196

ELSEVIER

Available online at www.sciencedirect.com

SCIENCE DIRECT®

JNMU

Journal of Nanjing Medical University, 2007, 21(3): 196-199

www.elsevier.com/locate/jnmu

Case Report

Valves replacement operation in treatment of electrical storm induced by rheumatic valves disease

Zhenqiang Chen*, Hui Zhang, Yang Zhao, Haiji Yang, Sheng'ai Ye, Liang Cheng, Ying Zhang

Department of Cardio-thoracic Surgery, the Traditional Chinese Medicine Hospital of Jiangsu Province, Nanjing 210029, China

Received 4 march 2007

Abstract

Objective: To present a case of electrical storm (ES) in a female patient with rheumatic valve disease. **Methods**: A female patient with severe rheumatic valve disease suffered an unexpected ES. She received more than 50 electrical shocks for repeated cardiac arrests due to ES over 16 hours. Then she received beta-blocking agent treatment and had an operation of double valves replacement. **Results**: ES was suppressed by sympathetic blockade with beta-receptor blocker and finally disappeared after the double pathological valves had been replaced. **Conclusion**: Increased sympathetic activity plays an important role in the genesis of electrical storm and sympathetic blockade may effectively suppress ES. However, the most important thing in the treatment of ES is to identify and eliminate the underlying cause of ES.

Keywords: electrical storm; rheumatic valve disease; sympathetic activity

INTRODUCTION

Electrical storm (ES) is a syndrome of recurrent ventricular tachycardia (VT) or ventricular fibrillation (VF), which is most common in patients who have had a recent myocardial infarction or ongoing myocardial ischemia. However, ES occurring in patients with rheumatic valves disease has been rarely reported. In this article we will present an unusual case of an adult female patient with rheumatic valve disease suffering from an unexpected electrical storm before double valves replacement.

CASE REPORT

A 38-year old female patient with no history of ischaemic heart disease was admitted to the hospital for severe rheumatic valves disease. Laboratory investigations showed normal electrolytes, normal cardiac enzyme and blood troponin I (TNI), normal liver and renal function, antistreptolysin "O" (ASO) 61.60 U/ml, rheumatoid factor (RF) < 20 U/ml, c-reactive protein (CRP) 1.0 mg/L. Arterial blood gases showed a pH of 7.38, pO2 of 85.3 mmHg, pCO_2 of 32 mmHg. Chest radiography revealed pulmonary oedema. Electrocardiogram (ECG) revealed a rhythm of atrial fibrillation with heart rate 127 bpm, low T waves in lead Ⅰ, Ⅱ, Ⅲ, aVL and aVF, inversed T waves in lead V4~6, ST segment depression by 0.05 mv to 0.075 mv and normal QT interval (***Fig 1***). Ultrasonic cardiography (UCG) showed rheumatic valves disease with moderate mitral stenosis, aortic regurgitation and tricuspid regurgitation, left atrial diameter (LAD) 49 mm, left ventricular end-diastolic diameter (LVDd) 55 mm, right atrial (RA) 39 × 62 mm, right ventricular diameter (RVD) 27 mm, mitral valve area 1.1 cm^2, cardiac ejection fraction (EF) 30%, pulmonary artery systolic pressure (PASP) 47 mmHg. The heart function classification is NYHA Ⅳ.

After admission, the patient received medication

*Corresponding author.

