



## ELECTRIC STORM IN A PATIENT WITH STEMI

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### ABSTRACT

A 57-year-old male patient was admitted to hospital with 2 hours history of right sided chest pain with diaphoresis. He was diagnosed with acute Inferior wall ST-elevated myocardial infarction and transferred to Cath-Lab for emergency PCI. During preparation for Trans-radial arterial puncture, patient went in pulse-less tachycardia and ventricular fibrillation (VF). During the following 2 hours, cardioversion was performed 72 times. The patient survived. CAG one week later showed triple-vessel disease. Electrical storm is an uncommon and dramatic but usually treatable syndrome of recurrent ventricular arrhythmias. Frequent precipitants of electrical storm include recent worsening heart failure, hypokalaemia, hypomagnesaemia and myocardial ischaemia. Amiodarone/ $\beta$ -blockers is the antiarrhythmic agent of choice and implantable cardioverter defibrillator improves long term outcome.

**Key words:** Myocardial Infarction, Electrical Storm, PCI ( Percutaneous cardiac intervention)

## INTROUCTION

Electrical storm is a life-threatening syndrome that involves recurrent episodes of ventricular arrhythmias. It is defined as 3 or more sustained episodes of ventricular tachycardia (VT), ventricular fibrillation (VF), or appropriate implantable cardioverter-defibrillator (ICD) shocks during a 24-hour period[1]. Sustained VT lasts 30 seconds, involves hemodynamic compromise, or requires intervention to terminate the episode. Management of electrical storm is challenging and requires an approach tailored to the underlying cause. It is known that myocardial ischaemia and infarction leads to severe metabolic and electrophysiological changes that induce silent or symptomatic life-threatening arrhythmias. The condition can manifest itself during the acute phase of a myocardial infarction (MI) and in the presence of structural heart disease, an ICD, or an inherited arrhythmic syndrome. The major symptoms are palpitations, dizziness, and often syncope. The clinical presentation might be dramatic and can involve cardiac arrest or multiple episodes of potentially fatal arrhythmias.

We present a case of a patient with AMI who developed electrical storm just before Primary Percutaneous Coronary Intervention (PPCI), which was suppressed with DC shocks, antiarrhythmic drugs and  $\beta$ -blocker.

## CASE REPORT

A 57 year old man with type 2 diabetes, ex-smoker was admitted in CCU with 2 hours history of severe ,intolerable right sided chest pain and diaphoresis. His vital signs upon arrival in CCU were; Temp: 36.7C, blood pressure 100/65 mmHg, heart rate 65bpm regular, respiratory rate was 18breaths/minute. Patient was conscious and oriented. Examination of head and neck was normal. The chest wall examination with cardiovascular and respiratory system was normal. Initial lab investigation showed CBC, Basic Metabolic Profile And Lipid Profile are in normal range. Cardiac enzymes are elevated- CK [TOTAL]- 5902 U/L CK-MB- 154U/L Troponin I: 17.4 ng/l.

Echocardiogram showed normal ventricular function and ejection fraction. An ECG was taken at the time of admission which showed sinus rhythm and ST elevation in leads II, III and avf lead[figure 5]. Patient was diagnosed as inferior wall STEMI. We planned for emergent PCI and initiated loading dose of aspirin, clopidogrel, atrovastatin at the time of admission.

In Cath lab, during preparation for Trans-radial arterial puncture, the patient had a cardiac arrest from recurrent ventricular tachycardia and ventricular fibrillation. Cardiopulmonary resuscitation was started and airway was maintained through endotracheal intubation. More than 72 Biphasic unsynchronized DC was given before achieving sustained sinus rhythm with cardiac output. Drug used during resuscitation was (a) atropine 2mg x IV bolus x 3 time at regular interval (b) Lidocaine 5 mg iv bolus x 5 times at regular interval (c) Amiodrone 150 mg iv bolus – 3 bolus dose at interval of 10 minutes and then Maintenance dose

of 300mg in 50ml NS @ 5ml/hr (d) Magnesium sulphate 20 ml iv X 2 times at regular interval (e) Esmolol 100 mg iv bolus X 2 times at regular interval (f) Adrenalin-1mg iv bolus x 5 times at regular interval (g) 5% HCO<sub>3</sub> 40 ml x iv bolus (h) Dopamine 10mcg/kg/minute x iv @ 3-6 ml/hr.

