Biomedical Research 2017; 28 (14): 6335-6338 *ISSN 0970-938X*
www.biomedres.info

The cause and emergency rescue of ventricular electrical storm during primary PCI for patients with acute ST-elevation myocardial infarction.

Riufang Liu[1], Fangxing Xu[1], Tongku Liu[2*], Hongyan Ma[2], Ming Gu[2], Fuxiang Ding[2], Shujie Wu[2], Lihua Xu[2]

[1]Department of Cardiology, Beijing Anzhen Hospital, Capital Medical University, Beijing, PR China

[2]The Center of Cardiology, Affiliated Hospital, Beihua University, Jilin, PR China

Abstract

Objective: To explore the cause and emergency rescue of Ventricular Electrical Storm (VES) during primary PCI for patients with acute ST-Elevation Myocardial Infarction (STEMI).
Method: Among 796 patients with STEMI undergoing primary PCI 39 patients (4.9%) who happened to the VES during PCI procedure were studied. The causes, the characteristics of the coronary artery lesions, DC shock and the use of anti-arrhythmic drugs for patients with VES were analysed.
Results: The coronary angiography (CAG) showed 100% occlusion of Infarction Related coronary Artery (IRA) in all patients with STEMI. In 39 patients with VES 28 patients (71.8%) were characterized by mental tension and fear during PCI, and 11 cases (28.2%) happened to VES because of reperfusion arrhythmia. VES occurred from 3-48 times during PCI procedure. All patients with VES were characterized by ventricular fibrillation and were treated with DC shock. An average of six times of DC shock per case was performed. At the same time of DC anti-arrhythmic drugs were used such as lidocaine, amiodarone and esmolol. The rescue success was 33 cases (84.6%). 6 cases died (15.4%) of cardiac shock. The tartrate metoprolol was used regularly oral to maintain steady rhythm for all survivors. VES did not happen again after metoprolol was used in all survivors.
Conclusion: VES was easy happening for patients with STEMI and with mental tension and fear during PCI procedure. DC shock for the converting VES are primary measures, and should be rapid implementation on the basis of anti-arrhythmic drugs (especially β-blockers).

Keywords: Ventricular electrical storm, Acute ST elevation myocardial infarction, Percutaneous coronary intervention, Rescue.

Accepted on June 16, 2017

Introduction

Ventricular Electrical Storm (VES), also call the sympathetic storm and is due to acute myocardial ischemia or inflammatory myocardial damage or necrosis which result from extreme instability of myocardial electrical activity and lethal malignant arrhythmia.

VES is defined as repeated occurrence (two or more times) of severe ventricular arrhythmias that is Ventricular Tachycardia (VT) and Ventricular Fibrillation (Vf) requiring multiple cardio version (such as electric converting or electrical shock) in 24 h by ACC/AHA/ESC 2006 guidelines for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death. VES is associated with hemodynamic instability. VES is of frequent occurrence in patients with acute myocardial infarction (AMI), especially in ST-segment elevation myocardial infarction (STEMI), and also can be found in patients with unstable angina and in patients with STEMI during percutaneous coronary intervention (PCI) procedure.

In this paper, a retrospective analysis of the characteristics, cause and emergency rescue of VES was performed as follow.

Clinical Data and Methods

Clinical data of the patients

769 patients were consecutively admitted to our hospital due to STEMI from Jan 2014 to Jun 2016. In all patients CAG showed the myocardial infarction related artery (IRA) was 100% occlusion and blood flow TIMI 0 grade (thrombolysis in myocardial infarction trial, TIMI). The patients with STEMI underwent emergency PCI treatment within 12 h from onset of chest pain symptoms to balloon dilatation. Among them VES happened in 39 cases (4.9%). The patient's mental state, the time of VES occurrence, electrocardiogram findings, and rescue measures were recorded. In 39 cases there were male 29

cases (74.4%) and female 10 cases (25.6. %). The patients age was 45-76 y old (an average of 62.7 ± 9.9 y old). Among cases there were 25 cases (64.1%) with hypertension, 25 cases (64.1%) with diabetes and 22 (56.4%) smokers.

Coronary angiography (CAG) and PCI procedure

All of the patients routinely accepted urgency CAG and PCI *via* the right radial artery or left radial artery. PCI preoperative 6 h or immediately after PCI were given 300 mg of aspirin and clopidogrel bisulphate 600 mg orally. During PCI procedure heparin sodium (100 u/Kg) was injected by the arterial sheath catheter. When the PCI procedure was lasting for each one hour heparin sodium added 1000 u to maintain ACT 350 s or over. In 4 h after PCI heparin sodium 1000 u/h was continuously drip intravenous lasting for a full 24 h. The heparin sodium 5000 u was injected 2 times daily in subcutaneous lasting for 3-4 d. All of the patients after PCI were orally to maintain the amount of aspirin 100 mg/d and clopidogrel bisulphate 75 mg/d for long-term (at least for 12 months). According to condition of the patient's isosorbide mononitrate, ACEI, beta blockers and statin therapy were used.

