Successful treatment of cardiac electrical storm in dilated cardiomyopathy using esmolol: A case report

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Abstract. The present study reports a case of electrical storm occurring in a 43-year-old woman with dilated cardiomyopathy. The patient suffered from a cardiac electrical storm, with 98 episodes of ventricular tachycardia rapidly degenerating to ventricular fibrillation in hospital. The patient was converted with a total of 120 defibrillations. Recurrent ventricular tachycardia/fibrillation was initiated by premature ventricular beats. The patient did not respond to the use of amiodaronum. However, the administration of esmolol stabilized the patient's heart rhythm. A moderate dose of the β-blocker esmolol, administered as an 0.5-mg intravenous bolus injection followed by an infusion at a rate of 0.15 mg/kg/min, inhibited the recurrence of ventricular fibrillation and normalized the electrocardiographic pattern. The results suggest that esmolol may be able to improve the survival rate of patients with electrical storm in dilated cardiomyopathy and should be considered as a primary therapy in the management of cardiac electrical storms.

Introduction

Electrical storm (ES) is a dramatic and life-threatening syndrome which is defined by three or more sustained episodes of ventricular tachycardia (VT), ventricular fibrillation (VF), or appropriate shocks from an implantable cardioverter-defibrillator within 24 h (1-3). This pathology is may cause fatal arrhythmia in certain patients with severe heart disease (4,5). Once an electrical storm occurs, the control of ventricular tachycardia/fibrillation using standard treatment alone is difficult (6-9). A schematic of the emergent treatment of ventricular arrhythmias is shown in Fig. 1. ES can manifest itself during post-infarction ischemic heart disease, various forms of cardiomyopathy, an implantable cardioverter-defibrillator, or an inherited arrhythmic syndrome, such as Brugada syndrome (1,10-16). ES typically has a poor outcome (17). The intravenous injection of class III antiarrhythmic drugs, including, amiodaronum and nifekalant, is used initially to inhibit ventricular arrhythmia in the majority of patients (18,19). However, electrical storms can show resistance to, or not be affected by these drugs, which is a major problem in the application of emergency medical care (20,21). In the present study, a case of ventricular electrical storm in a patient with dilated cardiomyopathy is reported, that was successfully treated by esmolol infusion.

Case report

A 43-year-old woman was referred to the Department of Cardiology, Jinan Central Hospital Affiliated to Shandong University (Jinan, China) on December 3, 2010. She underwent two defibrillator shocks within 24 h after having survived sudden cardiac death. The patient presented with a respiratory infection 20 days prior to admission. An intercritical electrocardiogram (ECG) was recorded upon admission using a 6-channel ECG system (ECG-1250P; Nihon Kohden, Inc., Tokyo, Japan) which detected a sinus rhythm and intraventricular block (Fig. 2). Transthoracic echocardiography revealed markedly enlarged left ventricular end-diastolic dimension [76 mm; normal range (NR), 42.5±7.5 mm] and lowered left ventricular ejection fraction (18%; NR, 65±5%), mild mitral regurgitation and mild elevation of pulmonary arterial pressure (pulmonary artery mean pressure, 34 mm Hg; NR, 10±5 mmHg). Dilated cardiomyopathy is a myocardial disease characterized by dilatation and impaired contraction of the left ventricle or both ventricles (22). The patient presented with sudden arrhythmic death. Transthoracic echocardiography revealed dilated cardiomyopathy with severe global left ventricular systolic dysfunction with an estimated left ventricular ejection fraction of 18%. These symptoms excluded ischemic heart disease and led to a diagnosis of dilated cardiomyopathy.

Initial laboratory tests revealed hypokalemia (2.9 mmol/l; NR, 4.4±0.9 mmol/l), high serum B-type natriuretic peptide (739 pg/ml; NR, <100 pg/ml), normal magnesium levels, no hypercalcemia, and normal troponin I and creatine phosphokinase enzyme levels. Following admission, the patient was under

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Key words: cardiac electrical storm, dilated cardiomyopathy, esmolol, ventricular tachycardia, ventricular fibrillation