E-mail address: chen_zq@sina.com

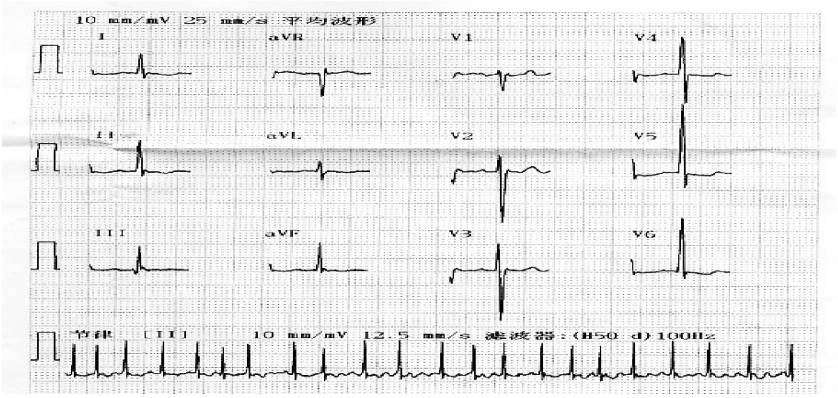

Fig 1 ECG on admission showed a heart rhythm of atrial fibrillation

of dopamine and milrinone and diuretics for heart failure, but the symptom has not improved. At 22:00 on the ninth day after admission, she experienced an unexpected asystolic cardiac arrest. The cardiopulmonary cerebral resuscitation was immediately performed. Fifty minutes later, the heartbeat automatically recovered. ECG monitoring revealed polymorphic ventricular premature beat, ventricular fibrillation. Recurrent pulseless VT and VF (***Fig 2***) occurred every 10 to 20 minutes, which necessitated electrical cardioversion or defibrillation to achieve sinus rhythm with a cardiac output. Continuous infusion of cordarone and lidocaine was administrated but proved futile. Ten hours after cardiopulmonary resuscitation, four times of 100 mg esmolol iv were administrated followed by its infusion at 100-180 mg/h. Repeated episodes of VT or VF was reduced to once every 1 to 2 hours. At the same time, inotropic therapy with milrinone and dopamine was given, as well as continued mechanical ventilation as she was hypotensive and oliguric. However, the patient still experienced multiple episodes of ventricular arrhythmia. During the sixteen hours after cardiopulmonary resuscitation, she had over fifty episodes of VT/VF and received 51 DC shocks. And then the episode of VT and VF stepped down after two times intravenous injection (15 mg betaloc) at the 17th hour. Premature ventricular beats frequently appeared (***Fig 3***) and the cardiac output was 1.8 L/min. At the 36th hour after the cardiac arrest, she successfully underwent a surgery of the mitral and aortic valves replacement and tricuspid valve annuloplasty. After the operation, Dopamine was continuously infused at 5-8 μg/(kg·min). With a bolus injection of 15 mg betaloc, it changed into oral medication at 75 mg twice a day. The VT and VF were thoroughly controlled. Only one episode of VF was found and reversed by defibrillation at the 5th hour after operation. The hemodynamic condition was stable with the heart rate around 60 bmp and the arterial pressure 100/65 mmHg. The cardiac output was improved from 3.0 L/min to 6.0 L/min, and the urine output increased also. EF was improved to 60%. At the third day after the operation, all the va-

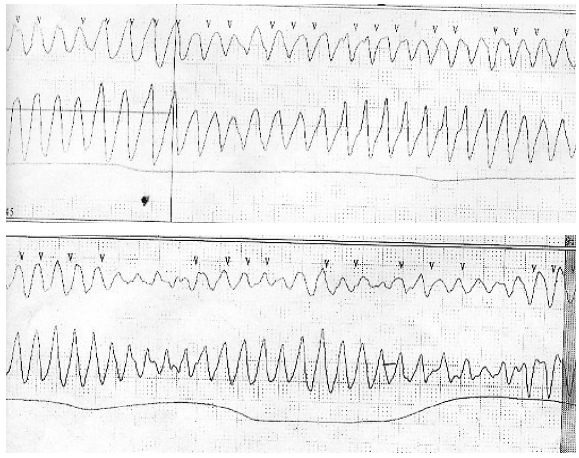

Fig 2 ECG showed recurrent VT/VF when the patient experienced ES

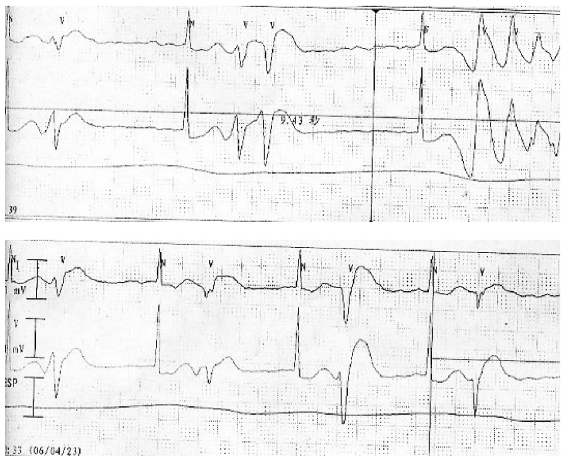

Fig 3 ECG of 24 h before valve replacement operation showed ES was suppressed however there were some premature ventricular beats