VES rescue

VT and Vf were happened in 4 cases with STEMI before and after CAG, and happened in 35 patients during PCI procedure or after opening of IRA. The patients with VT and Vf were treated by amiodarone, lidocaine and esmolol to control Vf repeatedly. If the drugs were ineffective during Vf episodes the artificial heart pressure *in vitro* was on going until recovering autonomous heartbeat. At the same time DC shock was immediately performed for patients with Vf. For patients with cardiac sinus arrest, significantly slow heart beat (bradycardia) and II or III degree atrioventricular block, emergency temporary artificial cardiac pacing was implanted with adjuvant therapy.

Statistical analysis

All data were analysed by using the statistical software SPSS 19.0. Measurement data use ($\bar{x}$ ± s) expression. The comparison between two groups used "t" test. Count data expressed as a percentage (%). The comparison between groups used X^2 test. The difference was statistically significant ($P<0.05$).

Result

The characteristics of the baseline data

According to the anatomy the coronary artery was divided into the Left Main Coronary Artery (LM), Left Anterior Descending Branch (LAD), Left Circumflex Branch (LCX) and Right Coronary Artery (RCA).The coronary artery stenosis 50%-100% in blood vessel diameter was considered as the lesion of pathological significance. In anyone lesion of LM, LAD, LCX and RCA was on record as single branch lesion (SBL), two vascular stenosis were double branch lesions (DBL), three vascular stenosis were recorded as three branch lesions (TBL). The larger diagonal branch lesion (>2.2 mm in diameter) was seen as LAD lesions. The big blunt edge branch lesion was considered as LCX lesions. LM stenosis was considered as double branch lesions. The distribution of coronary lesions and coronary vascular occlusion was shown in Tables 1 and 2.

Table 1. *The vascular distribution of coronary artery stenosis 50% to 100%.*

	SBL	DBL	TBL	Total
Male n (%)	11 (38.1)	9 (19.0)	9 (42.9)	29 (100)
Female n (%)	5 (50.0)	0 (0)	5 (50.0)	10 (100)
Total n (%)	16 (41.9)	9 (19.4)	14 (38.7)	39 (100)

Table 2. *The vascular distribution of coronary occlusions.*

	SBL	DBL	TBL	Total
Male n (%)	26 (90.5)	3 (4.8)	0 (0.0)	29 (100)
Female n (%)	10 (100.0)	0 (0.0)	0 (0.0)	10 (100)
Total n (%)	36 (90.3)	3 (6.5)	0 (0.0)	39 (100)

In 39 patients with STEMI there were 36 cases (92.3%) with single acute coronary artery occlusion, 3 cases (7.7%) with double coronary branch occlusion and 4 case (16.0%) with LM stenosis>50%-95%.The coronary vessels distribution of IRA was shown in Table 3.

Table3. *The vascular distribution of IRA.*

	Male (n=29)	Female (n=10)
LM n (%)	1 (3.4)	0 (0.0)
LAD n (%)	14 (48,3)	3 (30.0)
LCX n (%)	5 (17.2)	0 (0.0)
RCA n (%)	12 (41.4)	7 (70.0)

The patient's mental performance

Among 39 patients with STEMI 28 patients (71.8%) were characterized by mental tension and fear during PCI, the whole body sweating, hypotension (<90 mmHg) and rapid heartbeat (more than 100 times/min).

The relationship between VT, Vf, electric converting and gender

Vf occurred two times and over during 30 min PCI procedure. DC shock for patients with Vf was performed one time to 43 times. An average of six times of DC shock per case was performed. The branch number of coronary lesions (NCL), VT

and Vf, electric converting frequency and gender were showed in Table 4.

Table 4. *The relationship of NCL, VT and Vf, electric converting frequency and gender ($X^2 \pm S$).*

	Male (n=29)	Female (n=10)	t	P
NCL (n)	2.00 ± 0.89	1.750 ± 0.06	0.508	0.96
Vf (n)	5.57 ± 6.80	4.50 ± 4.24	0.441	0.563
VT (n)	4.10 ± 3.53	4.75 ± 3.86	0.335	0.898
DC shock	5.38 ± 6.42	5.25 ± 3.94	0.337	0.633
Death (n)	4 (13.8%)	2 (20.0%)	0.636*	0.174

Note: * is X^2.

Application of drugs in VES rescue

Application of anti-arrhythmic drugs during VES rescure was lidocaine 69.2% (27cases), atropine 56.4% (22 cases), epinephrine 51.3% (20 patients), amiodarone 23.1% (9 cases) and esmolol 74.4% (29 cases). In 26 patients (66.7%) atropine, lidocaine, adrenaline and amiodarone and esmolol were combined use to control VT and Vf repeatedly.