We explained the need for emergency percutaneous coronary angiography (CAG) and percutaneous coronary intervention (PCI) to his family, but they refused both. Patient was admitted in ICU and initiated conservative treatment for acute myocardial infarction. The treatment, however, did not improve his symptoms sufficiently, and he decided to undergo CAG and PCI on the 11<sup>th</sup> day of hospitalization. A coronary angiogram [figure 1 and 2] showed triple-vessel disease with diffuse stenosis seen in 1] Left Anterior Descending artery (middle and distal segment) 85% [2] Left proximal segment of OM1 circumflex 75% [3] Middle and Distal segment of Right Coronary Artery 95 %

1] Angioplasty first done in Right Coronary Artery (RCA), using 6F JR4 guiding catheter.

- ❖ Run through guide wire is passed through the catheter.
- ❖ Then used maverick dilatation balloon catheter of measuring 2.0 / 20 mm and dilated at 8-10 atm.
- ❖ We deployed BUMA drug eluting stent measuring 2.75 / 35 mm and 3.0/30mm at 10atm and 12 atm respectively at distal and proximal segment of RCA.

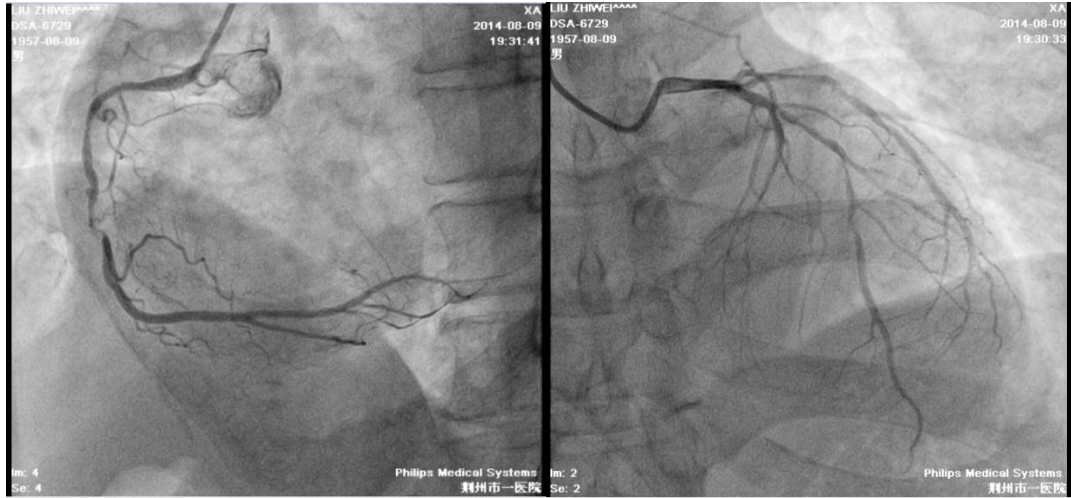
2] Angioplasty in Left anterior descending artery (LAD), using 6F XB3 guiding catheter

- ❖ BMW guide wire is passed through the catheter.
- ❖ We deployed BUMA drug eluting stent measuring 2.5 X 35 mm and 2.75X15mm at 10 atm and 12 atm respectively at distal and middle segment of LAD.
- ❖ Post dilatation done using QUANTUM balloon catheter measuring 2.5/12 mm and dilated at 16 atm.