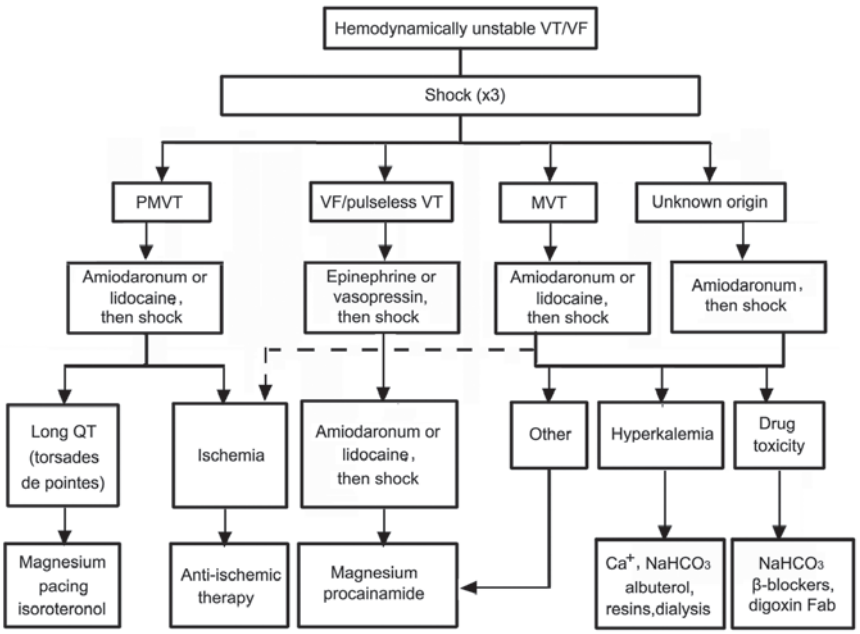

Figure 1. Schematic of the emergent treatment of VT/VF. VT, ventricular tachycardia; VF, ventricular fibrillation; PMVT, polymorphic VT; MVT, monomorphic VT; Fab, fragment antigen binding compound.

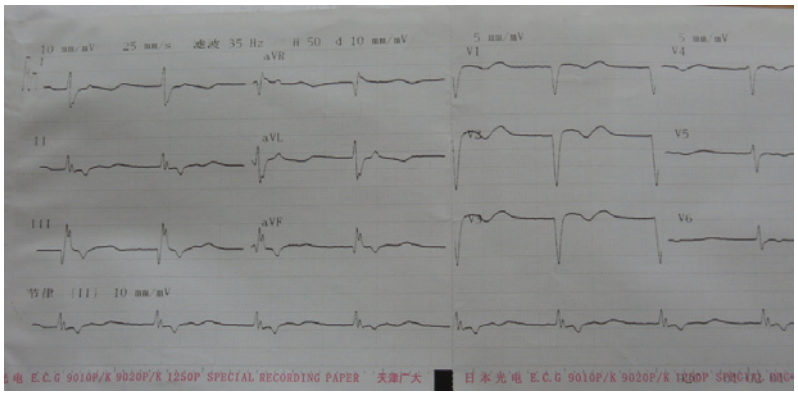

Figure 2. Electrocardiogram showing sinus rhythm and intraventricular block.

close observation. Genetic testing was not performed, and the patient had no family history of dilated cardiomyopathy. Informed consent was obtained from the patient on the day of admission. The present study was approved by the Ethics Committee of Jinan Central Hospital Affiliated to Shandong University.

In the following duration of hospital stay, 40 episodes of polymorphic ventricular tachycardia and ventricular fibrillation occurred within 24 h; recurrent polymorphic non-sustained and sustained ventricular tachycardia, triggered by ventricular premature beat, appeared incessantly and degenerated into polymorphic sustained ventricular tachycardia (Fig. 3). Repeated electrical cardioversion procedures were performed (54 in total). Firstly, the patient was prescribed high-dose amiodaronum (2 mg/min; ivp; Sanofi Pharmaceutical Co., Ltd., Paris, France), which is a class III antiarrhythmic drug, but gradual tapering of the amiodaronum infusion resulted in the recurrence of the electrical storm, which required frequent cardioversions. One day later, 58 episodes of polymorphic ventricular tachycardia/fibrillation occurred repeatedly, repeated electrical cardioversion procedures were performed (66 in total, indicating that the electrical storm exhibited resistance to amiodaronum. A number of clinical studies have demonstrated that β-blockers effectively suppress electrical storm (23-26); therefore, middle-dose esmolol (0.05-0.2 mg/kg/min; ivp; Oilu Pharmaceutical Co/. Ltd., Jinan, China), which is a β1-receptor blocker, was prescribed (20,21). Following an intravenous bolus injection of 0.5 mg esmolol, an infusion of esmolol at a rate of 0.15 mg/kg/min (ivp) was administered. No ventricular fibrillation occurred repeatedly during an interruption of the infusion for 24 h. The patient was free of ventricular tachycardia/fibrillation, although the premature ventricular beats persisted for 2 weeks following the application of esmolol (Fig. 4). The patient received oral bisoprolol fumarate (β-blockade; 5 mg/day; Merck Millipore, Darmstadt, Germany) to treat these premature ventricular beats until she was discharged on January 7, 2011.

During the treatment of the electrical storm, serum electrolytes (including Mg^{2+}, K^{+} and Ca^{2+}) were maintained at

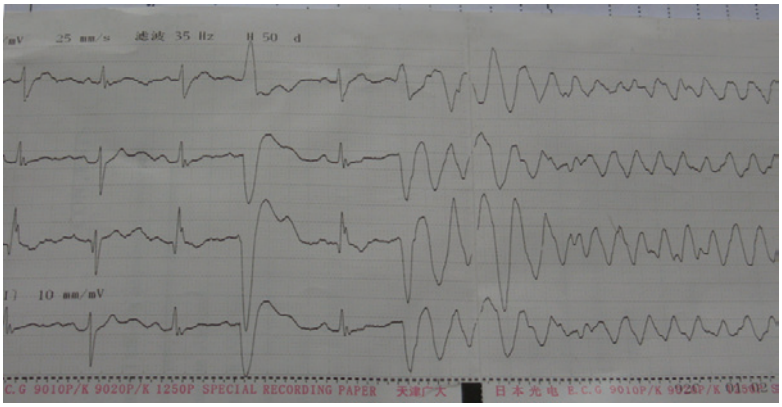

Figure 3. Electrocardiogram showing an episode of sinus rhythm with multiple ventricular premature beats spontaneously converted to ventricular tachycardia and ventricular fibrillation.