soactive drugs were withdrawn except oral betaloc (75 mg bid). Three days later, she moved out of the ICU. The ECG revealed sinus rhythm with premature ventricular beats once in a while. The UCG showed LAD 46 mm,LVDd 45 mm,RA 35 × 50 mm,RVD 22 mm,EF 52%. In the following 15 days, her sinus rhythm changed into atrial fibrillation. The 24-hour dynamic electrocardiogram taken on the 35th day after operation showed atrial fibrillation with 107 ventricular premature beats and 24 atrial premature beats. ECG taken on the 60th day revealed atrial fibrillation with low T wave in Ⅱ, Ⅲ,aVF,V5~V6 and reversed T wave in V4~V6, ST segment depression in V5~V6 by 0.05 mV. The UCG showed LAD 38 mm,LVDd 42 mm,RA 38×45 mm,RVD 21 mm,EF 62%,FS 32%。During the follow-up period, spironolactone and carvedilol were administrated for left ventricular dysfunction. ACE inhibitors were withheld in view of renal impairment.

DISCUSSION

ES is defined as recurrent, hemodynamically destabilizing ventricular tachycardia or ventricular fibrillation, occurring two or more episodes over a 24-hour period, and usually requiring electrical cardioversion or defibrillation[1]. Five clinical syndromes of electrical storm have been described [2]. These include: polymorphic VT/VF associated with myocardial ischemia or infarction; monomorphic VT in patients with structural heart disease; sustained nonsyncopal monomorphic VT; recurrent VT/VF with syncope or cardiac arrest; and arrhythmia with delayed repolarization (torsades de pointes).

Based on the previous studies, ES is found to occur most often in patients with coronary artery disease, including those with prior myocardial infarction, left ventricular dysfunction, and ventricular dilatation[3]. Although the exact mechanisms in triggering and termination of electrical storms are not well elucidated[4], it seems that three main mechanisms of arrhythmogenesis—re-entry, trigger activity and automatism—have all been implicated. Some evidence showed that chronic stretch of the scarred or viable myocardium changes the action potentially and facilitates the development of early and late after depolarizations[5,6].

This effect, called mechanical-electrical feedback, most frequently initiates both the triggered activity and re-entry tachycardias [7]. Moreover, various underlying factors [8], such as myocardial ischemia[9], cardiomyopathy[10,11], surgery[2] (especially after organ transplantation), drugs such as dobutamine, ICD implantation[12], recent worsening heart failure, neurohumoral disturbances and electrolytic abnormalities, may favor electrical instability and trigger ES in these patients. Some evidence indicates that it can be triggered by various factors including hypokalaemia and hypomagnesaemia. In this case, ES occurred in a patient with severe valve disease, which was rarely described before. We postulated that the recent worsening heart failure, which was induced by the severe pathological changes of the valves, activated the sympathetic nervous system, initiating ES.

In the treatment of ES, both prompt identification of the potential triggering factors and immediate management has major clinical significance. Once the patient is in cardiac arrest with pulseless VT or VF, electrical cardioversion or defibrillation is a priority. Then attempts must be made to find underlying causes and prevent further episodes. In this case, when the patient suffered from ES, immediate cardiopulmonary resuscitation was performed, which ensured the fundamental blood supply for the heart, the brain, the lung and other organs and avoided the development of severe complications. Then intravenous infusion of the beta-blockade agents, esmolol and betaloc combined with milrinone and dopamine was carried out to suppress the recurrent VT/VF and improved the cardiac function, which made the following operation feasible. From the case, we also found that conventional antiarrhythmic therapy is not the priority choice of treatment for ES because intravenous infusion of cordarone and lidocaine proved futile. These results are coherent to the previous studies which showed that treatment of ES with conventional antiarrhythmic drugs often yield a poor outcome[3,5]. The unfolding scenario is swift and desperate. Patients repeatedly go into VF, who are given antiarrhythmic medication serially, and receive repeated electrical shocks in an attempt to cardiovert the arrhythmia. Despite these efforts, most ES patients die—many within minutes or hours—especially if they have had a recent myocardial infarction or ongoing myocardial ischemia. However, sympathetic blockade may have a beneficial effect because increased sympathetic activity has been proved to be implicated in the generation of electrical storm [3]. In this case, as well as previous studies, the beta-blockade agents, could reduce the occurrence of ES[13-15].