3] Angioplasty in Left Circumflex Artery-

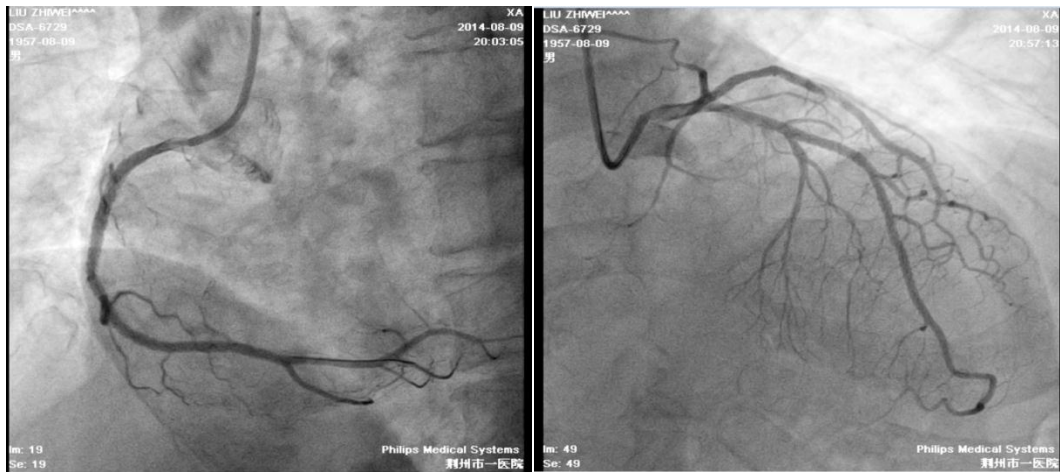
- ❖ RUNTHROUGH guide wire is passed through the catheter.
- ❖ Predilatation done with Maverick dilatation balloon catheter of measuring 2.0 X 20 mm and dilated into proximal circumflex to OM1.
- ❖ We deployed BUMA drug eluting stent measuring 2.5/35mm at 12 atm.
- ❖ Drug used during PCI - Heparin 8000 U /IV , TIROFIBAN-15ml [5mg/100ml]

All antiarrhythmic drugs, including amiodarone and lidocaine, were reduced gradually and ceased before discharge without recurrence of VT/VF. A total of more than 72 cardioversions were performed during admission. He was discharged with aspirin, clopidogrel, simvastatin. No major adverse cardiac events occurred during the following 3 months.



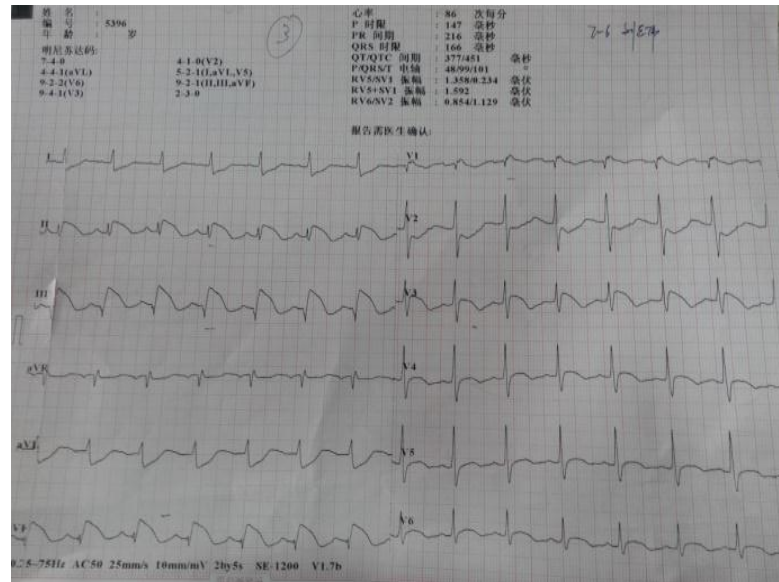
**Figure 1:** Right coronary angiogram before PCI

**Figure 2:** Left coronary angiogram before PCI



**Figure 3:** Right coronary angiogram after PCI

**Figure 4:** Left coronary angiogram after PCI



**Figure 5:** ECG at the time of admission.

## DISCUSSION

Electrical storm (ES) is a fatal condition where critical arrhythmia, such as VT and ventricular fibrillation, occurs incessantly. ES can be caused by structural heart disease, electrolyte imbalance, inherited arrhythmic syndrome, and myocardial infarction[2]. In patients who have bundle branch block, ventricular pre-excitation (Wolff-Parkinson-White syndrome), or rate-related aberrancy, supraventricular tachycardia (SVT) can resemble VT. Patients with VT may have minimal symptoms that prompt the erroneous diagnosis of SVT with aberrant conduction. For this reason, an ambiguous wide-complex tachycardia should be presumed to be VT, especially in patients who have structural heart disease. If this rule is followed, the diagnosis of electrical storm will be accurate in 80% of all patients with tachycardia and in 95% who have had a previous MI [3].