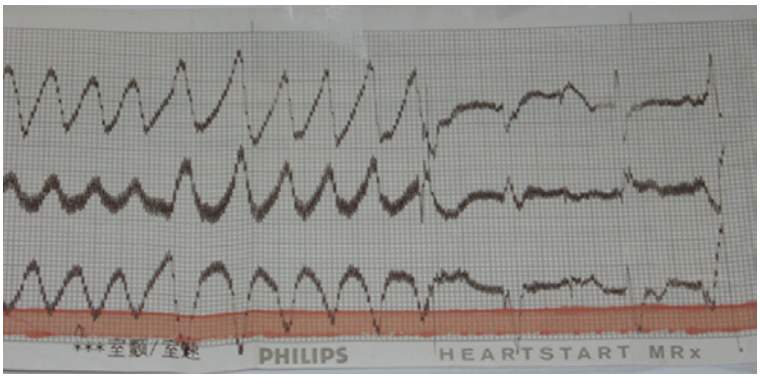

Figure 4. Electrocardiogram following the infusion of esmolol; a reduction in the occurrence of ventricular fibrillation was recorded. Although the premature ventricular beats persisted for several hours with the same coupling interval, the patient was free of ventricular fibrillation.

high levels by the infusion of intravenous electrolyte solution. When the acute phase of the electrical storm was controlled, the focus of treatment shifted toward maximizing heart-failure therapy and preventing subsequent ventricular arrhythmias. The patient was prescribed oral losartan (angiotensin receptor blocker; 50 mg/day; Novartis Pharma AG, Basel, Switzerland), oral bisoprolol fumarate (β-blockade; 5 mg/day), oral spironolactone (aldosterone antagonist; 20 mg twice daily), oral digoxin (digitalis; 0.125 mg/day; both Shanghai Xinyi Pharmaceutical Co., Ltd., Shanghai, China) furosemidum (loop diuretic; 40 mg twice daily; ivp; Shanghai He Feng Pharmaceutical Co., Ltd., Shanghai, China). Following this, the patient discharged on January 7, 2011 in a hemodynamically stable condition.

Discussion

Typically, electrical storms have a poor prognosis; they are defined as three or more distinct episodes of ventricular fibrillation, or hemodynamically destabilizing ventricular tachycardia occurring within a 24-h period, typically requiring treatment with electrical cardioversion or defibrillation (4).

Enhanced sympathetic nerve activity is associated with episodes of ES (27,28) and β-blockade has been demonstrated to reduce the risk of recurrent VT and VF (25). For patients with acute myocardial infarction, the use of β-blockade decreases the risk of sudden death, as β-blockers reduce mortality by preventing VT and VF (26). β-blockers treatment should be limited in patients with labile hemodynamic compensation or severe reduction of LV function. An electrical storm in patients with dilated cardiomyopathy occurs rarely with only a few reported cases. Once non-ischemic cardiomyopathy occurs, the heart experiences structural changes. Fibrosis results in scarring, which leads to regions of conduction block; however, groups of myofibrils are able to survive, particularly those surrounding the border of the scar. Slow conduction through these regions can facilitate electrically stable reentry (29-32).

It is acknowledged that amiodaronum and β-blockers, particularly the former, are able to treat arrhythmia effectively in the majority of patients. The present study presents an organized approach for effectively evaluating and managing electrical storms. β-blocker administration has antiarrhythmic and antiadrenergic effect. Its administration should be limited in patients with severe reduction of LV function or haemodynamic instability. Esmolol is a selective ultra short β1 blocker. The present study presents esmolol for effectively managing electrical storms in dilated cardiomyopathy with severe reduction of LV function or haemodynamic instability. Firstly, a high dose of amiodaronum was prescribed to treat cardiac electrical storm in a patient experiencing dilated cardiomyopathy. However, when gradual tapering of the amiodaronum infusion did not arrest the electrical storm, and frequent cardioversions were required, esmolol was administered in a bolus injection, followed by an infusion. Following this, there

were no further frequent repeats of ventricular fibrillation. This suggests that esmolol is effective in suppressing electrical storms, and that enhanced sympathetic nerve activity is involved in episodes of electrical storms (26,33).

In conclusion, the results strongly suggest that esmolol may improve the survival rates of patients experiencing electrical storms, and should be considered as a primary treatment option. However, as esmolol can exacerbate heart failure in patients with poor systolic function, its use in these patients should be closely monitored.

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