Successful resuscitation of a patient With electrical storm over 7 days: a case report

Yue-Ling Guo[1], Hai-ying Zhang[2], Wei Zhang[2], Yun-Feng Han[2], Shu-Lin Zhang[2]

[1.] Department of Medical Engineering, The Military General Hospital of Beijing PLA, Beijing 100070, China;
[2.] Department of Geriatrics, The Military General Hospital of Beijing PLA, Beijing 100070, China;
Correspondence author: Yun-Feng Han (hanyf001@126.com)
(The first two authors contributed equally to this work)

Abstract: A 75 year old woman with atrial fibrillation-related tachycardia-induced cardiomyopathy suffered ventricular fibrillation induced by dobutamine and was successfully defibrillated into atrial fibrillation in hospital. During the following 7 days, she suffered a cardiac electrical storm with 55 episodes of ventricular tachycardia rapidly degenerating to ventricular fibrillation and was converted with a total of 51 defibrillations and 4 chest compressions. Sinus rhythm was restored by electric cardioversion in the second episode of ventricular fibrillation. There was no response to the use of any recommended anti arrhythmic drugs. However, the use of chlorpromazine and promethazine surprisingly stabilized her heart rhythm. During a two-year follow-up period, the patient has remained free of ventricular fibrillation episodes and maintained sinus rhythm.
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Introduction

The term tachycardia-induced cardiomyopathy (TIC) or tachycardiomyopathy refers to impairment in left ventricular function secondary to chronic tachycardia, which is partially or completely reversible once the tachyarrhythmia is controlled. The incidence of TIC is unknown, but in selected studies of patients with atrial fibrillation, approximately 25% to 50% of those with left ventricular dysfunction had some degree of TIC[1,2]. Data on the prognostic significance of electrical storm strongly suggest that these patients have a poor outcome. Electrical storm might be an independent risk factor for cardiac death. In the AVID trial[3], patients with electrical storm had an increased risk of nonsudden cardiac death (risk ratio, 2.4). In the MADIT –II substudy, patients with electrical storm had a 7.4-fold higher risk of death than patients without electricalstorm[4].

The term electrical storm (ES) was introduced in the 1990s to describe a state of electrical instability of the heart characterized by a series of malignant ventricular arrhythmias in a short period of time[5]. This condition has been described in patients with post-infarction coronary artery disease, as well as in patients with various forms of cardiomyopathy, valvular disease, surgically corrected congenital heart disease, and genetically determined cardiac diseases without any apparent underlying structural disease, such as Brugada syndrome[6]. They need immediate resuscitative treatment, identification of the underlying cause and strategies for long term prevention of recurrence. We present a case of electrical storm when a woman with TIC was defibrillated 51 times over 7days but went on to make a good recovery.

CASE REPORT

A 75-year-old woman was admitted to our hospital due to worsening palpitation and dyspnoea. She had had hypertension for twenty years and atrial fibrillation for six month. The patient had no history of ischaemic heart disease, type 2 diabetes, hyperlipidaemia, chest pain, syncopal episode, or alcohol and drug abuse. She did not smoke and did not report any family history of syncope and sudden death. Blood pressure (BP) was 136/82 mmHg. Physical examination was unremarkable aside from a fast irregular pulse. Thyroid function tests were normal. Laboratory data were within normal limits. An electrocardiogram revealed atrial fibrillation with a ventricular rate of 107 beats per minute. The QT interval was 0.358s and no significant ST change was observed. The chest X-ray showed cardiomegaly (cardiothoracic ratio, 60%). The echocardiography revealed a dilated left ventricle with a diastolic diameter of 6.0cm and a reduced ejection fraction of 45%. A diagnosis of dilated cardiomyopathy was made. The patient was commenced on digoxin, valsartan, furosemide, spironolactone and metoprolol.

On the 5th hospital day, when dobutamine was infused at a rate of 1ug/kg/min for 3 hours, ECG monitoring revealed ventricular tachycardia (VT) rapidly degenerating to ventricular fibrillation (VF). Immediate chest compressions were started. Bolus infusion of 1 mg of adrenaline was given. Atrial fibrillation was restored by 3 electric cardioversion (200, 300, 360 Joule). Serum level of potassium was

in the normal range (4.1mmol/L). Initial laboratory findings revealed no significant abnormalities including electrolyte disturbance, troponin and CPK-MB enzymes. After 75mg of amiodarone was administered intravenously over 10 minutes, continuous intravenous administration (60mg per hour) was given.