We have described a patient with incessant VT/VF. Several antiarrhythmic drugs, electrical conversion, were required to suppress the electrical storm in this AMI patient. Persistent electrical storm is rare, but may be fatal in AMI. In patients with AMI, regional ischemia, combined with increased sympathetic activity, causes enlarged spatial and temporal dispersion of repolarization, which may be responsible for intraventricular reentry phenomena and VT/VF occurrence. Zipes reported that myocardial ischemia and infarction affected the denervation of sympathetic-parasympathetic fibers, which enhanced sympathetic activity, thereby increasing the propensity for ventricular arrhythmia[4]. Sympathetic blockade has been shown to prevent VT/VF. In the Canadian Amiodarone Myocardial Infarction Arrhythmia Trial (CAMIAT) and

the European Myocardial Infarct Amiodarone Trial(EMIAT)trials[5],patients on amiodarone who were also on  $\beta$ -blockers had a significant reduction in primary outcome events compared to patients not on.In our case,the patient was also treated with esmolol associated with amiodarone,lidocaine,which had shown the reduction in frequencies of VT/VF.As for approaches to antiarrhythmic drug therapy,several options have been exploited.The Adenosine-Triphosphate Sensitive ( $K_{ATP}$ )  $K^+$  channel is a metabolic sensor that opens during myocardial ischemia and has a key role in ischemic action potential duration shortening. Certain compounds can selectively block the sarcolemmal  $K_{ATP}$  channels and prevent associated malignant VT/VF. Cell-to-cell communications and their pharmacological manipulation have yielded new approaches to ischemia-associated uncoupling of gap junctions and its role in promoting malignant VT/VF. The identification of calstabin 2 depleted Ryanodine Receptor ( $RyR_2$ ) as a source of diastolic Sarcoplasmic Reticulum (SR)  $Ca^{2+}$  leak in catecholaminergic polymorphic ventricular tachycardia and in patients with heart failure susceptible to malignant VT/VF has led to the hypothesis that increasing calstabin 2 binding to  $RyR_2$  could be a potential new target to treat triggered malignant VT/ VF [6]. Whether this approach is helpful in arrhythmias secondary to acute ischemia is not known. In sustained VT or VF, the implantable cardioverter defibrillator (ICD) is highly effective in treating recurrences.

Despite advances in delivery of primary percutaneous coronary intervention (PPCI) for ST-segment elevation MI (STEMI), an unknown proportion of patients with STEMI experience VF prior to PPCI and thus may die before revascularization. Three prospective case-control studies of patients with STEMI and VF before PPCI identified associations between family history of sudden death, cumulative ST elevation, left coronary artery culprit lesions, and absence of pre-infarction angina with VF [7,8,9]. As in our case the patient did not had any past history of heart disease and developed VF prior to PPCI.In this situation the effective management of electrical storm requires an understanding of arrhythmia mechanisms, therapeutic options, indications for radiofrequency catheter ablation. Initial management involves determining and correcting the underlying ischemia, electrolyte imbalances, or other causative factors. In fact, electrical cardioversion (CV)/defibrillation and management of acute volume overload were the principal life-saving measures in the coronary care units before the advent of reperfusion therapies, beta-blockers, antithrombotic therapy, and statins [10].In our case ,initially we managed electrical storm with DC shocks,anti-arrhythmic medication and anti-thrombotic therapy. The causative factor for ES was underlying myocardial ischemia,so we explained the need for emergency percutaneous coronary angiography (CAG) and percutaneous coronary intervention(PCI) to his family.But they refused both and since patient was not hemodynamically stable so we decided to suspend the PPCI. The prognosis of patients with ES is poor due to the recurrence of arrhythmia and the progression of heart failure The risk of mortality is high, particularly in the first three months after the event[11]. ICD therapy improves survival in patients who have survived cardiac arrest and are at risk of sudden death—that is, with a history of myocardial infarction and reduced systolic left ventricular function[12].

## CONCLUSION

Electrical storm is a clinical emergency. Despite the lack of identifiable triggers for electrical storm in the majority of patients, triggering cause or exacerbating factors should be sought, including a systematic search for ischemia, decompensation of heart failure, changes to medications, bradycardia induced tachyarrhythmias, or other systemic illness. It has been estimated that 10-25% of may have reversible factors triggering the electrical storm episode [13]. We present this case to demonstrate the importance of recognizing sympathetically-mediated VT/VF in AMI. If the patient appears with arrhythmia just before PCI, use of  $\beta$ -blockers with amiodarone would be beneficial to prevent electrical storm and cardiac death, if PCI is not feasible.

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