In the management of the patient with electrical storm, the most importance thing is timely identification and treatment aiming at the underlying causes

of ES4. In this setting, the patient with rheumatic valve disease had an unexpected ES perhaps due to a recent worsening valves and heart failure. The VT/VF were stepped down after intravenous betaloc injection, but premature ventricular beats frequently appeared which indicated the patient would be still at risk of sudden death if the valves replacement operation was not carried out. So we took a great risk to perform the heart surgery. At last it turned out that the electrical storm disappeared and the cardiac function improved after mitral and aortic valves replacement and tricuspid valve annuloplasty. Over the 6 months of follow-up period, the patient was monitored and did not report the reoccurrence of VT/VF symptoms.

References

[1] Kowey PR, Levine JH, Herre JM. Randomized, double-blind comparison of intravenous amiodarone and bretylium in the treatment of patients with recurrent, hemodynamically destabilizing ventricular tachycardia or fibrillation. The Intravenous Amiodarone Multicenter Investigators Group. *Circulation* 1995;92:3255-63.

[2] Schmidt TD. and Muir AJ. A case of electrical storm in a liver transplant patient. *Transplant Proc* 2003;35:1437-8.

[3] Nademanee K. Treating electrical storm: sympathetic blockade versus advanced cardiac life support-guided therapy. *Circulation* 2000;102:742-7.

[4] Wilhelm H, Electrical storm: still a cryptogenic phenomenon? *European Heart Journal* 2006;27:2921-2.

[5] Vaidyanathan D. and Prabhakar D. Management of electrical storm--is long term antiarrhythmic therapy indicated? *Int J Cardiol* 2000;73:297.

[6] Okada.T, Yamada T, Murakami Y, Yoshida N, Ninomiya Y, and Toyama J. Mapping and Ablation of Trigger Premature Ventricular Contractions in a Case of Electrical Storm Associated with Ischemic Cardiomyopathy. *PACE* 2007;30:440-3.

[7] Babuty D, Lab M. Mechanoelectric contributions to sudden cardiac death. *Cardiovasc Res* 2001;50:270-9.

[8] Dorian P, Cass D. An overview of the management of electrical storm. *Can J Cardiol* 1997;13:13-7A.

[9] Perzanowski C. and Pai SM. Electrical storm after coronary artery bypass grafting due to a kinked left internal mammary artery graft. Pacing Clin. *Electrophysiol* 2004;27:545-6.

[10] Barriales V, Tamargo JA, Aguado MG, Martin M, Rondan J, Posada IS. Electrical storm as initial presentation of arrhythmogenic right ventricular cardiomyopathy in an elderly woman. *Int J Cardiol* 2004;94:331-3.

[11] Tanabe Y. Suppression of electrical storm by biventricular pacing in a patient with idiopathic dilated cardiomyopathy and ventricular tachycardia. Pacing Clin *Electrophysiol* 2003;26:101-2.

[12] Han SW. An electrical storm with more than 3000 shocks in a patient with an implantable cardioverter-defibrillator: is jet-lag a trigger? *Int J Cardiol* 2005;104:235-7.

[13] Arash A, Majid H, Mohammad RD, Amir FF, Nikoo MH, Bagherzadeh A. *et al*. Prevalence and Predictors of Electrical Storm in Patients With Implantable Cardioverter-Defibrillator, *Am J Cardiol* 2006;97:389-92.

[14] Connolly SJ, Dorian P, Roberts RS, Gent M, Bailin S, Fain ES. Optimal Pharmacological Therapy in Cardioverter Defibrillator Patients (OPTIC) Investigators. Comparison of beta-blockers, amiodarone plus betablockers, or sotalol for prevention of shocks from implantable cardioverter defibrillators: the OPTIC Study: a randomized trial. *JAMA* 2006;295:165-71.

[15] Susanne CC, Thomas K, Oliver M, Christian S, Stefan HH. Electrical Storm in Patients With Transvenous Implantable Cardioverter Defibrillators Incidence, *Management and Prognostic Implications JACC* 1998;32:1909-15.

CORRECTION

In the article *Effects of pioglitazone on the proliferation and differentiation of the human preadipocytes* (Journal of Nanjing medical University 2007; Vol 21 No 2, p99-103), the corresponding author is Xianghua Ma, E-mail address: xianghuama@sina.com.