Ten hours later after her first VF in the Ward, she suffered a new event of VT rapidly degenerating to VF and successfully defibrillated (200J) into sinus rhythm(70 times per minute). After intravenous bolus of 40mg of lidocaine and 75mg of amiodarone, continuous intravenous lidocaine (120mg per hour) and amiodarone (60mg per hour) was given. She suffered 2 episodes of VF and successfully defibrillated (200J) into sinus rhythm in next 4 hours. A diagnosis of ES was made. Immediately, metoprolol was administered intravenously 3 times (5mg/times) in one hour. Oral metoprolol was added to 50mg per day. The serum potassium level was kept at 4.0 mmol/L or higher.

The electrical storm persisted, despite the continuous infusion of lidocaine, amiodarone and repeated doses of metoprolol, magnesium and potassium. Her hemodynamic condition was stable and serum electrolytes were normal. She suffered 15-20 episodes of VT and 5--10 episodes of VF each day in the next 5 days. Each event triggered immediate chest compressions for 20-30s while charging the defibrillator. Sinus rhythm was restored by electric cardioversion(150-200J, total 51 times) or chest compressions(4 times) in each shock. The QT/QTc interval was 0.428s/0.426s and no significant ST change was observed, but she never suffered the event of *torsade de pointes* (TdP). Five days later after her first VF in the Ward, she was sedated by intramuscular chlorpromazine and promethazine. Surprisingly, the occurrence of VF was markedly decreased to 1--2 episodes of VF each day. After two days the occurrence of VT and VF terminated.

Ten days later after her first VF, sedation was discontinued and she regained consciousness with intact cerebral function. Coronary angiography showed normal coronary arteries. There was only a slight increase in cardiac enzymes (Troponin I: 0.2 ng/mL) during ES. The echocardiography revealed a dilated left ventricle with a diastolic diameter of 5.5cm and a reduced ejection fraction of 50%. Chest x-ray showed cardiomegaly (cardiothoracic ratio, 60%) and Cardiac MR showed dilated cardiomyopathy. No premature ventricular contractions were detected by 24-hour Holter monitoring. An implantable cardioverter defibrillator was not implanted due to economic reason.

Interestingly, upon discharge from the hospital the patient had no ventricular arrhythmias. At follow up six months later, a repeat echocardiogram confirmed that the left ventricular dimensions had returned to normal(diastolic diameter of 5.0cm) and the estimated ejection fraction was 70%. A diagnosis of atrial fibrillation-related TIC was made. During a two-year follow-up period, the patient has remained free of ventricular fibrillation episodes and maintained sinus rhythm. She has continued long-term treatment with metoprolol and amiodarone. At present, she is in excellent condition.

Discussion

ES is defined as the recurrence of hemodynamically unstable VT and/or VF, twice or more in 24 hours, requiring electrical cardioversion or defibrillation[7,8]. With the arrival of the ICD (implantable cardioverter defibrillator) this definition was broadened, and ES is also defined as the occurrence of three or more distinct episodes of ventricular tachycardia (VT) or ventricular fibrillation (VF) in 24 hours, requiring the intervention of the defibrillator [9]. It should be noted that this latter definition does not include the presence of hemodynamic instability. This condition has been described in patients with post-infarction coronary artery disease, as well as in patients with various forms of cardiomyopathy, valvular disease, surgically corrected congenital heart disease, and genetically determined cardiac diseases without any apparent underlying structural disease, such as Brugada syndrome[6].

