile the later patients have lower heart rate because of sinus node ischemia or atrioventricular block, which both increase QT interval discrepancy, and then cause ventricular arrhythmia. β-blocker should be used in early period of AMI, especially to those patients with anterior wall infarction, in order to correct the sympathetic-parasympathetic imbalance, which could decrease the rate of VF. For those patients showed who had bradycardia with RCA as IRA, artificial pacing maybe an effective method for preventing VF.

Limitation
The enrolled patients’ clinical data could not be get exactly as wish because the insufficient cases records, which limit the power of the conclusions.

Conclusion
The most common underlining disease causing IHVF is CAD and AMI in hospital; spatially those happened in emergency room. Keep [K⁺] above 4.5mmol/L could be an effective methods preventing IHVF, especially to those patients with AMI. The worse heart function means the higher rate of IHVF and higher mortality rate. Non-existing hypokalemia before IHVF and using adrenaline in resuscitation will predict the bad prognosis.

References