Monitoring features of electrocardiogram wave

In 29 (74.4%) of 39 cases electrocardiogram wave monitoring found that frequent ventricular premature beat, including 18 cases (46.2%) with ventricular wave R on T, before the onset of VES (VT and Vf). 12 cases (30.8%) of cardiac electric wave monitoring found Torsade de ventricular tachycardia. One patient suffered from Vf onset 48 times and received DC shock 30 times (200 joules per one time) until use of esmlolo to control Vf occurrence not repeatedly.

The right ventricle temporary pacing

The temporary artificial cardiac pacing was used in 13 patients with bradycardiac arrhythmia by way of femoral vein until stable cardiac rhythm.

Discussion

The cause and mechanism of VES

VES may take place in various organic heart diseases, especially acute coronary syndrome (ACS). Its mechanism is due to acute myocardial ischemia and reperfusion injury, and excessive activation of sympathetic and so on. Arya reported that 75% of VES patients were ischemic cardiomyopathy [1,2]. VES was also found in dilated cardiomyopathy, acute myocarditis, rheumatic heart disease, congenital heart disease, brugada syndrome and Long or short Q-T syndrome patients [3-9]. Those patients with VES in this study were all STEMI patients. VES was caused by acute myocardial ischemia, ventricular electrical instability and local current happening. Acute coronary arteries occlusion may make local myocardial ischemia, hypoxia and the membrane potential levels drop, the refractory period change of conduction system, forming local current to lead VT and VF. The patients with VES in this group were thought to be due to acute myocardial ischemia and reperfusion injury, a lot of catecholamine release and other factors. Hypoxia, hemodynamic disorder or electrolyte disorders, severe acidosis may induce ventricular electrical storm [10,11].

The rescue

VES has a very high mortality. Electrocardiogram wave monitoring is necessary for finding malignant arrhythmia in patients with STEMI. Once Vf happen chest compressions and DC shock are immediately performed. At the same time, the application of Anti-arrhythmic drugs is also necessary to maintain sinus rhythm. After DC shock the cerebrovascular protection, prevention and treatment of cerebral edema and high cranial pressure are important treatment. For patients with VES and with sympathetic activation, β-receptor blockers (such as esmolol or metoprolol) can control the excessive sympathetic activation to make cardiac rhythm stable. Cardiac sympathetic denervation in patients with refractory ventricular arrhythmias or electrical storm was effect. This result showed the sympathetic adrenal system affect the onset of VES. So that β-receptor blockers should be used to control VES continued.

Reference

1. Zipes DP, John Camm J, Borggrefe M, Zamorano JL. ACC/AHA/ESC 2006 guidelines for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death. J Am Coll Cardiol 2006; 48: 247-346.
2. Arya A, Bode K, Piorkowski C, Hindricks G. Catheter ablation of electrical storm due to monomorphic ventricular tachycardia in patients with nonischemic cardiomyopathy: acute results and its effect on long-term survival. Clin Electrophysiology 2010; 33: 1504-1509.
3. Chinushi M, Iijima K, Sato A , Furushima H. Short-coupling premature ventricular complexes from left ventricle triggered isoproterenol resistant electrical storm in a patient of Brugada syndrome. Heart Rhythm 2013; 10: 916-921.
4. Davis JE, Curtis LA, Rashid H. Idopathic cardiac electrical storm. J Emerg Med 2007; 37: 264-268.
5. Sessslberg HW, Moss AJ, Mcnitt S, Huang DT. Ventricular arrhythmia storms in post infarction patients with implantable defibrillators for primary prevention indications: a MADIT-II substudy. Heart Rhythm 2007; 4: 1395-1402.
6. Schweizer PA, Becker R, Katus HA. Successful acute and long-term management of electrical storm in Brugada syndrome using orciprenaline and quinine/quinidine. Clin Res Cardiol 2010; 99: 467-470.
7. Kramer DB, Zimetbaum PJ. Long-QT syndrome. Cardiol Rev 2011; 19: 217-225.

8. Bun SS, Maury P, Giustetto C. Electrical storm in short-QT syndrome successfully treated with isoproterenol. J Cardiovas Electrophysiol 2012; 23: 1028-1030.
9. Patel C, Yan GX, Antzelevitch C. Short QT syndrome:from bench to bedside. Circ Arrhythm Electrophysiol 2010; 3: 401-408.
10. Eifling M, Razavi M, Massumi A. The evaluation and management of electrical storm. Texas Heart Institute J 2011; 38: 111-121.
11. Vaseghi M, Gima J, Kanaan C, Ajijoia OA, Marnureanu A. Cardiac sympathetic denervation in patients with refractory ventricular arrhythmias or electrical storm: intermediate and long-term follow-up. Heart Rhythm 2014; 11: 360-366.

***Correspondence to**

Tongku Liu

The Center of Cardiology

Affiliated Hospital

Beihua University

Jilin

PR China