The patient reported to have atrial fibrillation six month prior to admission. The echocardiography revealed a dilated left ventricle with a diastolic diameter of 6.0cm and a reduced ejection fraction of 45% after admission. Six month after sinus rhythm was restored by electric cardioversion, a repeat echocardiogram confirmed that the left ventricular dimensions had returned to normal and the estimated ejection fraction was 70%. So, a diagnosis of atrial fibrillation-related TIC was made. Clinically, the most common cause of TIC is believed to be atrial fibrillation (AF), which is increasing in incidence with the aging of society. AF causes TIC via 2 distinct mechanisms: inadequate diastolic filling and tachycardia-induced systolic dysfunction. When AF develops, the active atrial contraction disappears in end-diastole, leading to a reduction in the cardiac output by 15–20%[10]. This insufficient diastolic filling is augmented by the shortening of diastolic filling time. Furthermore, sustained tachycardia results in impaired systolic function through a mechanism represented by TIC, which leads to a greater reduction in the cardiac output[10,11]. A recent report has suggested a risk of sudden death in this

particular group[12]. Our report is the first to document one patient with fibrillation-related TIC who suffered ES and has a good recovery.

It is noteworthy that, in spite of a detailed analysis of the electrocardiogram, haematological and biochemical examinations, and the patients'clinical symptoms, in only 36% was any triggering mechanism found that could provoke electrical storm. Those factors were acute ischemia, worsening heart failure, hypokalemia, hypomagnesemia, arrhythmogenic drug therapy, hyperthyroidism, and infection or fever. In this case, we found, CHF secondary to atrial fibrillation-related TIC, who developed ES during low-dose dobutamine infuse. Dobutamine is an inotropic pharmaceutical that improves the hemodynamic and clinical status of patients suffering from congestive heart failure (CHF) refractory to standard treatment[13]. The proarrhythmic effects of dobutamine are supported by several observations. It increases the dispersion of action potential duration in adjacent areas of ischemic and non-ischemic myocardium in experimental animals[14], and increases the incidence of VT in patients[15].

Electrical storm activates the sympathetic nervous system. β-Blockers play a key role in the management of ES. In a canine study[16], β-blockers increased the fibrillation threshold (that is, made the animals less susceptible
to fibrillation) 6-fold under ischemic and nonischemic conditions. Amiodarone is widely used in the treatment of electrical storm[6]. In acute amiodarone therapy, rapid intravenous administration blocks fast sodium channels in a use-dependent fashion (producing more channel blockade at faster heart rates), inhibits norepinephrine release, and blocks L-type calcium channels but does not prolong ventricular refractoriness. Conversely, in oral amiodarone therapy, prolonged ventricular refractory periods are seen over periods ranging from days to weeks[17]. Amiodarone has few negative inotropic effects and is safe in patients who have depressed systolic function. Amiodarone or β-blockers are generally accepted as the best available drugs for prevention of arrhythmic storm[18]. Class I antiarrhythmic drugs are used widely, with variable success rates, and can play a role in polymorphic ventricular arrhythmias[19]. But in our report, there was no response to the use of any above drugs in the patient.

The physical and emotional stress that patients experience in association with electrical storm and multiple electrical cardioversions often perpetuates arrhythmias. All patients who have electrical storm should be sedated. Short-acting anesthetics such as propofol, benzodiazepines, and some agents of general anesthesia have been associated with the conversion and suppression of VT[20]. Left stellate ganglion blockade and thoracic epidural anesthesia have also reportedly suppressed electrical storms that were refractory to multiple antiarrhythmic agents and β-blockade[21]. These therapeutic approaches directly target nerve fibers that innervate the myocardium, and a reduced adrenergic tone is most likely responsible for the reported efficacy[22]. In this article, we found, there was no response to the use of any recommended anti arrhythmic drugs. However, the use of chlorpromazine and promethazine surprisingly stabilized her heart rhythm.

The good neurological outcome in the patient was probably due to a number of positive factors. This case illustrates that immediate and high quality chest compressions is necessary. Each episode of VF initiated immediate manual chest compressions while charging the defibrillator, thus hands-off time was reduced to a minimum.

This case illustrates the importance of defibrillation, multiple anti-arrhythmic agents and sedation in management of electrical storm. Despite repeated defibrillations, she went on to make a good recovery. Every possible attempt should be made to reduce as far as possible the number of patients who undergo it.

Correspondence author:
Yun-Feng Han
Department of Medical Engineering,
The Military General Hospital of Beijing PLA,
Beijing 100070, China;
hanyf001@126.com.

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